MORBIDITY, AIR POLLUTION AND HEALTH STATISTICS

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Section I. Introduction

With the passage of the Clean Air Act Amendments of 1970, the Federal government committed the public and private sectors to billions of dollars in expenditure for pollution control. The authority provided by this Act, including the regulation of stationary and mobile sources of pollution, is scheduled for review by Congress this year. The underlying rationale for examining morbidity effects of air pollution comes from the Act itself, which states that national ambient air quality standards should be based on levels that will assure the protection of the public health.

The need for estimating the general population health effects from the criteria air pollutants subject to regulation became more urgent after the issuance of an Executive Order by President Reagan in February, 1981. The Order requires consideration of the potential benefits and costs of all major regulations. It is expected that the revision of primary air standards over the next two years will be interpreted as major regulations subject to the requirements of the Order.

Though air pollution is known to affect agricultural output, materials, and visibility, all available evidence points to the conclusion that a large portion of the benefits of air pollution control arise from improvements in the states of human health. Statistical measurement based on clinical and non-clinical experiments, has played a major role in the establishment of primary air quality standards in the United States. The evidence of the effects of air pollution on human health was initially determined

from a number of epidemiological investigations of air pollution episodes in the United States and Britain in the 1950s and 1960. Although there are a number of serious difficulties in determining the statistical relationship between air pollution and health, one can be optimistic about future scientific inquiry in this area. Since the initial studies of pollution episodes, new and better sources of information concerning health status and pollution exposure have been developed and statistical techniques more capable of dealing with this data have been developed.

Recently much research has been conducted to test empirically the claim that air pollution does affect the incidence of human mortality and morbidity. Most of these studies, however, have concentrated on the relationship of air pollution to mortality. The study results are then extrapolated to approximate the morbidity effects. Unfortunately, it is probably a significant distortion to use the statistically established relationship between air pollution and death to approximate the true effects of air pollution on disease. This assumption may have served to reduce the general acceptability of the empirical studies of health effects.

The analysis of morbidity effects themselves, should be an important area of concern. First, it requires a smaller sample size in order to obtain statistical significance since sickness occurs more frequently than death. Second, morbidity is probably a more sensitive indicator of pollution effects because of the immediacy of the effect. Third, there may be a good deal of acute illness resulting from air pollution which never result in death.

Fourth, the measurement of chronic and more severe morbidity can serve as a verification of the estimated mortality effects.

This paper attempts, in Section II, to provide a review of some of the statistical techniques that have been utilized to estimate the health effects of air pollution.

Section III highlights some of the reported results and Section IV suggests some of the problems inherent in this type of statistical analysis. Finally, Section V provides some new evidence of the morbidity effects from air pollution based on some recently developed data.

Section II. Statistical Techniques

This section reviews the four principal approaches that have been used to assess the impact of air pollution on human health.

The studies may be characterized as those involving animals and those with human subjects. Extensive use has been made of animal studies, especially with respect to mechanisms of damage to pulmonary macrophages. Animal studies have the great virtues of permitting measurement of long-term responses to relatively low levels levels of pollution and of permitting direct examination of damaged tissue. However, animal studies have the fundamental problem of extrapolation to man. With this link imprecise at best, animal studies can play only a minor role in the estimation of pollution control benefits.

Researchers have conducted at least three types of human studies - chamber experiments, statistical analysis of occupational exposure, and epidemiologic methods in general populations. Chamber studies on healthy and diseased subjects do reveal the levels at which various acute effects are observed in humans but have other problems such as the ethics of research and the difficulty of ascertaining chronic effects. Chamber studies can have great value in identifying thresholds for various subgroups of the population, but they can play only a limited role in assessing economic benefits because of the virtual omission of consideration of chronic effects. Studies of occupational exposures face problems of measuring actual exposures (particularly a problem for chronic effects resulting from past exposure) and the typical lack of compatability between the mix of occupational pollutants and

concentrations and those experienced by general populations. Moreover, there is substantial evidence of selection by industry and self selection by workers so that exposured industrial groups are not representative of the population. Therefore, occupational data will have limited relevance to the estimation of benefits of control of population exposures to the criteria air pollutants.

