A STATISTICAL STUDY OF THE MACROEPIDEMIOLOGY OF AIR POLLUTION AND TOTAL MORTALITY

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ABSTRACT

A statistical analysis of spatial patterns of 1980 U.S. urban total mortality (all causes) was performed, evaluating demographic, socioeconomic and air pollution factors as predictors. Specific mortality predictors included cigarette smoking, drinking water hardness, heating fuel use, and 1978-1982 annual concentrations of the following air pollutants: ozone, carbon monoxide, sulfate aerosol, particulate concentrations of lead, iron, cadmium, manganese, vanadium, as well as total and fine particle mass concentrations from the inhalable particulate network (dichotomous samplers). In addition, estimates of sulfur dioxide, oxides of nitrogen, and sulfate aerosol were made for each city using the ASTRAP long-range transport diffusion model, and entered into the analysis as independent variables. Because the number of cities with valid air quality and water hardness data varied considerably by pollutant, it was necessary to consider several different data sets, ranging from 48 to 952 cities.

The regression models and data were tested for outliers, influential observations, and behavior of residuals. The models which passed these tests showed sulfate aerosol, iron particles, and (to a lesser extent) total particle mass to be associated with mortality, although depending on the data set considered and the choice of variables versus factors to account for demographic effects on mortality, there were variations in the specific pollutants which showed statistical significance.

These results were compared to previous results and data for 1970. 1970 air pollution was not shown to be a better predictor for 1980 mortality than 1980 air pollution. Sulfate was not significant in 1970, but the large standard error for that period does not preclude the 1980 sulfate coefficient for that period as well.

Since the computed sulfate variable was so highly significant in the developmental regression models, the analysis was repeated using computed NO_x and SO_2 data as alternative independent variables. These variables were also highly statistically significant predictors for all-cause mortality. Because of the high degree of similarity of alternative regression models employing different pollutants as predictors, three different bases were employed to compare regression models. Using these criteria, SO_2 , sulfate aerosol, and manganese particles all showed indications of providing better regression fits than the other pollutant variables. Using a two-stage procedure employing mortality "adjusted" for demographics, smoking, poverty, etc., the differences in fit among the various sulfate measures, SO_2 , NO_x , Fe, Mn, fine particles and total particles were minimal. Thus it was concluded that statistical criteria alone will not suffice in defining air pollution relationships with total mortality.

The relatively strong associations (ca. 5 - 10%) shown for 1980 pollution with 1980 total mortality are generally not confirmed by independent studies, for example, in Europe. In addition, the US studies did not find those pollutants with known adverse health effects at the concentrations in question (such as ozone or CO) to be associated with mortality. The question of causality vs. circumstantial association must therefore be regarded as still unresolved.

EXECUTIVE SUMMARY

A statistical analysis of spatial patterns of 1980 U.S. urban total mortality (all causes) was performed, evaluating demographic, socioeconomic and air pollution factors as predictors. Specific mortality predictors that were considered included demographic factors, cigarette smoking, hardness of drinking water, heating fuel use, and annual concentrations of the following air pollutants for 1978-1982: ozone, carbon monoxide, sulfate aerosol, particulate concentrations of lead, iron, cadmium, manganese, vanadium, as well as total and fine particle mass concentrations from the inhalable particulate network (size selective samplers). In addition, estimates of sulfur dioxide, oxides of nitrogen, and sulfate aerosol were made for each of over 900 cities using the ASTRAP long-range transport diffusion model, and entered into the analysis as independent variables (SO_2 and NO_x were evaluated in a subsequent phase of the research). Because the number of cities with valid data on air quality and water hardness varied considerably by pollutant, it was necessary to consider several different data sets ranging from 48 to 952 cities.

The regression models and data were tested for outliers, influential observations, and behavior of residuals. The models which passed these tests showed sulfate aerosol, iron particles, and (to a lesser extent) total particle mass to be associated with mortality, although, depending on the data set considered and the choice of variables or factors to account for demographic effects on mortality, there were variations in the specific pollutants which showed statistical significance.

Table 1 lists the main variables used in this study and some of the statistical findings. The column, "% of mortality variance explained," indicates the degree of relationship between each variable and the mortality variable, without considering how these relationships change as the effects of age, race, poverty, etc., are taken into account through the use of multiple regressions. The regression coefficients express the change in mortality rate (deaths per thousand population) per unit of the variable in question ($\mu\text{g}/\text{m}^3$ for the pollutants). The elasticities are nondimensional regression coefficients, which relate the percentage change in mortality to a percentage change in the independent variable. Note that some of the elasticities express a degree of mortality dependence that is quite different from that given by the bivariate correlations. The range of elasticities is quite similar for most of the pollutants, for example, while the percentages of mortality variance explained vary considerably.

These results were compared to previous results and data for 1970. 1970 pollution was not a better predictor for 1980 mortality than 1980 pollution. The only coefficients which have undergone a statistically significant change since 1970 are those for poverty and the percentage of population 65 years of age or older (borderline significance). Since sulfate was not significant in 1970, the large standard error found does not preclude the 1980 coefficient for that period as well.

TABLE 1

MORTALITY CONTRIBUTIONS AND SENSITIVITIES OF VARIABLES USED

Variable (no. of obs. #)	% Mort. Variance Explained ⁺	Coefficient Range	Elasticity Range (%)
% > 65	69.7	0.54 - 0.83	68 - 104
Birth Rate	0.0	0.13 - 0.29	24 - 54
% Black	5.7	0.004 - 0.027	0.5 - 3.3
% Spanish	1.4	-0.08 - -0.02	-6.3 - -1.6
% Asian	4.4	0.04 - 0.08	1.0 - 2.0
% Poverty	8.1	-0.01 - 0.09	-1.3 - 7.1
% College Grad.	12.2	-0.08 - 0.0	-16.2 - 0
% Same Residence	0.72	0.011 - 0.027	7.2 - 18
% Pop. Change	13.1	-0.005 - -0.02	-1.6 - -7.3
Pop. Density	2.3	0 - 0.007	0 - 3.1
Smoking	2.6	0.007 - 0.018	14 - 37
Water Hardness (457)	1.5	-0.006 - 0.0	-3.3 - 0
% Oil Heat x °Day	2.5	*	*
Sulfate (54-914)	5.1	0.05 - 0.23	2.8 - 13
SO ₂ (914)	2.3	0.035 - 0.038	6.2 - 6.7
NO ₂ (914)	0.16	0.032 - 0.035	6.0 - 6.5
Iron (173)	1.0	0.07 - 0.44	0.8 - 5.1
Manganese (173)	2.9	3.0 - 12.0	1.2 - 4.7
Fine Particles (56)	7.8	0.04 - 0.05	7.8 - 9.7
Total Particles (56)	1.2	0.0 - 0.029	0 - 11.9

Data available for 952 cities unless otherwise indicated

⁺ Per cent of mortality variance explained by the variable, without considering other variables (bivariate correlation).

* Variable was never significant in multiple regressions

Since the computed sulfate variable was so highly significant in the developmental regression models, the analysis was continued using computed NO_x and SO_x data as alternative independent variables. All of the computed pollutant variables were highly significant predictors for all-cause mortality. Because of the high degree of similarity of alternative regression models employing different pollutants as predictors, three different bases were employed to compare regression models. Using these varying criteria, SO_2 , sulfate aerosol, and manganese particles all showed indications of providing better regression fits than the other pollutants. Statistical tests for the degree of fit showed that including a pollutant variable made a significant improvement, but that one could not distinguish among pollutants on this basis. Using a two-stage procedure employing mortality "adjusted" for demographics, smoking, poverty, etc., the differences in fit among the various sulfate measures, SO_2 , NO_x , Fe, Mn, fine particles and total particles were minimal. Further, visual comparison of residual patterns indicated only minimal differences, including comparison with the residuals obtained from a model with no air pollutants as predictors. Thus it was concluded that statistical criteria alone will not suffice in defining air pollution relationships with total mortality.

The relatively strong associations (ca. 5 - 10%) shown for 1980 pollution with 1980 total mortality are generally not confirmed by independent studies, for example, in Europe. In addition, the U.S. studies did not find those pollutants with known adverse health effects at current concentration levels (such as ozone or CO) to be associated with mortality. The question of causality versus circumstantial association must therefore be regarded as still unresolved.

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1.0 INTRODUCTION

The study of long term or chronic health effects of air pollution has long been fraught with difficulty. Early attention focused on air pollution episodes, such as Donora (PA) or the notorious London fogs of the 1950's and 60's, which dealt with short-term or acute effects, primarily SO_2 and particulates. In order to discern long-term effects, it was necessary to compare population health statistics in places with different environments, and such comparisons are often compromised by other population differences that may be related to the sources of air pollution (industrialization, for example). In addition, there may be many other attributes of place that relate to health, if the older literature is to be believed.¹

Lave and Seskin's 1970 cross-sectional (i.e., place-to-place variability) regression analysis² added a new dimension, that of appreciable long-term mortality effects at current U.S. ambient levels (about 9% of annual mortality associated with air pollution). These mortality effects were inferred from multiple linear regression analysis of annual mortality rates in a number of US standard metropolitan statistical areas (SMSAs) in relation to available center city annual air quality levels and a few other explanatory variables. The article talked mostly about "air pollution" as the causal agent; Lave and Seskin's later work³ focused more on the sulfate portion of total suspended particulates (henceforth referred to as "sulfate" or "sulfates" with the chemical symbol $\text{SO}_4=$) as the most important pollutant. This hypothesis gained widespread attention when long-range transport air quality models became available and were used to predict nationwide $\text{SO}_4=$ exposure levels and thus "excess deaths." 50,000 annual excess deaths was the figure often mentioned.

Subsequent cross-sectional regression studies both supported^{4,5} the importance of sulfur oxides and failed to do so,^{6,7} and the scientific community looked to other types of studies for possible confirmation. Even after years of animal and human clinical exposure studies, these long-term health effects are still a matter of debate.^{8,9}

This type of cross-sectional statistical analysis, the so-called ecologic regression analysis, was accepted by some critics and rejected by others. It makes inferences about the outcomes to individuals in terms of the behavior of groups, in this case entire SMSAs or cities, based on very limited information about individual exposures to potential causal agents. In addition, the types of group information that are available often do not include important lifestyle variables such as smoking, exercise, diet, etc. Insurance data have shown that mortality rates can vary by as much as a factor of two to three among groups differing in these lifestyle parameters.¹⁰ Cross-sectional regressions thus must address all the characteristics that vary regionally and which may affect longevity.

Spatial gradients in mortality have been noted by many authors, especially with respect to heart disease, which is the largest cause of death in most U.S. locations. Kleinman et al.¹¹ attempted to explain both regional and urban/suburban gradients in terms of three major risk factors: smoking, serum cholesterol, and blood pressure, based on individual survey data from about 4,500 individuals. They found that after accounting for these three risk

factors, heart disease mortality was 18-20% higher east of about the 96th meridian, and 5-12% higher in urban versus suburban locations. They concluded that "other environmental, life-style, medical care or genetic characteristics must be considered in trying to explain the geographic variations which have been observed for many years."

This report describes an attempt to incorporate the most recent (ca. 1980) mortality and air pollution data in the US, and applies more complete statistical analysis methods to this problem. We draw on a larger group of U.S. cities for our study population, and include additional air pollutants, smoking, and data on drinking water quality as well. However, the mortality data analyzed have been limited to the total deaths reported for each city (all ages, causes, etc.). A special effort was devoted to updating the estimates of the geographic distribution of cigarette smoking because of the established importance of smoking as a factor in both cancer and heart disease mortality. This effort is described in detail in a separate report.¹²

This report begins with a background discussion of cross-sectional regression techniques and the effects of various types of errors. Sources of data are described next, including data on mortality, demographics, smoking, and air pollution, and their descriptive statistics. The regression analysis is presented in three separate phases. Phase I, Section 4, is exploratory in nature and includes a factor analysis as a means of dealing with the many potential explanatory variables (especially the demographic and socioeconomic variables). Section 5, the Phase II regression analysis, presents the development of two specific mortality "models" which were optimized for subsets of cities having data on several pollutants. Section 6 compares 1970 and 1980 regression results, including a cross-validation. Section 7 presents the Phase III regression analysis, which includes two additional computed pollutant variables (SO_2 and NO_x) and develops some formal means of comparing alternative regression models. Section 8 discusses these results in the context of independent findings and similar studies for other time periods and locations. The report ends with the conclusions (Section 9), recommendations for further research (Section 10) and two Appendices. Appendix A presents the air pollution data base, and Appendix B is a discussion of air pollution health effects from a physiological perspective.

2.0 BACKGROUND OF CROSS-SECTIONAL STUDIES

2.1 GENERAL DESCRIPTION

The cross-sectional study attempts to define associations between variables through the analysis of spatial patterns. Typically, annual mortality rates are computed for a number of political subdivisions, appropriate demographic (control) variables are defined and data obtained from Census sources, and air pollution exposures are estimated from extant monitoring data, usually for the same time period. Multiple regression analysis then is used to postulate a "model" for mortality rates; such models have often been used to infer causal relationships between mortality and air pollution. These studies are usually regarded as investigating chronic effects, since no account is taken of daily variations in either mortality or pollution, except as reflected in the annual averages or totals.

Such cross-sectional studies are one of the few ways that chronic health effects in large populations may be studied at reasonable cost. However, there can be problems with this technique with respect to both the data used and the interpretation of results. The study design requires that the average properties of the population groups be interpreted as appropriate to each of the individuals comprising the group. All of the multiple regression variables refer to properties of the group, yet the group mortality rate stems from individuals who died for various individual reasons. This is often referred to as the "ecologic fallacy."¹³ For example, if a group has both a high rate of mortality and high consumption of tobacco (presumably because of an excess of heavy smokers), the ecologic assumption is that the heavy smokers in the group are the ones who died prematurely. An analysis of groups does not permit verification of such assumptions, except as inferred from statistical robustness. When responses are nonlinear, such that an average value of an independent variable does not correspond to the average response, this type of analysis may yield incorrect answers.

In addition, when only one year's data are analyzed it is generally not possible to distinguish between truly long term or chronic effects and the annual sum of short-term or acute effects. For truly long-term effects, previous or historic values of the independent variables (especially air pollution exposures) should be used. Time-series analyses are better suited for the study of short-term effects.

2.2 EFFECTS OF ERRORS IN ESTIMATES OF AIR POLLUTANT EXPOSURE

The accuracy of estimation of air pollution exposure will vary with the size of the political subdivision used and the pollutant. Some primary pollutants, such as TSP, CO, and SO₂, tend to be very local in nature, and concentrations may vary substantially within a few city blocks; in addition, concentrations may vary between indoors and outdoors. Secondary pollutants, such as oxidants and suspended sulfates, may exhibit less spatial variability, although ozone can be strongly attenuated by local NO_x sources.

The effect of errors in the measurement of a variable is to decrease both its statistical significance and its regression coefficient relative to the

true value, assuming that the effect was real in the first place (in the statistical sense). Since concentration patterns of many air pollutants often are collinear, this may mean that the pollutant with the least error in estimation of exposure may appear most statistically significant, even when the real situation is that a mix of pollutants is responsible for the observed effect. In such situations, measurement errors can mask the true effects. However, none of these considerations affects the overriding issue of causality versus association.

This type of error also is associated with the choice of political subdivision, since the larger the unit is in area, the larger are the likely errors in estimating true population exposures (assuming reasonably local pollution sources are present). For example, assume that there is a true relationship (association) between particle concentration and mortality (this need not be a causal relationship, since there may be other aspects of the pollution source to consider, such as occupational factors). Before implementation of the dichotomous sampler network, there were only two measures of particle concentration generally available: TSP (or total particle mass), which often reflects local pollution because it may include particles up to 50 μm in diameter which tend to remain close to the source; and sulfate, which is regional in extent since the particles are much smaller and thus travel further. When small areas (such as cities or census tracts) are used in the analysis, TSP exposures may be reasonably well represented and TSP may be statistically significant. On the other hand, if larger units are used, such as whole counties or Metropolitan Statistical Areas (MSAs), the true TSP effect will likely be masked by the exposure error, since a large number of people have now been "assigned" to the TSP monitor but live so far away that they are not actually exposed to the pollution measured there. Now, if at the same time there is a regional trend towards higher mortality (for whatever reason) in the region of high sulfates, sulfate may become the significant variable when MSAs are the observational unit. This result might appear to have a causal basis in physiology, since small particles such as sulfates can penetrate deeper into the lung, but in this case the result could have also appeared only because a regionally distributed pollutant was matched with a regionally distributed mortality trend. It should be obvious that an MSA-based analysis cannot capture local pollution effects, only regional ones, but that a city-based analysis should be able to detect either type. We chose cities as the observational unit for the study which is reported here. The challenge is to identify city-specific effects and to separate causal regional trends from circumstantial ones.

Mortality rates may be statistically unstable if the population base is too small. One solution to this problem is to use small geographic areas (i.e., central cities) and average the data over several years, which will improve the stability of estimates of both mortality and air pollution exposure. However, for cities over 25,000 population, as studied here, the variability of the annual numbers of deaths (ca. 250) should not be a problem; based on the median city size of about 57,000 people and the mean mortality rate, the sampling variance is only about 1.4% of the total observed variance.

2.3 EFFECTS OF OTHER ERRORS AND CONFOUNDING FACTORS

In order for error in measurement to increase the significance and regression coefficient for a variable, the error must have a selective bias and not just be random noise. An example might be better measuring instruments or characterization of exposure in areas of high mortality rates, compared to areas of lower mortality rates.

Patterns of population migration can also cause errors in estimated pollution exposures and can have confounding regression results. The confounding results from either selective migration of sick people or of the more economically advantaged. In either case, current (local) air quality may not represent the true long-term exposures of current residents.

Other problems can arise in these analyses when unadjusted total mortality data are used (all causes, all ages). Often, only total mortality data will be available for the smaller geographic subdivisions. Age adjustment is probably the most important correction to make, since the probability of dying increases exponentially with age above age 35. If mortality rates are available for detailed age groups, they can be combined into one age-adjusted total rate by reference to a standard population. On the other hand, if only total deaths are available but details are available on the population age distribution, then the expected total number of deaths may be computed on the same basis. In many cases, including the work described here, neither procedure has been followed but surrogate age adjustments were attempted by using a population age descriptor as an independent variable in the multiple regression. "Percentage of population aged 65 and over" is a common choice. If all populations have similarly shaped age distributions, such a choice may be acceptable, but a simple calculation will show, for example, that the regression coefficient for " $\% \geq 65$ " should be numerically equal to the mortality rate for this age group less the rate for the under-65 group. Many of the published studies do not meet this simple test. Similar considerations apply to explanatory variables such as "% nonwhite" or "% poverty."

Other problems have arisen due to collinearity between air pollution and demographic variables, which is always present to a certain extent. This is more of a problem in central cities than in MSAs, but the reduction in collinearity for MSAs is only apparent, not real, since it usually results from the use of central city air quality data to (inadequately) represent an entire SMSA. It is not possible a priori to establish acceptable limits on multicollinearity. In this case it arises from the fact that pollution sources have other types of impacts upon communities. Heavily industrialized cities tend to have demographically different populations than, say, bedroom suburbs for large metropolitan areas. These differences typically include education, smoking habits, race, and income, all of which can have independent effects upon average mortality rates. If such effects are not explicitly represented in the regressions, they may be erroneously (implicitly) represented in part by the air pollution variables. Such problems can only be handled by incorporating the appropriate independent variables in the analysis and then sorting them out by stepwise or other repetitive regression methods. Factor or principal component analysis may be another approach. However, when repetitive specification searches are made, statistical significance levels may be overstated (due to loss of independence).

Such regression methods often appear to be rather ad hoc and have been labelled as "data mining" or "fishing expeditions." The basic problem is lack of a theoretical a priori "model" (in the econometric sense) for human mortality and morbidity as well as lack of appropriate data bases for the known risk factors. For example, risk factors for heart disease, which accounts for a large fraction of all deaths, include smoking, diet, exercise, lung function, and perhaps drinking water hardness and ethnic background. A few of the well-known cross-sectional studies have attempted to represent some of these factors; most have not.

2.4 SPATIAL PATTERNS IN U.S. MORTALITY RATES

Spatial patterns of U.S. mortality rates show some well defined trends.¹⁴ Total mortality from "natural" causes (excluding accidents, suicides, homicide, etc.) is highest for white males in the southeast and in the northeast for females (Figure 1). Heart disease is higher east of the Mississippi and especially in the Southeast coastal plain (Figure 2). Lung cancer (Figure 3) shows very similar patterns, suggesting a common factor which might well be cigarette smoking. However, chronic respiratory disease deaths are higher in the West (Figure 4). These patterns suggest certain associations; the challenge to the analyst is to establish whether such associations are causal and not just circumstantial. Sauer's data are discussed in more detail in Section 8.6.1.

2.5 CRITERIA FOR CAUSALITY

There are no standard tests for causality, and Lave¹⁵ suggested that causality is only a theoretical construct, based on a consensus of scientific opinion. For long-term health studies, establishing causality requires that the clinical effects of the pollutant (see Appendix B, for example) be consistent with the health endpoint in question, that relationships based on spatial patterns be consistent with those based on temporal trends, and that all reasonably plausible confounding variables be accounted for. These caveats should be kept in mind in reviewing the following results.

(a) males

(b) females

Fig 1. Death rates for natural causes (ICDA 000-796) among whites aged 35-74 years (age-adjusted) in 50 lowest and 50 highest rate State economic areas United States, 1968-72 (from Sauer, 1980)

(a) males

(b) females

Fig 2. Death rates for cardiovascular diseases (ICDA 390-448, 780-796) among whites aged 35-74 years (age-adjusted) in 50 lowest and 50 highest rate State economic areas: United States, 1968-72 (from Sauer, 1980)

(a) males

(b) females

Fig 3. Death rates for respiratory cancer (ICDA 160-163) among whites aged 35-74 years (age adjusted) in 50 lowest and 50 highest rate State economic areas: United States, 1968-72 (from Sauer, 1980)

Figure 4. Death rates for chronic respiratory diseases (ICDA 490-493, 517-519) among white males aged 35-74 years (age-adjusted) in 50 lowest and 50 highest rate State economic areas: United States, 1968-72

3.0 DATA SOURCES, DESCRIPTIONS, AND METHODS

3.1 MORTALITY AND SOCIOECONOMIC DATA

Mortality and descriptive population data were obtained for 916 U.S. cities (having valid data entries) from the 1980 Census of Population, as listed in the magnetic tape file for the 1982 City and County Data Book (CCDB).¹⁶ The mortality counts are for 1979, but death rates (per 1000 population) are computed by dividing by the population as estimated on April 1, 1980, which could entail a slight error for cities with rapidly changing population.* The mortality variable used as the dependent variable throughout this analysis is based on all causes of death for all ages, races, and sexes, i.e., total mortality.

The following socioeconomic and demographic (independent) variables were extracted from the CCDB; the rationales for their inclusion are given below:

1. **% $\geq$ 65 (years of age).** Above age 35, mortality rates increase exponentially with age, but the percentage 65 and older is the only useful age statistic available from CCDB. The other age cuts (ages 5 and 25) are too low to influence mortality, and "median age" tends to be collinear with "% $\geq$ 65," which thus would add little new information where it is needed (the age distribution within the 65 and older group).
2. **Racial and ethnic distribution (% black, % Asian, % Hispanic).** Each of these groups tends to have mortality rates different from whites. Thus cities with higher than average percentages of these groups would be expected to have correspondingly different mortality rates for the total population.
3. **% below poverty level.** We feel this is a better income variable than median income, since any effect of income on mortality would be expected to be most obvious at the low end of the scale.
4. **% college graduates.** Education may be a better socioeconomic variable than income since some persons may have low income because they have poor health, not vice versa. Educational attainment, as a socioeconomic indicator, will not be changed by subsequent illness.
5. **% population change since 1970¹ (Δ pop.); % born in state of current residence (% resident).** These two variables are intended to capture population stability and migration, which can be important since ill health can be a factor in the decision to migrate and since long-term exposure to air pollution would be affected by migration.
6. **Ratio of males to females (%).** This variable could be important since males have higher mortality rates than females.

* The mortality rate listed in CCDB for Wilmington, DE (20.4 per thousand) was found to be in error and was adjusted to 14.4, based on local information. A similar error was found in the 1970 data for Wilmington.⁶

7. **Birth rate (per 1000 population).** Birth rates can influence mortality rates in two ways: first, through infant mortality. Second, if the census has undercounted the population base, the errors in population counts would inflate both birth and death rates by the same percentage. Both birth and death counts are believed to be accurate.
8. **% with homes heated by gas or by oil, times annual degree days.** These two variables are intended to capture space heating effects on air quality, including possible indoor air quality. These variables are really pseudo-pollution variables. Also, homes heated by gas are more likely to have gas cooking stoves, a possible source of indoor air pollution.
9. **City size variables: population density, log of population.** City size may have direct effects on mortality through changes in the pace of life, or indirect effects regarding the representativeness of monitoring data on air quality.

3.2 DRINKING WATER QUALITY DATA

Data on drinking water hardness and source (surface versus ground) were obtained from a data base compiled by the National Institutes of Health.¹⁷ These data were for ca. 1970 and earlier, but it was felt that the values would be reasonably stable over time. Data were available for 450 cities out of the above set of 916. Previous studies^{18,19} implicated soft water as a contributing factor in heart disease, primarily for males. Figure 5 is a map of the water hardness data, based on a subset of cities also having data on air quality.

3.3 AIR QUALITY DATA

3.3.1 Measured Data

Data on air quality were obtained from the U.S. Environmental Protection Agency as quarterly averages for the years 1978-82 from the Storage and Retrieval of Aerometric Data (SAROAD) system. Those quarters with enough data to provide valid quarterly averages then were combined to produce grand "annual" averages and medians for the entire period. This procedure maximizes the number of cities in the data set for each pollutant and is based on the premise that the long-term temporal trends are small. The only pollutant with obvious appreciable trends is lead. The following data sets resulted (number of cities in brackets):

1. **Ozone** [167] (annual means or medians of daily maximum hourly concentrations) (ppm). See Figure 6 for an isopleth map of these data.
2. **Carbon monoxide** [161] (ppm)
3. **Lead** [189] ($\mu\text{g}/\text{m}^3$)
4. **Vanadium** [167] ($\mu\text{g}/\text{m}^3$)
5. **Iron** [173] ($\mu\text{g}/\text{m}^3$) (Figure 7)

Figure 5. Total hardness of drinking water (ppm as CaCO_3).

Figure 6. Median daily maximum ozone (ppm).

Figure 7. Iron in suspended particulate ($\mu\text{g}/\text{m}^3$).
Note: distributions are not necessarily continuous.

6. **Manganese** [173] (ug/m^3)
7. **Cadmium** [173] (ug/m^3)
8. **Measured Sulfate** [138] (ug/m^3) (Figure 8)
9. Particle data from the dichotomous sampler network (1979-83): **Total mass** and **fine particle mass**, [n=56]; **total sulfate**, **fine particle sulfate** [n=54] (ug/m^3); samples taken every 3 or every 6 days.²⁰

Measurements of sulfate were restricted to 1978 and 1982, since the use of alkaline filters in 1979-81 created an artifact due to SO_2 conversion on the filter.²¹ Either median or mean concentrations of these measured pollutants were used in the regressions, depending upon which was more statistically significant. The final regression models used median values exclusively.

3.3.2 Modeled Air Quality Data

The intersecting set containing all these pollutants has too few cities for a robust statistical analysis (42 cities, listed in Table 1) so the data set was augmented with a sulfate variable that was estimated by model calculations using the ASTRAP model.²² Values were obtained by interpolation for all cities, except that measurements (California Air Resources Board) were used for Southern California since the ASTRAP model is not parameterized to reproduce the highly reactive atmosphere of the South Coast Air Basin. Figure 9 is a map of the ASTRAP data as originally computed.

In the Phase III Analysis (Section 7), computed values for SO_2 and NO_x were added to the analysis, also based on the ASTRAP model. ASTRAP computations deal only with anthropogenically generated pollutants, mainly from fuel combustion or smelter emissions sources. Measurements of course reflect all sources, including for example such natural sulfates as CaSO_4 , etc., which are less likely to affect health.

3.3.3 Visibility Data

Airport visibility data from 1976-81 for 129 cities were made available through the courtesy of Dr. Bart Ostro (EPA). Thirty-two of these locations corresponded to dichotomous sampler cities but the correlation between visibility and fine particulate concentrations was low (0.385). Dropping one outlier (Fresno) only improved the correlation to 0.626. We therefore decided not to use these data as surrogate measures of air quality.

3.4 CIGARETTE SMOKING DATA

Cigarette consumption was estimated from state sales taxes for three periods: 1955, 1969 and 1980.^{6,12} These estimates are based on regression analysis on state-level sales data (per capita for the population aged 18 and over), using various economic and demographic variables as predictors. Lower sales taxes in adjoining states were found to be an important factor in explaining cigarette sales differences. These regression results were then used to predict cigarette consumption in each state. We could not derive

Figure 8. 1978, 1982 composite mean measured sulfate (SAROAD stations) ($\mu\text{g}/\text{m}^3$).

Table 1. Forty-Two Cities Having Available Data on All 14 Pollutants

State	City
Alabama.....	Birmingham
Arizona.....	Phoenix
California.....	Bakersfield
	Fresno
	Los Angeles
	San Francisco
	San Jose
Colorado.....	Denver
Connecticut.....	Hartford
District of Columbia.....	District of Columbia
Florida.....	Tampa
Indiana.....	Gary
	Indianapolis
Kansas.....	Wichita
Minnesota.....	Minneapolis
Mississippi.....	Jackson
Nebraska.....	Omaha
Nevada.....	Reno
New Jersey.....	Camden
New Mexico.....	Albuquerque
New York.....	Buffalo
North Carolina.....	Charlotte
Ohio.....	Akron
	Cincinnati
	Cleveland
	Dayton
	Steubenville
	Youngstown
Oklahoma.....	Oklahoma City
Oregon.....	Portland
Pennsylvania.....	Bethlehem
	Philadelphia
Rhode Island.....	Providence
Tennessee.....	Nashville
Texas.....	Dallas
	El Paso
	Houston
Utah.....	Salt Lake City
Virginia.....	Norfolk
Washington.....	Seattle
	Spokane
West Virginia.....	Charleston

Figure 9. ASTRAP - computed sulfate aerosol ($\mu\text{g}/\text{m}^3$).

cigarette consumption data at finer geographic resolution and thus were forced to assume uniform consumption throughout each state with discontinuities at the borders. These errors are likely to lead to an underprediction of the effect of smoking on mortality.

Earlier analyses of smoking patterns²³ typically found large urban-rural differences, and it has long been assumed that city people are more likely to smoke. In the study of 1980 smoking data, SMSA tobacco sales from the 1977 Census of Retail Trade were compared to state-wide sales from the same source, and a consistent relationship was found, amounting to an annual urban-statewide difference of about 5 packs per year per person (out of 185). This small but statistically significant difference suggests that regional smoking patterns are now probably more important than urban-rural differences within regions, which supports the use of state-level data in the analysis of mortality effects.

Finally, a comparison was made of our estimates of cigarette consumption with independent state level surveys from 29 states on the percentage of people who smoke (smoking prevalence). The correlation coefficient relating these two measures was only about 0.5 (explaining 25% of the variance). Possible explanations for this poor correspondence include variations in amount consumed per smoker and underreporting by those responding to the survey. We prefer to use consumption data rather than prevalence, since heavy smokers have a much higher relative mortality risk than light smokers, and since consumption may reflect the possible effects of passive (involuntary) smoking. Cigarette consumption rates are analogous to air pollution emission rates.

For an analysis of chronic health effects, it is not clear whether current cigarette smoking rates or some time integral is the appropriate metric (the same question exists for air quality). For this reason, we considered two possible smoking variables: the 1980 data, as described above, and 1969 data.⁶ Because of strong collinearity between the two ($r=0.48$), regression models are limited to one or the other (or alternatively, the average, "SMOK78").

3.5 DESCRIPTIVE STATISTICS

3.5.1 Outliers

Descriptive statistics were obtained for each variable and the distributions were examined for bimodality and outliers. In Table 2 we present the descriptive statistics for the demographic variables and Table 3 contains these statistics for the pollutants. Outliers were identified by Tukey's exploratory criterion.²⁴ This procedure involves defining outliers in terms of the upper and lower quartiles, rather than by the standard deviation (see Figure 10) (which only applies to normally distributed variables). A value is considered to be a "possible outlier" if it is outside the range defined by $(LQP - 1.5 IQR, UQP + 1.5 IQR)$ where LQP and UQP denote the lower and upper quartile point respectively. A value is considered to be a "definite outlier" if it is outside the interval from $LQP - 3 IQR$ to $UQP + 3 IQR$. A variable is considered to have a bimodal distribution if more than 15 values were designated as "definite" outliers.