Because of the problems with the aforementioned approaches, assessment of general population benefits from controlling air pollution must rely on epidemiologic methods in general populations to detect chronic effects of long-term exposures to low levels of pollution. The epidemiologic approach has the great virtue that it can estimate the response to the full range of condition to which humans are actually exposed. The value of epidemiologic methods is enhanced if other risks can be controlled and potentially confounding variable can be included. Microepidemiologic studies using data on individuals are preferable to studies that use data on the means of large groups exposed to different levels of pollution. The reason is that data on individuals spans a far larger range of variation than does data on, say, metropolitan area averages. For example, cigarette smoking is a major contributor to adverse health states, probably much more important than air pollution parameters. The use of city-wide averages may obscure statistically significant dose-response relationships that exist among the non-smoking subset of the population. Despite the preferability of data on individuals, such data are not often available and many researchers have usually relied

upon average responses and average characteristics for population subgroups as the basis for analysis.

In the 1960s and into the 1970s, several epidemiologic studies and chamber experiments were reported. A number of the early epidemiologic studies of particulates and SO_2 focused on severe air pollution episodies - in London, New York City, and Donora Pennsylvania. In London, 4000 excess deaths were attributed to one severe episode in 1952; in New York some 200 excess deaths were attributed to an episode in November, 1953. These studies were reinforced by other evidence linking excess morbidity and lung function impairment in populations living in heavily polluted cites and towns.

Statutory obligations (in the Clean Air Act of 1970) forced regulators to set air pollution standards to provide a margin of safety in the protecton of sensitive groups. This statutory directive undoubtedly shifted research interests toward defining threshold levels for sensitive groups. For this purpose, chamber studies were indispensable. There continued to be, however, an interest in population epidemiologic approaches, principally because only through this latter method could one hope to evaluate the incremental impacts on the general population of alternative air standards. The next section summarizes some of the principal findings of these studies.

Section III. Some Previous Results

1. Mortality:

The effects of acute episodes of high level of particulates and sulfur oxides (the gas SO_2 and the particle SO_4) are well documented. Excess mortality has been demonstrated for episodes in London, New York, the Meuse Valley, and Donora, Pennsylvania. Demonstrating an effect at lower levels approximating present exposures has proven to be much more difficult and controversial. One attempt to document the effect of SO_2 on mortality used a time series analysis of mortality in Greater New York and data from one monitoring station to estimate exposure. Buechley, et. al.,F.N. showed that there was an excess in mortality after controlling for a number of potentially confounding variables at exposure levels of about 300 ug/m^3 of SO_2. This compares with the U.S. primary air standard of 80 ug/m^3 (.03 ppm) for annual mean 24 hour values and 365 ug/m^3 (0.14 ppm) for 24 hours not to be exceeded more than one per year.

Lave and Seskin F.N. have made extensive cross-sectional analyses of large Standard Metropolitan Statistical Areas (SMSA) in the U.S. as well as British data. They have attempted to

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F.N. We have concentrated on SO_2, SO_4 and particulates in this analysis because of the relative wealth of epidemiologic evidence linking them to chronic health effects in humans (as contrasted to CO, NO_x, or ozone).

Buechly, R. W., et. al., "SO_2 Levels and Particulates in Mortality - A Study in the New York-New Jersey Metropolis," Arch. Environmental Health 27:134 (1973).

Lave, L. B., and E. P. Seskin, "Air Pollution, Climate, and Home Heating: Their Effects on U.S. Mortality Rates," American Journal of Public Health 62:909 (1972).

control for several confounding variables, including age, income, home heating, race, and occupation. Three major problems exist with this approach. Although they recognize the potential confounding effect of smoking, they have not been able to control for it. Another problem, particularly in large SMSAs, is that monitoring data from one or a few monitors may not be very representative of outdoor exposures. And the problem of correlating this with indoor and total pollution exposure further complicates the quantitative assessment.[F.N.] A third potential problem with the Lave-Seskin approach is that it did not control for the availability of medical care. In Lave's most recent work,[F.N.] this third problem was addressed and found to not make a significant difference in the results. Lave found a statistically significant correlation between morbidity and exposure to SO_2 and SO_4 at current exposure levels but no association with particulates. Lave attributed the lack of association with particulate exposure to the fact that TSP as reported is a very crude index of the subcomponent of respirable particulates that are known to cause scarring of lung tissue and presumed to damage health.

Other studies using cross-sectional analyses of large metropolitan regions have been initiated by Crocker, et. al.[F.N.] Their

F.N. Spengler, et. al., (Feb. 1981, JAPCA) note that the correlation between indoor and outdoor pollution for the sample in their 6-city study was about .5 for SO_2. For non-smoking families, indoor particulate exposures were nearly identical to outdoor levels, but for smoking families indoor exposures were up to three times as high.

Lave, unpublished manuscript 1981.