Table 2. Descriptive Statistics for the Demographic and Socioeconomic Variables

Variables Considered	Mean	S.D.	Minimum	Maximum	Median	LQP	UQP	IQR	Outlier Limits*	
									Possible	Definite
Mortality rate (per 1000)	9.14	3.32	1.70	28.70	9.2	6.8	11.5	4.7	17.6	24.
% ≥ 65 years old	11.50	5.30	1.70	51.80	11.4	8.2	14.2	6.0	23.2	32.2
% Asian	2.27	6.28	0.00	79.80	0.9	0.5	1.9	1.4	4.0	6.1
% Spanish	7.20	12.23	0.10	93.00	2.3	1.0	8.0	7.0	18.4	28.0
% Black	11.18	15.57	0.00	95.60	3.6	0.9	15.9	15.0	38.4	60.0
% Poverty	11.90	6.75	1.40	43.30	11.1	6.8	15.9	9.1	29.6	43.2
% College Graduate	18.57	10.06	2.40	68.70	15.8	11.8	22.4	10.6	38.3	54.2
% Population change since 1970	28.94	227.57	-27.20	6346.	4.8	-5.8	22.4	28.2	(-48.01, 64.60)	106.9
Population density	40.05	34.08	0.50	397.10	30.6	20.5	48.6	28.1	90.8	132.9
Sex ratio (%)	92.45	7.79	74.70	220.50	91.8	87.9	96.3	8.4	108.8	121.4
% Resident in same state	59.62	16.78	5.50	90.80	62.9	47.7	72.6	24.9	5.5	90.8
Birth rate (per 1000)	16.99	5.34	2.90	52.90	16.5	13.6	19.6	6.0	28.6	37.6
Size of population (log pop.)	4.76	0.34	4.40	6.85	4.7	4.5	4.9	0.4	5.4	6.1
Size of city (log land area)	1.30	0.40	0.00	3.20	1.2	1.0	1.5	0.5	(0.25, 1.8)	3.0
1969 Smoking (pack/person yr)	194.96	24.81	115.00	303.00	202.0	174.0	209.0	35.0	(121.5, 261.5)	31.4
1980 Smoking (pack/person yr)	186.43	23.15	125.21	324.55	181.9	174.5	198.6	24.1	(138.2, 234.9)	271.1
% Gas heat x 10 ⁴	31.3x10 ⁴	20.7x10 ⁴	0.00	90.1x10 ⁴	24.4x10 ⁴	14.9x10 ⁴	52.6x10 ⁴	37.7x10 ⁴	109.3x10 ⁴	105.8x10
% Oil heat x 10 ⁴	66.4x10 ⁴	11.6x10 ⁴	0.00	70.7x10 ⁴	0.49x10 ⁴	201	7.2x10 ⁴	7.2x10 ⁴	18x10 ⁴	28.8x10
Water Hardness (ppm)	113.29	102.50	4.00	626.00	86.0	38.5	144.0	105.5	302.3	460.5

* Lower outlier limits are zero unless indicated.

Table 3. Descriptive Statistics for the Pollution Variables

Variable Considered	Mean	S.D.	Minimum	Maximum
Computed $\text{SO}_4^{=}$	5.1	3.5	0.1	14.8
Computed SO_2	16.1	10.9	0.28	46.4
Computed NO_x	17.0	10.6	0.73	50.9
SAROAD SO_4 (1)	9.5	3.5	2.392	34.2
SAROAD SO_4 (2)	9.2	3.3	2.000	34.2
SO_4 Fine (Dichot.) (1)	4.9	2.9	0.820	12.1
SO_4 Fine (Dichot.) (2)	3.9	2.3	0.840	11.6
SO_4 Total (Dichot.) (1)	5.3	3.0	0.860	12.9
SO_4 Total (Dichot.) (2)	4.3	2.4	1.030	12.3
Cadmium (1)	0.005	0.010	0	0.079
Cadmium (2)	0.004	0.008	0	0.040
Carbon monoxide (1) (ppm)	1.966	0.942	0.300	5.91
Carbon monoxide (2) (ppm)	1.916	0.989	0.300	7.00
Iron (1)	1.118	1.685	0.2	4.3
Iron (2)	1.052	0.611	0.23	4.3
Lead (1)	0.460	0.272	0.098	2.760
Lead (2)	0.418	0.265	0.088	2.760
Manganese (1)	0.040	0.032	0.020	0.219
Manganese (2)	0.036	0.027	0.020	0.188
Ozone (1) (ppm)	0.048	0.017	0.019	0.195
Ozone (2) (ppm)	0.047	0.017	0.018	0.195
Vanadium (1)	0.025	0.04	0.002	0.027
Vanadium (2)	0.019	0.034	0.002	0.345
Fine Particle Mass (Dichot.) (1)	20.5	6.4	10.3	42.0
Fine Particle Mass (Dichot.) (2)	17.7	6.0	7.2	37.1
Total Particle Mass (Dichot.) (1)	41.2	12.4	20.4	94.6
Total Particle Mass (Dichot.) (2)	37.5	11.9	21.9	98.6
Fine Particle Lead (Dichot.) (1)	0.3	0.30	0.010	2.6
Fine Particle Lead (Dichot.) (2)	0.2	0.11	0.010	2.6

Note: (1) Mean of site data

(2) Median of site data (all value in $\mu\text{g}/\text{m}^3$ except as noted).

Table 3. Descriptive Statistics for the Pollution Variables (Cont'd)

Variables Considered	Median	LQP	UQP	IQR	Outlier Limits	
					Possible	Definite
Computed $\text{SO}_4^{=}$ (1)	4.2	2.0	7.7	5.7	16.25	24.
SAROAD $\text{SO}_4^{=}$ (1)	9.98	7.80	12.7	4.9	20.	27.5
SAROAD $\text{SO}_4^{=}$ (2)	9.80	7.50	12.0	4.5	18.8	26.8
SO_4 Fine Dichot (1)	4.395	2.192	7.110	4.918	14.487	21.9
SO_4 Fine Dichot (2)	3.750	2.005	5.260	3.255	10.143	15.0
SO_4 Total Dichot (1)	4.970	2.567	7.487	4.930	14.892	22.3
SO_4 Total Dichot (2)	3.965	2.232	5.697	3.465	10.894	16.1
Cadmium (1)	0.002	0.001	0.003	0.002	0.006	0.009
Cadmium (2)	0.001	0.001	0.003	0.002	0.006	0.009
Carbon Monoxide (1)	1.868	1.233	2.580	1.347	4.600	6.6
Carbon Monoxide (2)	1.800	1.100	2.600	1.500	4.900	7.1
Iron (1)	0.922	0.675	1.298	0.623	2.233	3.2
Iron (2)	0.907	0.656	1.270	0.614	2.191	3.1
Lead (1)	0.401	0.289	0.562	0.273	0.9715	1.38
Lead (2)	0.359	0.259	0.495	0.236	0.809	1.20
Manganese (1)	0.029	0.023	0.040	0.017	0.066	0.09
Manganese (2)	0.027	0.020	0.037	0.017	0.0625	0.088
Ozone (1)	0.045	0.040	0.051	0.011	0.0625	0.084
Ozone (2)	0.046	0.036	0.053	0.017	(0.011, 0.079)	0.10
Vanadium (1)	0.010	0.005	0.036	0.031	0.083	0.12
Vanadium (2)	0.006	0.005	0.021	0.029	0.045	0.069
Mass Fine Dichot (1)	19.6	15.3	24.5	9.2	38.3	51.9
Mass Fine Dichot (2)	16.9	12.9	21.6	8.7	34.7	47.8
Mass Total Dichot(1)	38.6	33.5	46.1	12.6	65.1	84.0
Mass Total Dichot(2)	34.6	30.6	43.2	12.5	61.9	80.7
Lead Time Dichot (1)	0.220	0.150	0.300	0.150	0.525	0.75
Lead Time Dichot (2)	0.170	0.125	0.245	0.120	0.425	0.60

Note: (1) Mean
 (2) Median
 (3) (all value in $\mu\text{g}/\text{m}^3$ except as noted).

Figure 10. Nomenclature for quartile analysis.

Several demographic variables had a large number of definite outliers, examples being the oil heat variable with 90 and % Asian with 63. In the case of the pollutants, cadmium and manganese had a large number of cities which were definite outliers. In future analyses we might consider these variables as categorical, i.e., high and low cadmium, as well as analysing the values themselves. For those variables having fewer than 15 outliers one could consider regression analysis with jackknifing (omitting one outlying city at a time).

3.5.2 Correlations.

The next task was to obtain the pairwise correlations for the demographic set and for the pollution set. These are reported in Tables 4 through 6 respectively. Before obtaining these correlations a factor analysis was done (with a Varimax rotation), ordering the correlation matrix so that sets of collinear variables appear together. It is interesting that on combining pollutants with demographic variables we observed % resident and % Asian to be correlated with SO₄⁼ variables, and that cadmium is correlated with % Spanish (both high in Texas) and birthrate with % college graduate. Several pollutants are not highly correlated with demographic variables, including NO_x, iron, manganese and total particle mass. Correlations among the pollutant variables are discussed below.

3.6 DATA SETS ANALYZED

As is often the case with this type of analysis, the analysis had to be performed on various subsets because data were missing for one or more variables. For the Phase I analysis, these subsets were arranged to permit hypothesis testing on each of the limiting variables, generally one at a time. The usual hypothesis was that a given variable has a statistically significant effect on mortality in the direction expected (positive for air pollutants; negative for drinking water hardness). A few corollary hypotheses were examined, dealing with possible sources of confounding such as regional trends or interactions among variables.

The bulk of the Phase II statistical analysis was performed for a set of 159 cities that had relatively complete air monitoring data in addition to water hardness data. These data are listed in Appendix A. Large cities that are missing from this list (primarily because of missing air quality data) include: Boston, Washington, Miami, New York, Pittsburgh, St. Louis, Austin, Richmond and Milwaukee. However, the median population of the group is about 130,000; the total population studied is about 41 million or about 25% of the total U.S. urban population.

The data set of 159 cities was selected primarily for the availability of metals, which were complete for Mn, Fe, Pb, and Cd; only six cities were missing vanadium data. However, 41 cities lacked CO data; 48, ozone data; 106, total and fine particulate mass; 108, total and fine sulfur portions of particulate mass. These latter two items were obtained from the dichotomous sampler network.

Twenty-two smaller cities (but greater than 100,000 population) were excluded because of missing air quality data. The 53 cities having dichotomous

Table 4. Correlation Matrix (All Variables)

	MR	BR	A65	BLK	ASN	SPN	POOR	GRAD	DELPOR	MF	RES	DENS	POP	LAND	SMOK69	SMOK80	SMOK78		
MR	MORTALITY RATE	1.0000	.0031	.8347**	.2381**	-.02112**	-.01178**	.2850**	-.03489**	-.03618**	-.05798**	.0850*	.1530**	-.0308	-.0654	.1317**	.1442**	.1599**	
BR	BIRTH RATE	.0031	1.0000	-.02530*	.1765**	.0353	.4125**	.3053**	-.03266**	.1492**	.0938*	-.0839*	-.0783*	.0268	.0607	-.01466**	-.01494**	-.01719**	
A65	% > 65 YEARS	.8347**	-.02530**	1.0000	.0664	-.01584**	-.01329**	.1152**	-.02360**	-.0805*	-.05459**	-.0898*	.1242**	.0034	-.0794**	.1594**	.2275**	.2205**	
BLK	% BLACK	.2381**	.1765**	.0664	1.0000	-.01005**	-.01043**	.6144**	-.01796**	-.0656	-.02993**	.0970*	.1204**	.1585**	.1065**	-.04089	.0419	-.0058	
ASN	% ASIAN	-.02112**	.0353	-.01584**	-.01005**	1.0000	.1262**	-.01024**	.0964*	.0072	.1848**	-.01242**	.1143**	.0225	-.01071	-.01185**	-.02707**	-.02202**	
SPN	% HISPANIC	-.01178**	.4125**	-.01329**	-.01043**	.1262**	1.0000	.1985**	-.01843**	-.0092	.1191**	-.02982**	.3142**	.0819*	.0012	-.0963*	-.01165**	-.0073	
POOR	% POVERTY	.2850**	.3053**	.1152**	.6144**	-.01024**	.0964*	1.0000	-.01237**	-.0939*	-.01776**	.0893*	.1285**	.1284**	.0586	-.01559**	-.0222	-.01048**	
GRAD	% COLLEGE GRADUATES	-.03489**	-.03266**	-.02360**	-.01796**	.0964*	-.01843**	-.01237**	1.0000	.0379	.1850**	-.02268**	-.01293**	-.01088	.0086	-.00538	-.0453	-.0570	
DELPOR	% POPULN CHANGE SINCE 1970	-.03618**	.1492**	-.0805*	-.0656	.0072	-.00092	-.0939*	.0379	1.0000	.0765*	-.01041**	-.0700	-.0237	.0225	-.0227	.0260	.0009	
MF	MALE/FEMALE RATIO	-.05798**	.0938*	-.05459**	-.02993**	.0970*	.1848**	-.01242**	.1143**	.0225	1.0000	-.02597**	-.01026**	-.0304	.0615	-.0228	-.01015**	-.00698	
RES	% RESIDENT IN SAME STATE IN 1970	.0850*	-.0839*	-.0898*	.0970*	-.02982**	.0893*	-.02268**	-.01041**	-.02597**	1.0000	-.01152**	-.0333	-.0333	-.0551	-.02849**	-.01107**	-.02303**	
DENS	POPULATION DENSITY	.1530**	-.0783*	.1242**	.1204**	.1143**	.3142**	.0819*	.0012	-.0963*	.1285**	1.0000	.2163**	-.01406**	.2402**	-.01058**	.0840*	.0840*	
POP	1980 POPULATION	.0308	.0268	.0034	.1585**	.1065**	.0225	.0819*	.0012	-.0963*	.1285**	1.0000	.2163**	-.01406**	.2402**	-.01058**	.0840*	.0840*	
LAND	LAND AREA	-.0654	.0607	-.0794**	.1594**	.2275**	.2205**	.0058	-.0073	-.00698	-.0222	-.01048**	-.0330	1.0000	.3603**	-.0093	-.0074	-.0096	
SMOK69	1969 SMOKING	.1317**	-.01466**	.1594**	-.04089	-.01185**	.0963*	-.01559**	-.00538	-.0227	.0228	-.02849**	.2402**	-.0093	1.0000	.5201**	.8812**	.8812**	
SMOK80	1980 SMOKING	.1442**	-.01494**	.2275**	.0419	-.02707**	.0963*	-.0227	-.00538	-.0227	.0228	-.02849**	.2402**	-.0093	.5201**	1.0000	.8621**	.8621**	
SMOK78	AVERAGE SMOKING	.1599**	-.01719**	.2205**	-.0058	-.02202**	.0963*	-.00538	-.0227	.0228	-.02849**	.2402**	-.0093	-.0093	.8812**	.8621**	1.0000	.8812**	
GASDAY	% GAS HEAT x DEGREE DAYS	-.01328**	-.00970*	-.01559**	-.01794**	-.01870**	-.02836**	-.0259	-.0874*	.0306	.4143**	-.00572	-.0370	-.0073	-.0074	-.0076	-.00265	-.0589	
OILDAY	% OIL HEAT x DEGREE DAYS	.1580**	-.02646**	.1581**	.0068	-.01152**	-.01168**	.0378	-.01000*	-.0701	-.0189**	.2167**	.2603**	.0233	-.0504	.1279**	.0022	.0747	
HARD	WATER HARDNESS	-.01231*	.2003**	-.01121*	-.01305*	-.00948	.1052	-.01238*	-.00379	.0767	.0937	-.00011	-.01108*	.0120	-.00524	-.00330	0.0497	0.0497	
SO4C1	COMPUTED SO4= (1)	.2118**	-.02821**	.1057**	.2295**	-.02089**	-.02580**	.0875*	-.01106**	-.0375	-.02002**	.3300**	.2366**	.0514	-.00898*	.1962**	.2024**	.2284**	
SO4C2	COMPUTED SO4= (2)	.2255**	-.03365**	.1124**	.2031**	-.02264**	-.02401**	.0892*	-.01540**	-.0636	-.02622**	.4358**	.3063**	.0397	-.00982*	.1884**	.1214**	.1790**	
SO2	COMPUTED SO2	.1506**	-.02633**	.0399	.1156**	-.01306**	-.00730	-.0533	-.01473**	-.0614	-.01614**	.3017**	.3850**	.0551	-.01189**	.2827**	.0811*	.2127**	
NOX	COMPUTED NOX	-.0400	-.01230**	-.01040**	-.00052	-.0222	.2433**	-.01809**	-.00467	-.0653	.0078	.0040	.4681**	.0894*	-.01177**	.3380**	-.00678	.1631**	
SO4M	SAROAD SO4=	.4331**	-.0918	.2733**	.3003**	-.02802**	-.02150*	.3335**	-.04361**	-.03884**	-.03694**	.4398**	.1088	-.0298	-.01894*	.1505	.1260	.1530	
SO4IP	IP SO4=	.3195*	-.01470	.1815	.5974**	-.02418	-.02280	.3686**	-.02933*	-.04546**	-.03967**	.3037*	.2621	.1437	-.00888	.1225	.2348	.1953	
FP	FINE PARTICLES (IP)	.2788*	-.0841	.1957	.2747*	-.01677	-.01267	.1285	-.01495	-.03585**	-.03570**	.2344	.1720	.1167	-.01086	.1657	.2555	.2377	
TP	TOTAL PARTICLES (IP)	.1085	.2445	-.03352	.0291	.1790	.5268**	.1222	-.0504	.0552	.0823	-.02606	.0994	.2324	.1262	.0442	-.0480	.0050	
FPB	FINE LEAD (IP)	-.02068	.2203	-.02489	.0291	.1790	.5268**	.1222	-.0504	.0552	.0823	-.02606	.0994	.2324	.1262	.0442	-.0480	.0050	
PB	TOTAL LEAD (SAROAD)	.0613	.1528	.0208	.0606	.1003	.2019*	.1071	-.0018	.0219	-.00781	-.02130**	.2221**	.1741*	.0489	.0513	-.01525	-.00572	
FE	IRON (SAROAD)	.0990	.1607	-.00299	.0144	-.0181	.1958*	.0051	-.02624**	.0286	.0932	-.01400	-.00397	.1479	.0424	-.00335	.0067	-.0157	
MN	MANGANESE (SAROAD)	.1696	-.00213	.0630	.0496	-.0161	-.0168	-.0416	-.03135**	-.01183	-.00495	.0550	.0080	-.00581	.0306	.0568	.0500	.0500	
CAD	CADMIUM (SAROAD)	-.02567**	.2172*	-.02370**	.0013	-.00749	.4757**	.1467	-.00671	.1526	.1709	.1013	-.01971*	.0651	.0999	-.02422**	.1427	-.00598	
O3	OZONE (SAROAD)	-.01434	.1148	-.00918	-.01493	.0621	.3197**	-.01197	.0617	.1881*	.0673	-.01752*	-.00430	-.00124	-.00354	.0953	-.00802	.0059	
CO	CARBON MONOXIDE (SAROAD)	-.01534	-.01229	-.00573	-.00964	.1149	.1403	-.01152	.1567	.0504	.0702	-.03083**	.0599	.0360	-.00118	.1134	.0443	.0898	
		GASDAY	OILDAY	HARD	SO4C1	SO4C2	SO2	NOX	SO4M	SO4IP	FP	TP	FPB	PB	FE	MN	CAD	O3	CO
MR		-.01328**	.1580**	-.01231*	.2118**	.2255**	-.01506**	-.0400	.4331**	.3195*	.2788*	.1085	-.02068	.0613	.0990	.1696	-.02567**	-.01434	-.01534
BR		-.00970*	-.02646**	.2003**	-.02821**	-.03365**	-.02633**	-.01230**	-.00918	-.01470	-.0841	.2445	.2203	.1528	.1607	-.00213	.2172*	.1148	-.01229
A65		-.01559**	.1581**	-.01121*	.1057**	.1124**	.2031**	-.01040**	.2733**	.1815	.1957	-.00352	-.02489	.0208	-.00299	.0630	-.02370**	-.00918	-.00573
BLK		-.01794**	-.0068	-.01305*	.2295**	.2031**	.1156**	-.00052	.3003**	.5974**	.2747*	-.00010	.0291	.0606	.1003	.0490	.0013	-.01493	-.00964
ASN		-.01870**	-.01152**	-.00948	-.02089**	-.02264**	-.01306**	.0222	-.02802**	-.02418	-.01677	-.01351	.1790	.0552	-.00181	.01611	-.00749	.0621	.1149
SPN		-.02836**	-.01168**	.1052	-.02580**	-.02401**	-.00730	.2433**	-.02150*	-.02280	-.01267	.2286	.5268**	.2019*	.1958*	.00168	.4757**	.3197**	.1403
POOR		-.02393**	.0378	-.01238*	.0875*	.0892*	-.0533	-.01809**	.3335**	.3686**	.1285	.0124	.1222	.1071	.0051	-.00416	.1467	-.01197	-.01152
GRAD		.0259	-.01000*	-.00379	-.01106**	-.01540**	-.00467	-.04361**	-.02933*	-.04546**	-.03967**	-.0504	-.0018	-.02624**	-.03135**	-.00671	.1567	.1567	.1567
DELPOR		-.0874*	-.00701	.0767	-.00375	-.00636	-.00614	-.00653	-.03884**	-.04546**	-.03585**	-.00280	.0552	.0219	.0286	-.01183	.1526	.1881*	.0504
MF		.0306	-.01989**	.0937	-.02002**	-.02622**	-.01614**	.0078	-.03694**	-.03967**	-.03570**	-.00626	.0823	-.00781	.0932	-.00495	.1709	.0673	.0702
RES		.4143**	.2167**	-.00011	.3300**	.4358**	.3017**	.0040	.4398**	.3037*	.2344	.0049	-.02606	-.02130**	-.01400	.0550	.1013	-.01752*	-.03083**
DENS		-.00572	.2603**	-.01108*	.2366**	.3063**	.3850**	.4681**	.1088	.2621	.1720	-.00778	.0994	.2221**	-.00397	-.00589	-.01971*	-.00430	.0599
POP		-.00370	.0233	-.00294	.0514	.0397	.0551	.0894*	-.00298	.1437	.1167	.0210	.2324	.1741*	.1479	.0080	.0651	-.00124	.0360
LAND		-.00073	-.00504	.0120	-.00898*	-.00982*	-.01189**	-.01117*	-.01894*	-.00888	-.01086	-.00114	.1262	.0489	.0424	-.00581	.0999	-.00354	-.00118
SMOK69		-.00746	.1279**	-.00524	.1962**	.1884**	.2827**	.3380**	.1505	.1225	.1657	.1012	.0442	.0513	-.00335	.0306	-.02422**	.0953	.1134
SMOK80		-.00265	-.00022	-.00330	.2024**	.0811*	-.00678	.1260	.2348	.2555	.1371	-.00480	-.01525	.0067	.0568	.1427	-.00802	.0443	.0443
SMOK78		-.00589	.0747	-.00497	.2284**	.1790**	.2127**	.1631**	.1530	.1953	.2377	.1354	.0050	-.00572	-.00157	.0500	-.00598	.0059	.0059
GASDAY		1.0000	.1765**	.3973**	.2290**	.2290**	.2204**	-.00690	.0663	.0709	.0073	-.0277**	-.01336	.1048	.3318**	-.02783**	-.02831**	-.01636	.01636
OILDAY		-.01765**	1.0000	-.03485**	.2558**	.2558**	.2558**	-.00907	.0663	.0709	.0073	-.0277**	-.01336	.1048	.3318**	-.02783**	-.02831**	-.01636	.01636
HARD		.3973**	-.03485**	1.0000	-.0387	-.0387	-.0387	-.00603	-.00832	.0045	-.00054	.1230	.2452	.0169	-.00509	.1317	.1887*	.1235	.0353
SO4C1		.2290**	.2558**	-.0387	1.0000	.8597**	.8010**	.3513**	.7227**	.7234**	.5634**	.1074	-.01351	.0684	.0361	.1935*	-.03165**	-.02296**	-.00481
SO4C2		.1879**	.4938**	-.02000**	.8597**	1.0000	.8840**	.4549**	.7063**	.7775**	.5989**	.0938	-.01065	.0769	.0057	.1492	-.03140**	-.00993	.0053
SO2		.2204**	.2880**	-.00603	.8010**	.8840**	1.0000	.7077**	.7223**	.7831**	.6572**	.2125	-.00363	.1439	.1573	.2807**	-.02787**	-.0264	.0208
NOX		-.00690	.1922**	-.00832	.3513**	.4549**	.7077**	1.0000	.3925**	.5365**	.5109**	.1998	-.01748	.2383**	.0840	.0516	-.00392	.2731**	.1402
SO4M		.0663	-.00907	.0045	.7227**	.7063**	.7223**	.3825**	1.0000	.7343**	.7146**	.2453	-.00289	.0193	.1664	.2351*	-.01330	-.01100	-.01546
SO4IP		.0709	.1163	-.00054	.7234**	.7775**	.7831**	.5365**	.7343**	1.0000	.7								

Table 5. Pollutant Correlation Matrix
(Number of city pairs given in parenthesis)

	Sh. SO4	SAR. SO4	Dich. FSO4	Dich. TSO4	Dich. FMass	Dich. TMass	Iron	Manganese	Cadmium	Vanadium	Lead	Ozone	CO
Sh. SO4	1.0	0.760 (128)	0.702 (54)	0.696 (54)	0.590 (56)	0.059 (56)	0.046 (163)	0.202 (163)	-0.319 (163)	0.268 (157)	0.008 (163)	-0.239 (114)	-0.065 (126)
Sar. SO4		1.0	0.725 (48)	0.724 (48)	0.697 (50)	0.220 (50)	0.166 (138)	0.235 (138)	-0.133 (138)	0.133 (138)	-0.088 (138)	-0.105 (103)	-0.155 (110)
Dich. FSO4			1.0	0.994 (54)	0.750 (54)	0.245 (54)	0.207 (54)	0.413 (54)	-0.167 (54)	0.243 (54)	-0.130 (54)	-0.119 (47)	-0.170 (51)
Dich. TSO4				1.0	0.762 (54)	0.271 (54)	0.211 (54)	0.403 (54)	-0.166 (54)	0.260 (54)	-0.122 (54)	-0.129 (47)	-0.160 (51)
Dich. FMass					1.0	0.605 (56)	0.331 (56)	0.401 (56)	-0.112 (56)	0.028 (56)	0.194 (56)	0.133 (49)	-0.028 (53)
Dich. TMass						1.0	0.686 (56)	0.528 (56)	0.079 (56)	-0.144 (56)	0.275 (56)	0.188 (49)	-0.073 (53)
Iron							1.0	0.780 (173)	0.074 (173)	0.132 (167)	0.307 (173)	-0.081 (119)	0.111 (126)
Manganese								1.0	-0.041 (173)	0.241 (167)	0.079 (173)	-0.269 (119)	-0.065 (126)
Cadmium									1.0	0.083 (167)	-0.092 (173)	0.054 (119)	-0.263 (126)
Vanadium										1.0	0.005 (167)	-0.076 (119)	0.027 (126)
Lead											1.0	0.281 (119)	0.274 (126)
Ozone												1.0	0.167 (106)
CO													1.0

Table 6. Correlation Between Selected Pollutants and Demographic Variables

	Computed SO ₄ ⁼	Measured SO ₄ ⁼	Computed NO _x	Total Particle Mass	Iron	Ozone	CO
% Resident in same State	0.38	0.40	0.00	0.005	-0.14	-0.18	-0.32
% Asian	-0.25	-0.29	0.02	-0.14	-0.02	0.06	0.12
% Black	0.23	0.	-0.01	0.0	0.01	-0.15	-0.10
% > 65	0.12	0.26	-0.10	-0.04	-0.03	-0.09	-0.05
Birthrate	-0.31	-0.07	-0.12	0.24	0.16	0.11	-0.13
Log Population	0.01	-0.12	0.09	0.07	0.01	-0.06	0.07
Smoking	0.19	0.16	0.16	0.10	-0.13	0.09	0.12
Water Hardness	-0.13	-0.07	-0.08	0.24	0.13	0.06	-0.14
% Poor	0.23	0.33	-0.18	0.01	0.14	-0.12	-0.13
% College	-0.13	-0.37	-0.05	-0.28	-0.26	0.06	0.15
Pop. Density	0.26	0.12	0.47	-0.08	-0.04	-0.04	0.06

samplers in addition to SAROAD and water hardness data are also listed in Appendix A. These include both major and smaller cities and cover most regions of the country fairly well, with the possible exception of New York State and New England. The total population of the 53 cities is 23 million people, or about 13% of the total U.S. urban population.

3.7 RELIABILITY OF AIR QUALITY EXPOSURE ESTIMATES

Appendix A gives the numbers of observations and the numbers of monitoring sites for the SAROAD air quality data. These vary from thousands of observations for the gaseous pollutants with hourly data (CO and O₃), to fewer than ten observations for some of the metals and for measured sulfate. The numbers of monitoring stations assigned to a city range from one to seventeen; the average number of monitoring sites was less than three for all measured pollutants. The dichotomous sampler data are generally from only one or two samplers per city,* with some exceptions.²⁰

3.7.1 Measured versus Computed Sulfate

One way to assess the adequacy of monitoring coverage from a statistical point of view is to use the number of observations or monitors as a weighting factor in the regression analysis. Another method is to examine the correlation between the mean value and the number of observations, i.e., do high concentration values occur in locations with fewer or more observations than average? This was found to be the case for cadmium (positive correlation) and

*Cities with more than one dichotomous sampler include Birmingham (5), Phoenix (3), Los Angeles (4), San Jose (2), San Francisco (2), Denver (2), Hartford (2), Chicago (3), Boston (2), Baltimore (2), Detroit (2), Minneapolis (2), St. Louis (2), Durham (2), Buffalo (3), Cleveland (3), Portland (2), Philadelphia (14), Pittsburgh (3), Houston (3), Salt Lake City (2), Seattle (2).

vanadium (negative correlation). Only for cadmium was the number of monitor locations correlated (positively) with the mean value. Use of weighting factors in regression is limited to a single choice of weighting factor per regression, which could lead to difficulties in choosing the "best" weight factor and regression. As an exploratory step, we calculated the correlation between ASTRAP computed $\text{SO}_4^{=}$ and measured (SAROAD) values, using four alternative weighting factors:

Correlations Between Computed and Measured $\text{SO}_4^{=}$ Values

<u>Weighting Factor</u>	<u>Correlation Coefficient</u>
none	0.760
no. measured observations	0.743
(no. measured observations) ^{1/2}	0.783
no. monitor sites	0.761
(no. monitor sites) ^{1/2}	0.762

Since none of these correlation coefficient values are statistically different from one another, we conclude that random noise in the sulfate measurements is not a major consideration in comparison to the spatial variation from city to city. The weighted mean values were not significantly different from the unweighted values.

3.7.2 Other Correlations Among Pollutants

Other correlations among pollutants measures are given in Table 5, for the "159" city data set. They are grouped according to particle characteristics or source type, for example, all the various sulfate measures and industrial sources: total mass (dichotomous) iron, and manganese. Manganese is also significantly correlated with sulfates and small particles. Cadmium and manganese are largely uncorrelated with the other pollutants, with the exception of manganese versus vanadium. Vanadium is associated with sulfate, perhaps reflecting heavy fuel oil as a common source (or, more speculatively, catalytic action of manganese in oxidizing SO_2 to $\text{SO}_4^{=}$).

Lead, ozone, and carbon monoxide are all associated with mobile sources, although not exclusively so. The correlations among this group are much more modest than in the other groups. Lead was also associated with total particle mass and with iron, perhaps reflecting a coarse particle mode.

3.7.3 Cities with High Levels of Specific Pollutants

Based on the 159 city data set, the following cities rank high in terms of median pollutant concentration levels.

CO: Los Angeles, Pasadena, San Jose, Denver, Des Moines, Boise, Indianapolis, Chattanooga, Spokane. This may well be as a result of the monitor locations, in relation to traffic density.

Ozone: San Bernadino, Burbank, Los Angeles, Pasadena. Those rankings result from the well-known tendency for Southern California to have high photochemical reactivity.

Lead: Ashland (KY), Grand Rapids, Ogden, Salt Lake City. These high values appear to be mostly due to industrial (rather than mobile) sources.

Vanadium: This appears to be a bimodal data set; cities with heavy fuel oil use, mostly along the Eastern Seaboard have higher values than those in the interior or on the West coast. Exceptions are Bakersfield (CA), several cities in Illinois, and Greensboro (NC).

Iron: High values are found in Phoenix, Bakersfield, Granite City (IL), Ashland (KY), Detroit, Steubenville, and Youngstown, OH. Most of these cities have heavy industrial manufacturing.

Manganese: High values are found in Granite City (IL), East Chicago (IN), Gary (IN), Hammond (IN), Ashland (KY), Detroit, Steubenville (OH), Youngstown (OH) and Sioux Falls (SD). These are largely manufacturing cities, as was the case with iron.

Cadmium: The highest values are all found in Texas; it is not known whether this may be an artifact of either sampling or analytical procedures.

Measured SO₄: The highest values are found in Gadsden (AL), Atlanta, Evansville, and New Albany (IN), Terre Haute (IN), Ashland (KY), New Orleans; Flint, Grand Rapids and Lansing (MI); Akron, Canton, Cleveland, Steubenville, Toledo, Youngstown (OH); Chattanooga, Norfolk, Kenosha (WI), and Charleston (WV). As can be seen by comparing Figures 8 and 9, the patterns of measured sulfate are much less uniform than the computed values. It is not known whether these local differences are real or artificial.

Total Particle Mass (Dichotomous Samples): The highest values are found in Phoenix, Bakersfield, Pasadena, Denver, Gary (IN), and Ashland (KY). These locations appear to have a mixture of natural and anthropogenic sources of particles.

Fine Particle Mass (Dichotomous Samples): The highest values are found in Birmingham, Pasadena, Gary (IN), Ashland (KY), Akron and Cleveland, which are mostly manufacturing locations.

Total & Fine Particle Sulfur Mass (Dichotomous Samples): The highest values are found in Gary (IN), Buffalo, Akron, Cleveland, and Chattanooga, all manufacturing locations.

3.8 STATISTICAL METHODS

Most of the statistical computations upon which this analysis is based were performed at the State University of New York at Stony Brook using standard statistical package programs: BMDP²⁵ and SPSS.²⁶ These calculations include bivariate correlations, multiple linear regressions, factor analysis, and analysis of the frequency distributions of the residuals from these regressions. The spatial autocorrelation analysis was performed at Brookhaven.

For the Phase I analysis, the regressions were run in the stepwise mode, both forward and backward, with a tolerance of 0.15 on the significance of a variable for retention in the prediction equation. This allows a 15% chance of falsely rejecting a variable in the backward mode (in which all variables are included initially and then the least significant ones are sequentially eliminated), or a 15% chance of wrongly entering a variable in the forward mode. Because of collinearity in the data set, backward and forward modes do not always end up with the same model specification.

For the regressions, we require a probability less than 5% that the results might be due to chance for a result to be "statistically significant." Some cases where $0.05 < p < 0.10$ are described as "borderline significant."

As seen in Tables 4, 5 and 6, there are some collinear variables in this data set, which requires special care in selecting candidate regression models. Factor analysis was one of the statistical methods used as a result of this situation. More details on the factor analysis procedure are given below.

4.0 REGRESSION ANALYSES ON THE 1980 DATA SET: PHASE I

In the first phase of the analysis, each pollutant was considered separately. This was done for several reasons, the first being that the ratio of the number of variables (35) to the number of cities having complete data sets (42) or data on at least two pollutants (about 160 cities on average) was far less than the desired rule of thumb value of 5. Moreover, some of these pollutants were known to be collinear. In particular, SAROAD sulfate, computed sulfate, total sulfur (dichotomous) and fine particle mass all measure the level of sulfate and hence were not considered simultaneously. To further allow for the number of variables considered, two regressions were run for each pollutant. The first involved stepwise regression of all demographic variables plus each pollutant, and the second involved stepwise regression of the demographic factor scores plus each pollutant. Each of these pairs of regressions was done on the following two data sets: (1) the cities having data on the pollutant (one at a time) and (2) the cities having data on the pollutant of interest plus values for drinking water hardness. Thus a total of four regression analyses were done for the data sets defined by each pollutant.

4.1 FACTOR ANALYSIS

The factor analysis approach was used to reduce the very large and partially collinear set of demographic variables to a smaller set of uncorrelated variables, called factor scores. The factor analysis results were based on the correlations between the demographic variables obtained from the entire set of cities using "pairwise deletion". This means that wherever a city had information on two variables, that city was included in the computation of the correlation for these two variables. The resulting correlations then were based on the maximum possible number of observations. Two factor analyses were done. The first used data from all the 916 cities and included all demographic variables. The second was done using the 450 cities for which data on water hardness were available; these data were included in the demographic variable set.

The factor analysis (set (1) above) which excluded the hardness variable resulted in seven significant factors. After VARIMAX rotation of the factors, we could consider the loadings for each variable on each factor as reflecting what that factor is measuring. The first factor had highest loadings on log of the land area and log of the city population, hence was named "the city size" factor. The second factor loads highest on percentage over 65 (negative loading) and ratio of males to female (positive loading) and is the "age/sex" factor. The third factor loads highest on percentage black and percentage below poverty level and is the black/poverty factor. The fourth had high negative loadings on the gas heat variable and percentage of long term residents, the fifth factor was the population density/oil heat factor and the sixth the smoking/Asian factor (since it assigns essentially equal weight to the 1969 and 1980 smoking figures and a smaller negative loading on the percentage of Asians). The seventh factor could be seen as the birthrate factor since the loadings are high and positive for percentage Spanish and the birthrate and negative for percentage of college graduates.

The factor analysis of the cities with water data available gave somewhat similar results; the city size factor was present with high loadings on the

same variables observed above on exclusion of water. The same was true for the black/poverty, age/sex, smoking/Asian, and birthrate factors. However, the addition of water hardness resulted in density and population change forming a factor and in water hardness and oil heat forming an additional factor.

4.2 REGRESSION RESULTS

In Table 7 we report the results of stepwise regressions on the variables and on the factors. On comparing the two columns, with the exception of fine particle mass and fine lead, one sees essentially the same results for each pollutant regardless of whether factor scores are used to represent the demographic variables or the demographic variables themselves. That is, significant positive coefficients are observed for iron, manganese, SAROAD sulfate, computed sulfate, total sulfate and fine sulfate with both approaches. For the regression which included computed sulfate, the analyses with factor scores estimated significant coefficients for all factors except city size. All coefficients were positive except the coefficient for the age/sex factor scores. The adjusted R^2 was 0.68. On using the individual demographic variables, significant ($p < 0.10$) positive coefficients are observed for percentage over 65, the birthrate, smoking in 1969, percentage poor, and percentage of long-term residents. Significant negative coefficients are observed for the change in population, the male to female ratio, percentage Spanish, gas heat and oil heat variables. The resulting adjusted R^2 was 0.87.

Table 7. Results of the Regressions on

(a) All Demographic **Factors** plus One Pollutant at a Time
(b) All Demographic **Variables** Plus One Pollutant at a Time

All Cities With Given Pollutant Available

Pollutant*	(a) Using Demographic Factors Coefficient $\pm$ SE (p)	(b) Using Demographic Variables Coefficient $\pm$ SE (p)	n
Cadmium	-66 $\pm$ 11 (0.000)	-1.9 $\pm$ 9.2 (0.84)	173
CO	-0.268 $\pm$ 0.12 (0.031)	-0.160 $\pm$ 0.09 (0.082)	194
Fe	0.59 $\pm$ 0.16 (0.000)	0.56 $\pm$ 0.10 (0.000)	172
Mn	12.8 $\pm$ 3.5 (0.000)	10.6 $\pm$ 2.29 (0.000)	172
Pb	0.49 $\pm$ 0.46 (0.28)	0.35 $\pm$ 0.33 (0.29)	214
O ₃	-30.6 $\pm$ 6.90 (0.000)	-13.7 $\pm$ 5.7 (0.017)	211
Vanadium	-1.7 $\pm$ 4.8 (0.73)	0.70 $\pm$ 3.1 (0.82)	165
Fine Particle Mass	0.026 $\pm$ 0.03 (0.39)	0.036 $\pm$ 0.02 (0.086)	81
Total Particle Mass	0.002 $\pm$ 0.01 (0.89)	0.01 $\pm$ 0.01 (0.34)	68
Fine Pb	-4.40 $\pm$ 1.68 (0.011)	-0.06 $\pm$ 1.4 (0.97)	77
SO ₄ (SAROAD)	0.129 $\pm$ 0.04 (0.001)	0.100 $\pm$ 0.03 (0.000)	202
SO ₄ (Computed)	0.081 $\pm$ 0.02 (0.000)	0.084 $\pm$ 0.02 (0.000)	872
Total SO ₄ (dich.)	0.272 $\pm$ 0.07 (0.001)	0.188 $\pm$ 0.05 (0.000)	78
Fine SO ₄ (dich.)	0.276 $\pm$ 0.08 (0.002)	0.200 $\pm$ 0.05 (0.000)	78

* Median readings used

p = level of statistical significance.

In Table 8 we report the results of stepwise regressions for the data set for cities with drinking water data. There are significant positive coefficients for iron, manganese, computed sulfate, total sulfate, and fine sulfate with both demographic variables and factor scores as the covariates. Vanadium has significant positive coefficients only if factor scores are used as the covariates, and fine mass and SAROAD sulfate have significant positive coefficients only if the demographic variables themselves are used as covariates. The regression for computed sulfate included all factors except the hardness/oil heat and the density/population change factors. All other factors had significant positive coefficients except the long term resident/gas heat and the city size factor. The adjusted R^2 was 0.79. In the analysis of these cities with water data which included all demographic variables plus hardness values plus computed sulfate, four demographic variables had significant positive coefficients. They were (in order of entry) percentage over age 65, birthrate, percentage poor, and the 1969 smoking variable. Five variables had significant negative coefficients. These were the percentage Spanish, the percentage college graduate, oil heat, hardness, and population change variables. The adjusted R^2 was 0.87.

Table 8. Results of the Regressions on

(a) All Demographic **Factors** plus One Pollutant at a Time
 (b) All Demographic **Variables** Plus One Pollutant at a Time

All Cities With **Water Hardness Data** Plus Pollutant Available

Pollutant*	(a) Demographic Factors Coefficient $\pm$ SE (p)		(b) Demographic Variables Coefficient $\pm$ SE (p)		n
Cadmium	-60.5 $\pm$ 11.5	(0.000)	-7.96 $\pm$ 10.29	(0.4)	169
CO	-0.14 $\pm$ 0.1	(0.18)	-0.15 $\pm$ 0.09	(0.091)	185
Fe	0.48 $\pm$ 0.1	(0.001)	0.55 $\pm$ 0.1	(0.000)	168
Mn	8.2 $\pm$ 3.2	(0.011)	10.5 $\pm$ 2.3	(0.000)	168
Pb	0.71 $\pm$ 0.4	(0.110)	0.35 $\pm$ 0.35	(0.31)	214
O ₃	-13.9 $\pm$ 4.9	(0.005)	-9.99 $\pm$ 4.2	(0.018)	199
Vanadium	4.6 $\pm$ 2.7	(0.087)	-0.03 $\pm$ 3.24	(0.992)	161
Mass Fine	0.023 $\pm$ 0.02	(0.30)	0.03 $\pm$ 0.02	(0.08)	68
Mass Total	0.016 $\pm$ 0.01	(0.14)	0.01 $\pm$ 0.01	(0.10)	68
Fine Pb	1.35 $\pm$ 1.37	(0.33)	-0.19 $\pm$ 1.1	(0.86)	64
SO ₄ (SAROAD)	0.04 $\pm$ 0.03	(0.22)	0.04 $\pm$ 0.02	(0.052)	197
SO ₄ (Computed)	0.15 $\pm$ 0.02	(0.000)	0.12 $\pm$ 0.02	(0.000)	418
Total SO ₄ (dich.)	0.18 $\pm$ 0.07	(0.08)	0.19 $\pm$ 0.05	(0.001)	65
Fine SO ₄ (dich.)	0.16 $\pm$ 0.07	(0.023)	0.18 $\pm$ 0.05	(0.001)	65

* Median readings used

p = level of statistical significance.

4.3 CONCLUSIONS FROM PHASE I ANALYSIS

Taken individually, many of the pollutant variables are significant with positive coefficients, although some are significant and negative. Clearly,

both results cannot be valid (at least in a causal sense) and multiple pollutant regressions are the next step to clarify the findings. The most significant pollutants (+), sulfate, iron, manganese, are consistent across all four sets of regressions, although some of the negative ones also display this consistency.

None of the mobile-source pollutants showed significant positive coefficients. In the case of lead and CO, the monitoring data could be from sites not representative of true population exposures. Ozone is highest in Southern California, where age-adjusted mortality rates are average. It is also possible that the ozone monitoring data are not representative, since they are average daily maximum values (as opposed to annual averages) and since urban ozone may be depressed by local NO_x sources.

5.0 PHASE II REGRESSION ANALYSES

The exploratory and regression analyses described above were followed by investigations of the outliers and of the birthrate variable, and by a set of "planned" stepwise regression analyses. Outliers which could be seen as resulting from clearly unreliable data or as being artifacts were deleted from the data set. All subsequent analyses reported in this section were done on this reduced data set. The birthrate variable which was originally included to account for an undercount of the population in the low income cities was investigated for the validity of this hypothesis. The planned regressions were built around stepwise entry of pollutants based on physical significance as well as statistical significance. These analyses and the results are described below.

5.1 DELETION OF OUTLIERS

Table 9 shows the outliers which were deleted and the reason for doing so. Then, pairwise correlations were obtained for the relevant variables and compared to those obtained prior to the deletion of these cities. In general, removal of these values made little change in the pairwise correlations. When differences were observed, they were usually in the form of an increase in the absolute value of the correlation coefficient. This is as one would expect, since errors in data usually result in an increase in the proportion of variance due to deviations from the linear model, which causes a decrease in correlation.

Table 9. Outliers Deleted From Second Stage of Analysis

City	Variable Deleted	Reason for Deletion
Burbank, California	CO	Data anomalously high, probably due to sampler being in a high traffic location, not representative of residential areas.
	Pb	Only 1 year of data; Suspiciously high value.
Glendale, California	O3	Only 1 year of data; Suspiciously high value.
Granite City, Illinois	Vanadium	Only 1 year of data; Suspiciously high value.
Lawrence, Indiana	All	Anomalously low birth and death counts. No measured air quality data available.
Norwalk, Connecticut	CO	Data anomalously high, probably due to sampler in high traffic locations, not representative of residential areas.
Stamford, Connecticut	CO	Data anomalously high, probably due to sampler in high traffic locations, not representative of residential areas.

5.2 INVESTIGATION OF THE BIRTHRATE VARIABLE

The estimated effect of birthrate on mortality was nearly an order of magnitude higher than expected if it were representing solely the census undercount (20-30% versus a few percent). If this variable is indeed proportional to census undercount then one would expect it to be a better predictor of mortality in cities with high poverty than in very affluent cities. It was also of interest to determine whether the significance of the pollution variables was dependent on the birthrate variable.

To investigate the effect of the birthrate variable in various strata, we determined the top, middle and bottom 33rd percentiles of the poverty and Spanish variables. We then defined a city to be in the High Poor/High Spanish stratum if its poverty and Spanish variables were both in the top 33% of the observed values. The values for these percentiles were 14% and 5%, respectively. Similarly the cut-off values for the Low Poor/Low Spanish groups were 8% and 1.4%, respectively.

Separate regressions were run on the High Poor/High Spanish cities and the Low Poor/Low Spanish cities with all demographic variables (excluding % Poverty and % Spanish variables). In the 92 Low Poor/Low Spanish cities, the birthrate variable was non significant, as might be expected if the effect being measured was indeed a surrogate for undercount. In the 106 High Poor/High Spanish cities, the birthrate coefficient was significant ($b + S.E. = 0.12 \pm 0.025$; $p < 0.00005$), also in accordance with this hypothesis.

This apparent interaction (between poverty and birthrate) was not observed in regressions in the marginal strata. In the 304 cities in the bottom 33% of the sample with respect to the percentage Spanish (less than 1.4% Spanish population), Low Spanish cities, the estimated birthrate coefficient is 0.18 ± 0.02 ; $p < 0.0005$ whereas in cities in the top 33rd percentile for % Spanish (more than 5% Spanish population), the coefficient is quite similar at $(0.14 \pm 0.01, p < 0.00005)$. Similarly, in the 288 cities in the bottom 33% of the sample with respect to poverty, the estimated birthrate coefficient is 0.12 ± 0.02 ($p < 0.0005$), essentially identical to the estimate for the 317 High Poor cities in the top 33rd percentile for poverty (0.12 ± 0.01 ; $p < 0.00005$). Moreover, the interaction terms birth x Spanish and birth x Poverty were not significant, in a separate linear regression. In addition, all of the birthrate coefficients for these subsets were significantly lower than those for the entire data set.

Thus, while comparison of the first two regressions (High Poor/High Spanish versus Low Poor/Low Spanish) indicated that the birthrate variable might in some way account for undercount in the poor cities, the results of the second set of regressions are more equivocal, so that it may be of interest to investigate the results of regressions which do not include the birthrate variable. Stepwise regressions with all demographic variables except birthrate, plus each of the $SO_4^{=}$ measures, gave no consistent pattern. On excluding the water variable ($n = 872$) and the birthrate variable, the computed $SO_4^{=}$ term was nonsignificant ("beta value" = 0.02; $p = 0.21$), whereas when birthrate was included, a highly significant coefficient for the computed $SO_4^{=}$ is estimated (0.08 ± 0.02 ; $\beta = 0.08$; $p < 0.0005$). However, including the water variable (418) and excluding the birthrate variable resulted in only a slight drop in the computed $SO_4^{=}$

coefficient from that observed in the corresponding analysis with birthrate (from 0.113 to 0.08 ± 0.02): the coefficient remains significant in both cases. Interestingly, the water variable never enters as a significant term, leading us to conclude that the relationship between computed $\text{SO}_4^{=}$, birthrate, and mortality is different in the cities for which we have water data than in those for which we do not. The coefficients for Total Sulfur Mass (STD) and Fine Particulate Mass (MFD) are unaffected by the inclusion or exclusion of the birthrate variable. In the analyses which included the birthrate variable the coefficients for these pollutants are significant both in the entire set (of 78 cities for STD and 81 cities for MFD) and in the subset with water data (65 for STD and 68 for MFD). On exclusion of the birthrate variable from the four regressions the coefficient shows little change and remains significant. We thus concluded that subsequent models should include the birthrate variable, even if its meaning is not fully understood, since it contributes substantially to the overall goodness of fit.

5.3 STEPWISE ENTRY WITH RESPECT TO POLLUTANT TYPE

Rather than build a model based solely on statistical significance, it was decided to build a model based on pollutant variables selected from groups of similar species. This was done in part because of the large amount of missing data for many pollutants, which reduced the data set to a sample of 42 cities, if all pollutants are considered simultaneously. Pollutants were entered in a stepwise manner as illustrated in the flow chart given in Table 10.

The pollutants were grouped according to their source (as in Table 5), the four groups being (1) Fine Particles (sulfates) (2) Large Particles (3) Industrial Particles (4) Heating Fuel Variables and (5) Mobile Source (Automotive) Pollutants. The intent was to select the "best" pollutant variable from each group based on availability of data and on statistical significance. Each group was investigated in a stepwise manner, beginning with the fine particles (sulfates). During this phase of the analysis, the demographic variables were entered in a stepwise fashion, as needed to optimize the overall model fit. These regression analyses were done with and without the water variable. The results are summarized in Tables 11 to 15. The resulting "final" model included computed $\text{SO}_4^{=}$ and iron, in agreement with the Phase I analysis. Both pollutants were available in 159 cities. Table 16 shows the final model obtained from this analysis; most of the variables are significant and the pollutant variables are highly significant.

5.4 INVESTIGATION OF THE FINAL PHASE II MODEL

5.4.1 All Possible Subset Regression Analysis

The final model was investigated first by doing "all possible subset" regression on the 159 cities having data on iron, computed $\text{SO}_4^{=}$ and water hardness (listed in Appendix A). All demographic variables were considered, one change being that the average of the 1969 and the 1980 smoking values were used instead of two separate variables. All subsequent analyses followed suit since regressions using either 1969 smoking, 1980 smoking or the average resulted in models with identical R^2 values and significant and essentially equal coefficients for whichever smoking variable was included. Moreover, earlier factor analysis had indicated that equal weighting of these two values was appropriate.

Table 10. Flowchart For Planned Stepwise Regressions

I. Stepwise Regressions with all demographic variables plus a selected sulfate variable.

Possible sulfate variables are:

- (1) Computed SO₄ (NSO₄): n=872 without inclusion of water variable;
n=418 on inclusion of water variable
- (2) SAROAD SO₄(LSO₄): n=187 without inclusion of water variable;
n=182 on inclusion of water variable
- (3) Fine Particle Mass (Dichotomous Samplers) (MFD):
n=81 without inclusion of water variable;
n=68 on inclusion of water variable
- (4) Sulfate Portion of Total Particle Mass (Dichotomous Samplers)(STD):
n=78 without inclusion of water variable;
n=65 on inclusion of water variable

I. Select Best Sulfate Variable

(A) Is NSO₄ coefficient significant?

No Yes

(B) Is LSO₄ coefficient significant?

No Yes

(C) Is MFD coefficient significant?

No Yes

(D) Is STD coefficient significant?

No Yes

SULFATE=NONE

SULFATE=STD

SULFATE=MTD

SULFATE=LSO₄

SULFATE=NSO₄

To Step II

To Step II

To Step II

To Step II

To Step II

Table 10. Flowchart For Planned Stepwise Regressions (cont.)

II. Stepwise Regressions with all demographic variables plus
SULFATE VARIABLE PLUS (Total Mass) PARTICLE VARIABLE. (MTD)

(A) Is MTD Coefficient Significant?

III. Stepwise Regressions with all demographic variables plus
SULFATE AND PARTICLE VARIABLES PLUS INDUSTRIAL POLLUTANT VARIABLE.
INDUSTRIAL POLLUTANT VARIABLES ARE:

- (1) Iron (Fe)
- (2) Manganese (Mn)

(A) Is Fe coefficient significant?

Table 10. Flowchart For Planned Stepwise Regressions (cont.)

IV. Stepwise Regressions with all demographic variables plus
SULFATE, PARTICLE, INDUSTRIAL POLLUTANT VARIABLE, PLUS FUEL VARIABLE.
FUEL VARIABLES ARE:

- (1) Vanadium(VANADIUM)
- (2) Gas Heat(GASDAY)
- (3) Oil Heat(FUELDAY)

Are any fuel variable coefficients significant?

V. Stepwise Regressions with all demographic variables plus
SULFATE, PARTICLE, INDUSTRIAL VARIABLES AND FUEL VARIABLES PLUS AUTOMOTIVE
POLLUTANT

AUTOMOTIVE POLLUTANTS ARE:

- (1) Lead (Pb)
- (2) Carbon Monoxide (CO)
- (3) Ozone (O₃)

Are any coefficients significant?

Table 11. Results of Planned Stepwise Regressions:
Results of Stepwise Entry of Demographic Variables
Plus Various Sulfate Measures, One at a Time¹

Sulfate Coefficient $\pm$ S.E. (level of significance, ² sample size)			
Data Set-----> Sulfate	Cities With Water Data	All Available Cities	Elasticity
(1) Computed SO₄ (NSO ₄)	0.11$\pm$0.02 (0.000, n=418)	0.08$\pm$0.02 (0.000, n=872)	0.061 0.045
(2) SAROAD SO ₄ (LSO ₄)	0.07 $\pm$ 0.02 (0.004, n=182)	0.10 $\pm$ 0.02 (0.000, n=187)	0.07 0.10
(3) Fine Particle Mass (MFD)	0.05 $\pm$ 0.02 (0.000, n= 68)	0.04 $\pm$ 0.02 (0.000, n= 81)	0.096 0.077
(4) Total Sulfate Mass (STD)	0.19 $\pm$ 0.05 (0.000, n= 65)	0.20 $\pm$ 0.05 (0.000, n= 78)	0.088 0.094

¹See Table 10 for description of planned stepwise regressions.

²Level of significance = 0.000 denotes $p < 0.0005$

³Elasticity for variable $x_i = b_i \left(\frac{x_i}{y_i} \right)$

Variable in bold selected for next step

Table 12. Results of Planned Stepwise Regressions:
Results of Stepwise Entry of Demographic Variables
Plus Computed Sulfate Plus Particle Mass Variable¹

Pollutant Coefficients $\pm$ S.E. (if Variable Enters Stepwise) (level of significance ² , sample size)			
Data Set----->	Cities With Water Data	All Available Cities	
Coefficient for: Computed SO ₄	0.10 $\pm$ 0.03 (0.001, n=67)	0.12 $\pm$ 0.03 (0.000, n=80)	
Total Particle Mass (MTD)	(+) ³ (0.37, n=67)	(-) (0.535, n=80)	

¹See Table 10 for description of planned stepwise regressions

²Level of significance = 0.000 denotes $p < 0.0005$

³Sign of partial correlation reported if variable did not enter.

Table 13. Results of Planned Stepwise Regressions:
Results of Stepwise Entry of Demographic Variables
Plus Computed Sulfate Plus Industrial Pollutants One at a Time¹

Coefficients $\pm$ S.E. (if Variable Enters Stepwise) (level of significance ² , sample size)					
Data Set----->	Cities With Water Data		All Available Cities		Elasticity
Regr. with---->	Iron	Manganese	Iron	Manganese	
<u>Coefficient for:</u>					
Computed SO ₄	0.09 $\pm$ 0.02 (0.000, n=159)	0.11 $\pm$ 0.02 (0.000, n=159)	0.08 $\pm$ 0.02 (0.000, n= 163)	0.11 $\pm$ 0.02 (0.000, n=163)	0.06
Industrial Pollutants:					
(1) Iron	0.45 $\pm$ 0.10 (0.000, n=159)	X ³	0.41 $\pm$ 0.11 (0.000, n=163)	X	0.05
(2) Manganese	X	9.9 $\pm$ 2.25 (0.000, n=159)	X	9.3 $\pm$ 2.23 (0.000, n=163)	0.04

¹See Table 10 for description of planned stepwise regressions

²Level of significance = 0.000 denotes p<0.0005

³X denotes variable not entered

Variables in bold selected for next step.

Table 14. Results of Planned Stepwise Regressions:
Results of Stepwise Entry of Demographic Variables
Plus Computed Sulfate and Iron Plus Fuel Variables, One at a Time¹

Coefficient $\pm$ S.E. (if Variable Enters Stepwise) (level of significance, ² sample size)						
Data Set----->	Cities With Water Data			All Available Cities		
Regression with--> Coefficient for:	Vanadium	Gas Heat	Fuel Heat	Vanadium	Gas Heat	Fuel Heat
Computed SO ₄ =	0.08 $\pm$ 0.02 (0.000,n=153)	0.09 $\pm$ 0.02 (0.000,n=159)	0.09 $\pm$ 0.02 (0.000,n=159)	0.08 $\pm$ 0.02 (0.000,n=157)	0.08 $\pm$ 0.02 (0.000,n=163)	0.08 $\pm$ 0.02 (0.000,n=163)
Iron	0.43 $\pm$ 0.10 (0.000,n=153)	0.45 $\pm$ 0.10 (0.000,n=159)	0.45 $\pm$ 0.10 (0.000,n=159)	0.41 $\pm$ 0.10 (0.000,n=157)	0.41 $\pm$ 0.11 (0.000,n=163)	0.41 $\pm$ 0.11 (0.000,n=163)
Fuels:						
(1) Vanadium	(+) ³ (0.541,n=153)	X ⁴	X	(+) (0.68,n=157)	X	X
(2) Gas Heat	X	(+) (0.56,n=159)	X	X	(+) (0.81,n=163)	X
(3) Oil Heat	X	X	(+) (0.96,n=159)	X	X	(-) (0.98,n=163)

¹See Table 10 for description of planned stepwise regressions

²Level of significance = 0.000 denotes $p < 0.0005$

³Sign of partial correlation reported if variable did not enter

⁴X denotes variable not entered.

Table 15. Results of Planned Stepwise Regressions:
Results of Stepwise Entry of Demographic Variables
Plus Computed Sulfate and Iron, Plus Automotive Pollutant Variables, One at a Time¹

Coefficient $\pm$ S.E. (if Variable Enters Stepwise) (level of significance, ² sample size)						
Data Set----->	Cities With Water Data			All Available Cities		
Regression with-->	Lead	CO	Ozone	Lead	CO	Ozone
Coefficient for:						
Computed SO ₄	0.10 $\pm$ 0.02 (0.000,n=159)	0.06 $\pm$ 0.26 (0.03,n=116)	0.06 $\pm$ 0.03 (0.024,n=111)	0.11 $\pm$ 0.02 (0.000,n=163)	0.05 $\pm$ 0.03 (0.07,n=119)	0.06 $\pm$ 0.03 (0.000,n=114)
Iron	0.48 $\pm$ 0.10 (0.000,n=159)	0.44 $\pm$ 0.11 (0.0001,n=116)	0.52 $\pm$ 0.11 (0.000,n=111)	0.46 $\pm$ 0.10 (0.000,n=163)	0.46 $\pm$ 0.11 (0.000,n=119)	0.46 $\pm$ 0.11 (0.000,n=11)
Automotive Pollutants:						
(1) Lead	-0.64 $\pm$ 0.22 (0.005,n=159)			-0.68 $\pm$ 0.22 (0.002,n=163)		
(2) Carbon Monoxide		(-) (0.426,n=116)			(-) (0.493,n=119)	
(3) Ozone			(-) (0.68,n=111)			(-) (0.97,n=114)

¹See Table 10 for description of planned stepwise regressions

²Level of significance = 0.000 denotes $p < 0.0005$

Table 16. Final Model After Residual Evaluation
(n=159)

Variable (followed by Order of Entry)		Coefficient±S.E.	(level of significance)
% age 65 or over ¹	(1)	0.699 ± 0.028	(0.0000) ¹
Birthrate	(2)	0.21 ± 0.02	(0.0000)
% Spanish	(4)	-0.04 ± 0.007	(0.0000)
% Black	(5)	0.01 ± 0.006	(0.077)
% Asian	(10)	0.05 ± 0.04	(0.12)
Smoking (1969)	(6)	0.015 ± 0.003	(0.0000)
% Poverty	(8)	0.04 ± 0.02	(0.072)
% Long-term Res.	(9)	0.015 ± 0.002	(0.033)
Iron	(7)	0.45 ± 0.10	(0.0000)
Computed SO ₄ =	(3)	0.09 ± 0.02	(0.0002)
Constant		-7.68 ± 0.95	(0.0000)
Adjusted R ² =0.88			

¹Level of significance; 0.000 indicates p<0.0005

In all regression of possible subsets, all subsets of 1,2,3...21 independent variables are considered. The optimal subset is that which results in the highest "CP" value, "CP" being a statistic which takes into account the fact that non-nested models are being compared. Compared to Table 16, the optimal model resulted in essentially identical estimates for the computed sulfate coefficient (0.11 ± 0.02 , $p < 0.0005$), a slightly lower coefficient for the iron variable (0.34 ± 0.11 , $p = 0.008$) and a few different demographic variables. As expected, water hardness entered with a negative coefficient (-0.0010 ± 0.0006 , $p = 0.002$), as did the percentage of college graduates (-0.02 ± 0.01 , $p = 0.004$). Those variables which were common to both models had essentially the same estimated coefficients as in the planned stepwise regression model. These included birthrate (0.22 ± 0.02 , $p < 0.0005$), percentage age 65 and over (0.665 ± 0.03 , $p < 0.0005$), percentage Asian (0.07 ± 0.03 , $p = 0.004$), percentage Spanish (-0.04 ± 0.01 , $p = 0.004$) and percentage below poverty level (0.048 ± 0.02 , $p = 0.002$). The variables % Black and % long term residents did not enter. The adjusted R² value for this optimal model was 0.87, essentially identical to that of the model resulting from the planned stepwise regressions. We thus concluded that our normal stepwise regression procedures were resulting in near-optimum models.

5.4.2. Robustness to Demographic Variable Set and Data Set.

In addition to supporting the hypothesis that computed SO₄⁼ and iron are reasonable predictors of mortality, the planned regressions and the subsequent "all possible subset regression analysis" indicated several other aspects of this data set. It became clear that the demographic variable set which was

included in the model was partly dependent on the data set (i.e., selection of cities) and the pollutants being studied and that the pollutants selected might depend in turn on the demographic variables chosen. Further analyses supported this finding. For example, whereas the planned analysis of 67 cities with water data, computed $\text{SO}_4^{=}$ data, and total particle mass (MTD) data indicated that MTD was not a significant predictor, a subsequent analysis of 53 cities which also had iron data found MTD to be a significant predictor. Iron was not significant, and computed $\text{SO}_4^{=}$ was only borderline significant ($p = 0.10$). Moreover, in this data set, the $\text{SO}_4^{=}$ coefficient was estimated at 0.057 ± 0.03 , half the value obtained in all previous analyses. The important point here is that iron was not entered in the model, merely available in the data set, yet we obtained totally different conclusions with respect to the relative importance of the sulfate and particle variables, apparently because of the unique characteristics of this particular set of 53 cities.