Crocker, et. al., Methods Development for Environmental Benefits Assessment, for U.S. EPA, 1981

work demonstrates a much weaker link between mortality and air pollution than Lave found for sulfates or S02. Methodological problems with the Crocker study indicate that caution must be exercised in interpreting the results. It is unreasonable to use either of these principal mortality studies as indicating a major effect between mortality and SO_x at current exposure levels. A small effect may exist, but cigarette consumption and better information on actual exposures would have to be known first before that effect could be quantified with any precision.

2. Morbidity

Several of the same pollution episodes that provided evidence on the acute mortality effects of high levels of pollution also revealed substantial increases in morbidity. For example, analysis of the impacts of the 1952 London fog made use of data on applications for admissions to hospital for non-accident cases. Other studies have used consultation rates with general practitioners as an indication of adverse effects of high concentrations of air pollution. Another source of data on acute effects are industrial sickness absence records. Holland's detailed review of the literature on short-term effects of air pollution on morbidity concluded that while most of the studies showed some association, it was very difficult to asses the results in a truly quantitative fashion. The early (pre-1970) studies were all carried out without the objective of providing information for setting standards. In the early studies, only limited attempts were made to control for other factors such as weather or influenza that would also have affected short-term responses. In more recent studies,

Holland attributes the failure to establish clear links between air pollution and acute effects to the fact that major efforts to reduce pollution had been made so that daily variation in the range of pollution now encountered is not large enough to lead to effects that can be distinguished from background levels.[F.N.]

Epidemiologic methods have been used to assess long-term (chronic) effects from low levels of exposure to SO_2 and particulates. One of the problems faced by researchers has been the general lack of availability of suitable morbidity data. While morbidity represents a more sensitive change in health state in response to an environmental insult than does death, the subtleness of various impacts make them much more difficult to identify and quantify. A number of researchers have resorted to expensive case control studies in which individuals are monitored over relatively long periods for effects such as cough, sputum, and respiratory disease. These studies have shown demonstratable correlations between the frequency of symptoms and disease of the repiratory tract and air pollution levels. One such study in Genoa showed a very significant (r=0.98) correlation between the frequency of bronchitis and annual mean sulfux dioxide levels.[F.N.] The effect persisted for levels of exposure only slightly above the present U.S. primary air standard (105 ug/m^3 or 0.037 ppm).

F.N. Holland, et. al., "Health Effects of Particulate Pollution: Reappraising the Evidence," American Journal of Epidemiology 110:525-659 (1979)

Petrilli, et. al.,

Data from several other studies (in New Hampshire, Nashville, Australia, Great Britain, Chicago and Germany) further indicates an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and various health effects. For sulfur dioxide, adverse health effects were noted at levels only slightly above the present primary air standard. For particulate, Holland concluded that "it is not possible to find any reasonable evidence that would justify the (U.S.) primary air standard of 75 ug/m^3 for total suspended particulate." In a rebuttal, Shy argued that there were sound reasons to disagree with the assessment.[F.N.]

Epidemiologic evidence has played a central role in the establishment of primary air standards in the U.S. Because of the continuing controversy over chronic morbidity effects at exposure levels near or below the present U.S. standard, we have sought and obtained access to much better data than has heretofore been analyzed.

F.N. Shy, C. M., "Epidemiologic Evidence and the United States Air Quality Standards," American Journal of Epidemiology 110:661-671 (1979).

Section IV. Research Design

The aim of this section is to provide an overview of the statistical problems associated with the epidemiological approach to estimating the morbidity effects of air pollution. Some of these problems are common to almost all areas of statistical inquiry. Others are more specifically related to the uncertainty in the measurement of air pollution and health. The statistical problems can perhaps be generalized into three different areas: questions of proper functional form, data and measurement problems, and specification problems and certainties.

Most of the epidemiological research on the health effects of air pollution have assumed a linear dose-response relationship. The additive linear functional form implies that each marginal improvement in air quality results is a constant improvement in health. In addition, it posits that there is no interactive effect among pollutants or between pollution and other variables, such as weather conditions. Unfortunately, there is little theoretical or empirical justification for this functional form. Most clinical research has generated an S-shaped (or logistical) dose-response relationship. However, the linear form may be an acceptable approximation of the true form over a certain range of air pollution values. For larger changes in air pollution, the linear approximation may be a less accurate estimate of the health effects, relative to some non-linear specification.

There are two other potential problems with the linear form. First, it can predict negative values for the dependent variable,

even if the dependent variable is always observed to be non-negative. Second, it structurally assumes that the explanatory variables will have a similar effect over the entire range of the dependent variable. These two points are discussed in more detail in Section V.