5.4.2.1 53-City Data Set

The final model for the 53 cities included variables for water hardness and % college graduates, which were excluded from the analysis of 159 cities. However, the 53 city model excluded % Spanish, % Black and % at the Poverty Level, variables included in the analysis of 159 cities (having iron, computed $\text{SO}_4^{=}$, and water hardness but no data on particle mass). Moreover, this model fit as well as the model which excluded the particle mass variable and included the iron variable. The adjusted R^2 was 0.89 as compared to 0.88 for the iron and $\text{SO}_4^{=}$ model. Thus the discrepancies in the pollutant coefficient estimates obtained in the analyses could be due in part to the selection of different demographic variable sets for the different data sets.

Although the objective of this project is to estimate the effects of air pollution on mortality, we found that the results may be sensitive to both the selection of cities for analysis (i.e., the data set) and the selection of non-pollution variables to "adjust" the mortality rates for demographic and socio-economic effects (i.e., the demographic variable set or DVS). Often, the availability of pollution data defines the (maximum) data set, but we have provided enough demographic variables for any number of DVS's to be selected in a given situation. For the smaller data sets, this is a matter of concern, since it was shown that allowing too wide a choice in stepwise regression can lead to R^2 inflation.

Accordingly, we attempted to define a "best" demographic model for each of the two major data sets of interest, $n = 159$ (water hardness, computed sulfate, and iron) and $n = 53$ (the previous variables plus total particle mass), using stepwise regression. To check the robustness of the results, we then cross-validated the two models, i.e., evaluated the 159 city DVS with the 53 city data set (Table 17) and vice versa (Table 18).

Two approaches were used to investigate this problem and to obtain a final model. A first approach was to do factor analysis (with VARIMAX rotation)²⁶ of the demographic variables in the 159 cities with data on computed sulfate, water hardness and iron. Our objective was to reduce this extensive demographic variable set to a smaller set of demographic factors which would not be collinear. The factor analysis was done solely on the 159 city data set, since

Table 17. Regression Results for Demographic Model Defined by 159-City Data Set

MODEL #	1-1	1-2	1-3	1-4	1-5	1-6	1-7	1-8	1-9	1-10	1-11	1-12
$\bar{X} \geq 65$	0.699** (.032)	0.698** (.033)	0.704** (.031)	0.790** (.068)	0.793** (.067)	0.794** (.067)	0.821** (.068)	0.809** (.064)	0.829** (.065)	0.822** (.068)	0.668** (.016)	0.544** (.012)
Birth Rate	0.222** (0.01)	0.223** (0.01)	0.216** (0.01)	0.288** (0.01)	0.287** (0.01)	0.290** (0.01)	0.288** (0.01)	0.294** (0.01)	0.293** (0.01)	0.288** (0.014)	0.176** (0.004)	0.158** (0.005)
% Black	0.009 (0.01)	0.011 (0.01)	0.008 (0.01)	0.020 (0.01)	0.019 (0.01)	0.021 (0.01)	0.024 (0.01)	0.022 (0.01)	0.027 (0.01)	0.023 (0.014)	0.006 (0.004)	0.004 (0.005)
1969-80 Smoking	0.012** (0.00)	0.011** (0.00)	0.016** (0.003)	0.015* (0.005)	0.015* (0.01)	0.015* (0.01)	0.017* (0.005)	0.015* (0.005)	0.017** (0.005)	0.017** (0.004)	0.008* (0.002)	0.004 (0.003)
% College	-0.009 (0.01)	-0.017 (0.01)	-0.021* (0.01)	-0.061* (0.03)	-0.065* (0.03)	-0.061* (0.03)	-0.076* (0.03)	-0.071* (0.024)	-0.077* (0.03)	-0.078* (0.03)	-0.020* (0.007)	-0.019* (0.006)
Pop. Density	0.004 (0.002)	0.003 (0.002)	0.007* (0.003)	0.007 (0.004)	0.006 (0.004)	0.007 (0.004)	0.007 (0.004)	0.006 (0.004)	0.007 (0.004)	0.006 (0.004)	-0.0001 (0.001)	0.004* (0.002)
% Spanish	-0.042** (0.01)	-0.037** (0.01)	-0.051** (0.01)	-0.022 (0.02)	-0.02 (0.02)	-0.02 (0.02)	-0.02 (0.02)	-0.016 (0.02)	-0.078 (0.02)	-0.019 (0.02)	-0.03** (0.006)	-0.029** (0.006)
% Poor	0.029 (0.02)	0.018 (0.02)	0.050* (0.02)	-0.051 (0.05)	-0.051 (0.05)	-0.055 (0.05)	0.009 (0.02)	-0.064 (0.05)	-0.050 (0.05)	-0.043 (0.05)	0.055** (0.01)	0.046** (0.01)
Computed SO ₄ ⁻	0.093** (0.02)	0.104** (0.02)	X	0.068 (0.04)	0.067 (0.04)	0.070 (0.04)	X	0.070 (0.04)	X	X	0.130** (0.02)	0.126** (0.02)
Iron	0.376** (0.11)	X	0.429** (0.11)	0.100 (0.22)	X	0.147 (0.77)	0.073 (0.23)	X	0.151 (0.18)	X	X	X
Total Particle Mass	X	X	X	0.005 (0.02)	0.010 (0.01)	X	0.009 (0.02)	X	X	0.013 (0.01)	X	X
Adj. R ² n	0.88 159	0.87 159	0.86 159	0.87 53	0.88 53	0.88 53	0.87 53	0.88 53	0.87 53	0.87 53	0.86 418	0.80 872

* denotes $p < 0.05$ ** denotes $p < 0.001$

() denotes standard error of coefficient

X denotes variable not entered

Table 18. Regression Results for Demographic Model Defined by 53-City Data Set

MODEL #	2-1	2-2	2-3	2-4	2-5	2-6	2-7	2-8	2-9	2-10	2-11	2-12
% ≥ 65	0.730** (.035)	0.727** (.036)	0.754** (.037)	0.750** (.053)	0.750** (.052)	0.762** (.053)	0.769** (.051)	0.768** (.053)	0.787** (.051)	0.769** (.050)	0.689** (.017)	0.577** (.011)
Birth Rate	0.185** (0.02)	0.187** (0.02)	0.169** (0.02)	0.267** (0.03)	0.267** (0.03)	0.278** (0.25)	0.268** (0.03)	0.283** (0.25)	0.281** (0.03)	0.269** (0.03)	0.185** (0.02)	0.160** (0.01)
% College Grad.	-0.0002 (0.01)	-0.009 (0.01)	-0.009 (0.01)	-0.078* (0.02)	-0.076* (0.02)	-0.07* (0.02)	-0.089* (0.02)	-0.078* (0.02)	-0.082* (0.02)	-0.086* (0.02)	-0.009 (0.008)	-0.004 (0.006)
1969-80 Smoking	0.012** (0.004)	0.011* (0.004)	0.018** (0.004)	0.019** (0.005)	0.019** (0.005)	0.019** (0.005)	0.021** (0.005)	0.019** (0.01)	0.021** (0.005)	0.021** (0.004)	0.009* (0.003)	0.006* (0.003)
% Black	0.020** (0.005)	0.020** (0.005)	0.027** (0.005)	0.004 (0.008)	0.005 (0.008)	0.008 (0.008)	0.008 (0.008)	0.006 (0.008)	0.012 (0.008)	0.009 (0.007)	0.020** (0.003)	0.020** (0.003)
% Resident in State of Birth	0.015* (0.007)	0.011 (0.007)	0.021* (0.008)	0.021* (0.01)	0.024* (0.01)	0.021* (0.01)	0.027* (0.01)	0.021* (0.01)	0.024* (0.01)	0.027* (0.01)	0.017** (0.004)	0.022** (0.004)
% Asian	0.047 (0.04)	0.048 (0.04)	0.039 (0.04)	0.08* (0.04)	0.080* (0.04)	0.072 (0.04)	0.08* (0.04)	0.07 (0.04)	0.07 (0.04)	0.080* (0.04)	0.072* (0.03)	0.040** (0.02)
Water Hardness	-0.009 (0.007)	-0.008 (0.008)	-0.009 (0.008)	-0.003* (0.001)	-0.003* (0.002)	-0.002 (0.002)	-0.003* (0.002)	-0.002 (0.002)	-0.002 (0.002)	-0.003 (0.002)	-0.001* (0.0005)	X
Computed SO ₄ ⁻	0.115** (0.024)	0.123** (0.02)	X	0.048 (0.04)	0.049 (0.04)	0.057 (0.04)	X	0.058 (0.04)	X	X	0.136** (0.02)	0.123** (0.02)
Iron	0.344* (0.12)	X	0.407* (0.13)	-0.063 (0.21)	X	0.164 (0.16)	-0.08 (0.21)	X	0.169 (0.16)	X	X	X
Total Particle Mass	X	X	X	0.026 (0.02)	0.023* (0.01)	X	0.029 (0.02)	X	X	0.025* (0.01)	X	X
Adj. R ²	0.85	0.85	0.83	0.89	0.89	0.89	0.89	0.89	0.89	0.89	0.85	0.80
n	159	159	159	53	53	53	53	53	53	53	418	872

* denotes $p \leq 0.05$ () denotes standard error of coefficient
 ** denotes $p \leq 0.001$ X denotes variable not entered

the 53 city data set is too small (the number of observations divided by the number of variables was well below the rule of thumb value of 5). This was followed by stepwise regression of the demographic factors plus water hardness, the average smoking variable, iron and computed $SO_4^{=}$. The results are given in Table 19. The first factor, which accounted for the largest proportion of the variance, loaded highest on the ratio of males to females, the percentage of long term residents, the percentage of Asians, the percentage of college graduates, and log of the population. This factor entered stepwise in the analysis done without the pollutants and had a significant negative coefficient in the forced entry regression which included iron, computed sulfate and hardness as well as the significant demographic factors. The second factor, which loaded highest on density, population change since 1970, and percentage over the age of 65, also was a significant predictor of mortality, with a positive coefficient. The fourth factor, percentage Spanish and Birthrate, was also a significant positive predictor. The third factor, percentage black and percentage below the poverty level, was not a significant predictor, nor was the fifth (percentage of gas heating degree days and percentage of fuel heating degree days). If we use adjusted R^2 as a criterion, the factor model does not fit these data as well as

Table 19. Regression Results for Demographic Factor Model

MODEL	3-1	3-2	3-3
Factor 1	-1.102** (.15)	-1.084** (0.165)	-1.150** (0.14)
Factor 2	0.938** (0.162)	0.909** (0.166)	1.006** (0.14)
Factor 4	-0.338* (0.15)	-0.225 (0.15)	-0.358* (0.15)
1969-80 Smoking	0.007 (0.006)	0.006 (0.007)	0.009 (0.006)
Water Hardness	-0.003* (0.001)	-0.002 (0.001)	-0.003* (0.001)
Computed $SO_4^{=}$	0.040 (0.05)	0.064 (0.05)	X
Iron	0.689* (0.21)	X	0.719** (0.21)
Adj. R^2 n	0.49 159	0.45 159	0.44 159

X denotes variable not entered.

* denotes $p < 0.05$

** denotes $p < 0.001$

() denotes standard error of coefficient

the models which use the individual demographic variables. The adjusted R^2 is 0.49 for the model using factors, compared to 0.85 for most models using the demographic variables themselves. This is likely due to the fact that factor model does not fit the individual variables perfectly and thus has a greater error of measurement than the variables per se. On the other hand, the factor analysis model is the more appropriate in the case of many collinear variables and it is likely to be more robust when a large number of predictor variables must be considered relative to the number of cities. For this reason we should note carefully that only iron appears to be a significant pollutant in the factor model, that the sulfate coefficient is greatly reduced and nonsignificant both with and without the inclusion of the iron variable.

5.4.2.2 Additional Demographic Variables

While factor analysis is considered as a direct method of variable reduction, it yielded a model with much lower predictive power (lower R^2). Thus a second approach was to obtain two other demographic variable sets (DVS) through stepwise regression and to use these DVS in forced entry regression in several data sets defined by pollutant and availability of data on water hardness. The first DVS was obtained by doing stepwise regression of all demographic variables in the 159 cities having computed $SO_4^{=}$, iron, and water hardness data and is referred to as the 159 City Demographic Variable Set (or 159 CDVS) (Table 17). The second DVS was obtained by stepwise regression of all demographic variables in the 53 cities having data on computed $SO_4^{=}$, iron, and total particle mass and hence is referred to as the 53 city DVS (Table 18). After obtaining these CDVS, regressions with forced entry of the two DVS plus computed $SO_4^{=}$, iron, total particle mass, water hardness mass, and average smoking were done on the several data sets depending on their availability. The data sets considered were:

- (1) the 53 city data set having values for total particle mass, computed $SO_4^{=}$, iron, and water hardness,
- (2) the 159 city data set with values for iron plus computed $SO_4^{=}$ plus water hardness, (n = 418).
- 3) the city data set having values for computed $SO_4^{=}$ plus water hardness,
- (4) the city data set having data on MTD, computed SO_4 , and iron,
- (5) the city data set having information on iron plus computed $SO_4^{=}$,
- (6) the city data set having information on computed $SO_4^{=}$.

Tables 17 and 18 show that the coefficients for the demographic variables depend greatly on the data set. The pollutant coefficients depend to some extent on the DVS but to a greater extent on the data set. In any case all models fit equally well from the vantage point of R^2 . In the case where the 159 city demographic variable set is considered, the sulfate variable is significant at the 0.05 level in all data sets but the 53 city data set (in which particles are considered) and the estimate ranges from 0.067 to 0.104. Where the 53 CDVS

is used the computed sulfate coefficient is again significant in all data sets except the 53 city data set. The particle mass variable is significant only in the 53 city data set and only in the case where the 53 city demographic variable set is used.

5.5 INVESTIGATION OF RESIDUALS FOR INFLUENTIAL OBSERVATIONS AND VIOLATION OF REGRESSION ASSUMPTIONS

The residual analyses described in this section were done for two data sets for two models. The models considered were (1) the model obtained using the 159 cities having data on computed sulfate, hardness and iron plus the 159 city demographic variable set, and (2) the model obtained using the 53 cities having data on computed sulfate, iron, Total Particle Mass (MTD) Dichotomized, and hardness. These models are given then in Table 17 as Model 1-1 and in Table 18 as Model 2-5. The residuals were evaluated through identification of outliers, tests of homogeneity of mean and variance, and tests for spatial autocorrelation. Tests for linearity with respect to the sulfate variable were also done and we also to determined whether transformation of the mortality variable would produce a better model.

The residual plots were inspected visually for outliers. The following statistics were calculated for each city: (1) the standardized residual, (2) the standardized deleted residual, (3) Mahalanobis Distance.²⁶ The first two statistics evaluate the residual for a given city, the first being the residual divided by the standard error of the estimate and the second being the residual on deleting the city, and recalculating the coefficients. The third statistic measures the extent to which the vector of measures for the independent variables is an outlier, and hence the extent to which the city might be an influential observation. On plotting these values and, in the case of standardized residuals, taking into account the fact that there were 159 cities, only one city (Bakersfield, CA) appeared to be an outlier with respect to lack of fit. This city also appeared to be an influential observation. Deletion of this city from the 159 city data set slightly lowered the sulfate coefficient (from 0.09 ± 0.02 to 0.06 ± 0.02), but also reduced the birthrate coefficient considerably (from 0.22 ± 0.02 to 0.15 ± 0.02). Deletion of Bakersfield did not seem to affect the results in the 53 data set. Deletion of the city with the second highest deleted residual and the second highest distance value had no effects in either data set.

The normality of the residuals was evaluated by computation of the coefficient of skewness and kurtosis. These were non significant for the 53 city residuals (skewness = -0.29, kurtosis = 3.54) and non significant after deletion of the Bakersfield residual from the 159 city data set (skewness = 0.23, kurtosis = 3.06).

The assumption that the mean residual equalled 0.0 and the variance was constant was tested by grouping the cities with respect to the each of the following variables: (1) region, (2) the value of computed $\text{SO}_4^{=}$, (3) the value of the iron variable, and (4) the mortality value predicted by the model ($\hat{y}$). For each list of these variables, analyses of variance of the residuals were done, 95% confidence interval estimates of the mean residual were obtained, and a Bartlett test for homogeneity of variance was done. In every

case the analysis of variance indicated equality of the mean residual with respect to the variable being considered. In Figure 11 we plot the 95% confidence intervals obtained for the 5 groups defined for each of the above mentioned variables in the 158 cities. With the exception of values of y in the interval from 4.4 to 7.0, the mean residual equals zero. Hence we conclude that at worst we have very slight evidence that the model for the 158 cities underestimates mortality. The Bartlett test for homogeneity of variance was significant (0.02) with respect to region indicating that the model might have slightly better precision as a predictor for cities in the north central and southern (standard deviation = $s = 0.62$, $n = 55$ north central cities; $s = 0.66$, $n = 47$ southern cities), intermediate precision in the northeast cities ($s = 0.8$, $n = 28$) and the worst precision for the western cities ($s = 0.99$, $n = 28$ cities). However, these estimates are all inversely proportional to the size of the sample from each region and hence we do not read too much into this calculation. A similar explanation cannot be given for the observation of borderline heteroscedasticity with respect to the value of y . The standard deviation of the residuals is lowest ($s = 0.49$ as compared to 0.63, 0.67, and 0.93) in the 11 cities having the lowest predicted values. The precision of the model appears to be completely independent of the value of the sulfate variable ($p = 0.16$) and of the value of the iron variable $p = 0.26$) indicating that the dependence on y must reflect variability in precision with respect to some demographic variable. Similar investigations were done for the 53 city data set after grouping the cities with respect to each of the following variables: (1) region, (2) the predicted value, y , (3) the value of total particle mass MTD, and (4) the value of the computed sulfate (Figure 12). In this case the Bartlett test was always nonsignificant as were the the four tests of equality of mean residual. However, this could result simply from our having lower power to detect departures from the regression assumptions with only 53 values.

A test for linearity with respect to the sulfate variable was done for the 159 city model in the 158 city data set (less Bakersfield, CA). We tested the linearity of the relation between Y_i , mortality for the i th city, adjusted for all independent variables but the sulfate variable and X_i' , the i th cities sulfate value after adjusting for all other predictor variables. The cities were grouped with respect to their value of adjusted sulfate value by dividing the range of X' into 5 intervals of equal width (Figure 13). We then did an analysis of variance of the five means and partitioned the sum of squares between groups to estimate the mean squared error due to deviations from linearity. On comparing this term to the mean square error within groups, we conclude that there is no evidence of nonlinearity ($F(3,153) = 0.38$, $p = 0.91$).

The Bartlett test cited above indicated that some power transform of the mortality itself might result in a model with homogeneity of variance. Therefore, the Box-Cox approach was used to determine the optimal power transformation which would stabilize the residual variance so that it was uncorrelated with the mean predicted value. The estimated power transform was 0.5 for the 159 city data set and 0.1 for the 53 city data set indicating that the square root of mortality might be a better dependent variable than mortality itself in the first case and the tenth root, or the log of mortality might be more appropriate dependent variables.

Figure 11. 95% confidence limits for regression residuals (n = 158).

95 % CONFIDENCE LIMITS FOR RESIDUALS

n=53

quartile group

geographic region

Figure 12. 95% confidence limits for regression residuals (n = 53).

Figure 13. Linearity plot for sulfate (n = 158).

5.6 CONSIDERATION OF SPATIAL AUTOCORRELATION

Least squares models of the type considered here assume that the data are generated by adding a normally distributed error term to a linear combination of predictors. At each point measured, the error term is assumed to be independent of, and distributed identically to, the error terms at all other points. If this assumption is not met, the least squares estimation method may not be appropriate. Under the model, the residuals are estimates of the error terms. We can therefore get some idea of whether the least squares assumptions are met by inspecting the residuals which should have little or no systematic behavior when plotted against the predictors (Figures 11 and 12). In addition, there should be no spatial pattern to the residuals. This last requirement may be tested by inspecting the spatial autocorrelation of the residuals. Spatial autocorrelation may be thought of as a measure of the similarity between residuals as a function of their geographic distance. Cliff and Ord²⁷ give a method suitable for testing for spatial autocorrelation in a set of least squares residuals which is a generalization of the well-known Durbin-Watson test for autocorrelation of residuals in time series data.

Models 1-1 to 1-3 (Table 17) were evaluated* using the Cliff and Ord procedures, in addition to a regression employing the same demographic predictors but no air pollution variables. Spatial autocorrelation was not significant for any of the four models ($p = 0.6-0.8$); that is, spatial autocorrelation is not present regardless of whether air pollution is used as a predictor variable.

* Analysis courtesy of Dr. Neal Oden, Brookhaven National Laboratory.

6.0 COMPARISON OF 1970 TO 1980: CROSS VALIDATION AND EVALUATION OF LAG EFFECTS OF THE SULFATE VARIABLE

6.1 COMPARISON OF 1970 TO 1980 DATA AND MODELS

1970 data* were available for 122 of the 159 cities defined above. In Table 20 we list the variables for which the 1970 measures correspond to the 1980 measures. In the second column we give the estimated coefficient using the 1980 value to predict 1980 mortality in this data set and in the third column, the estimated coefficient on using the 1970 value to predict 1970 mortality. The sulfate, manganese, birthrate, nonwhite and log population density coefficients appear to increase in absolute value while the iron, % over 65, % college, % poor, smoking and hardness coefficients appear to have decreased in absolute value. Of these, particular attention should be paid to the sulfate coefficient which was not a significant predictor in 1970 but became significant in 1980, and to the birthrate coefficient, which appears to have increased. Simple t tests to compare these coefficients, which ignore the correlation between the estimates, indicate that only the change in the poverty coefficient is significant. This conclusion results in part from the relatively large standard errors in 1970.

Table 20. Results of Cross Validation Study: Coefficients Obtained
for 122 cities having Pollution Data for 1970 and 1980 (n=122)

Variable	Coefficient $\pm$ SE (p)			
	1980 Data Set		1970 Data Set	
SO ₄ =	0.053 $\pm$ 0.03	(0.06)	0.007 $\pm$ 0.03	(0.80)
Fe	0.053 $\pm$ 0.10	(0.78)	0.168 $\pm$ 0.09	(0.05)
Mn	2.90 $\pm$ 5.07	(0.57)	1.6 $\pm$ 1.17	(0.28)
% >65	0.740 $\pm$ 0.03	(0.000)	0.774 $\pm$ 0.03	(0.000)
Birth rate	0.147 $\pm$ 0.02	(0.000)	0.101 $\pm$ 0.03	(0.002)
% College	-0.029 $\pm$ 0.01	(0.05)	-0.052 $\pm$ 0.02	(0.015)
% Poor	0.019 $\pm$ 0.02	(0.41)	0.134 $\pm$ 0.04	(0.0001)
Non-White	0.022 $\pm$ 0.01	(0.001)	0.014 $\pm$ 0.01	(0.11)
Log Pop. Density	0.081 $\pm$ 0.28	(0.77)	0.061 $\pm$ 0.13	(0.64)
Smoking	0.008 $\pm$ 0.002	(0.005)	0.009 $\pm$ 0.003	(0.006)
Water Hardness	-0.0006 $\pm$ 0.0008	(0.39)	-0.001 $\pm$ 0.0008	(0.18)
Constant	-3.86 $\pm$ 1.19	(0.002)	-2.46 $\pm$ 17	(0.038)
Adj. R ²	0.88		0.88	

p = level of statistical significance

*1969 mortality data with demographic data from the 1970 Census and measured air quality data from 1969-71, taken from Lipfert.⁶

To test a general hypothesis that the set of coefficients has not changed, we regressed the change in mortality rate (1980 minus 1970, see Table 21) on the change in each independent variable (for all variables but water hardness, which did not change) plus the 1980 values. This is done because the coefficients of the 1980 value estimate the difference in the coefficient (1970-1980) as follows:

Let $Y_{j,1980}$ denote the mortality for city j in 1980, $B_{i,1980}$ the coefficient for X_i in 1980, and let $X_{ij,1980}$ denote the value for variable i in city j in 1980, we have

$$Y_{j,1980} = a_{1980} + B_{i,1980} X_{ij,1980} + e_{ij,1980}$$

Similarly, we define $Y_{j,1970}$, $B_{i,1970}$ and $X_{ij,1970}$ and note that

$$Y_{j,1970} = a_{1970} + B_{i,1970} X_{ij,1970} + e_{ij,1970}$$

We can then define for all variables where $C_i = B_{i,1980} - B_{i,1970}$ and $A = a_{1980} - a_{1970}$:

$$Y_{j,1980} = A + a_{1970} + (B_{i,1970} + C_i)X_{ij,1980} + e_{j,1980}$$

Subtracting (2) from (3) and setting $DY_j = Y_{j,1980} - Y_{j,1970}$, and $DX_{ij} = X_{ij,1980} - X_{ij,1970}$, we obtain

$$DY_j = A + C_i X_{ij,1980} + B_{i,1970} DX_{ij} + (e_{j,1980} - e_{j,1970})$$

Thus to test the hypothesis that there has been no change in the regression coefficients, we first regress change in mortality versus DX , the change in the values of the independent variable and then regress change in mortality versus the 1980 values plus the changes in the independent variable. In the case of hardness we have $DX_{ij} = 0$ and hence no DX_{ij} variable in the first regression. We then test the significance of the C_i terms in the usual manner. On doing this we obtain $F(11,100) = 1.7$, and $0.10 > p > 0.05$, borderline significance for the 159 city data set. Inspection of the appropriate terms indicates a significant change in the poverty coefficient ($p = 0.04$) and borderline significance for the percentage over age 65 ($p = 0.07$).

In Table 22 we show similar results from including only cities having total particle mass data as well as data on sulfate, iron, and water hardness. In general, by inspection one can see very little change in the coefficients. This is substantiated through t-tests comparing the individual coefficients and through the above described analysis, in which the set of coefficients which measure change in the coefficients from 1970 to 1980 contribute little to the prediction of change in mortality.

6.2 CROSS-VALIDATION WITH A 10-YEAR LAG

Just as 1969 cigarette smoking was shown to be an acceptable, if not better, predictor for 1980 mortality than 1980 smoking, it is possible that 1970 pollution might similarly be a better predictor than 1980 data. As a check, the 1980 cross-validation model was evaluated with 1970 values for

Table 21. Mean Values for 122 Cities in 1980 and 1970

Title	1980	1970	Difference
Mortality rate (deaths/1000)	10.37	11.15	-0.78
Log pop. density	1.55	1.39	0.15
% $\geq$ 65	12.56	10.98	1.59
% poor	15.61	10.43	5.22
% college	16.5	10.9	5.6
Birth rate (births/1000)	17.8	19.1	-1.27
SO ₄ (μg/m ³)	6.1	8.95	-2.86
Total particle mass (μg/m ³)	37.1	88.4	-57.6
Fe (μg/m ³)	1.044	2.040	-0.991
Mn (μg/m ³)	0.035	0.080	-0.045
Smoking (packs/person yr)	187	192	-4.7

Table 22. Cross Validation Results for 1980 and 1970
(50 cities having particle mass data)

Variable	1980 Data Set		1970 Data Set	
SO ₄	0.032 ± 0.04	(0.40) ^a	-0.054 ± 0.04	(0.23)
Total particle mass	0.018 ± 0.014	(0.21)	0.002 ± 0.009	(0.85)
Fe	-0.112 ± 0.32	(0.73)	-0.017 ± 0.13	(0.90)
Mn	-2.28 ± 8.50	(0.79)	4.64 ± 1.8	(0.01)
% $\geq$ 65	0.715 ± 0.05	(0.00)	0.715 ± 0.05	(0.000)
Birth rate	0.154 ± 0.04	(0.002)	0.166 ± 0.05	(0.002)
% College	0.07 ± 0.03	(0.013)	0.064 ± 0.04	(0.10)
% Poor	-0.011 ± 0.05	(0.80)	0.094 ± 0.05	(0.07)
Non-White	0.018 ± 0.01	(0.14)	0.019 ± 0.01	(0.12)
Log Pop. Density	0.65 ± 0.38	(0.095)	0.18 ± 0.21	(0.36)
Smoking	0.010 ± 0.004	(0.03)	0.011 ± 0.004	(0.008)
Water Hardness	-0.002 ± 0.001	(0.18)	-0.003 ± 0.002	(0.08)
Constant	-3.845 ± 1.85	(0.045)	-2.543 ± 1.75	(0.154)
Adjusted R ²	0.89		0.89	

^aStatistical significance level

SO₄⁼ and TSP. Run in the stepwise mode for 122 cities, 1970 measured sulfate was just significant, with a coefficient of $0.05 \pm .024$ ($p = 0.04$). This is virtually the same coefficient found using 1980 computed sulfate for this data set. TSP was not significant ($p = 0.47$). However, when regressed against all the demographic variables in addition to both 1970 SO₄ and TSP, neither was significant, although TSP was close ($p = 0.12$). This finding thus confirms the finding of some measure of sulfate aerosol as a predictor for 1980 mortality, and fails to indicate any importance for lagged rather than same year pollution.

Since same-year pollution data is implicated if not favored, it is also possible that the effects on mortality are acute (short-term) rather than chronic (long-term). However, the magnitude of the air pollution effect indicated by this study (ca. 10%) is much larger than what has been found in most studies of short term or episodic effects (typically 2-4%). This hypothesis could be checked by examining the seasonal trends in mortality in high pollution areas versus those in low pollution areas.

The final cross-validation check consisted of regressing 1970 mortality against 1970 demographic variables, but with 1980 computed sulfate to see if the lack of measurement error inherent in the computed data might be more important than the inconsistent time periods. The result was non-significant.

6.3 REGRESSIONS INCLUDING "1980" TSP DATA

Attempts to evaluate total particle mass as a predictor for 1980 mortality were limited by the small number of cities with the required data (53). Two other data sets were evaluated for this purpose. Dropping the iron variable increases the dichotomous sampler data set to 67 cities. For this data set, the coefficient for computed SO_4 was about the same as for 159 cities, 0.096 ± 0.031 ($p = 0.003$). The total particle mass coefficient was greatly reduced with respect to the $n = 53$ case and non-significant, 0.007 ± 0.009 ($p = 0.45$). Dropping the outlier city of Bakersfield, CA made only slight changes:

Computed SO_4 =:	0.073 ± 0.029	($p=0.014$)
Total Particle Mass:	0.010 ± 0.008	($p=0.21$)

The final step in this part of the analysis was to use a preliminary set of (1982) TSP measurements, extracted manually from published SAROAD data. The 1979-81 data were avoided because of the filter problems mentioned earlier. This gave a data set of 143 cities. The number of TSP monitors in each city used in compiling these data varied from 1 to 26.

The results of these regressions, run as forced entry using the 159 city DVS (Model 1-1 in Table 17) were to reduce the iron coefficient slightly and increase the computed SO_4 coefficient and its level of significance, with marginal significance for TSP (coefficients from 0.005 to 0.012, lowest p value of 0.08. We thus tentatively conclude that it is the 53 city data set which led to reduced significance for SO_4 , not the inclusion of a total particle mass variable.

7.0 PHASE III REGRESSION ANALYSIS

The computed sulfate variable obtained from Shannon's ASTRAP model²² was perhaps the most successful air pollution variable in the Phase I-II analyses, for two possible reasons:

- o concentration values were estimated for every city in the contiguous United States.
- o the values were all self-consistent with no random measurement error, having been generated by a model providing smooth contours.

The question arises as to how much the success of this variable stems from these properties, as opposed to the intrinsic nature of any sulfate aerosol health effects per se.