If one is attempting to estimate the probability of death or illness from air pollution and/or wishes to utilize a non-linear functional form, there are a number of probabilistic models available including logit, probit and Tobit. Each carries its own assumptions about the shape of the dose-response function and about the error term. With the uncertainty intrinsic to an area of inquiry such as air pollution and health, it is extremely important that alternative function forms be tested so that the goodness-of-fit, compatability and predictive results can be con-pared.

The second major statistical problem germane to the study of air pollution and health, is that of availability and accurate measurement of the necessary data. Of obvious importance is the availability of accurate measures of air pollution exposure. There are three major concerns here: which pollutant to measure, the relationship of ambient levels to exposure, and the time structure of pollutants exposure.

The measurement of ambient air pollution is obtained pri-marily through Environmental Protection Agency monitors sited throughout the country. The measurement techniques have improved dramatically over the last two decades and are becoming greater more accurate and specific. For example, EPA is moving towards

the measurement and standard setting for inhalable particulates (those less than 3 microns) rather than total suspended particulates. The former are now believe to be most responsible for generating deleterious effects on the respiratory system. Among the other pollutants, however there is still question as to which are the most important precursors of health effects. Only further clinical study will reduce uncertainty in this area.

Even the most accurate measurement of pollution at the monitoring site may not represent the measure of actual pollution exposure, however. First, there can be significant spatial variation of the pollutant and the potential receptor from the source of measurement. Second, individuals working in other areas or in closed environments will receive different exposures for at least part of the day. Third, actual exposure will vary according to the time spent inside, the degree of insulation and ventilation, and the prevelant pollutant in the area. For example, carbon monoxide easily penetrates all structures, while large order particulates and reactive pollutants such as sulfur dioxide and ozone do not. Researchers are usually left with making the simplifying assumption that, on average, the monitored air quality level is somewhat representative of exposure. Random measurement errors of air pollution exposure will lead to an underestimate of the air pollution effect.

Another question relating to the use of ambient levels as a proxy for exposure is that of which statistical measure to use. The mean, maximum and minimum pollution level all have been used in the past. Each is suggestive of a different kind of

relationship between air and health. Satisfactory answers to this question would help the policy makers decide if it is chronic doses above some minimum level or acute doses at high levels that generate serious health effects.

Finally, there is a question of the time lag of air pollution induced health effects. Health effects may well be related to current levels of pollution or they may be a result of cumulative exposure over a number of years. If the latter is the case, the use of current levels may lead to a biased estimate of the pollution effect.[F.N.]

The choice of the health measure also presents a problem for morbidity research. Although there are a number of sources of data on illness and hospital visits, few have the standardization and sample size necessary for a cross-sectional analysis. Thus, most of the morbidity studies have been either time series analyses for a given city, studies of emergency room utilization, or simple two city or city/rural comparisons using analysis of variance. In addition, there have been surveys which have attempted to link overt effects - eye stinging, sneezing, coughing, breathing - with recorded levels of air pollution. Recently, some other data bases, which include questions about health care utilization and health status, have been used. Included are the Michigan Survey Panel Data, and the National Center for Health Statistics Health Interview Survey (HIS).

F.N. See Daniel M. Violette, "Estimating the Human Health Benefits of Improved Air Quality," prepared for the National Commission on Air Quality Benefits Estimation Panel, January, 1980. PP 189-193.

The latter has many possibly useful indicators of health status. For acute illness, it measures restricted activity days, work loss days, school loss days, bed days, and hospital days. Restricted activity days (RAD) is the inclusive term for all the ways in which one can react to acute illness. It is officially defined in the HIS as a day in which "a person cuts down on his usual activities for the whole of that day because of an illness or injury.... It does not imply complete inactivity, but it does imply only the minimum of usual activities." In addition, the HIS reports the health condition or diagnosis that is believed responsible for each RAD.

The variable measuring work loss days is based on the response to the survey question asking how many days in the last two weeks did illness or injury prevent one from working. Obviously, the amount of pain or discomfort tolerated by an individual before missing work is a very subjective decision and may have little to do with any objective measure of illness. In addition, reported or actual WLD may be affected by other unmeasured factors such as response to the survey or attitude towards work. Part of the decision to miss work, however, will be based on socio-economic and job related factors which can be measured or approximately empirically. The statistician can only assume that there is an underlying distribution which determines the health effects threshold. For each chronic illness, the HIS records the duration of limitation, the degree of limitation and the diagnosis.

The measurement of other, potentially confounding variables is also important to the study of air pollution and morbidity especially since the "true" causative variables to describe morbidity are unknown. Omission of variables that explain the variation in the dependent variable can lead to serious estimation problems, as discussed below.

Much of the previous health effects research has utilized aggregate data to proxy socio-economic variables. For example, in their mortality study, Lave and Seskin use variables such as the percentage of population 65 or older, the percentage of the population who are non-white, and the percentage with income below poverty. Individual data presents a distinct improvement and allows the researcher to disaggregate the analysis and discern the variation in the pollution effect across categories, such as age, race, and sex.

There exist a number of other variables which may vary collinearily with air pollution and may also affect health status. Those most frequently cited include occupational exposure, smoking, migration, indoor pollution, diet, exercise, risk attitude, weather and "urbanness".[F.N.] Again, some of these confounding effects can be eliminated through use of individual data, if available. By stratifying the sample one can explicitly account for the effects of occupation, smoking, indoor exposure and various geographic factors.

F.N. For a detailed description of the problems generated by these factors and attempts to reconcile them in mortality studies see Richard Wilson, et al., Health Effects of Fossil Fuel Burning, Ballinger Press, 1980, PP. 191-214.

For some factors, such as diet, exercise, and attitude towards health care, direct measurement through survey will probably not be economically feasible. However, many of these influences can be proxied by socio-economic surrogates. A statistically significant pollution effect can be generated artificially only if these factors vary with air pollution and not with the socio-economic proxies.

The third major statistical problem relevant to the study of the morbidity effects of air pollution is that of econometic specification. Even if acceptable data on pollution exposure, health status and their potentially confounding factors is available, improper specification of an estimated equation can seriously bias the coefficients. Three different specification problems may be relevant to this area of research: multicollinearity, omitted variables and simultaneity.

Since the "true" model of health status is far from certain, one can only make reasonable guesses about the variables that should be included in a regression equation explaining illness. There is a trade off involved. As explanatory variables are added, multicollinearity may become a problem; specifically, variables that vary with air pollution may be included so that the estimated effect of pollution becomes confounded. To limit the number of explanatory values, however, is to open up the possibility of omitted variable bias, discussed below.

Multicollinearity can exist, and usually does, among air pollution variables. Particulates, sulfur dioxide and sulfates are all generated from fossil fuel combustion by stationary

sources. On the other hand, hydrocarbons, carbon monoxide, nitrous oxides and ozone are the result of fuel combustion from mobile sources. Multicollinearity can also arise because of the relationship between air pollution are the other explanatory variables including socio-economic and urbanization variables. To the extent that these factors vary systemically (e.g., both air pollution and urbanization may increase as we move from the Southwestern to the Northeastern United States), there will be difficulty discerning the independent influences of air pollution.

Another potentially serious specification error occurs when a non-random explanatory variable, correlated with air pollution, is omitted from the estimated equation. The included independent variables then take on explanatory "noise" from both the excluded variable and the error term and will have biased estimated coefficients. The degree of the bias will be proportional to two factors. First, the collinearity between the excluded and air pollution variables; and second, the importance of the omitted variable in explaining the dependent variable.

A final specification problem is that of simultaneity. This would occur if, for example, the explanatory variable 'physicians per capita' is used to explain the variation in health status. If health status in turn influences the locational decision of physicians, the estimated coefficients will be biased and inconsistent. A technique, such as two stage least squares, could be used to reduce this simultaneity problem.

Section V. Some Preliminary Results

The data set which comes closest to meeting many of the needs outlined above is the annual Health Interview Survey (HIS) conducted by the National Center for Health Statistics. This is a scientific survey of 50,000 households comprising roughly 120,000 persons. Besides basic demographic and economic characteristics of the respondents, the survey includes data on acute and chronic illness (identified by diagnosis), disability days for those in and out of the labor force, work and school loss days due to illness, measures of health care utilization, height and weight, family income, occupation and industry of employment, and individual cigarette consumption. The availability of the latter makes the data set superior to many others and mitigates the separation of the health effects of cigarette smoking versus air pollution.

To provide a preliminary assessment of the effects of air pollution on morbidity, a data base was created that provides detailed information about the individuals and their health status, the levels of several pollutants to which they are exposed, their climate, and the area within which they live. Thus, the HIS results for 1976 were merged with: 1976 EPA data on ambient levels of particulates (TSP), sulfur dioxide (SO_2), and sulfates (SO_4); NOAA data on wind, temperature and precipitation; and Census data on density and other urban characteristics. For this analysis, 130 cities of medium size (population of 100,000 - 500,000) were pre-selected in order to reduce the intra-city variation of the air pollution measures.