7.1 NEW AIR POLLUTION VARIABLES

To test the hypothesis that computed pollutants are more successful than measured pollutants, regardless of composition, distributions of two other pollutants were calculated by Shannon using the ASTRAP model: SO₂ (gas) and NO_x (NO + NO₂, gas). Values were computed for the centroids of each of the 952 cities in the data set, although the implied concentration differences between locations closer than the ASTRAP grid size (ca. 120 km) were not considered meaningful. At the same time, SO₄⁼ concentrations were recomputed for these same locations ("new" SO₄⁼), to preclude the interpolation errors that might have been introduced by the previous (1st and 2nd phases) data treatment. These computations were based on ca. 1980 SO_x and NO_x emission rates, but 24-year average meteorology was used. Thus they should be considered estimates of long-term spatial average concentrations. The pertinent statistics are given in Table 23, which shows that all three sulfur measures are highly

Table 23. Statistics of Computed Air Quality Variables (g/m³)

Variable	Mean	Std. Dev.	Min.	Max.	Correlations			
					New SO ₄ ⁼	SO ₂	NO _x	Measured (SAROAD) SO ₄ ⁼
"old" SO ₄ ⁼	4.81	3.60	0	14.78	0.860	0.801	0.353	0.760
"new" SO ₄ ⁼	5.78	3.81	0	13.08	1.0	0.884	0.455	0.706
SO ₂	15.94	10.97	0	46.38		1.0	0.708	0.722
NO _x	16.89	10.63	0	50.89			1.0	0.382

correlated, but that NO_x has distinctly different spatial patterns from SO_x. This results from the additional emissions sources for NO_x which emit negligible SO_x, including transportation sources and natural gas combustion. Although the "old" SO₄⁼ values correlate somewhat better with the SAROAD measurements (128 observations), these correlation coefficients (0.706 - 0.760)

are not statistically different. The differences in $\text{SO}_x\text{-NO}_x$ correlations among the SO_x variables imply that there are some differences among the SO_x variables as well.

7.2 REGRESSION ANALYSIS

7.2.1 Stepwise Regressions

Following the analyses used in Phases I and II, the initial exploration of these new data sets was by stepwise regressions, taking one pollutant at a time. The measured pollutant data from Phases I and II were included in these trials. These calculations were made for the largest possible data sets and for the data sets limited by the cities having water hardness data. The results are given in Table 24, and show virtually identical results for all four computed pollutants.

Even for identical "fits", the magnitude of a regression coefficient b_1 will vary according to the mean values of the variable x_1 , which have a considerable range for the pollutants shown in Table 24. Computing the elasticity at the mean ($E_1 = b_1 \bar{x}_1 / \bar{y}$) is one way to remove these scale effects. These elasticity values range from 0.033 for manganese to 0.087 for measured $\text{SO}_4^{=}$ and may be interpreted as the fraction of the mean mortality rate associated with the pollutant. The last entry in Table 24, ($\text{SO}_4^{=}\text{*O}_3$), examined the interaction between these two pollutants, if any. As can be seen, this combined variable was not significantly associated with mortality variations and thus was not considered further.* An additional interaction variable ($x_1\text{*}\%$ (prior) resident), was also investigated as a means of implying air pollutant dose rather than concentration.* These "dose" results were not significantly better than concentrations and thus were not considered further.

Although the overall regressions for (new) $\text{SO}_4^{=}$ and NO_x provided virtually identical fits, there were some interesting differences in the models defined by the final steps and in the stepwise regression sequences. Both models had the same ($p < 0.05$) regression coefficients for the following variables:

- % > 65
- birth rate
- population change
- smoking
- % (prior) residents

The coefficients for the two stepwise models were statistically different for % poor and % Spanish (both larger in absolute magnitude for the NO_x model), and % black and % Asian failed to enter in the NO_x model. Table 25 gives the results for the stepwise sequences of variable entry. $\text{SO}_4^{=}$ enters on the third step with a very high β (partial regression coefficient), which then decays as additional variables enter the model. On the other hand, NO_x entered

*Interaction variables $X_1 * X_j$ were entered into regressions along with the separate variables X_1 and X_j , to test for the significance of interactions vis-a-vis the separate contributions.

Table 24. Phase III Stepwise Regression Results
(each pollutant entered separately)

Full Data Set					Water Hardness Data Set			
Pollutant(x_i)	n	Adj. R^2	b_i (S.E.)	E_i	n	Adj. R^2	b_i (S.E.)	E_i
Old SO_4 =	908	0.814	0.085(.016)	0.045	454	0.853	0.098(.016)	0.052
New SO_4 =	908	0.817	0.103(.016)	0.066	454	0.854	0.123(.017)	0.081
SO_2	908	0.816	0.035(.005)	0.061	454	0.854	0.037(.005)	0.063
NO_x	908	0.817	0.035(.005)	0.065	454	0.848	0.032(.006)	0.050
SAROAD SO_4 =	187	0.831	0.100(.024)	0.087	185	0.858	0.082(.022)	0.071
IP SO_4 =	78	0.813	0.195(.052)	0.083	68	0.883	0.177(.053)	0.077
Fine Part.	80	0.806	NS	[0.059]*	68	0.867	NS	[0.059]*
Total Mass	80	0.806	NS	[0.047]*	68	0.867	NS	[0.027]*
Fe	172	0.851	0.401(.115)	0.041	172	0.851	0.401(.115)	0.041
Mn	172	0.849	9.60(2.54)	0.033	172	0.849	9.61(2.54)	0.033
SO_4 = O_3	211	0.806	NS	[0.018]*	201	0.849	NS	[0.007]*

() = standard error of regression coefficient

E_i = elasticity = $b_i \frac{x_i}{y}$ (evaluated at the mean)

NS = not significant (pollutant did not enter stepwise regression)

[]* = elasticity if pollutant had entered.

its model on the 7th step and its value improved with each succeeding step, ending with a value identical to sulfate. Thus, if the analysis had been terminated on the 8th step or earlier, the conclusions from the comparison of the two variables might have been quite different ($\text{SO}_4^{=}$ appearing to be "better" than NO_x).

The water hardness variable usually had a negative coefficient (as expected), but never achieved statistical significance. Thus the chief difference between the two data sets was in the selection of cities, not the effects of the water hardness variable per se. As discussed above, the "water hardness cities" tended to be larger in population.

Table 25. Sequences of Stepwise Regressions

Regression Step	Partial Regression Coefficients	
	β_{SO_4}	β_{NO_x}
2	0.119	0.057
3	0.212 *	0.087
4	0.160	0.059
5	0.140	0.059
6	0.134	0.067
7	0.127	0.058*
8	0.125	0.085
9	0.133	0.110
10	0.117	0.117

$$\beta_1 = b_1(\bar{x}_1/\bar{y})$$

* boldface type indicates that variable has entered the regression model.

7.2.2 Fixed Model Regressions

Based on the preliminary results of the stepwise regression analysis, a fixed demographic model was selected to further examine pollutant effects. Stepwise regression models for the three computed pollutants ($\text{SO}_4^{=}$, SO_2 , NO_x) were examined. From the list of all demographic variables entering for each of three pollutants, six were chosen for the fixed model: % of population aged 65 and over, birth rate, % population change since 1970, % of population of Hispanic origin, average of 1969 and 1980 cigarette smoking estimates (packs per person per year), and % of population classified as below the poverty line. Three other variables were not included in the fixed model: % of population classified as black or Asian, which entered only with one or two of the computed pollutants and were not significant when they entered; % of population residing in their state of birth was significant only with NO_x . Results of the fixed-model regressions are shown in Table 26. The pollutant regression coefficients are not significantly different from those given in Table 24; adjusted R^2 values are slightly lower for the fixed model regressions, as expected, since they are not "optimal" models.

Table 26. Results from Fixed Demographic Model Regressions

MODEL #	4-1	4-2	4-3	4-4	4-5	4-6
% $\geq$ 65	0.546** (0.010)	0.547** (0.010)	0.551** (0.010)	0.670** (0.015)	0.670** (0.015)	0.672** (0.016)
BIRTH RATE	0.187** (0.011)	0.182** (0.011)	0.180** (0.011)	0.219** (0.014)	0.209** (0.014)	0.203** (0.014)
Δ POP.	-0.013** (0.001)	-0.013** (0.001)	-0.014** (0.001)	-0.010* (0.004)	-0.009* (0.004)	-0.014** (0.004)
% SPANISH	-0.025** (0.004)	-0.031** (0.004)	-0.042** (0.005)	-0.031** (0.005)	-0.035** (0.005)	-0.042** (0.005)
SMOKING 78	0.009** (0.002)	0.008** (0.002)	0.009** (0.002)	0.011** (0.003)	0.011** (0.003)	0.013** (0.003)
% POOR	0.041** (0.008)	0.053** (0.008)	0.064** (0.008)	0.067** (0.009)	0.076** (0.009)	0.086** (0.009)
WATER HARDNESS	X	X	X	-0.0006 (0.0005)	-0.001 (0.0005)	0.0007 (0.0005)
COMPUTED SO ₄ =	0.112** (0.015)	X	X	0.109** (0.016)	X	X
COMPUTED SO ₂	X	0.038** (0.005)	X	X	0.038** (0.005)	X
COMPUTED NO _x	X	X	(0.038)** (0.005)	X	X	0.032** (0.006)
Adj. R ² n	.813 916	.814 916	.813 916	.850 459	.851 459	.845 459

* denotes $p < 0.05$ ** denotes $p < 0.001$

() denotes standard error of coefficient

x denotes variable not entered

7.2.3 Subset Analysis

Because of the implied differences due to selection of cities, several different subsets were analyzed. The criteria for subset selection were:

- o availability of measured air pollution data
- o city size
- o level of (computed) pollutant concentration
- o geographic region

These subsets were investigated using stepwise regressions to allow different demographic models for each subset. The largest data sets, employing the computed pollutant variables, were most useful, since quartiles with over 100 observations each could be defined. The ability to perform such sub-set analyses is another advantage for the computed air pollutant variables.

7.2.3.1 City Size Subsets

Table 27 presents the results of stepwise regressions for mortality rate by city size. The full data set was divided into four quartiles, as shown. As city size increased, the percentages of minorities increased as did the birth rate and poverty fraction, whereas there was a slight decrease in the percentages of long term residents. Sulfate was statistically significant in three out of the four quartiles; the coefficients were not significantly different among quartiles. It is also interesting to note that the average death rate shows an increase with city size (whereas the percentage over 65 does not), and the regression coefficient of mortality on % ≥ 65 also increases, implying that people over 65 live longer in smaller cities.

City size quartiles could not be used with the measured pollutant data, since these locations overwhelmingly fell into the largest city size quartile. The final portion of the city size analysis used weighted regressions to deal with possible heteroscedasticity. Weights used were the number of deaths in each city and the square root of that number. These regression results were within the confidence limits implied by Table 24 for the full data set.

7.2.3.2 Pollutant Level Subsets

As discussed above, many of the pollutants in this study are highly correlated with each other. One way to examine the interrelationships is to study the variation of one pollutant within quartiles of another. Accordingly, stepwise regressions were run for all pollutants, one at a time, for subsets of the full data set defined by quartiles of each of the computed variables: "new" $\text{SO}_4^{=}$, SO_2 , and NO_x . This analysis explored the possible presence of pollution thresholds as well as the collinearity issue.

If a pollutant threshold were present, regression coefficients would lose significance or take on lower values in the lower quartiles. To understand how use of pollutant quartiles may help to deal with collinearity between pollutants, consider a matrix (ij) of cities grouped according to their SO_2 (i) and NO_x (j) values. The collinearity would appear on the diagonal of this matrix. However, a regression along a row or column would consider the effect of varying SO_2 at constant NO_x or vice versa, and thus should be influenced by collinearity less than the full data set.

The demographic characteristics of the cities grouped according to pollutant level also showed some interesting trends. Death rates increased with pollutant level for SO_2 and $\text{SO}_4^{=}$, but not with NO_x . City size (population) was constant. The age distributions showed mixed trends; birth rate tended to decrease. Population change showed a strong decrease with pollution level. The percentage of black population increased with SO_x level, but not with NO_x . The percentage of Hispanics and Asians was generally mixed; % Hispanics

Table 27. Stepwise Regressions of Mortality on SO₄= By Quartiles of City Size

Quartile	1		2		3		4	
	Coeff.(b ₁) (SE)	Mean ($\bar{x}_1$)	Coeff.(b ₁) (SE)	Mean ($\bar{x}_1$)	Coeff.(b ₁) (SE)	Mean ($\bar{x}_1$)	Coeff.(b ₁) (SE)	Mean ($\bar{x}_1$)
avg. death rate/1000 pop.		9.00		9.07		9.22		9.27
avg. city population		28,595		38,101		58,105		166,708
% 65	0.537 (0.019)	11.7	0.533 (0.023)	11.4	0.582 (.021)	11.8	0.607 (0.014)	11.4
birth rate	0.166 (0.020)	16.3	0.218 (0.028)	16.6	0.122 (0.017)	17.0	0.232 (0.015)	18.1
Δpop.	-0.023 (0.0036)	14.8	-0.0156 (0.0036)	20.9	-0.0052 (0.0021)	15.0	-0.01 (0.0026)	9.7
Computed SO ₄ =	0.0842 (0.029)	6.0	0.139 (0.040)	5.9	[0.045] (0.033)	5.9	0.0948 (0.020)	5.5
% poor	0.0481 (0.015)	10.7		11.8		11.8		13.9
Smoking78	0.0122 (0.0048)	189.0		192.	0.0142 (0.0047)	190.0	0.0068 (0.0029)	190.0
% black		8.1		10.1	0.0366 (.0059)	10.6	0.0170 (0.0038)	17.0
(log) land area		10.8		13.1	-0.309 (0.126)	17.2		46.1
% Resident in 1970		61.0		61.0	0.0356 (0.0058)	60.5		57.3
% Asian		1.4		1.6		2.1	0.050 (0.019)	2.2
% Spanish		4.76		6.6		7.9	-0.029 (0.0047)	9.95
Adj. R ²	0.827		0.740		0.843		0.916	
Std. Error	1.45		1.90		1.241		0.853	
n	231		224		228		235	

() = standard error of regression coefficient

[] = coefficient if pollutant had entered the regression

blank indicates variables not entered.

decreased with $SO_4^{=}$ level. The age coefficient (mortality attributed to those 65 and over) increased sharply with SO_x level and was mixed for the four NO_x levels.

Table 28 presents elasticities for the pollution coefficients by pollution quartile, for those pollutants that were significant for the full data set, based on stepwise regressions. The 10% significance level was selected to compensate for the smaller data sets in each quartile. It is seen that almost all pollutants are significant in the third quartile (next to highest concentrations). Within the same pollution quartile, SO_2 was significant in 3 out of the 4 $SO_4^{=}$ quartiles. NO_x and $SO_4^{=}$ were no more significant within quartiles than might be expected by chance, even though they had previously been found significant for the full data set. However, NO_x was significant at the 0.15 level in 8 out of 12 instances. Overall, there are 72 possibilities for statistical significance in Table 28; at the 10% level, 7 entries would be expected. 19 entries are shown, which is substantially more than would be expected due to chance alone.

NO_x tends to be significant within the lower pollution quartiles, whereas $SO_4^{=}$ and SO_2 were significant for the higher pollution quartiles. SO_2 was especially significant within quartiles other than SO_2 as expected; the regression coefficients were not statistically different between quartiles. $SO_4^{=}$ regression coefficients also were not statistically significantly different between quartiles, but had some resemblance to a conventional dose-response relationship (higher values at higher $SO_4^{=}$ levels).

Table 28. Elasticities of Pollutant Variables Within Pollution Quartiles
(Stepwise Regressions, one pollutant at a time)

Quartiles		Pollutants						
		Computed			Measured (SAROAD)			
		SO ₄ ⁼	SO ₂	NO _x	Fe	Mn	SO ₄ ⁼	O ₃
SO ₂	1	(-.037)	(0.003)	(0.056)	(0.01)	(0.002)	(0.05)	(-0.06)
	2	(0)	(0.03)	0.050*	(0)	(0.004)	(0.16)	(-0.08)
	3	(0.021)	0.079*	0.076*	0.13*	0.15*	(0.09)	(-0.08)
	4	(0.048)	(0.01)	(0.017)	(0.01)	(0.05)	0.08*	(-0.12)
SO ₄ ⁼	1	(-.06)	(0.01)	(0.06)	(-.05)	(0.05)	(-0.07)	(-0.11)
	2	(0.03)	0.09*	0.11*	(0.08)	(0.02)	(-0.02)	(0)
	3	0.05*	0.10*	(0.09)	0.24*	0.16*	(0.04)	(-.07)
	4	0.11*	0.07*	(0.05)	(0.02)	(0.05)	(0.09)	(-.18)
NO _x	1	(0.05)	(0.02)	(0.04)	(0.14)	(0.09)	(0)	(-0.01)
	2	(0.04)	(0.04)	0.05*	(0.06)	(0.07)	(0.06)	(-0.02)
	3	0.16*	0.14*	(0.04)	0.23*	0.26*	0.15*	(-0.11)
	4	(0.05)	(0.05)	(0.02)	(-0.12)	(-0.05)	(0.12)	(-0.10)

* = regression coefficient significant at the 0.10 level or better

() non significant elasticity, calculated as if pollutant had entered regression.

In summary, the pollutant quartile analysis achieved a partial separation of the collinear pollutants, in that NO_x showed statistical significance in several instances when the other pollutants did not. The technique did not distinguish among Fe, Mn, $\text{SO}_4^{=}$ or SO_2 , nor between $\text{SO}_4^{=}$ and SO_2 .

7.2.3.3 Regional Subsets

Subsets by geographic regions were defined as follows; West: west of 92° W. longitude. For the remainder of the US, east of 92° W. longitude, Northeast: north of 42° N. latitude; Southeast: south of 37° N. latitude; Midsection: between 35° and 42° N. latitude (Fig. 14). In many cases, these subdivisions are less arbitrary than state boundaries, and were also chosen with a view towards equalizing the numbers of cities in the three eastern subdivisions. The results are given in Table 29. Only the computed pollutants showed statistical significance for these subsets; it was not possible to determine whether the lack of significance of Fe, Mn, and measured $\text{SO}_4^{=}$ was due to the reduced number of observations in each subset or due to collinearity between other regional factors and pollutant levels.

It should also be recognized that the use of state-wide average smoking data may have compromised this regional analysis, because there may not be an adequate range for the smoking variable in all regions, since local city differences are not accounted for.

Table 29 indicates that the pollution regression coefficients are reasonably consistent across regions (with the exception of a high value for NO_x in the Northeast). All four regions showed some sensitivity to pollution, but the Southeast was the least sensitive (the Northeast and Midsections being the most). By pollutant, all three computed pollutants were significant in 5 out of 8 possible entries. $\text{SO}_4^{=}$ was never significant in the West; SO_2 and NO_x were never significant in the Southeast. Ozone generally showed negative regression coefficients within regions, including the West. To estimate between-region pollution effects, the mean values for the demographic variables were used together with their corresponding regression coefficients to calculate a predicted regional average mortality rate, without considering air pollution. The resulting residuals (observed-predicted) then were regressed against regional average SO_2 , $\text{SO}_4^{=}$, and NO_x values ($n=4$). Most of the results were not statistically significant, but the coefficients compared well with the values obtained within regions. An alternative approach towards investigating the influence of unspecified regional factors uses dummy variables, $m-1$ variables for m regions. The dummy variable takes on the value 1.0 if the city is in the region, 0 if it is not. Regressions were performed on this basis using a fixed model regression and both the full data set and the subset having water hardness data. The results were:

- o increases in the coefficients for computed SO_2 and $\text{SO}_4^{=}$
- o decreases, but still significant values, for computed NO_x , Fe, and Mn.
- o loss of statistical significance for SAROAD $\text{SO}_4^{=}$, fine particles, and total particles.

In the full data set the coefficient for $\text{IPSO}_4^{=}$ decreased and lost significance, while in the water hardness data set, this coefficient became only slightly smaller with the addition of the regional dummy variables. In

Figure 14: Regional definitions used in Section 7.2.2.3

Table 29. Pollution Regression Coefficients By Region
(Stepwise regressions with one pollutant at a time)

		Region			
		Northeast	Mid-section	Southeast	West
(a) full data set	n	137	260	128	383
	SO ₄ =	0.162 (0.043)	0.154 (0.034)	0.168* (0.092)	
	SO ₂	.089 (0.012)	0.040 (0.010)		0.025 (0.009)
	NO _x	0.108 (0.015)	0.030 (0.010)		0.021 (0.005)
(b) cities having water hardness data	n	88	175	59	132
	SO ₄ =	0.165 (0.044)	0.146 (.039)	0.220 (0.073)	
	SO ₂	0.059 (0.014)	0.037 (0.011)		
	NO _x	0.068 (0.018)	0.027 (0.011)		

() = standard error of regression coefficient

blank: indicates variable did not enter stepwise regression

* this coefficient is not statistically significant but is provided for reference.

general, the "midsection" dummy variable was most often statistically significant, with coefficients ranging from 0.4 to 1.4 (in mortality rate units). Values for dummy variable coefficients correspond to the average mortality rate increment relative to the Western US (the region for which no dummy variable was entered). Since the South is often mentioned as the region having different dietary patterns and lifestyle parameters, it was of interest that the "South" dummy variable was statistically significant in only one out of the 18 regressions. Evidently, the model adequately accounts for any differences between the South and the rest of the U.S. in terms of their effects on total mortality.

In summary, this regional and subset analysis indicated that most pollutants were significant within regions (as well as between regions). There was no firm evidence that the indicated pollution effects in the national data sets were surrogates for regional differences.

7.2.4 Multiple Pollutant Regressions

The subset analyses suggested that different pollutants could have their primary effects in different parts of the country. Accordingly, some multiple pollutant regression models were evaluated. For the combination of computed SO₄= and NO_x, ozone, and Fe (n=119), ozone did not enter, SO₄= and NO_x showed reduced significance (p = 0.0377 and 0.0652, respectively), and Fe showed

increased significance. However, the combined elasticity, $13.5 \pm 6\%$, was not significantly greater than that given by $\text{SO}_4^{=}$ alone. This result suggested that Fe was the most important pollutant for this set of cities. When manganese was substituted for iron, $\text{SO}_4^{=}$ became less significant and NO_x more so. SO_2 and iron showed equal significance, with NO_x becoming non-significant. A similar calculation for 172 cities, not using ozone, resulted in similar findings. It was difficult to conclude that one pollutant was more important than another; however, either Fe or Mn consistently showed higher statistical significance when entered in combination with the computed pollutants.

7.3 EXAMINATION OF RESIDUALS

The validity of hypothesis tests on regressions requires that the distribution of residuals be normal and that residuals be independent. In this section, we consider the degrees to which these criteria were met.

7.3.1 Frequency Distributions

As in the second phase of the analysis, examination of the residuals from the various regressions revealed a few outliers (greater than 3 standard deviations from the mean). As a result, the frequency distribution of residuals has too few values in the mid-range and too many on the tails. This situation persisted when outliers were dropped; the standard deviation was reduced and different observations became "outliers," preserving the same general shape of the frequency distribution of residuals. It is not known what effect this slightly non-normal distribution has on hypothesis testing based on the regressions, but it is believed that the effect is minor except perhaps for marginal significance levels.

7.3.2 Spatial Autocorrelation

Spatial autocorrelation was not apparent from visual observations of the maps of residuals (Figures 15-17).^{*} The next step was to test the distributions of residuals for spatial autocorrelation. This was done through the cooperation of Dr. N. L. Oden of BNL, using the method developed by Cliff and Ord.²⁴ This procedure computes a spatial autocorrelation coefficient I and its significance level. The results are tabulated in Table 30. The parameters of spatial autocorrelation are the correlation coefficient I and its statistical significance level. Significance depends on properties of the spatial autocorrelation matrix in addition to the number of observations, but the results of Table 30 indicate a tendency for statistical significance to increase with n. Spatial autocorrelation may still be present at lower values of n but the test may not be capable of discerning it. None of the regional data sets created by subdividing the water hardness data set showed significant spatial autocorrelation; all of the subsets based on the full data set did. However, all of the magnitudes of the spatial autocorrelation coefficient are quite low, so that the practical effects on the validity of hypothesis tests are not believed to be serious.

^{*} The consequences of the "hidden observations" on Figures 15-17 are discussed below.

Figure 15. Map of residuals from multiple regression of mortality on computed SO₄= concentrations. RSO₄= observed - predicted mortality rate, where 5 = highest value and 1 = lowest value. 470 observations are hidden.

Figure 16. Map of residuals from multiple regression of mortality on computed NO_x concentration. RNO_x = observed - predicted mortality rate, where 5 = highest value and 1 = lowest value. 470 observations are hidden.

Figure 17. Map of residuals from multiple regression of mortality on computed SO₂ concentration. RSO₂ = observed - predicted mortality rate, where 5 = highest value and 1 = lowest value. 470 observations are hidden.

Table 30. Results of Spatial Autocorrelation Analysis

Data Set	n	Pollu- tants Inclu- ded	Spatial Autocorrelation Coefficient (I)*	Significance Level
Full	914	SO ₄ =c	0.082 - 0.086	0.0000
Water Hardness	457	SO ₄ =c	0.085 - 0.087	0.0000
SAROAD SO ₄ = data	136	SO ₄ =	0.045 - 0.056	0.02 - 0.04
IP Data Set	75	SO ₄ =	0.008	0.008
West (full)	383	SO ₄ =c	0.072	0.0000
West (water hardness)	131	SO ₄ =c	0.014 - 0.035	0.14 - 0.34
Southeast (full)	131	SO ₄ =c	-0.12 - -0.13	0.01 - 0.03
Southeast (water hardness)	60	SO ₄ =	-0.06 - -0.18	0.24 - 0.94 (NS)
Northeast (full)	140	SO ₄ =c	0.12 - 0.21	0.0000
Northeast (water hardness)	90	SO ₄ =c	-0.020 - -0.023	0.76 - 0.82 (NS)
Mid Section (full)	260	SO ₄ =c	0.02 - 0.04	0.01 - 0.02
Mid Section (water hardness)	176	SO ₄ =c	0.006 - 0.009	0.31 - 0.34 (NS)

* Ranges in (I) are due to alternative definitions of "neighboring" locations in the matrix.

7.3.3 Outlier Cities

Residual plots were visually examined for outliers and their locations were identified. All the large negative residuals (observed << expected) were located in Florida, and were probably retirement communities. In contrast, North Miami Beach was a large positive outlier (observed >> expected). Many of the other positive outliers were heavy industrial cities, including Wheeling, WV; Steubenville, OH; Pittsburgh, PA; and Johnstown, PA. However, Hammond, IN was a negative outlier. Other positive outliers included Easton and Wilkes-Barre, PA (possible coal mining impact), Zanesville, OH; Watertown, NY; Atlantic City, NJ; Rockville, MD; Suffolk, VA; and Terre Haute, IN. No justification was apparent to delete any of these cities from the analysis.

7.4 TWO-STAGE REGRESSION ANALYSIS

All of the environmental variables are associated to some degree with the demographic variables (Tables 4 and 6). To eliminate as much of this effect as possible in the regression coefficients, a two-stage regression procedure was employed. The previously determined demographic models were run for the full data set and the water hardness data set, each without any pollution variables in the model. The residuals from these regressions, which may be thought of as mortality rates "adjusted" for demographics, smoking, etc., then were used as the dependent variable with each pollutant, taken one at a time. This procedure was thought to result in assigning the minimum mortality effect (for a given data set) to each pollution variable. Further, it assumes that the same demographic models (and coefficients) are used for each pollutant. The fact that we have derived two such "true" models (or adjustment procedures) indicates the uncertainties about the non-pollution effects on mortality.

CORRELATION COEFFICIENTS

ELASTICITIES, ABOUT THE MEAN

Figure 18. Correlation coefficients and elasticities for various pollutants vs. residuals from fixed demographic models (2-stage regressions).

Figure 19. Map of residuals from mortality model with no air pollution variables. "5" denotes highest mortality (and residual); "1" denotes lowest mortality (negative residual).

This suggests the presence of some other urban mortality factors not accounted for by this model.*

The regression coefficients in the fixed demographic model are of course free to adopt whatever values result in the minimum squared error for the pollutant. The ranges of these coefficient values as the nine pollutants are introduced, one at a time, are therefore of some interest:

% > 65:	0.543 to 0.702
smoking 78:	0.007 to 0.016
birth rate:	0.160 to 0.212
% poor:	0.041 to 0.079
population change:	-0.003 to -0.015
% Spanish:	-0.019 to -0.04

The variables are listed in order of their effect on mortality rate ($b_i * y$); the ranges shown represent considerable uncertainty for the first three variables (10-20%). However, at least part of the differences in coefficient values is associated with the restriction of the data sets for some pollutants to larger cities, which tend to result in larger coefficients for the age and smoking variables. The regression coefficients that resulted from use of city size as a weighting factor were nearly identical to the unweighted results.

7.5 COMPARISON OF MODELS

In Section 7.2, pollutant effects on mortality were considered by allowing the demographic model to be freely adjusted, both in terms of the variables selected and their coefficients, in order to maximize adjusted R^2 for the pollutant. The regression R^2 or the t value for the pollutant then becomes the figures of merit to be used to compare pollutants. In Section 7.4, we used those same figures of merit, but insisted that the demographic model be fixed for all pollutants, both in terms of the variables selected and their coefficients. In the following section, we present a third technique, in which the demographic model has a fixed structure but varying coefficients for all pollutants. Test of significance are developed to test whether differences in regressions (unexplained variance) are statistically significant.

7.5.1 Methodology for Comparisons

Use of related pollutants in the regression models for mortality rates gives rise to multi-factor models which differ very little from one another, as seen in Table 24, for example. There is always a temptation to declare the model with the highest adjusted R^2 value or the highest t statistic for the

* These maps encompassing the whole US tend to have about 50% hidden observations, since adjacent locations cannot be displayed separately at this scale. To test the hypothesis that the hidden observations are chosen randomly and do not result in a biased data presentation, we enlarged the section defined by 37° latitude $< 42^\circ$, 80° longitude $< 92^\circ$ (Ohio, Indiana, Illinois). The distribution of 1's, 2's etc. was no different than in Figure 19, using a χ^2 test ($p = 0.25$). We thus concluded that it was safe to use residual maps with a large number of hidden observations, for visual evaluation.

pollution variable as "best," (however close its competitors might be). This practice ignores the fact that a given data set represents only one realization from the universe of possible data, and that its regression statistics thus all carry confidence limits. If the models tested in Table 24 were independent, one could use the conventional confidence limits for R^2 as a guide towards defining statistically significant differences between models. However, since these models generally only differ in the pollution variables chosen, they are not independent and special techniques are required to test the differences for statistical significance.

Snedecor and Cochran²⁸ give such a method, which requires the correlation between sets of residuals from alternative models, R_r , in order to compare two correlated variances (in this case the residuals from competing models for predicting the same set of y 's). Their procedure appears to be similar to that of Wolfe.²⁹

Snedecor and Cochran pose this problem in terms of a variance ratio

$$\phi = \sigma_1^2 / \sigma_2^2,$$

where σ is the (true) standard error of estimate from a regression. For the case for which the models have the same numbers of explanatory variables and thus degrees of freedom for error:

$$\phi = \frac{1 - R_1^2}{1 - R_2^2}$$

For any single data set purporting to be a realization of the true relationship, we have a regression standard error of estimate s which is an estimate of σ . Let $F = s_1^2 / s_2^2$; then confidence limits for ϕ are given by

$$\phi = F(K \pm \sqrt{K^2 - 1})$$

$$\text{where } K = 1 + \frac{2(1 - R_r)t^2}{n-2},$$

R_r is the correlation between sets of residuals for competing models 1 and 2, and t corresponds to the desired confidence interval for $n-2$ degrees of freedom.

In order to test the hypothesis that $\phi = 1$, we are interested in confidence limits that include unity. The above expressions can be solved to yield the value of F that would have to be observed in a given realization in order to find a statistically significant difference between models 1 and 2. The parameters are the correlation between sets of residuals R_r and the number of degrees of freedom ($\sim$ the number of observations). The resulting expression for $K = 1$ is:

$$\phi = F(1 \pm 2t) \sqrt{\frac{1 - R_r^2}{n-2}}$$

thus for $R_r = 0$ (uncorrelated residuals),

$$\phi = F(1 \pm 2t (n - 2)^{-1/2} ,$$

which provides very nearly the same confidence limits for the regression correlation coefficients as does the standard approach using the z transform (Snedecor and Cochran,²⁸ p. 185). However, for the case of $R_r \neq 0$, especially $R_r = 1.0$, the expression for ϕ provides much tighter confidence limits. The correlation R_r between alternative sets of residuals will be high when the residuals are controlled by either random factors or explanatory factors not included in the model, as opposed to the explanatory factors that distinguish model 1 from model 2.

7.5.2 Results

Comparisons of the regression standard errors and thus degrees of fit were made according to the above protocol for all possible combinations of pollutants, taken one at a time. This required consideration of four different data sets, defined by the availability of data on the various pairs of pollutants:

- o computed $\text{SO}_4^{=}$, SO_2 , and NO_x : 914 cities,
- o computed pollutants and SAROAD measured pollutants (Fe, Mn, $\text{SO}_4^{=}$): 137 cities,
- o computed pollutants and IP network pollutants (FP, TP, $\text{SO}_4^{=}$): 77 cities,
- o all pollutants: 48 cities (maximum coincident data set).