The initial work concentrated on the determination of the contribution of air pollution to acute illness in adults. The sample of all male non-smokers was used to estimate the variation in work loss days (WLD). This group was chosen for a number of reasons. First, the sample size of males is greater than that of females. Second, by using only non-smokers the air pollution effects cannot be attributed to the impact of cigarette smoking.* Also, cigarette smoking may be determined simultaneously by those variables that are used to explain health status. If smoking were included as an explanatory variable, it would necessitate a slightly more complex and less easily interpreted model. Third, males tend to have less family and child-rearing responsibilities outside of work. Therefore, there is less of a possibility of non-health related work loss days occurring. Work loss may be a more accurate indicator of illness for males than for females. Finally, work loss measurement is more conducive to a monetary evaluation of losses.

The dependent variable was hypothesized to be a function of ambient air pollution levels, various demographic and socio-economic variables, the existence of chronic disease, climate conditions, and measures of "urbanness".

Basically, two pollution variables, total suspended particulates TSP and sulfates were used. They were selected because of their use in the Lave and Seskin study, because of the preponderance of clinical evidence cited above concerning their health

*There still remains the possibility that non-smokers living in a household with a smoker will be the recipient of a smoking affect. This possibility will be considered in subsequent work.

effects and because their measurement tends to be of acceptable consistency. The correlation coefficient of these two variables was .2.

A number of demographic and socio-economic variables including age, race, family income, family size, physicians per 100,000 people, blue or white collar worker, and whether or not the individual was married and currently living with spouse were all employed to explain the variation in WLD. These variables were believed to be important factors in measuring the degree of and response to pollution exposure, and the ability to partake in preventive care including direct physician access, housing and sanitary conditions, diet, exercise, and occupational exposure. Data limitations preclude a determination of the degree to which diet and exercise, for example, may affect health. It is believed however, that the included independent variables are ample proxies for the measurement of access to and use of preventive care while at the same time independent enough to preclude problems with multicollinearity.

The existence of chronic disease (a binary variable) will probably play an important role in determining the frequency of work loss or activity restriction and was included in the estimation. The climatic conditions faced by individuals, including precipitation and average temperature or number of degree days were included because of their potential effect on WLD and RAD. Finally, a measure of the general urban structure, population density, was included.

Multiple regression was selected as the appropriate statistical tool because of its ability to control for many factors in

the analysis. A major uncertainty in the estimation, however, was the exact form that the statistical model should take. A special problem exists in that the dependent variable is truncated at zero and that a large percentage of the health status observations (between 70 and 95%) are zero. For this reason, three different models were tested. Each has different characteristics and assumptions about the structural nature of the explanatory variables and each generates a different shape for the dose-response relationship.

First, the ordinary least squares (OLS) method was used. Although cheaper to run and computationally simpler, this technique ignores the zero truncation and can possibly predict negative values for WLD. In addition, it has the implicit structural assumption that the same factors that cause the existence of any work loss day (the movement from zero to one or more) also explain the particular number of WLDs, given that at least one WLD has actually occurred. One advantage to this technique is that the linearity makes extrapolation easier. The estimated equation took the following form:

$$(1) \quad W_1 = b_o + b_1 D + b_2 A + b_3 C + b_4 M + b_5 U + u$$
$$= Xb + u$$

where W_1 = Number of work loss days

b_i = Estimated co-efficients

D = Demographic and socio-economic characteristics

A = Air pollution measures

C = Chronic condition

M = Meteorologic variables

U = Urban structure variables

X = Matrix of above independent variable

The partial derivation of work loss days with respect to the air pollution variable is:

(2) $\partial W_1 / \partial A = b_2$

An alternative technique was to use the Tobit model. This technique constrains the dependent variable to be non-negative but still implies the structual assumption described above. An additional problem is that the shape of the resulting dose-response curve will have positive first and second derivatives (convex from below) which is contrary to the generally accepted shape of the curve.

The stochastic model underlying the Tobit estimation is:

(3) $W_2 = Xb + u \quad \text{if } Xb + u > 0$

$W_2 = 0 \quad \text{if } Xb + u \leq 0$

with $u \sim N(0, \sigma^2)$

where W_2 = proportion of all work days that are lost days.

The model assumes that there is an underlying stochastic index $I = Xb + u$ which is observed only when it is positive.

The expected value of W_2 in the model is:

(4) $EW_2 = Xb\, F(Z) + \sigma f(Z)$

where $Z = Xb/\sigma$, $f(Z)$ is the unit normal density and $F(Z)$ is the cumulative normal distribution.

The expected value of W_2 for observations above the limit, W_2^*, is expressed by:

$$(5) \quad EW_2^* = Xb + \sigma f(Z)/F(Z)$$

The latter term is the expected value of the truncated normal error term. The meaning of F(Z) is simply the probability of the value Z being above zero.

Following McDonald and Moffitt,[1] the relationship between the expected value of all the observations, W_2, the expected value of those values above zero, W_2^*, and F(Z) is:

$$(6) \quad EW_2 = F(Z)\ EW_2^*$$

The partial derivative of the expected value of all observations expressed in (6) with respect to air pollution is:

$$(7) \quad \partial EW_2/\partial A = F(Z)(\partial EW_2^*/\partial A) + EW_2^*\ (\partial F(Z)/\partial A)$$

or the change in W_2 for those observations above zero weighted by the probability of being above the limit plus the change in the probability of being above zero weighted by the expected value of W_2 if above zero. With estimates of b and σ both of the right hand side terms can be calculated.

The third technique used was the logit-OLS model. In this case, a logit model was first used to determine the probability of an individual having at least one WLD in the survey period.

[1] John F. McDonald and Robert A. Moffitt, "The Uses of Tobit Analysis," Review of Economics and Statistics 62(2), May 1980.

In the second stage, OLS is used to determine whether air pollution influences the number of WLDs, given an individual has had at least one. This method has the advantage of consistency with statistical characteristics of the data. First, it truncates the dependent variable at zero (and one) by turning the frequency into a probability. Second it enables the use of different structual forms to explain the probability of a WLD episode (one or more) and the number of WLDs. Third, the estimated equation will assume the form of the logistic curve, the functional form that is believed to be typical of many dose-response relationships.

The estimated equation of the logit model is:

$$(8) \quad \log [(W_3/(1 - W_3)] = Xb$$

where W_3 is the probability that WLD > 0 in the two week survey period. The left hand side of (8) is simply the log of the odds of a work loss day. The change in W_3 due to a change in A is:

$$(9) \quad \partial W_3/\partial A = b_2 \cdot W_3 (1 - W_3)$$

The equation can also be expressed in terms of probability:

$$(10) \quad W_3 = (1 + e^{-Xb})^{-1}$$

The change in the probability of a WLD due to a change in pollution is:

$$(11) \quad \partial W_3/\partial A = b_2 e^{-Xb} (1 + e^{-Xb})^{-2}$$

The expected number of work loss days are the product of the probability of a non-zero WLD times the number of WLD,

$$E(W) = W_3 \cdot E(W_1 / W_1 > o)$$

The regression results for the three models using the sample of all male non-smokers, age 18-65, are presented in Table 1.

Table 1

The Estimation of WLD For Male Non-Smokers (N=4473)

	(A) OLS	(B) TOBIT	(C) LOGIT	(D) OLS(W>l)
CONSTANT	-.29	-1.2	-3.97	-.61
PMEN	.00177[b]	.00228[b]	.00614[b]	.0032
SULF	-.0083	-.013	-.033	-.0055
AGE	.004[a]	-.0003	-.0076[c]	.0746[a]
CHRON	.22[a]	.16[a]	.35[a]	1.25
RACE	-.02	-.027	-.04	-.36
MARR	-.03	-.0012	.37[a]	-.9[a]
INC	-4.5×10^{-6}	-1.4×10^{-6}	-2.3×10^{-6}	4.1×10^{-6}
TEMP	.0063[b]	.0014	.005	.088[a]
PRECIP	-.0013	.0024	.009	-.04[c]
DENS	4.0×10^{-6}	6.9×10^{-6}	3.0×10^{-6}	-2.7×10^{-5}
BLUE	-.06[c]	-.017	.157[c]	-1.33[a]
F	4.07[a]	--	--	6.38[a]
χ^2	--	113.2[a]	25.8[a]	--
mean dep	.2355	.061	.055	4.07
R^2	.01	--	--	.19
$\overline{W}$*	--	.2735	--	--

a = Significance at 1% level
b = Significance at 5% level
c = Significance at 10% level

PMEN = annual arithmetic mean of particulates

SULF = annual arithmetic mean of sulfates

AGE = age

CHRON = number of chronic conditions

RACE = $\begin{cases} 1 & \text{if nonwhite} \\ 0 & \text{if white} \end{cases}$

MARR = $\begin{cases} 1 & \text{if married and living with spouse} \\ 0 & \text{if unmarried or married and not living with spouse} \end{cases}$

INC = family income

TEMP = annual mean temperature

PRECIP = annual precipitation

DENS = population density

BLUE = $\begin{cases} 1 & \text{if blue collar worker} \\ 0 & \text{if not} \end{cases}$

The results of the three estimates are generally consistent with a priori expectations. In all three models, particulates are shown to be related in a positive and signficant way to work loss days. The mean level of sulfates does not appear to affect WLD. This result was confirmed when each of the pollution variables was run separately in the regression. There may be a number of explanations for this result. First, the sulfate measurement techniques are not believed to be very accurate. The errors in measurement may lead to serious underestimation of the coefficient. Second, the particulate measure may be proxying a number of variables; it measures coarse and inhalable particles as well as sulfate and nitrate particles. Third, these may be estimation problems due to collinear or omitted variables.