Only 6 out of the 69 comparisons were statistically significant ($p < 0.05$): both computed SO_2 and $\text{SO}_4^{=}$ provided better fits than FP, TP, or $\text{SO}_4^{=}$ (IP), for the 77 city data set. All other pollutant/data set combinations provided essentially equivalent regression fits, according to this statistical criterion. These findings are displayed graphically in Figure 20, in which a "+" entry indicates that the pollutant heading the column results in a lower regression standard error than the pollutant at the left end of the row. When this difference is statistically significant, the symbol is entered in bold face. Of course, selection of a different required level of significance could have resulted in different pollutants being judged as providing better regression fits. Note that 6 out of 69 at the 0.05 level is only marginally more than the 3 or 4 that would be expected due to chance.

Figure 20 indicates that some pollutants seem to have more than a random share of "wins." The scores are as follows:

- o Computed SO_2 : better fits in 19 out of 20 comparisons,
- o Manganese: better fits in 12 out of 13 comparisons,
- o Computed $\text{SO}_4^{=}$: better fits in 12 out of 20 comparisons,
- o Fine particles: better fits in 6 out of 13 comparisons,

- o Iron: better fits in 5 out of 13 comparisons,
- o Computed NO_x: better fits in 7 out of 20 comparisons,
- o Measured (SAROAD) SO₄⁼: better fits in 4 out of 13 comparisons,
- o Measured (IP) SO₄⁼: better fits in 2 out of 13 comparisons,
- o Total particles: better fit in 1 out of 13 comparisons.

	[n = 48]								
	NO _x	SO ₂	SO ₄ C	SO ₄ M	SO ₄ IP	FP	TP	FE	MN
NO _x	.	+	+	-	-	+	-	-	+
SO ₂	-	.	-	-	-	-	-	-	-
SO ₄ C	-	+	.	-	-	-	-	-	+
SO ₄ M	+	+	+	.	-	+	-	-	+
SO ₄ IP	+	+	+	+	.	+	+	+	+
FP	-	+	+	-	-	.	-	-	+
TP	+	+	+	+	-	+	.	+	+
FE	+	+	+	+	-	+	-	.	+
MN	-	+	-	-	-	-	-	-	.

	[n = 77]					
	NO _x	SO ₂	SO ₄ C	SO ₄ IP	FP	TP
NO _x	.	+	+	-	-	-
SO ₂	-	.	-	-	-	-
SO ₄ C	-	+	.	-	-	-
SO ₄ IP	+	+	+	.	-	-
FP	+	+	+	+	.	-
TP	+	+	+	+	+	.

	[n = 137]					
	NO _x	SO ₂	SO ₄ C	SO ₄ M	FE	MN
NO _x	.	+	+	+	+	+
SO ₂	-	.	-	-	-	+
SO ₄ C	-	+	.	-	+	+
SO ₄ M	-	+	-	.	+	+
FE	-	+	-	-	.	+
MN	-	-	-	-	-	.

	[n = 914]		
NO _x	.	+	+
SO ₂	-	.	-
SO ₄ C	-	+	.

+ = statistically significant

Figure 20. Regression fit comparison chart for single pollutants entered into a fixed demographic model. Bold = statistically significant.

The chi-squared distribution (or a sign test) can be used to test if a given pollutant's successes are more frequent than would be expected due to chance. The null hypothesis here is that the occurrence of a better regression fit is a random event. For $p < 0.05$ and 20 possibilities, more than 15 "wins" (or fewer than 5) would be statistically significant. For 13 possibilities, statistical significance would require more than 10 or fewer than 3. Thus, according to this criterion, measured (IP) $\text{SO}_4^{=}$ and total particles result in poorer fits more often than would be expected due to chance, and computed SO_2 and manganese provide better regression fits more often. When SO_2 is compared to manganese, there were no statistically significant differences, and SO_2 provided a slightly better fit for the 48 city data set while the reverse was true for the 137 city data set. Thus, based on this criterion we have no statistical basis for preferring one of these two pollutants over the other.

This analysis leads to a hierarchy of pollutants, with regard to their regression performance in the linear model described above:

- o Better regression fits: computed SO_2 and manganese,
- o Indeterminate fits: computed $\text{SO}_4^{=}$, and measured (SAROAD) $\text{SO}_4^{=}$, computed NO_x , fine particles, iron,
- o Worse regression fits: measured (IP) $\text{SO}_4^{=}$ and fine particles.

Note that this hierarchy differs from the results of Table 24 and from those of Figure 18.

7.6 SUMMARY OF PHASE III FINDINGS

The Phase III regression results confirmed that mortality is statistically significantly associated with air pollution in US cities, using ca. 1980 data. The association has practical significance, representing from about 5-10% of all-cause mortality. These findings were robust across city size, by region, and generally were stronger for the higher levels of pollution. Thus, they do not appear to be artifacts of the analysis, within the constraints imposed by use of total (all cause, all ages) mortality.

However, the Phase III analysis also established that it is not possible to assign a specific air pollutant to this association with certainty. Some pollutants either showed no association or a negative one, including ozone, lead, and carbon monoxide, and thus could be eliminated from further consideration, based on the available data. However, of those pollutants which showed significant positive associations with mortality, it was considerably more difficult to determine which were "best" in order to suggest candidates for further investigations of possible causal relationships. Such determinations were hindered by collinearity among pollutants and by the large numbers of cities lacking measured values of pollutant concentrations.

In stepwise regressions which allow the demographic model to be adjusted (both different variables and different coefficients), the largest effects on mortality were seen for the three sulfate variables. However, the three computed pollution variables (SO_2 , $\text{SO}_4^{=}$, and NO_x) all tended to have the largest

t values (most statistically significant). Differences among these three were quite small, which led to the search for tests to determine whether small differences between regressions were statistically significant.

Taken one at a time in a fixed demographic model (varying coefficients) for the various possible data sets, the only statistically significant differences among the nine pollutants compared were that both computed $\text{SO}_4=$ and SO_2 provided better regression fits than any of the three pollutants from the inhalable particulate network (fine particles, total particles, and sulfate). However, considering that all of the 69 pairs of regressions compared in this way comprise a set of random results, both SO_2 and manganese appeared superior to the others more often than chance would allow ($p < 0.005$), and measured (IP) sulfate and total particles appeared worse. All other pollutants were indistinguishable from one another on this basis.

Using a two-stage regression approach which forces each pollutant into a fixed demographic model with the same variables and coefficients (but different numbers of cities according to the availability of pollutant data), manganese, iron, and SAROAD $\text{SO}_4=$ provided the best regression fits, but the elasticities of all nine pollutants were essentially the same (about 5%). Note that this comparison uses mortality rates "adjusted" for demographics, smoking, and population change.

Thus among the three comparison techniques, no one pollutant is clearly "superior." In multiple pollutant models, significance was shown for either iron or manganese in combination with the computed pollutants (one at a time). It thus appears that statistical criteria alone will not suffice in defining air pollution relationships with total mortality.

8.0 DISCUSSION OF REGRESSION RESULTS

8.1 INTERPRETATION OF THE AIR POLLUTION RESULTS

The preceding regressions showed a consistent positive association between total mortality and concentrations of sulfur oxides, nitrogen oxides, particulate trace metals, and fine particles. We need to consider the strength of these associations and possible alternative interpretations before speculating about causality, especially given the lack of specificity of the results.

One way to quantify the strength of the association is by calculating the elasticity, which has been defined as

$$E_i = b_i \frac{\bar{x}_i}{\bar{y}}$$

which can be interpreted as a dimensionless regression coefficient. It represents the percentage that the dependent variable would change for a given percentage change in the independent variables. Thus, for the computed sulfate variable ($\bar{x}=5$), a coefficient of 0.10 indicates a 5% effect on mortality ($\bar{y}=10$). Thus, each percentage change in sulfate would result in a 0.05% change in mortality, or that complete elimination of sulfate would result in a 5% improvement in mortality. All of these statements are conditional on a causal relationship, which has not been established.

The sum of the air pollution elasticities derived in this study range from about 0.05 to over 0.20 (the higher figures based on inclusion of 1978 or 1982 TSP). These are very large figures, considering the modest concentration levels involved. By comparison, the elasticity for cigarette smoking ranges from about 0.18 to about 0.34, depending on the regression; drinking water hardness, only about 0.04. Elasticities for demographic variables also vary substantially: for % ≥ 65 , 0.60-0.80; for poverty, about 0.05; % long term resident, 0.10.

From the standpoint of the model fit, i.e., explaining the variance or spatial variation in mortality rates, the variables may rank somewhat differently. The measure here is a standardized partial regression coefficient, defined as

$$\beta_i = b_i \frac{\sigma_{xi}}{\sigma_y}$$

By this measure, demographic factors are most important, explaining over 90% of the variance. Smoking only explains about 2%, because of the relatively low spatial variation in smoking across the U.S. (σ_x). Socioeconomic factors account for about 0.5% and air pollution, about 3%. Use of age-race-sex adjusted or specific mortality rates would thus create an entirely different regression situation, in that a much smaller degree of spatial variation would have to be explained by socioeconomic and pollution factors. Lower R^2 's would be expected, but the possibilities for confounding between demographic and pollution factors would be greatly reduced.

Table 32 lists previous estimates of regression coefficients for some of the variables used in this study. Agreement appears satisfactory, with the possible exception of % $\geq$ 65 and birthrate. The manganese coefficient in the present study appears to have a substantially higher coefficient than found in previous work, in spite of a much lower average concentration at present. This may be an indication of surrogate behavior, which would be characterized by constant elasticity.

8.2 OTHER LIFESTYLE PARAMETERS

Smoking is only one of a number of personal or lifestyle variables that can affect mortality rates. Others may include exercise, diet, obesity, ethnic origin, and alcohol consumption. Few quantitative studies have been done on these effects, either separately or jointly. In any event, the lack of city-specific measures of these factors makes their consideration for the present study design somewhat academic.

Alcohol consumption has been found to have mixed effects on mortality, small amounts being beneficial and large amounts harmful.³⁰ Thus it is difficult to use gross consumption data on an average per capita basis as an index variable. Since heavy alcohol consumption appears to often be accompanied by smoking (cirrhosis of the liver and respiratory cancer are correlated), the cigarette consumption variable may include some effects of alcohol use as well, through collinearity.

Body weight has similar effects; the lowest total mortality rates occur at average or slightly below average weights.²⁸ However, heart disease had more of a monotonic relationship with weight. For heart disease and total deaths, persons weighing more than 40% above average had approximately twice the average risk of dying.

Part of the unexplained variance in total mortality rates may be due to such lifestyle factors. If there are systematic regional variations in these results, some confounding may have occurred, for example with respect to the sulfur variables, which also have a regional distribution.

The behavioral risk factor survey also asked questions about body weight and exercise. Among the fragmentary results available, there did not seem to be regional patterns of excessive risk. Also, males were often less likely to be overweight or sedentary, which does not explain their excess mortality relative to females. Thus these factors may not be terribly important with respect to longevity.

8.3 DRINKING WATER VARIABLES

The region of very soft drinking water lies along the Eastern seaboard, including most Atlantic coast and some Gulf coast cities. Some West coast cities also have soft water. The high hardness region includes the Mid-west and parts of the Southwest. Some consumers in hard water areas use commercial softeners for laundry and bathing, but often not for cooking and drinking water.

The regression results for this variable are equivocal. Water hardness was not selected as a significant predictor variable for the n=159 city set,

Table 32. Independent Estimates of Regression Coefficients

Variable	Est. Coeff. (External Data)	Basis	Results from this Study			
			n=916	n=459	n=159	n=48-53
% $\geq$ 65	0.465	Difference in total US mortality rates for % $\geq$ 65 to % $<$ 65	0.54	0.67	0.70 to 0.75	0.58 to 0.83
% Black	0.0276	Difference in age-adjusted total US rates, white-black			0.008 to 0.027	0.004 to 0.027
% Asian	0.005	Difference in age-adjusted total US rates, white-"all other"			0.04 to 0.05	0.07 to 0.08
% Spanish	-0.017	Difference in age-adjusted total US rates, Mexican, Puerto Rican or Cuban born, all whites + all blacks adjusted for poverty ³⁴	-0.02 to -0.04	-0.04	-0.04 to -0.05	-0.02 to -0.08
Smoking	0.012	Relative risk, by amount smoked (Surg. Gen. Rpts) ¹⁰	0.009	0.011 to 0.013	0.011 to 0.018	0.007 to 0.019
Water Hardness	-0.0015 -0.0072 -0.004	Great Britain ³⁷ Italy ³⁶ Great Britain ³⁷	--	-0.006 to -0.001	-0.0009	-0.002 to -0.003
% Poverty	0.11-0.17 0.02-0.06 0.034	1970, cities ⁶ 1970, SMSAs ¹⁸ 1970 expected value ³⁸	0.04 to 0.06	0.07 to 0.09	0.018 to 0.05	-0.05 to -0.009
Birth Rate	0.05-0.12 0.01-0.02	1970, cities ⁶ Census undercount	0.18	0.21	0.17 to 0.23	0.13 to 0.29
% College Graduates	-0.03 to -0.09	1970, cities ⁶			0 to -0.02	-0.06 to -0.08
Sulfate	-0.04 to +0.12	Various studies cited ⁹			0.07 to 0.23	0.05 to 0.18
Iron	0.30	1970, weighted regression for cities with Fe as sole pollutant ⁶			0.34 to 0.44	0.07 to 0.44
Manganese	-0.017	1970, cities ⁶			3.0 to 12.0	12.0

which were constructed specifically for this purpose. When included by means of "forced entry", the variable was not significant, regardless of which air pollutants were included in the regression. However, the variable was just significant ($p = 0.05$) for the $n = 53$ data set, including the total particle mass variable, only when the total mass variable was included in the regression. (Conversely, the total particulate mass variable was only significant for this data set when the water hardness variable was included.) Water hardness was rarely significant in the Phase III analysis. The values of the water hardness coefficient were consistent with previous studies,^{19,32} corresponding to about 1-3% of total mortality, based on the elasticity at the mean. Thus, water hardness appears to be less important than air pollution, at least for total mortality. Water hardness is expected to be more important for mortality from heart disease.

8.4 ETHNIC ORIGIN

There are also sizable mortality differentials in the US population by country of origin, as noted by Kitagawa and Hauser³³ for the 1960 period. These reflect the same trends shown by the native countries, i.e.,

- o U.K., Ireland and Finland; higher mortality,
- o Mexico and Mediterranean countries; lower mortality,
- o Austria and Eastern European countries; higher mortality.

These countries of origin are reflected in the U.S. Census Divisions as follows, based on the 1970 Census (1980 data not given in CCDB):

Region	Leading Country of Origin for Foreign Stock	% of Total (Region) Population
New England	- Canada	(7.9%)
Middle Atlantic	- Italy	(6.1%)
East North Central	- Germany	(2.4%)
West North Central	- Germany	(2.9%)
South Atlantic	- Germany	(0.9%)
East South Central	- Germany	(0.4%)
West South Central	- Mexico	(3.7%)
Mountain	- Mexico	(2.4%)
Pacific	- Mexico	(4.3%)
* percentage of "foreign stock" population times percentage for leading country of origin.		

Thus one can see that entire regions are only slightly affected by the foreign born population (actually 1st and 2nd generations). However, some major cities may be slightly more affected; for example, in 1970 New York City had 8.6% Italian "foreign stock", while Chicago had 5.7% Polish. This difference might be enough to contribute a small mortality differential in favor of New York.

8.5 OTHER ETHNIC GROUPS

8.5.1 Asian

The regression coefficient for the variable "% Asian" was found to be positive, that is, after controlling for all other factors, cities with relatively high percentages of Asians and Pacific islanders had higher mortality rates, which appears somewhat counterintuitive. First of all, because of the ecologic nature of the regressions, this result does not necessarily imply that those particular groups had higher mortality rates themselves, only that those cities with higher percentages of Asians had higher overall mortality rates when all variables were accounted for. 1980 death rates for detailed racial groups are not yet available; Kitagawa and Hauser³³ found that in 1960 Asian Indians had 32% higher mortality than whites; Japanese, 30% lower; and Chinese, 18% higher. If these groups are weighted according to their population numbers in 1980, the result is a 2.7% increase in mortality relative to whites, in accordance with the present regression results. However, there are also sizable numbers of Filipinos, Koreans, and Vietnamese to be considered, for which no mortality data are available.

A further consideration is socioeconomic status and income. According to the 1980 Census, Asians and Pacific islanders had a higher median income than whites, but also a higher proportion earning under \$5000 annually. They also tended to be better educated. Thus, to the extent that education or income variables are used in the same regression with "% Asian", there could be some interactions present.

8.5.2 Spanish

The Hispanic population tends to be poorly defined and includes both whites and blacks. Mortality statistics are not normally tabulated on this basis, but Rosenwaike³⁴ provided age-adjusted rates for three Hispanic subgroups on the basis of place of birth: 1979-81 deaths to those born in Cuba (20% of Rosenwaike's study group), Mexico (56%), and Puerto Rico (24%). The age-adjusted mortality rate for this group was about 12% lower than for the weighted average for all whites and blacks, with notable reductions in heart disease and cancer (increase in accidents, homicides, and liver disease). Rosenwaike noted that this large improvement in mortality among aged Hispanics was achieved in spite of a somewhat disadvantaged economic status (including substantially less use of the U.S. health care system).³⁵ Taking the poverty statistics into account, the mortality rate for this Hispanic subpopulation appeared to be about 19% lower than expected. This differential translates into an expected value for the "% Spanish" regression coefficient of -0.017, which is somewhat lower than that derived from these regressions, but still within range. Rosenwaike points out that the Spanish population may be particularly prone to Census undercount, which would tend to inflate their mortality statistics and thus bring the exogenous coefficient more into line with these results.

We can only speculate about reasons for the reduced heart disease and cancer rates for Hispanics. Possibilities include selective migration of the most fit (especially for Cubans, who had the lowest rates of the three subgroups, by far) and lifestyle parameters such as diet and smoking.

8.6 DISCUSSION - OTHER RELEVANT STUDIES

Our analysis is intended to provide estimates of the effects of air pollution on total mortality, but also to give insights into other contributing factors that must be understood as part of the analytical process. In so doing, the work of previous authors must be considered.

8.6.1 Sauer's Correlation Analysis of 1960's and 1970's Mortality

Sauer¹⁴ presents data by states and for 510 State Economic Areas for a number of cause-of-death categories for 1968-72 mortality, taking into account age, race, and sex. Age was accounted for by presenting age-adjusted rates for persons aged 35-74 years. Although no regression models are used, he gives a number of interesting correlations that could shed some light on the possible sources of spatial variations in mortality. The diseases (causes of death) of primary interest to this study are:

major cardiovascular (MCV)	(ICD*-390-458, 582-4, 780-796)
ischemic and other heart disease (IHD)	(ICD 410-429)
lung cancer (LC)	(ICD 160-163)
influenza and pneumonia (FLU)	(ICD 470-486)
chronic respiratory disease (CRD)	(ICD 490-3, 517-19)
suicide (S)	(ICD E950-E959)

*ICD = International Classification of Diseases.

The hypotheses that could be tested by means of Sauer's correlation analyses are:

- o Are the relative rankings by state or SEA stable in time (1959-61 versus 1968-72)?
- o Do blacks and whites have similar patterns of mortality?
- o Do males and females have similar patterns of mortality?
- o Do these relationships vary by metropolitan versus non-metropolitan residence?

Formally, we postulate a relationship for each age-adjusted, sex, or race specific group and cause of death such as:

$$\text{death rate} = a_0 + a_1 (\text{genetic factors}) + a_2 (\text{personal habits}) + a_3 (\text{local environment}) + a_4 (\text{regional factors}). \quad (1)$$

Sauer's correlations, which represent partial derivatives of death rates for specific disease groups with respect to several of the above factors, can be used to hypothesize which of the above factors may be most important, and which may have environmental factors (relating to location). Suicide was included in the cause of death categories above as an example which is unlikely to have an environmental influence. One should also keep in mind that environmental conditions were changing in many cities from 1960 to 1970, usually for the better.

Since Sauer's data comprise a large number of observations (510 for whites), the usual significance criteria for correlation are not very useful; a coefficient of 0.1, which only explains 1% of the variance, is statistically significant. For this reason, we will arbitrarily define a correlation coefficient greater than 0.7 as "high" (explains more than half of the variance), between 0.5 and 0.7 as "moderate," and less than 0.5 as "low" (less than 25% of the variance).

8.6.1.1 1959-61 versus 1968-72.

The following SEA correlations were presented by Sauer with respect to time:

	White males		White females	
	Metro.	Non-metro.	Metro.	Non-metro.
major cardiovascular (MCV)	0.85	0.86	0.84	0.82
ischemic heart disease (IHD)	0.80	0.76	0.84	0.79
lung cancer (LC)	0.54	0.78	0.32	0.34
influenza and pneumonia (FLU)	0.50	0.36	0.38	0.32
chronic resp. dis. (CRD)	0.70	0.66	0.48	0.42
suicide (S)	0.55	0.55	0.40	0.29

"High" correlations over time indicate that the relative rankings of all the factors in Eq. (1) have remained stable in various locations over time, as is the case for all categories of heart disease and for lung cancer in non-metropolitan males. On the other hand, all other causes of death for females had low correlations, indicating changing causal factors. Temporal correlations do not give any hints as to the origins of the spatial patterns, nor whether the rates have changed in magnitude over time. Note that since changes in local environmental conditions have not been uniform from 1959 to 1972, those diseases with strong (local) environmental factors would be expected to show lower temporal correlations (lung cancer and "FLU").

8.6.1.2 Blacks versus Whites

The following SEA correlations were shown by Sauer for blacks versus whites for 1968-72. Only SEA's with a black population greater than 10,000 were included (n = 91 metropolitan, 64 non-metropolitan).

	Males		Females	
	Metro.	Non-metro.	Metro.	Non-metro.
major cardiovascular (MCV)	0.73	0.62	0.33	0.42
ischemic heart disease (IHD)	0.72	0.60	0.47	0.58
lung cancer (LC)	0.10	0.24	0.25	0.45
influenza and pneumonia (FLU)	0.39	0.38	0.50	0.33
chronic resp. dis. (CRD)	0.04	0.19	0.21	0.17
suicide (S)	-0.03	0.26	0.12	-0.09

Blacks and whites in a given SEA may be expected to have differing genetic and personal predisposing factors and differing local environments, but the same regional environment. Thus high black-white correlations indicate that regional factors may be more important than local or genetic factors. This was the case only for metropolitan male heart disease deaths. Smoking habits are known to differ between blacks and whites, and probably explain the low correlations for lung cancer, CRD, and perhaps female heart disease, together with the possible effects of local environmental conditions. The relatively low numbers of observations for black-white correlations weakens these conclusions somewhat.

8.6.1.3 Males versus Females

The following male/female correlations were presented for SEAs for 1959-61 and 1968-72.

	1959-61		1968-72	
	Metro.	Non-metro.	Metro.	Non-metro.
major cardiovascular (MEV)	0.63	0.69	0.78	0.74
ischemic heart disease (IHD)	0.72	0.68	0.81	0.77
lung cancer (LC)	0.21	0.32	0.49	0.46
influenza and pneumonia (FLU)	0.57	0.40	0.58	0.52
chronic resp. dis. (CRD)	0.67	0.35	0.58	0.44
suicide (S)	0.42	0.32	0.50	0.25

Except for one-person households, males and females in a given SEA would be expected to have the same local and regional environments, somewhat similar personal factors, but differing genetic factors. The 1968-72 heart disease correlations imply such a finding, and the 1959-61 values are nearly large enough to be classified as "high." None of the other cause of death categories do, although it is interesting that all the male-female correlations are higher than the black-white correlations. The relatively larger correlations for "FLU" may reflect the contagious nature of the disease, for example, and for CRD, the higher correlations in metropolitan areas may reflect the influence of local environmental factors.

In summary, the above three tabulations of correlations imply that spatial patterns of heart disease deaths have been stable in time, may be influenced by local environmental factors, and perhaps by regional ones. Genetic and personal lifestyle factors would seem to be less important. These conclusions are admittedly speculative, but the marked differences between the heart disease correlations and those for other causes of death with respiratory connotations strongly suggest that the current air pollution mortality analysis be repeated for specific causes of death.

8.6.1.4 Correlation Between Disease Categories

One way to hypothesize about the potential nature of causal factors is to examine the correlations between these causes of death, as given below in Table 33 for white males and females in 1968-72 SEAs.

Table 33. Correlations Between Major Causes of Death for Whites 1968-72

	<u>MCV</u>	<u>IHD</u>	<u>LC</u>	<u>FLU</u>	<u>CRD</u>	<u>S</u>
MCV males	1.0	*	0.61	0.46	0.21	NS
females	1.0	*	0.22	0.40	NS	NS
IHD males		1.0	0.58	0.41	0.17	NS
females		1.0	0.25	0.25	NS	NS
LC males			1.0	0.37	0.25	NS
females			1.0	0.18	0.28	0.28
FLU males				1.0	0.30	NS
				1.0	0.20	NS
CRD males					1.0	0.38
females					1.0	0.33
S males						1.0
females						1.0

*Correlation not relevant since IHD is a subset of MCV
 NS denotes $r < 0.17$ ($p > 0.001$).

The strongest correlation is for male heart disease versus lung cancer, which suggests smoking as a common (personal) factor (also see Ref. 36). The lack of correlation between CRD and either heart disease or lung cancer suggests the influence of other factors (local) for CRD, but since CRD rates are low (~1.5% of total) there may also be a problem with sampling variability. The low correlations for females would seem to argue against an important common environmental factor for these diseases.

8.6.1.5 Comparison of Average Rates

To get some insight into plausible hypotheses for local versus regional causal factors, it is necessary to consider the average rates, again as given by Sauer¹⁴ (Table 34). In general, the differences between race and sex groups tend to far outweigh those due to metropolitan status, except for ischemic heart disease. Acute deaths (heart attacks) are higher in non-metropolitan areas, whereas chronic deaths are just the opposite (these two subsets had been combined in the previous analysis). However, the large differences between death rates for chronic ischemic heart disease among the four groups imply that regional factors may operate as multipliers rather than as additives. The higher rates for non-metropolitan acute deaths argue against local air pollution as a common factor. Clearly a regression analysis for individual causes of death vs. various explanatory factors is warranted; Sauer's use of combined chronic and acute ischemic heart disease deaths may have masked some important relationships. Wallace and Gill⁴⁰ have confirmed Sauer's findings with a county-level correlation analysis.

Table 34. U.S. Age Adjusted Death Rates (Age 35-74) for 1968-72

	(Deaths per 100,000 population)							
	White				Black			
	Male		Female		Male		Female	
	Metro	Non-Metro	Metro	Non-Metro	Metro	Non-Metro	Metro	Non-Metro
MCV	818	813	350	336	1120	1244	759	846
IHD	622	593	224	206	672	617	413	394
Acute	401	463	130	140	306	386	170	213
Chronic	221	130	94	66	369	201	243	180
LC	120	108	26	19	161	113	28	19
FLU	37	32	18	16	92	70	41	35
CRD	53	59	14	13	53	39	16	11
S	28	32	14	10	13	11	4	2

8.6.1.6 Sauer's Explanatory Hypotheses

Sauer identifies the Southeast coastal plain as an area of high cardiovascular mortality for males, but advances no causal hypotheses. He finds high rates for all causes (males and females) dating as far back as 1949-51 in areas with mining activities or with a history of mining. Elevation above sea level is negatively associated with heart disease (as in 1949-51), as is lung cancer. However, CRD is positively associated. He also notes that the relationship with height above sea level is not borne out by international comparisons. Sauer identifies a weakening trend over time of the influence of population density, although suburban counties generally have lower death rates than do center-city counties, as was the case for 1949-51. The effect of medical care resources was mixed, depending on the type of medical practitioner. Sauer concludes that if the whole country could achieve the lowest rates shown, 170,000 fewer deaths (14%) would occur each year (to those under 75 years of age).

8.6.1.7 Conclusions From Reanalyzing Sauer's Data

In addition to Sauer's own findings, the following results are relevant to this inquiry.

- o Patterns of heart disease and to a lesser extent, respiratory disease, have been stable in time.
- o There are regional factors for heart disease (primarily ischemic) affecting males and females (and black males).
- o Local factors may influence deaths from chronic respiratory disease.
- o Smoking may be a common factor linking heart disease and lung cancer (males).

8.6.2 Cross-Sectional Studies in Other Countries

The persistence of geographic patterns of mortality in the U.S. makes it impossible to achieve a truly independent verification of the regression findings simply by analyzing a different time period. However, there is no reason a priori to expect the various factors affecting mortality to have similar interdependence in other countries, and thus confirming evidence should be sought from international studies.

8.6.2.1 Chinn et al. (UK)⁴¹

Chinn et al. analyzed mortality in 116 counties and boroughs in England and Wales during the period 1969-1973.

- o Dependent variables were mortality for infants (aged 0-1) and for males and females aged 45-54, 55-64, 65-74 -- for all causes, stomach cancer, lung cancer, flu, pneumonia, bronchitis, hypertension, and ischaemic heart disease.
- o Independent variables were (then-current) SO₂ and particulate (British smoke) levels, various socioeconomic variables, latitude, temperature, rainfall, water hardness, and smoking (limited data).

They concluded that there was "no consistent relation of smoke or sulphur dioxide with mortality from all causes or with mortality from specified causes postulated a priori to be related to pollution. In particular there was no significant association between smoke and mortality rates for respiratory illness. Comparison with results from similar analyses of data from two previous decades suggested that a decline in the strength of associations had occurred in parallel with declining levels of the pollution." They also found:

- o No associations of infant mortality with smoke or SO₂.
- o Some sensitivity of results to the selection of data sets, especially for all-cause and heart disease mortality. Deleting the London boroughs, which were the highest in SO₂, strengthened some of the SO₂ correlations, especially for females and for deaths from heart disease. Several of these became significant ($p < 0.05$).
- o Including either water hardness or smoking variables weakened the associations of male mortality with SO₂.
- o Even in 1948-50, female mortality from heart disease was not associated with air pollution (as an index variable).
- o Associations were shown more often for the 45-64 age group than for the 65-74 age group.

The strengths of this study include:

- o The geographic areas considered were quite small by U.S. standards (maximum of 70 square miles) and each contained four monitoring sites,

on average. Thus exposure to the specific air pollutants considered appears to have been much better characterized than in most comparable U.S. studies.

- o Both age-specific and cause-specific deaths were considered, and five-year averages (1969-73) were used to obtain numerical stability.
- o The data for both SO₂ and smoke contained an adequate range to be able to detect an effect, if one were to exist. Levels ranged from essentially background to almost twice the U.S. ambient standard. SO₂ and smoke were only weakly correlated ($r=0.25$).
- o Both drinking water hardness and smoking were included, although the smoking analysis was quite crude.

The weaknesses included:

- o Only winter pollution levels were used, since summer levels were "uniformly low." If the latter were strictly true, the degrees of association would not be affected but (nonstandardized) regression coefficients (not given) would be too low. Use of year-round data would have provided a more straightforward comparison with other studies.
- o No data were included on frequency of fog, sulfate particles, oxidants, or acid aerosols.
- o SO₂ was correlated with male smoking, making it difficult to separate the effects of each.
- o No attempt was made to construct long-term average levels of pollution from the available historical data.
- o Although the paper presents regressions of current mortality on indices of past pollution levels (back to about 1950), it gives no indications as to what typical concentrations might have been at that time.

The findings of this study are in contradiction to many of the U.S. cross-sectional studies, in that there is no association of small particles (British smoke) with mortality. It confirms the importance of considering smoking and drinking water hardness. The study provides no confirmation of the hypothesis that the very young and the very old are most susceptible to the effects of air pollution. The instability of the SO₂ results and the correlation of SO₂ with smoking (to the extent it could be shown with the limited smoking data available) suggest that the SO₂ findings may be compromised through collinearity with smoking (when smoking was not included).

8.6.2.2 Göttinger (West Germany)⁴²

Census tracts in Munich were studied for deaths to males and females over 65 from all causes. Control variables included climate, education, and population density. Air pollution variables were SO₂, particulates (TSP), and CO. Only TSP was significant ($p < 0.05$) for both males and females.

8.6.2.3 Krzyzanowski and Wojtyniak (Poland)⁴³

This was a 10 year follow up study of 4355 residents of Cracow, selected randomly. TSP and SO₂ were measured in 20 locations, and the subsets were classified as to high (TSP = 180 ug/m³ and SO₂ = 114 ug/m³) and low (TSP = 109 ug/m³ and SO₂ = 53 ug/m³) levels of pollution. Pollution was measured during the first 5 years of the 10-year study. Pollution was marginally significant for males (p = 0.051), more so for smokers (p = 0.029). For females, air pollution was significant but negative (p = 0.024), but smoking was not significant. It was not possible to separate the effects of TSP from SO₂. No explanations were offered for the negative finding for females.