The models also show that chronic illness is associated with more WLDs. The OLS model has age and average temperature related positively to WLD and blue collar employment related negatively to WLD. By comparing this model to the Logit-OLS models some interesting distinctions can be made.

Estimation (C) suggests that air pollution, measured by particulate levels, will affect the probability of a WLD episode. However, estimation (D) suggests that air pollution does not influence the number of days lost given an episode has occurred. Further evidence of this result is obtained by applying Eq(7) to the Tobit estimates. The result, after taking the partial derivative, indicates that the first term in the right hand side of Eq(7) - the change in WLD for those observations above zero - is small (.0000175) relative to the second term - the change. in the

probability of being above zero (.0001). Thus, the total effect of air pollution on WLD is driven more by adding to the probability of an episode than by affecting the actual number of WLD.

Another interesting result shown by the estimated equations (C) and (D) is that being married and working in a blue collar job both increase the probability of a work loss episode but have a negative effect on the number of days lost. Age has the reverse affect: it slightly decreases the probability of an episode but has a strong positive influence on the number of days lost. The latter result is confirmed by the OLS estimate (A).

The sensitivity of the variables was further tested by considering various other subsamples. For example, the model was estimated for those aged 45-65 and for those with chronic conditions. In each the magnitude of the estimated air pollution coefficient increased and remained significant. In the estimate of males who smoke, however, the air pollution variable was no longer significant. In fact, the F statistic testing whether all of the independent variables together are different from zero was not significant. This result suggests that for smokers, the variance in WLD, especially as it relates to air pollution, may be harder to explain.

For comparative purposes, estimates of the expected annual work days lost for the urban population can be derived from each of the models. Equations (2),(7) and (9) were used to estimate the partial effect on the WLD variable due to a change in air pollution. To generate the annual estimate, two assumptions were used: First, following Freeman[2] a 20% reduction in air pollution

from baseline level as a result of air pollution legislation is assumed. Second, it is assumed that of the approximately 100 million workers in the U.S., 75% are located in urban areas and can be affected by reductions in air pollution. With these assumptions, the OLS model (A) estimates that 52 million WLD are prevented due to the 20% reduction in air pollution[3]. The Tobit model suggests a reduction in WLD of 45.1 million while the logit model suggests 49.6 million less WLD annually due to pollution control. It should be acknowledged that for the latter estimate the effect of pollution on the number of WLD given an episode has occurred, was assumed to be zero following the results of estimation (D).

The similarity of the results from the alternative models lends strong support to the belief that ambient levels of air pollution can significantly affect the incidence of morbidity.

[2] A. Myrick Freeman III, The Benefits of Air and Water Pollution Control: A Review and Synthesis of the Recent Estimates, December 1979.

[3] The details of these calculations are presented in Appendix I.

Appendix I

OLS

From the results of estimation (A):

$b_2 = .00177 \quad \overline{PMEN} = 76.8 \quad W_1 = .2355$

Therefore, the pollution elasticity = .577 and a 20% decrease in particulates would reduce WLD by 11.54%.

There were a total of 530 million WLD in 1976 for those 18-65 years old. If this were the result of a 11.54% reduction, it means that 69 million WLD were saved. Taking 75% of that, for the urban population, totals 52 million WLD.

Tobit

From Eq(7), the change in the probability of any one day being a work loss, due to a change in air pollution is .000118. Therefore, multiplying

.000118 (prob. of a WLD from a unit of air pollution)	x	19.6 (20% decrease for pollution)	X	75 million (urban workers)	x	260 (work days a yr)	=	45.1 million WLD

Logit

From Eq(9).

$dW_3 = b_2 \cdot A \cdot (1 - A)dA$

$= (.00614)(.055)(.945)(19.6) =$

$= .00625 =$ probability of an episode in the 2 week period.

In a year:

$.00625 \times 26 \times 75$ million $= 12.197$ million
$\underline{\times\ 4.07}$ average WLD/episode
49.64 million WLD