8.6.2.4 Comparisons of Individual Mortality Experience

Hanis et al.⁴⁴ performed a retrospective study of refinery and chemical plant workers at three locations: Baton Rouge (LA), Baytown (TX), and Bayway/Bayonne (NJ), controlling directly for smoking and occupational exposures. For all causes of death, the differences among the three locations were negligible (0.25%) and all three were lower than the corresponding rate for the U.S. as a whole (probably a manifestation of the "healthy worker effect"). "Exposed" workers (to petroleum, petrochemicals, and related substances) had higher death rates than nonexposed workers for all causes (20%) and for heart diseases (40%). New Jersey workers had slightly higher cancer rates including lung cancer, but substantially lower deaths from respiratory disease. Baton Rouge workers had slightly lower (4%) heart disease deaths. The results of this study suggest that regional mortality variations are small when like, controlled populations are compared.

8.6.2.5 Trend Analysis

Another largely unexplained phenomenon in US epidemiology is the nationwide decline in coronary heart disease mortality, which began in about 1968.⁴⁵ The trend is stronger for acute deaths (37.4% decline) than for chronic (14.1% decline), and for all age groups for acute but mainly older groups (65+) for chronic. Various life style factors have been considered as possible explanations (better diet, more exercise, etc.), but environmental factors have not yet been considered. The finding by Sauer of higher acute heart disease mortality in non metropolitan areas would appear to argue against improvements in local air quality as an explanation.

8.7 EVIDENCE FOR CAUSALITY - PHYSIOLOGICAL BASIS

The Phase III analysis concluded that SO₂, SO₄⁼, and Mn were all associated with mortality, and that criteria other than statistical were needed to consider causality. Appendix B discusses the known physiological effects of some of these pollutants, notably SO₂ and NO_x. We note that time-averaged population exposure to SO₂ will generally be poorly represented by outdoor concentration levels, since this reactive gas is strongly adsorbed by indoor surfaces. In addition, SO₂ is usually absorbed by the moist upper airways and is not known to have long term health effects at the low concentrations involved here.

Sulfate particles are generally small enough to penetrate deeply into the lung, where they may be dissolved in mucus. If these particles are sufficiently acidic, some changes to respiratory mechanics may result. However, normally there is sufficient ammonia present in situ to neutralize the small particles characteristic of secondary aerosols.

Manganese particles are usually small and are not known to be toxic at the concentration levels involved here. Not only is Mn an essential trace element,⁴⁶ but also, intake through food and water is likely to be larger than through inhalation. These findings of an association between mortality and Mn may indicate a surrogate for some aspect of heavy industry (which produces airborne Mn). Analysis of mortality rates by cause and sex would be helpful in evaluating this hypothesis.

In summary, although each of the pollutants showing the strongest associations with mortality have been linked to adverse health effects in some way, none of them is known to have such an association on a physiological basis at the concentrations in question.

9.0 CONCLUSIONS

This analysis shows that concentrations of certain air pollutants may be predictors of 1980* mortality in U.S. cities. The pollutants include sulfur oxides, oxides of nitrogen, particulate trace metals (Fe and Mn), and fine particles; it is not possible to define the most important pollutants with certainty. The strength of the association between air pollution and mortality is about 5-10%, which is somewhat higher than similar estimates for 1970 mortality. No reasons are evident for this increase, since air concentrations of pollutants have decreased since 1970. No such associations were found for ozone, carbon monoxide, or lead particles; it is not known to what extent these negative findings are due to poor estimates of actual exposures to these pollutants. The analysis also showed that some of the pollutant/mortality associations are sensitive to the selection of data sets for analysis and to the structure of the models used to account for demographic and socioeconomic factors.

This inquiry was conducted in three phases. In the first phase, the pollutants considered included a number of measured pollutants and one species, sulfate aerosol, for which concentrations were obtained from a numerical model. This procedure allowed a large number of cities (over 900) to be considered, since the limitations of measurement networks were avoided. In the second phase, the unimportant pollutant variables were dropped and a number of detailed statistical checks were made on the validity of the regressions.

Since the first two phases of this analysis concluded that the computed sulfate variable was usually statistically significant and thus very important, the analysis was extended in a third phase to include additional computed air pollutants, SO_2 and NO_x , in order to test the hypothesis that the success of the sulfate variable may have been due to the use of computed rather than measured values, per se. Although the correlation between computed values of $\text{SO}_4^{=}$ and NO_x was only about 0.45, there were no statistically significant differences in regressions employing any of the three computed pollutant variables (one at a time), either for all available cities or for various subset combinations. When these statistical comparisons of fixed demographic model regressions were extended to compare results for nine separate pollutants (one at a time), it became apparent that SO_2 and manganese provided the lowest regression errors more often than would be predicted by chance, in contrast to sulfate and NO_x .

The analysis also indicates that the usefulness of sulfate aerosol as a predictor of total mortality in cities may have increased since 1970 (and presumably earlier). The change may either be due to reduced variance (which could have resulted either from use of a computed measure of sulfate aerosol in the present study rather than actual measurements, or from changes in the covariance structure of the data set), or from an actual change in mortality.

These findings were shown to be generally robust to the presence of influential observations, although not necessarily to major changes in the data sets

*Actual 1979 calendar year deaths divided by the population estimated as of April 1, 1980.

sets under consideration. Tests for behavior of the regression residuals (homogeneous variance) were satisfactory. Thus, on statistical grounds, the mortality models which were developed appear to be acceptable.

This analysis did not deal directly with the issue of causality, nor can any study of this type provide such information directly. Neither is it possible to separate acute effects from chronic effects: at best, the study provides hypotheses for further evaluation. However, it should be mentioned that the other variables in this model predict directions and magnitudes of effects due to demographic variability (age and race), cigarette smoking, and drinking water hardness which are in agreement with previous independent estimates. Thus there is no a priori reason to suspect that the air pollution effects shown are artifacts of the analysis. Further investigation appears warranted. On the other hand, cross-sectional analyses necessarily deal with the places of death and not the causes. No causal, i.e., physiological, mechanisms have been proposed which would explain the health effects mechanisms for long term exposure of these pollutants at the relatively low concentrations experienced ca. 1970-1980. Indeed, those pollutants with known adverse health effects at current ambient concentrations, such as lead, CO, and ozone were not shown to be predictors of increased mortality, and some may have even indicated "beneficial" effects, a counterintuitive finding. In addition, there is no strong statistical evidence that past exposures (i.e., 1970) are better predictors of 1980 mortality than 1980 exposure estimates. Further, there is no confirmatory evidence available from international studies. Since the geographic pattern of "unexplained" mortality is not greatly changed by the introduction of air pollution variables into the model, it is possible that some important causal variable(s) may have been omitted from the analysis.

10.0 RECOMMENDATIONS

The finding of possibly significant associations of human mortality with air pollution at current ambient concentrations requires further investigation and confirmation. First, those findings relating to the U.S. should be reviewed for consistency with respect to specific causes of death. Then, the analysis should be repeated for subsets of the population stratified by age, sex,* and race, to preclude the need for demographic adjustment variables. Such an analysis will require partitioning of statewide smoking rates into age, race and sex specific rates, using newly available survey data on behavioral risk factors. Use of age as a stratifying variable rather than as a control variable will reduce the variance of the dependent variable and allow consideration of lifetime exposures to pollution. Elimination of racial control variables will increase the number of degrees of freedom, and should help compensate for the "noiser" mortality rates incurred when population subsets are used.

Finally, the findings should be confirmed in the light of international mortality experience, the only truly independent data available. This in turn may require inclusion of country of origin as a control variable in the U.S. studies.

* For example, white male mortality rates tend to be high in the Southeast U.S., whereas white female rates are high in the Northeast.

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APPENDIX A

**AIR QUALITY AND
DRINKING WATER HARDNESS**

DATA BASE

(173 cities)

Cities in the 53-city Data Set

(dichotomous samplers)

'AL'	BIRMINGHAM
'AL'	MOBILE
'AZ'	PHOENIX
'AR'	LITTLE ROCK
'CA'	FRESNO
'CA'	BAKERSFIELD
'CA'	LOS ANGELES
'CA'	PASADENA
'CA'	SAN FRANCISCO
'CA'	SAN JOSE
'CO'	DENVER
'CT'	HARTFORD
'FL'	TAMPA
'GA'	ATLANTA
'ID'	BOISE
'IL'	CHICAGO
'IN'	GARY
'IN'	INDIANAPOLIS
'KS'	WICHITA
'KS'	TOPEKA
'KY'	ASHLAND
'MN'	MINNEAPOLIS
'MN'	ST. PAUL
'MS'	JACKSON
'MT'	BUTTE
'NE'	OMAHA
'NV'	RENO
'NJ'	CAMDEN
'NJ'	JERSEY CITY
'NM'	ALBUQUERQUE
'NY'	BUFFALO
'NC'	DURHAM
'NC'	CHARLOTTE
'OH'	CLEVELAND
'OH'	CINCINNATI
'OH'	STEUBENVILLE
'OH'	DAYTON
'OH'	AKRON
'OH'	YOUNGSTOWN
'OK'	OKLAHOMA CITY
'OR'	PORTLAND
'PA'	BETHLEHEM
'PA'	PHILADELPHIA
'RI'	PROVIDENCE
'TN'	NASHVILLE
'TN'	CHATTANOOGA
'TX'	HOUSTON
'TX'	DALLAS
'UT'	SALT LAKE CITY
'VA'	NORFOLK
'WA'	SEATTLE
'WA'	SPOKANE
'WV'	EL PASO

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	WATER HARD. (ppm)	ASTRAP SO_4	CO (ppm)			IRON			LEAD			SAROAD SO_4		
				MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b
AK	ANCHORAGE	.	.	2.30	14560	4	1.28	53	1	0.47	354	3	2.70	53	1
AL	BIRMINGHAM	59	5.09	2.10	11592	7	1.34	82	2	0.81	149	3	10.40	20	1
AL	GADSDEN	72	6.32	.	.	.	1.43	75	2	0.25	75	2	13.00	15	1
AL	MOBILE	20	4.78	.	.	.	0.66	56	1	0.33	56	1	.	.	.
AR	LITTLE ROCK	18	2.76	.	.	.	0.59	67	1	0.53	98	2	.	.	.
AZ	PHOENIX	244	1.35	2.20	23351	6	2.89	927	4	0.74	940	4	5.10	372	4
AZ	TUCSON	138	1.49	2.00	12576	3	0.93	130	1	0.31	647	6	4.70	186	5
CA	BAKERSFIELD	108	1.89	1.20	4273	1	2.23	51	1	0.55	375	2	.	.	.
CA	BERKELEY	26	1.95	.	.	.	0.44	57	1	0.42	57	1	5.00	24	1
CA	BURBANK	207	2.08	.	.	.	1.55	8	1	.	.	.	4.70	8	1
CA	FRESNO	119	0.73	1.80	14942	3	1.66	56	1	0.73	235	2	5.10	24	1
CA	LONG BEACH	62	2.08	2.50	6836	1	0.85	8	1	0.64	153	2	8.10	8	1
CA	LOS ANGELES	125	2.08	3.30	6143	4	1.58	96	2	0.90	417	3	10.80	185	3
CA	OAKLAND	26	1.95	1.40	3617	2	1.21	8	1	0.95	8	1	4.80	8	1
CA	PASADENA	126	2.08	3.20	5189	2	0.81	7	1	0.80	164	3	7.00	6	1
CA	SACRAMENTO	21	0.74	0.70	6251	3	1.02	111	1	0.38	282	3	3.00	24	1
CA	SAN BERNARDINO	133	1.11	2.50	5544	2	1.90	51	1	0.67	261	2	11.10	24	1
CA	SAN DIEGO	207	2.27	1.10	12652	3	1.29	116	1	0.48	303	2	9.80	24	1
CA	SAN FRANCISCO	58	0.39	2.20	5539	2	0.81	61	2	0.30	235	2	5.40	8	1
CA	SAN JOSE	194	0.39	3.00	3453	1	1.14	126	1	0.46	429	2	3.10	24	1
CA	SANTA ANA	141	4.13	.	.	.	1.06	51	1	0.59	137	1	8.65	16	1
CO	DENVER	116	1.95	3.70	20951	4	1.61	27	1	0.18	67	2	6.00	734	6
CT	BRIDGEPORT	29	9.88	2.70	5459	1	0.71	71	1	0.43	266	3	8.40	29	1
CT	HARTFORD	12	7.91	3.10	7890	2	0.93	21	1	0.41	216	4	9.70	21	1
CT	NEW HAVEN	43	9.48	2.20	3139	1	1.26	53	1	0.61	181	2	8.50	13	1
CT	WATERBURY	24	9.48	3.10	897	1	1.31	70	1	0.41	385	5	8.80	30	1
DC	WASHINGTON	102	11.79	1.80	13243	7	0.88	37	1	1.00	554	9	11.70	144	8
DE	WILMINGTON	48	12.08	1.60	8482	1	0.44	30	1	0.40	210	3	.	.	.
FL	JACKSONVILLE	274	4.89	1.50	24201	7	0.45	107	1	0.36	277	3	11.40	19	1
FL	ORLANDO	123	4.27	1.90	688	1	0.54	50	1	0.31	50	1	.	.	.
FL	PENSACOLA	20	4.37	0.60	3963	2	0.33	59	1	0.22	59	1	.	.	.
FL	TAMPA	81	8.43	1.20	21823	5	0.84	129	1	0.53	662	6	9.00	138	4
GA	ATLANTA	20	6.75	2.10	8632	5	0.87	99	1	0.52	99	1	13.30	20	1
GA	COLUMBUS	25	4.98	.	.	.	0.52	82	1	0.21	82	1	.	.	.

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	WATER HARD. (ppm)	ASTRAP SO_4	CO (ppm)			IRON			LEAD			SAROAD SO_4		
				MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b
IA	CEDAR RAPIDS	99	2.99	1.20	4546	1	0.70	102	1	0.25	102	1	6.90	31	1
IA	DAVENPORT	128	5.01	1.40	4817	1	0.80	151	2	0.35	195	3	7.40	28	1
IA	DES MOINES	78	2.67	3.10	11515	3	0.92	123	1	0.39	462	3	5.60	22	1
IA	DUBUQUE	282	3.84	1.80	4598	1	0.37	73	1	0.13	110	1	9.30	20	1
IA	WATERLOO	241	2.99	.	.	.	0.81	45	1	0.13	45	1	.	.	.
ID	BOISE CITY	85	0.49	3.00	8108	2	1.06	26	1	0.66	160	3	2.00	26	1
IL	CHICAGO	133	7.65	2.40	19332	9	1.86	58	2	0.35	2530	18	.	.	.
IL	GRANITE CITY	182	8.97	2.00	6130	1	3.52	18	1	0.44	18	1	.	.	.
IL	JOLIET	361	7.65	1.30	4129	3	0.62	17	1	0.25	17	1	.	.	.
IL	PEORIA	425	4.99	1.90	3416	2	0.99	25	1	0.28	25	1	.	.	.
IL	ROCKFORD	352	3.99	2.40	3288	1	0.49	39	1	0.19	39	1	.	.	.
IN	BLOOMINGTON	111	10.25	.	.	.	0.57	23	1	0.63	23	1	.	.	.
IN	EAST CHICAGO	133	7.19	1.90	1215	2	3.29	31	3	0.67	728	5	10.40	62	4
IN	ELKHART	203	7.52	.	.	.	0.95	11	1	0.37	11	1	.	.	.
IN	EVANSVILLE	100	7.70	1.70	2417	2	0.68	105	1	0.28	105	1	13.60	20	1
IN	FORT WAYNE	76	7.18	.	.	.	0.92	80	1	0.45	248	2	12.90	25	1
IN	GARY	136	7.19	0.70	1094	2	3.07	64	2	0.33	189	3	9.20	91	6
IN	HAMMOND	136	7.19	0.90	67	1	1.74	119	3	0.58	816	8	9.40	103	8
IN	INDIANAPOLIS	241	8.76	3.00	11452	4	0.64	110	3	0.47	990	16	10.40	6	1
IN	KOKOMO	402	8.76	.	.	.	1.46	19	1	0.45	109	1	.	.	.
IN	LAFAYETTE	402	8.56	.	.	.	0.99	23	1	0.57	23	1	.	.	.
IN	MARION	138	8.76	.	.	.	0.65	24	1	0.42	24	1	.	.	.
IN	NEW ALBANY	96	10.86	.	.	.	0.93	31	2	0.46	131	2	14.00	18	1
IN	RICHMOND	274	8.27	.	.	.	1.11	44	2	0.64	44	2	.	.	.
IN	SOUTH BEND	403	6.97	1.10	898	1	0.82	149	2	0.30	186	5	8.60	19	1
IN	TERRE HAUTE	208	10.25	.	.	.	0.82	99	2	0.20	99	2	13.50	24	1
KS	KANSAS CITY	276	3.30	1.50	6581	2	1.70	186	2	0.29	247	3	8.00	36	2
KS	TOPEKA	100	1.93	.	.	.	0.93	84	2	0.25	84	2	6.60	21	1
KS	WICHITA	86	1.78	1.60	7841	3	0.65	37	2	0.29	37	2	5.10	21	1
KY	ASHLAND	93	9.70	1.50	7866	4	4.33	16	1	1.03	16	1	17.50	16	1
KY	COVINGTON	112	11.42	2.00	1974	1	1.26	116	1	0.38	116	1	14.50	18	1
KY	LOUISVILLE	90	10.86	2.50	10784	3	0.76	62	1	0.36	62	1	.	.	.
LA	BATON ROUGE	38	3.67	0.60	882	1	0.46	104	1	0.51	104	1	10.50	5	1
LA	NEW ORLEANS	74	4.34	0.80	3446	2	0.69	113	1	0.32	113	1	13.00	5	1

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	MANGANESE			MAX. OZONE (ppm)			CADMIUM			VANADIUM			DICHOT. PARTICULATE			
		MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	FINE	TOTAL	FINE-S	TOT-S
IA	DAVENPORT	0.036	151	2	.	.	.	0.005	151	2	0.005	151	2	.	.	.	.
IA	DES MOINES	0.032	123	1	0.045	259	2	0.001	123	1	0.005	123	1	.	.	.	.
IA	DUBUQUE	0.020	73	1	.	.	.	0.001	73	1	0.005	73	1	.	.	.	.
IA	WATERLOO	0.029	45	1	.	.	.	0.001	45	1	0.005	45	1	.	.	.	.
ID	BOISE CITY	0.023	26	1	.	.	.	0.001	26	1	0.005	26	1	12.120	33.010	1.040	1.430
IL	CHICAGO	0.077	58	2	0.035	5223	16	0.003	58	2	0.005	58	2	21.040	45.190	5.040	5.160
IL	GRANITE CITY	0.188	18	1	0.027	635	1	0.009	18	1	.	.	.	.	.	.	.
IL	JOLIET	0.020	17	1	0.054	168	1	0.002	17	1	0.178	17	1	.	.	.	.
IL	PEORIA	0.037	25	1	0.050	1175	1	0.002	25	1	0.091	25	1	.	.	.	.
IL	ROCKFORD	0.027	39	1	0.038	927	2	0.006	39	1	0.055	39	1	.	.	.	.
IN	BLOOMINGTON	0.028	23	1	.	.	.	0.002	23	1	.	.	.	.	.	.	.
IN	EAST CHICAGO	0.185	31	3	0.039	1053	2	0.003	618	5	0.011	31	3	.	.	.	.
IN	ELKHART	0.034	11	1	.	.	.	0.001	11	1	.	.	.	.	.	.	.
IN	EVANSVILLE	0.023	105	1	0.054	586	2	0.001	105	1	0.005	86	1	.	.	.	.
IN	FORT WAYNE	0.033	80	1	0.057	928	2	0.001	165	2	0.005	56	1	.	.	.	.
IN	GARY	0.154	64	2	0.026	483	2	0.002	183	3	0.009	64	2	25.240	60.440	11.62	12.32
IN	HAMMOND	0.103	119	3	0.036	2039	2	0.003	456	7	0.006	84	1	.	.	.	.
IN	INDIANAPOLIS	0.021	110	3	0.038	5571	6	0.004	280	6	0.005	84	3	21.090	34.010	4.890	5.220
IN	KOKOMO	0.091	19	1	.	.	.	0.004	70	1	.	.	.	.	.	.	.
IN	LAFAYETTE	0.036	23	1	.	.	.	0.000	23	1	.	.	.	.	.	.	.
IN	MARION	0.045	24	1	.	.	.	0.001	24	1	.	.	.	.	.	.	.
IN	NEW ALBANY	0.030	31	2	0.038	427	1	0.001	31	2	0.005	18	1	.	.	.	.
IN	RICHMOND	0.064	44	2	.	.	.	0.001	44	2	.	.	.	.	.	.	.
IN	SOUTH BEND	0.044	149	2	0.046	1110	1	0.001	149	2	0.006	123	1	.	.	.	.
IN	TERRE HAUTE	0.020	99	2	.	.	.	0.001	99	2	0.005	84	1	.	.	.	.
KS	KANSAS CITY	0.040	186	2	0.030	798	2	0.002	186	2	0.005	186	2	.	.	.	.
KS	TOPEKA	0.028	84	2	.	.	.	0.001	84	2	0.005	84	2	10.330	26.860	2.210	2.440
KS	WICHITA	0.020	37	2	0.048	1432	1	0.001	37	2	0.006	37	2	13.590	33.200	2.740	2.920
KY	ASHLAND	0.111	16	1	0.034	1039	2	0.003	16	1	0.010	16	1	33.350	65.700	.	.
KY	COVINGTON	0.062	116	1	0.032	687	1	0.002	116	1	0.005	116	1	.	.	.	.
KY	LOUISVILLE	0.024	62	1	0.050	1575	1	0.002	62	1	0.005	62	1	.	.	.	.
LA	BATON ROUGE	0.020	104	1	0.050	2088	2	0.001	104	1	0.005	104	1	.	.	.	.
LA	NEW ORLEANS	0.020	113	1	0.040	2327	3	0.001	113	1	0.012	113	1	.	.	.	.
LA	SHREVEPORT	0.028	105	1	.	.	.	0.001	105	1	0.005	105	1	.	.	.	.
MA	BOSTON	0.020	95	1	0.040	503	1	0.001	95	1	0.047	95	1	.	.	.	.

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	MANGANESE			MAX. OZONE (ppm)			CADMIUM			VANADIUM			DICHOT. PARTICULATE			
		MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	FINE	TOTAL	FINE-S	TOT-S
MA	BROCKTON	0.027	30	1	.	.	.	0.002	30	1	0.041	30	1	.	.	.	.
MA	CAMBRIDGE	0.020	52	1	.	.	.	0.001	52	1	0.036	52	1	.	.	.	.
MA	FALL RIVER	0.020	63	1	0.052	89	1	0.001	63	1	0.059	63	1	.	.	.	.
MA	LAWRENCE	0.020	40	1	0.043	250	1	0.001	40	1	0.051	40	1	.	.	.	.
MA	LOWELL	0.020	26	1	.	.	.	0.002	26	1	0.051	26	1	.	.	.	.
MA	NEW BEDFORD	0.020	81	1	.	.	.	0.001	81	1	0.047	81	1	.	.	.	.
MA	SPRINGFIELD	0.020	95	2	0.053	504	1	0.002	95	2	0.062	95	2	.	.	.	.
MA	WORCESTER	0.020	113	2	0.051	731	3	0.001	113	2	0.053	113	2	.	.	.	.
MD	BALTIMORE	0.056	53	1	0.033	2052	4	0.002	53	1	0.031	53	1	.	.	.	.
ME	PORTLAND	0.020	107	2	.	.	.	0.001	107	2	0.062	107	2	.	.	.	.
MI	ANN ARBOR	0.020	30	1	.	.	.	0.001	30	1	0.005	30	1	.	.	.	.
MI	DETROIT	0.106	95	2	0.031	2841	3	0.006	95	2	0.007	95	2	.	.	.	.
MI	FLINT	0.035	87	2	0.049	1220	1	0.001	87	2	0.005	87	2	.	.	.	.
MI	GRAND RAPIDS	0.041	22	2	0.032	1128	2	0.003	22	2	0.005	22	2	.	.	.	.
MI	LANSING	0.020	73	1	0.047	655	2	0.001	73	1	0.005	73	1	.	.	.	.
MN	DULUTH	0.032	114	2	.	.	.	0.001	114	2	0.005	114	2	.	.	.	.
MN	MINNEAPOLIS	0.025	170	4	0.018	177	1	0.001	170	4	0.005	170	4	13.680	29.230	2.080	2.560
MN	ST PAUL	0.034	60	2	.	.	.	0.003	60	2	0.005	60	2	12.920	25.470	2.150	2.300
MO	RAYTOWN	0.035	29	1	.	.	.	0.001	29	1	0.005	29	1	.	.	.	.
MS	JACKSON	0.030	69	2	0.057	1253	1	0.002	69	2	0.005	69	2	15.710	31.920	4.780	5.140
MT	BILLINGS	0.040	224	2	0.033	494	2	0.002	203	2	0.010	172	2	.	.	.	.
MT	BUTTE-SILVER BO	0.059	553	6	0.030	527	1	0.008	570	6	0.010	289	4	7.210	21.910	1.030	1.030
MT	GREAT FALLS	0.023	117	1	.	.	.	0.002	115	1	0.010	105	1	.	.	.	.
NC	CHARLOTTE	0.020	115	1	0.054	2286	6	0.001	115	1	0.014	115	1	22.600	35.760	4.470	4.910
NC	DURHAM	0.020	137	1	.	.	.	0.002	137	1	0.018	137	1	16.790	25.060	5.230	5.420
NC	GREENSBORO	0.020	44	1	.	.	.	0.001	44	1	0.032	44	1	.	.	.	.
NC	RALEIGH	0.020	42	1	.	.	.	0.003	42	1	0.006	42	1	.	.	.	.
NC	WINSTON-SALEM	0.020	111	2	.	.	.	0.001	111	2	0.011	111	2	.	.	.	.
NE	LINCOLN	0.025	72	2	0.034	1212	1	0.001	72	2	0.005	72	2	.	.	.	.
NE	OMAHA	0.026	130	1	0.031	3079	3	0.001	130	1	0.005	130	1	13.070	39.900	2.020	2.240
NH	CONCORD	0.020	37	1	.	.	.	0.001	37	1	0.031	37	1	.	.	.	.
NJ	BAYONNE	0.020	72	1	0.054	1291	1	0.004	72	1	0.036	72	1	.	.	.	.
NJ	CAMDEN	0.035	99	2	0.057	1333	1	0.003	99	2	0.030	99	2	19.330	33.680	5.240	5.870
NJ	ELIZABETH	0.020	81	1	.	.	.	0.004	81	1	0.025	81	1	.	.	.	.
NJ	JERSEY CITY	0.020	95	2	.	.	.	0.004	95	2	0.046	95	2	17.340	32.050	4.360	4.760

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	WATER HARD. (ppm)	ASTRAP SO_4	CO (ppm)			IRON			LEAD			SAROAD SO_4		
				MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b
LA	SHREVEPORT	68	2.66	.	.	.	1.53	105	1	0.37	105	1	.	.	.
MA	BOSTON	12	6.01	3.10	16726	4	1.08	95	1	0.56	120	2	10.70	101	5
MA	BROCKTON	11	6.15	.	.	.	0.57	30	1	0.33	30	1	8.40	13	1
MA	CAMBRIDGE	56	6.01	2.70	197	1	0.61	52	1	0.39	52	1	10.20	36	1
MA	FALL RIVER	15	6.15	.	.	.	0.82	63	1	0.61	63	1	11.20	61	1
MA	LAWRENCE	44	6.72	.	.	.	0.43	40	1	0.30	40	1	8.75	14	1
MA	LOWELL	42	6.01	4.00	3717	1	0.99	26	1	0.52	54	1	7.35	14	1
MA	NEW BEDFORD	15	6.15	.	.	.	0.39	81	1	0.29	81	1	11.00	81	1
MA	SPRINGFIELD	12	7.29	1.80	7917	3	0.78	95	2	0.67	138	3	8.00	175	5
MA	WORCESTER	9	7.29	1.50	6749	2	1.04	113	2	0.42	143	2	9.50	98	2
MD	BALTIMORE	50	11.79	1.80	14741	6	1.31	53	1	0.56	688	5	12.00	152	4
ME	PORTLAND	14	5.07	.	.	.	0.52	107	2	0.40	456	6	8.80	26	1
MI	ANN ARBOR	80	7.43	.	.	.	0.58	30	1	0.20	30	1	.	.	.
MI	DETROIT	98	7.43	1.70	20903	4	2.26	95	2	0.32	95	2	.	.	.
MI	FLINT	86	5.69	.	.	.	1.03	87	2	0.15	87	2	13.20	22	1
MI	GRAND RAPIDS	130	5.39	0.70	3925	2	1.08	22	2	1.00	22	2	14.10	22	2
MI	LANSING	86	6.20	.	.	.	0.55	73	1	0.16	73	1	13.80	23	1
MN	DULUTH	44	1.34	2.90	3488	1	1.08	114	2	0.18	522	5	5.50	109	2
MN	MINNEAPOLIS	68	1.72	1.80	10524	6	0.66	170	4	0.51	1311	9	6.60	110	2
MN	ST PAUL	58	2.16	2.10	12051	5	0.82	60	2	0.38	1603	9	7.10	357	6
MO	RAYTOWN	86	3.30	.	.	.	1.52	28	1	0.24	88	1	8.50	29	1
MS	JACKSON	47	3.64	0.90	3542	1	0.91	69	2	0.37	236	4	9.00	45	2
MT	BILLINGS	148	1.07	1.30	3306	1	0.55	67	1	0.27	242	2	6.60	116	1
MT	BUTTE-SILVER BO	56	0.74	1.50	1286	1	1.13	313	8	0.28	610	8	5.20	743	6
MT	GREAT FALLS	151	0.93	1.10	4004	1	0.23	23	1	0.16	112	1	3.60	19	2
NC	CHARLOTTE	22	9.19	2.40	28937	8	0.71	115	1	0.44	115	1	11.10	30	1
NC	DURHAM	41	8.83	3.10	4632	1	0.59	137	1	0.39	189	2	10.00	20	1
NC	GREENSBORO	40	8.83	.	.	.	0.52	44	1	0.25	162	1	9.90	21	1
NC	RALEIGH	30	7.37	3.30	2681	2	0.37	42	1	0.29	133	1	.	.	.
NC	WINSTON-SALEM	34	8.25	1.50	6165	2	0.61	111	2	0.34	214	2	11.10	29	1
NE	LINCOLN	206	1.76	1.10	13167	2	0.72	72	2	0.29	72	2	4.90	25	1
NE	OMAHA	236	2.16	1.90	9442	4	0.81	130	1	0.36	178	4	7.30	15	1
NH	CONCORD	12	6.65	.	.	.	0.24	37	1	0.14	37	1	6.40	24	1
NJ	BAYONNE	25	9.88	0.90	7376	1	0.92	72	1	0.35	72	1	8.60	23	1

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	WATER HARD. (ppm)	ASTRAP SO_4	CO (ppm)			IRON			LEAD			SAROAD SO_4		
				MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b
NJ	CAMDEN	80	12.15	1.50	16074	3	0.91	99	2	0.43	155	2	11.60	18	1
NJ	ELIZABETH	151	9.88	2.90	15152	2	0.90	81	1	0.74	81	1	10.50	15	1
NJ	JERSEY CITY	36	9.88	4.00	7125	1	1.06	95	2	0.50	155	3	9.70	6	1
NJ	NEWARK	26	9.88	1.90	7843	1	0.89	101	1	0.54	162	2	10.25	26	1
NJ	PERTH AMBOY	65	11.34	2.70	9509	1	1.25	41	1	0.67	41	1	10.10	28	1
NM	ALBUQUERQUE	112	1.98	1.80	15711	5	1.29	101	1	0.59	519	6	2.80	47	5
NV	RENO	40	0.29	2.60	2971	2	1.25	143	1	0.32	143	1	1.65	26	1
NY	ALBANY	54	7.62	.	.	.	0.51	75	2	0.22	75	2	9.10	79	2
NY	BUFFALO	124	8.78	1.40	7982	3	0.94	94	2	0.30	142	5	11.10	254	5
NY	NIAGARA FALLS	126	7.66	0.80	9258	1	0.78	122	2	0.19	122	2	10.50	182	3
NY	ROCHESTER	82	7.71	1.60	9492	3	0.92	89	1	0.23	136	3	9.30	668	9
NY	SYRACUSE	109	7.29	1.00	7347	3	0.69	91	2	0.34	91	2	9.10	108	2
NY	UTICA	16	6.85	0.50	3067	1	0.62	82	1	0.32	82	1	8.50	154	2
NY	YONKERS	61	9.88	.	.	.	0.59	45	1	0.51	45	1	8.40	24	1
OH	AKRON	106	9.26	1.70	8750	3	1.12	135	1	0.26	135	1	14.70	750	8
OH	CANTON	413	9.26	1.10	4604	1	1.78	108	1	0.25	108	1	13.10	270	3
OH	CINCINNATI	115	11.42	1.60	7406	4	1.28	217	2	0.48	217	2	12.70	44	2
OH	CLEVELAND	128	9.26	1.90	6808	2	1.87	143	2	0.34	143	2	16.00	128	2
OH	COLUMBUS	90	8.31	1.70	8567	3	0.88	127	1	0.33	127	1	12.70	221	4
OH	DAYTON	358	8.27	1.30	10692	4	0.82	104	2	0.42	104	2	11.65	84	2
OH	MANSFIELD	229	9.26	.	.	.	1.38	91	1	0.17	91	1	12.00	116	3
OH	PORTSMOUTH	96	9.97	.	.	.	0.86	113	1	0.31	113	1	12.50	141	1
OH	STEUBENVILLE	111	12.85	2.60	3566	1	2.88	97	1	0.46	97	1	17.40	126	1
OH	TOLEDO	170	7.83	1.20	8326	2	0.77	103	1	0.21	103	1	13.90	206	2
OH	YOUNGSTOWN	86	12.19	2.10	2859	1	2.25	125	1	0.32	125	1	15.50	319	2
OK	OKLAHOMA CITY	90	1.73	0.70	13357	3	0.82	37	1	0.20	199	4	5.90	37	1
OK	TULSA	91	1.90	2.30	6520	2	0.68	13	1	0.27	45	2	5.60	8	1
OR	PORTLAND	9	0.47	2.90	18175	5	1.57	130	2	0.34	234	4	6.80	91	2
PA	ALLENTOWN	157	12.15	1.00	5606	1	0.74	46	2	0.45	46	2	9.70	129	2
PA	BETHLEHEM	6	9.18	1.20	1793	1	1.74	33	2	0.27	192	3	12.40	249	3
PA	ERIE	129	8.16	0.60	1139	1	0.84	19	2	0.36	428	5	11.60	376	7
PA	HARRISBURG	13	10.89	1.50	4617	2	1.15	38	1	0.82	117	3	12.90	157	3
PA	PHILADELPHIA	75	12.15	1.80	44511	13	0.94	82	2	0.46	6497	24	.	.	.
PA	READING	88	12.15	1.10	6393	1	0.65	60	2	0.39	385	3	11.60	311	5

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	MANGANESE			MAX. OZONE (ppm)			CADMIUM			VANADIUM			DICHOT. PARTICULATE			
		MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	FINE	TOTAL	FINE-S	TOT-S
NJ	NEWARK	0.020	101	1	0.045	594	1	0.003	101	1	0.033	101	1	.	.	.	.
NJ	PERTH AMBOY	0.029	41	1	.	.	.	0.002	41	1	0.029	41	1	.	.	.	.
NM	ALBUQUERQUE	0.028	101	1	0.050	3239	4	0.001	101	1	0.005	101	1	8.950	31.430	1.670	1.950
NV	RENO	0.027	143	1	0.047	1016	2	0.001	143	1	0.007	143	1	11.820	32.650	1.100	1.240
NY	ALBANY	0.020	75	2	.	.	.	0.001	75	2	0.112	75	2	.	.	.	.
NY	BUFFALO	0.031	94	2	0.036	323	1	0.001	94	2	0.006	94	2	23.530	43.260	5.940	7.080
NY	NIAGARA FALLS	0.054	122	2	0.034	1334	1	0.001	122	2	0.008	122	2	.	.	.	.
NY	ROCHESTER	0.020	89	1	0.036	1691	2	0.001	89	1	0.017	89	1	.	.	.	.
NY	SYRACUSE	0.020	91	2	0.037	2349	4	0.001	91	2	0.008	91	2	.	.	.	.
NY	UTICA	0.024	82	1	0.039	1646	1	0.001	82	1	0.031	82	1	.	.	.	.
NY	YONKERS	0.020	45	1	.	.	.	0.002	45	1	0.048	45	1	.	.	.	.
OH	AKRON	0.033	135	1	0.034	1294	3	0.002	135	1	0.005	135	1	24.600	44.330	8.270	8.770
OH	CANTON	0.083	108	1	0.030	1070	3	0.003	108	1	0.005	108	1	.	.	.	.
OH	CINCINNATI	0.039	217	2	0.037	4379	3	0.002	217	2	0.005	217	2	23.120	39.570	6.470	6.910
OH	CLEVELAND	0.078	143	2	0.043	1313	1	0.004	143	2	0.005	143	2	24.580	47.870	7.090	8.060
OH	COLUMBUS	0.024	127	1	0.029	4065	3	0.004	127	1	0.005	127	1	.	.	.	.
OH	DAYTON	0.029	104	2	0.034	2790	2	0.002	104	2	0.005	104	2	18.800	33.340	4.490	4.580
OH	MANSFIELD	0.045	91	1	.	.	.	0.002	91	1	0.005	91	1	.	.	.	.
OH	PORTSMOUTH	0.055	113	1	.	.	.	0.001	113	1	0.005	113	1	.	.	.	.
OH	STEUBENVILLE	0.118	97	1	0.045	1687	1	0.004	97	1	0.014	97	1	23.090	44.960	4.030	4.110
OH	TOLEDO	0.024	103	1	0.037	3264	2	0.001	103	1	0.006	103	1	.	.	.	.
OH	YOUNGSTOWN	0.094	125	1	0.030	1517	2	0.003	125	1	0.005	125	1	20.230	43.050	6.050	6.850
OK	OKLAHOMA CITY	0.024	37	1	0.049	1256	1	0.002	37	1	0.005	37	1	15.860	40.020	4.310	4.310
OK	TULSA	0.029	13	1	0.053	1483	1	0.001	13	1	0.005	13	1	.	.	.	.
OR	PORTLAND	0.049	130	2	0.023	226	1	0.003	130	2	0.023	130	2	14.660	38.010	2.030	2.600
PA	ALLENTOWN	0.032	46	2	0.033	1455	1	0.005	46	2	0.069	46	2	.	.	.	.
PA	BETHLEHEM	0.094	33	2	0.049	1499	1	0.010	33	2	0.043	33	2	17.880	30.830	5.480	5.640
PA	ERIE	0.020	19	2	0.040	1392	1	0.002	19	2	0.006	19	2	.	.	.	.
PA	HARRISBURG	0.037	38	1	0.044	1540	2	0.003	38	1	0.046	38	1	.	.	.	.
PA	PHILADELPHIA	0.039	82	2	0.033	7363	10	0.002	82	2	0.023	82	2	21.040	38.110	5.780	6.360
PA	READING	0.033	60	2	0.029	1552	1	0.002	60	2	0.055	60	2	.	.	.	.
PA	SCRANTON	0.020	8	1	0.054	1377	2	0.001	8	1	0.020	8	1	.	.	.	.
RI	PROVIDENCE	0.020	23	1	0.049	1425	2	0.003	23	1	0.036	23	1	12.890	22.490	3.890	4.050
SC	COLUMBIA	0.020	44	1	.	.	.	0.001	44	1	0.006	44	1	.	.	.	.
SD	SIOUX FALLS	0.100	22	1	.	.	.	0.001	22	1	0.005	22	1	.	.	.	.

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	MANGANESE			MAX. OZONE (ppm)			CADMIUM			VANADIUM			DICHOT. PARTICULATE			
		MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	FINE	TOTAL	FINE-S	TOT-S
TN	CHATTANOOGA	0.049	205	2	0.042	755	1	0.002	205	2	0.005	205	2	16.640	22.320	9.850	9.930
TN	KNOXVILLE	0.038	80	2	0.035	1477	2	0.001	80	2	0.005	80	2	.	.	.	.
TN	MEMPHIS	0.026	99	3	0.039	1424	2	0.001	99	3	0.005	99	3	.	.	.	.
TN	NASHVILLE-DAVID	0.027	75	1	0.036	1486	2	0.001	75	1	0.005	75	1	20.450	32.990	3.610	3.870
TX	ABILENE	0.020	353	2	.	.	.	0.030	353	2	0.002	353	2	.	.	.	.
TX	BEAUMONT	0.030	294	4	0.048	608	2	0.030	294	4	0.004	294	4	.	.	.	.
TX	DALLAS	0.020	278	2	0.046	3085	4	0.030	278	2	0.002	278	2	16.480	34.740	2.730	3.300
TX	EL PASO	0.050	478	7	0.047	2100	4	0.040	807	17	0.005	478	7	15.740	57.950	2.280	2.860
TX	FORT WORTH	0.030	564	3	0.053	2360	2	0.030	564	3	0.002	564	3	.	.	.	.
TX	GALVESTON	0.020	50	1	.	.	.	0.030	50	1	0.003	50	1	.	.	.	.
TX	HOUSTON	0.024	1530	25	0.055	5011	8	0.030	263	3	0.006	263	3	13.370	32.440	3.380	3.460
TX	LAREDO	0.020	98	1	.	.	.	0.030	98	1	0.003	98	1	.	.	.	.
TX	LUBBOCK	0.020	305	2	.	.	.	0.030	305	2	0.003	305	2	.	.	.	.
TX	PORT ARTHUR	0.020	167	4	0.051	231	1	0.030	167	4	0.006	167	4	.	.	.	.
TX	SAN ANTONIO	0.020	453	3	0.043	2578	3	0.030	453	3	0.002	453	3	.	.	.	.
TX	SEABROOK	0.030	85	1	0.061	380	1	0.030	85	1	0.004	85	1	15.390	34.850	1.860	2.040
TX	TYLER	0.020	223	1	.	.	.	0.030	224	1	0.002	223	1	.	.	.	.
TX	WACO	0.020	235	1	0.041	235	1	0.030	235	1	0.002	234	1	.	.	.	.
TX	WICHITA FALLS	0.030	180	1	.	.	.	0.030	180	1	0.003	180	1	.	.	.	.
UT	OGDEN	0.028	29	1	0.052	1468	1	0.001	29	1	0.005	29	1	.	.	.	.
UT	SALT LAKE CITY	0.046	23	1	0.049	1484	1	0.001	23	1	0.005	23	1	15.390	34.850	1.860	2.040
VA	NORFOLK	0.022	146	2	0.037	443	1	0.001	146	2	0.074	146	2	16.910	30.250	5.880	6.730
VT	BURLINGTON	0.027	14	1	0.049	527	2	0.001	14	1	0.026	14	1	.	.	.	.
WA	SEATTLE	0.024	242	3	0.037	176	1	0.001	242	3	0.009	242	3	11.870	25.950	1.670	2.140
WA	SPOKANE	0.036	108	1	0.041	587	1	0.002	108	1	0.005	108	1	9.380	35.480	1.050	1.420
WA	TACOMA	0.032	141	2	0.037	156	1	0.002	141	2	0.010	141	2	.	.	.	.
WI	EAU CLAIRE	0.020	74	1	.	.	.	0.001	74	1	0.005	74	1	.	.	.	.
WI	KENOSHA	0.020	85	1	0.057	584	3	0.002	85	1	0.005	85	1	.	.	.	.
WI	MADISON	0.020	139	2	0.044	782	3	0.001	139	2	0.005	139	2	.	.	.	.
WI	RACINE	0.032	87	1	0.050	1380	1	0.001	87	1	0.005	87	1	.	.	.	.
WI	SUPERIOR	0.092	76	2	.	.	.	0.001	76	2	0.005	76	2	.	.	.	.
WV	CHARLESTON	0.042	63	2	0.048	1615	1	0.001	63	2	0.005	63	2	20.100	37.400	4.710	4.710

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	WATER HARD. (ppm)	ASTRAP SO ₄	CO (ppm)			IRON			LEAD			SAROAD SO ₄		
				MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b
PA	SCRANTON	26	9.18	2.00	5824	1	0.64	8	1	0.23	76	2	11.10	221	2
RI	PROVIDENCE	24	7.25	1.60	5570	2	0.63	23	1	0.79	500	4	9.60	112	1
SC	COLUMBIA	29	7.09	1.40	2446	1	0.45	44	1	0.34	251	2	.	.	.
SD	SIOUX FALLS	484	1.52	.	.	.	1.10	22	1	0.37	22	1	4.60	22	1
TN	CHATTANOOGA	59	7.25	3.20	2466	1	1.44	205	2	0.34	205	2	13.90	29	1
TN	KNOXVILLE	116	8.22	3.00	5250	2	1.16	80	2	0.66	80	2	12.50	29	1
TN	MEMPHIS	38	3.91	1.60	10579	5	0.66	99	3	0.47	134	5	.	.	.
TN	NASHVILLE-DAVID	90	7.73	3.00	12410	5	0.67	75	1	0.38	2572	14	7.50	2001	14
TX	ABILENE	194	1.36	.	.	.	0.90	353	2	0.16	353	2	.	.	.
TX	BEAUMONT	66	2.52	0.50	5069	2	0.76	294	4	0.27	294	4	6.20	5	1
TX	BROWNSVILLE	266	0.39	.	.	.	1.08	314	3	0.25	314	3	.	.	.
TX	DALLAS	66	2.28	0.70	12348	4	0.72	887	16	0.59	969	16	11.60	11	1
TX	EL PASO	146	2.45	1.70	15222	4	2.20	477	7	0.99	934	17	8.50	5	1
TX	FORT WORTH	144	1.65	0.80	17425	2	1.21	564	3	0.44	652	4	5.20	5	1
TX	GALVESTON	115	1.45	.	.	.	0.71	50	1	0.18	50	1	5.60	41	1
TX	HOUSTON	73	1.79	0.90	27445	4	1.88	263	3	0.61	1558	25	8.20	5024	30
TX	LAREDO	328	0.47	.	.	.	1.43	98	1	0.11	98	1	.	.	.
TX	LUBBOCK	422	1.73	.	.	.	1.43	305	2	0.12	305	2	.	.	.
TX	PORT ARTHUR	45	2.52	.	.	.	0.58	167	4	0.18	167	4	.	.	.
TX	SAN ANTONIO	223	0.97	0.90	11183	3	0.64	453	3	0.45	515	3	5.40	166	7
TX	SEABROOK	94	1.45	0.30	1509	1	0.63	85	1	0.09	85	1	.	.	.
TX	TYLER	71	2.37	.	.	.	1.29	224	1	0.35	224	1	.	.	.
TX	WACO	156	1.79	.	.	.	0.55	235	1	0.17	235	1	.	.	.
TX	WICHITA FALLS	67	1.48	.	.	.	0.98	180	1	0.26	180	1	3.40	5	1
UT	OGDEN	158	2.91	2.40	7867	2	1.25	29	1	1.21	29	1	3.80	29	1
UT	SALT LAKE CITY	186	2.91	2.70	4962	2	1.87	23	1	1.44	23	1	5.50	23	1
VA	NORFOLK	74	9.02	1.70	6610	1	1.13	146	2	0.25	205	3	13.00	107	2
VT	BURLINGTON	56	5.35	0.90	7679	1	0.83	14	1	0.60	14	1	6.80	14	1
WA	SEATTLE	18	2.41	2.90	29978	8	1.06	242	3	0.43	242	3	6.10	30	1
WA	SPOKANE	156	0.71	3.00	16913	7	1.64	108	1	0.34	108	1	4.00	27	1
WA	TACOMA	21	2.41	2.80	4443	1	1.33	141	2	0.46	141	2	5.70	30	1
WI	EAU CLAIRE	54	2.38	.	.	.	0.28	74	1	0.16	74	1	7.40	25	1
WI	KENOSHA	132	5.60	.	.	.	0.50	85	1	0.28	85	1	13.00	22	1
WI	MADISON	345	3.99	.	.	.	0.46	139	2	0.16	139	2	9.20	29	1
WI	RACINE	130	5.60	0.80	9178	1	0.77	87	1	0.23	87	1	9.10	29	1
WI	SUPERIOR	50	1.62	.	.	.	1.57	76	2	0.24	76	2	7.50	26	1
WV	CHARLESTON	32	11.17	1.10	3923	1	1.05	63	2	0.29	285	3	15.50	35	2

^a Total number of observations included

^b Number of monitoring sites supplying data.

AIR QUALITY DATA - MEDIAN CONCENTRATIONS, IN $\mu\text{G}/\text{M}^3$ EXCEPT AS NOTED

STATE	CITY	MANGANESE			MAX. OZONE (ppm)			CADMIUM			VANADIUM			DICHOT. PARTICULATE			
		MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	MED.	#OBS ^a	#MON ^b	FINE	TOTAL	FINE-S	TOT-S
AK	ANCHORAGE	0.032	53	1	.	.	.	0.001	53	1	0.005	53	1	.	.	.	.
AL	BIRMINGHAM	0.055	82	2	0.057	269	1	0.002	82	2	0.005	82	2	24.510	48.260	4.850	5.270
AL	GADSDEN	0.060	75	2	.	.	.	0.001	75	2	0.008	75	2	.	.	.	.
AL	MOBILE	0.025	56	1	.	.	.	0.001	56	1	0.005	56	1	20.890	38.840	4.600	5.000
AR	LITTLE ROCK	0.020	67	1	0.046	991	1	0.001	67	1	0.006	67	1	17.840	33.820	2.690	2.960
AZ	PHOENIX	0.065	940	4	0.049	4649	7	0.001	894	4	0.005	102	1	15.240	56.520	2.470	2.820
AZ	TUCSON	0.020	130	1	0.051	2864	2	0.001	249	6	0.005	130	1	.	.	.	.
CA	BAKERSFIELD	0.038	51	1	0.067	1732	1	0.001	51	1	0.038	51	1	19.560	57.810	4.800	5.420
CA	BERKELEY	0.020	57	1	.	.	.	0.001	57	1	0.005	57	1	.	.	.	.
CA	BURBANK	0.030	8	1	0.079	1521	1	0.003	8	1	0.006	8	1	.	.	.	.
CA	FRESNO	0.035	56	1	0.061	5321	4	0.001	56	1	0.006	56	1	10.320	42.060	2.120	2.200
CA	LONG BEACH	0.023	8	1	0.040	1331	1	0.001	8	1	0.010	8	1	.	.	.	.
CA	LOS ANGELES	0.027	96	2	0.070	3126	5	0.002	96	2	0.009	96	2	21.810	44.900	4.480	5.310
CA	OAKLAND	0.032	8	1	0.022	1472	2	0.001	8	1	0.010	8	1	.	.	.	.
CA	PASADENA	0.023	7	1	0.097	1496	2	0.001	7	1	0.007	7	1	31.070	55.110	.	.
CA	SACRAMENTO	0.020	111	1	0.048	2842	3	0.001	111	1	0.005	111	1	.	.	.	.
CA	SAN BERNARDINO	0.044	51	1	0.109	2038	2	0.001	51	1	0.007	51	1	.	.	.	.
CA	SAN DIEGO	0.023	116	1	0.057	3403	2	0.001	116	1	0.009	116	1	.	.	.	.
CA	SAN FRANCISCO	0.020	61	2	0.024	1332	2	0.002	61	2	0.009	61	2	12.160	26.830	1.820	2.540
CA	SAN JOSE	0.023	126	1	0.049	2770	2	0.001	126	1	0.005	126	1	12.440	34.210	1.260	1.860
CA	SANTA ANA	0.020	51	1	0.066	866	1	0.001	51	1	0.008	51	1	.	.	.	.
CO	DENVER	0.042	27	1	0.041	3584	4	0.004	27	1	0.005	27	1	16.090	57.250	2.200	2.460
CT	BRIDGEPORT	0.020	71	1	0.062	795	1	0.003	71	1	0.026	71	1	.	.	.	.
CT	HARTFORD	0.020	21	1	0.055	941	1	0.002	21	1	0.050	21	1	14.770	33.080	5.320	5.970
CT	NEW HAVEN	0.026	53	1	0.057	920	1	0.002	53	1	0.033	53	1	.	.	.	.
CT	WATERBURY	0.023	70	1	.	.	.	0.003	70	1	0.019	70	1	.	.	.	.
DC	WASHINGTON	0.020	37	1	0.035	4315	6	0.001	37	1	0.047	37	1	22.510	36.500	6.970	7.410
DE	WILMINGTON	0.025	30	1	.	.	.	0.001	30	1	0.021	30	1	.	.	.	.
FL	JACKSONVILLE	0.020	107	1	0.049	1859	3	0.001	107	1	0.030	107	1	.	.	.	.
FL	ORLANDO	0.020	50	1	.	.	.	0.001	50	1	0.010	50	1	.	.	.	.
FL	PENSACOLA	0.020	59	1	0.053	1978	2	0.001	59	1	0.005	59	1	.	.	.	.
FL	TAMPA	0.020	129	1	0.046	2672	4	0.001	129	1	0.014	129	1	11.420	34.110	3.360	3.880
GA	ATLANTA	0.020	99	1	.	.	.	0.002	99	1	0.005	99	1	20.290	30.920	5.620	5.970
GA	COLUMBUS	0.020	82	1	0.049	416	1	0.001	82	1	0.005	82	1	.	.	.	.
IA	CEDAR RAPIDS	0.039	102	1	0.043	1676	1	0.001	102	1	0.005	102	1	.	.	.	.

^a Total number of observations included

^b Number of monitoring sites supplying data.

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APPENDIX B

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PATHOPHYSIOLOGICAL EFFECTS OF AIR POLLUTION

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APPENDIX B

PATHOPHYSIOLOGICAL EFFECTS OF AIR POLLUTION

ANATOMY AND FUNCTION OF THE RESPIRATORY SYSTEM

The respiratory system's major function is to supply the human body with oxygen and to eliminate carbon dioxide. The uppermost part of the system is the pharynx, which includes the mouth and nose and extends to the trachea. In this part of the system inhaled air is warmed to body temperature and saturated with water vapor, preventing dessication of and damage to the lower respiratory tissues. The nasal mucosa, with a surface area of about 160 square centimeters, is covered with glands which secrete the fluid necessary to moisten the air. Sneezing is a forceful way of eliminating trapped particles and irritating substances.

The airway consists of the trachea, bronchi, and bronchioles. The trachea divides into the two major bronchi, which divide into smaller bronchi, on down to the bronchioles. At each division the diameter decreases. The larger airways have rigid cartilage rings which keep them from collapsing. The smaller branches lack cartilage, but have a strong muscular lining. The muscle can contract and relax and thus regulate the air flow to lower regions.

The interior of the trachea and bronchi are covered with mucous glands and ciliary cells. The latter carry a number of small fibers that sweep mucous fluid and particles outward where they are expectorated or swallowed. Mucous which has collected in the bronchi as a reaction to infection or to substances in the inhaled air is expelled by coughing and by the ciliary action. This clears the passages and prevents noxious particles from reaching the more delicate lower tissues.

The bronchioles divide further and finally end in ten to twenty alveoli. These are thin membranes in which a dense network of capillary vessels is embedded. The gas exchange takes place here while the red blood cells are squeezed through the narrow vessels. The hydrodynamic force necessary to pump the blood through the capillaries comes from the contraction of the right ventricle of the heart.

The interior of the alveoli are covered with a thin layer of fluid, which acts as a surfactant and keeps the surface tension low. The elastic fibers in the space between the alveolar sacs create a tendency of the alveoli to collapse during the expiration phase. Inhalation creates a tension of about -3 mm mercury in the alveoli. The low surface tension allows air to be drawn in without collapse of the alveoli.

REACTION TO POLLUTANTS

Particulates and gaseous substances in the air exert their effect mostly on the respiratory system and heart. An effect on the skin and mucous

membranes, such as the conjunctivae, can occur, but is of lesser importance as a possible contributory factor to ill health and premature death.

The ways in which air pollution could potentially affect life expectancy are numerous, and are outlined below.

- I. Effects on Pulmonary Function
 - A. Interference with clearance mechanisms resulting in secondary pneumonia
 - B. Inciting immunologically mediated reactions
 - C. Inciting non-immunologically mediated inflammation
 - D. Asphyxiation
 - E. Carcinogenesis or co-carcinogenesis
- II. Effects on Cardiovascular Function
 - A. Pulmonary blood vessels
 - 1. Primary destruction or thickening of vessel walls
 - 2. Destruction of pulmonary vascular bed secondary to fibrosis or emphysema
 - B. Coronary and Systemic Vasculature
 - 1. Induction of vascular spasm
 - 2. Acceleration of atherosclerosis
 - C. Myocardium
 - 1. Metabolic poisoning
 - 2. Induction of arrhythmias
 - a. directly
 - b. through mechanisms of hypoxia
- III. Effects on other systems
 - A. Inhibition of metabolism
 - B. Distant carcinogenesis
 - C. Induction of metabolic enzymes

Examples of a number of these possible mechanisms can be found by examining the reaction to various types of particulate and gaseous pollutants found in the air. Some of the examples are from occupational exposures and may not affect the mortality patterns of the general population. However, they do serve as examples of the potential mechanisms by which air pollutants might affect mortality.

We will review mechanisms and examples under each of the types of pollutants that are known to exist. It should be recognized, however, that there is reason to believe that certain combinations of pollutants may in fact have synergistic effects. For example, certain small particles of metallic origin such as iron or manganese may catalyze the oxidation of sulfur dioxide to sulfur trioxide, the precursor of sulfuric acid. Thus, while sulfur dioxide by itself may have mildly irritating effects, its presence when combined with that of certain small particulates might increase or otherwise alter its effect on the respiratory system.

Reduced pulmonary function has been recognized as a risk factor for coronary atherosclerosis,¹ separate from the effects of cigarette smoking. Although detailed mechanisms for these increased risks have not been well established, it is possible that air pollution exposure is a contributing factor.

DUSTS

If the ciliary lining of the upper part of the bronchial tree functions well, particles greater than 5 microns in diameter are removed from the upper part of the respiratory system. Smaller particles may reach lower branches of the bronchial tree and particulates less than one micron in diameter can be deposited in alveoli. Very small particles behave like gases and remain suspended in the air by Brownian motion. Some of these very small particles will be deposited eventually on the walls of the terminal alveoli.

In the alveoli, particles may be phagocytosed by white blood cells or macrophages; moved outwards and be removed by the ciliary lining of the larger bronchioli; accumulate in the connective tissues filling up the space between the alveolar sacs; or enter the lymphatic system to be carried away and captured in the regional lymph glands. The particles may reduce the surface tension maintained by the surfactant. They may activate enzymes such as elastase, present in macrophages, which could then destroy alveolar tissue. Some insoluble particles such as carbon, quartz, silica and asbestos, may become trapped in the interstitial lung tissue. Soluble particles would not be trapped; their effects would depend on chemical reactions within the system.

Inflammatory reactions occurring as a result of activation of enzymes or as result of trapping in the interstitial lung tissue can result in destruction of alveolar tissue. With destruction of lung tissue in the alveoli, changes occur which lead to disease and disability. Reduction of the alveolar capillary bed increases the resistance to the blood flow from the right side of the heart. The flow is maintained by increase in pressure in the pulmonary vascular system. The increased pressure leads to hypertrophy of the heart muscle. With continued increases in resistance, eventually the heart is no longer able to generate the necessary pressure and the heart muscle begins to fail.

Reduction of the exchange surface area of the alveoli results in poor oxygenation of the blood. The hypoxia can aggravate the induced pulmonary hypertension as well as failing to provide adequate oxygenation for normal cell function throughout the body. Destruction of elastic fibers between the alveolar sacs leads to air trapping in the alveoli, thus contributing to poor gas exchange.

The association of various pneumoconioses and occupational exposure to dusts has long been recognized. In most of these pneumoconioses the dust gives rise to a fibrosis in the spaces surrounding the alveoli and bronchioles. The fibrosis is the end result of repair of tissue damage.

This damage could occur directly (as in silicosis), as a result of foreign body reaction (as in asbestosis), or as a result of an immunologic reaction (as in bagassosis). The progressive fibrosis leads to a reduction of the volume of the pulmonary capillary bed, as well as to a decrease in gas exchange across the alveolar membranes. The diminished gas exchange and diminished volume lead to progressive hypoxia, which may further aggravate pulmonary hypertension, predispose to cardiac arrhythmias, and diminish functional respiratory reserve. Death follows as a result of pulmonary failure, which may be precipitated by infection, or as a result of right heart failure.

GASES

The locus of action of gaseous substances depends on their solubility in the mucous fluids lining the respiratory tract and the concentration in the inspired air. Highly water soluble gases (SO_2) are absorbed in the upper airways. Less soluble gases (CO , O_3) reach the more vascularized lower regions of the respiratory system and can damage the alveolar tissue, or enter the blood stream and exert their toxic effect on remote organs, such as liver, kidney, bones, nervous system, or heart.

Hydrocarbons

Hydrocarbons, in particular the polynuclear aromatic hydrocarbons such as benzo(a)pyrene, are frequently implicated as carcinogens based on animal tests. Oxygenated derivatives such as formaldehydes may act as allergens or irritants which affect the pulmonary system or as carcinogens. Aromatic hydrocarbons such as benzene may also have significant toxicity to the bone marrow.

Sulfur Oxides

Acute episodes of high pollution with an associated dramatic increase in the number of deaths among high risk individuals have occurred in Belgium,² London,³ Pennsylvania,⁴ Japan, and New York City. Follow up of the population of Donora, Pennsylvania, which was exposed in 1948 to an acute sulfur dioxide fog, demonstrated a continued higher mortality in the decade following the exposure.⁴ However, animal experiments showed that sulfur dioxide was only a mild irritant and concentrations up to 1 ppm do not cause pulmonary damage. As mentioned before, sulfur dioxide may be converted to sulfur trioxide which forms sulfuric acid in the presence of water. Sulfuric acid is a very potent irritant and can cause severe bronchial constriction and coughing. In higher concentrations (at the milligram/ m^3 level), it can lead to necrosis with a resulting fibrotic reaction. Some sulfates may act chemically on cell membranes, causing release of histamines, thus initiating bronchiolar constriction.

Nitrogen Oxides

Nitrogen oxides, particularly nitrogen dioxide, have been demonstrated to cause pulmonary disease in animals after chronic exposure. Changes observed included loss of ciliary action in the bronchial tree with resulting impaired ability to combat infections, as well as distended alveoli leading to emphysematous changes. In animals, systemic effects such as weight loss and increased methemoglobin levels have been observed.⁵ In humans a study performed by the USPHS in Chattanooga, Tennessee suggested that observed episodes of bronchitis might be associated with higher levels of nitrogen dioxide.⁶

Carbon Monoxide

Carbon monoxide compromises cardiovascular system function by forming a tightly bound complex with hemoglobin, called carboxyhemoglobin. Hemoglobin has an affinity for carbon monoxide 200 to 400 times greater than its affinity for oxygen. Carboxyhemoglobin decomposes slowly, thus impairing the oxygen carrying capacity of the blood. Chronic low levels of carbon monoxide exposure may accelerate atherosclerosis and/or precipitate coronary vessel spasm.⁷ Shinshu disease, in a Japanese population habitually exposed to high carbon monoxide concentrations, included angina pectoris, heart valve lesions, and premature atherosclerosis.⁸

Ozone

Ozone is a strong oxidant and an irritant to the upper part of the respiratory system. Acute exposures to very high levels of ozone can precipitate pulmonary edema. Whether chronic exposures to low levels can be harmful is more questionable. In animals ozone has been implicated in pulmonary fibrosis, tumor induction, bronchitis, acceleration of aging, and in reduction of the ability to combat infection.⁹ These effects have not been demonstrated in man and a diminished sensitivity to ozone has been observed in persons chronically exposed to low levels of ozone.¹⁰⁻¹³

SUMMARY

Most available information about the effects of air pollution on health refers to the effects on the respiratory system. These effects range from bronchiolar constriction and coughing induced by irritants to pulmonary fibrosis. Direct effects upon the heart as well as more distant target organs may be suspected, but evidence is lacking. The heart might also be affected by its attempts to compensate for pulmonary disease, or by hypoxia occurring as a result of lung disease.

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Notice to Users of BNL 52122

"A Statistical Study of the Macroepidemiology of Air Pollution and Total Mortality"

Please note that, since publication of this report, errors have been discovered in the long-range transport model used to estimate expected concentrations of SO_2 , SO_4^{2-} , and NO_x , such that spatial bias may be present in these estimates. Accordingly, mortality regressions based on these estimates ($n=916$) should not be used. Estimates based on actual air quality measurements are not affected.

Frederick W. Lipfert