Effects of Air Pollution on Health Outcomes

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PREFACE

This report documents the findings and recommendations of a Rand Corporation study on the health effects of air pollution. The study was funded by the U.S. Environmental Protection Agency under Cooperative Agreement No. CR811040-01-0. It represents the first phase of a three-phase project evaluating the relative abilities of several analytical approaches to measure pollution effects. The data analyzed were collected in Seattle and Dayton during the Health Insurance Experiment (HIE) that Rand conducted for the Department of Health and Human Services. The second phase will extend the analysis to HIE data from other cities and to other data sets. These results will then be used in the third phase to measure the health effects of air pollution.

Those effects are important for formulating federal programs for pollution control. Comparing the benefits with the costs of control will enable federal lawmakers and regulators to decide on a cost-effective level of control. Since the study considers several pollutants, Phase II output may be helpful to regulators who must decide where to concentrate scarce pollution control resources. Our methodology and findings should also be of interest to several other groups:

- All parties interested in air quality, especially in decisions regarding the Clean Air Act and regulations issued under its authority.
- Epidemiologists interested in the health effects of ozone and other pollutants.
- Statisticians and social scientists interested in the application of statistical procedures to panel data, especially procedures designed to draw precise inferences from limited data.

SUMMARY

BACKGROUND

It is widely known that air pollution adversely affects health. The "killer fogs" of London amply demonstrated that very high concentrations of air pollutants can kill people. Controlled laboratory studies have also identified adverse short-term effects of high levels of pollutants on lung function and other physiological indicators. An analysis done in Southern California found that people living in a highly polluted area had poorer health than those in a cleaner location. However, studies of pollution at the more moderate levels encountered by almost all Americans have been much less conclusive. Some have not been able to detect significant health effects; others have yielded mixed results--relationships in the expected direction in some cases, associations between increasing pollution and improving health in others.

The question, then, is why research into air pollution has not been more successful at measuring health effects. One possible reason is that many of the studies done to date have had one or more methodological shortcomings. Some, for instance, have confounded the effects of air pollution with those of risk factors such as smoking. Others have not accounted for the possibility that people with respiratory problems might move to places with cleaner air, leaving a healthier population behind in the polluted area. Generally, data have not been available for analyzing day-to-day health responses of general populations to pollution episodes. Finally, there has been some difficulty in deciding what diseases to look for as evidence of health effects.

Whatever its cause, the lack of reliable assessments of the health effects of air pollution has hampered regulatory agencies interested in comparing the value of health improvements obtained through good air quality with the costs of controlling pollution.

OBJECTIVES AND APPROACH

This study has two objectives: to examine the health effects of air pollution on a general population in moderately polluted cities, and to apply a battery of disparate analytical approaches to an especially attractive set of data collected with the same data methods in two widely separated cities, Seattle and Dayton.

The data we analyzed were collected during The Rand Corporation's Health Insurance Experiment (HIE). This data set is attractive for our present purpose for several reasons:

- It is a sample of the general population, not of some group selected for a particular characteristic, e.g., susceptibility to air pollution.
- Data were collected in cities with pollution levels typical of U.S. cities in general.
- Several general health measures, such as use of medical services and time lost to illness, were recorded daily for several thousand people over three to five years.
- These were supplemented by other general measures, such as overall health status and lung function, in addition to data on specific diseases and chronic health problems.
- The data included information on smoking and other risk factors, and other potential confounding variables and risk factors.

We employed a simple cross-sectional analysis and three panel analyses. The cross-sectional analysis estimated pollution effects by pairing all individual yearly responses (e.g., time lost to illness over the course of a year) with the corresponding individual yearly pollution exposures. This analysis treated all responses the same; yearly responses from the same person and from different people were all regressed together. The panel analyses, on the other hand, tracked responses from the same individual or population over time. These analyses were as follows:

- An analysis that used the aggregated exposure and response of the whole population on a daily basis. Comparing the whole fixed population with itself from one day to the next eliminated any bias resulting from geographical sorting on the part of sickly people.
- An analysis that took advantage of within-city data on variations in pollution by using the day-to-day health responses of individuals. This approach employed recently developed statistical methods designed to draw more precise and consistent estimates. It eliminated geographical sorting bias by estimating responses for each person separately; thus, each person acted as his own control. The individual responses were then analyzed together.
- An analysis that estimated the change in individuals' health over the entire course of the HIE as a function of their cumulative exposure to pollution over that period. Using change in individual health status as the dependent variable should reduce the sorting bias.

RESULTS

We found the individual day-to-day approach to be the most promising. It yielded negative associations of air pollution with health for all pollutants examined except for ozone. Half of those associations were significant at the 1 percent level. Because this approach, unlike the others, was applied only in Seattle and because it is quite expensive, we recommend that it be tested further.

The aggregated day-to-day method yielded a number of counterintuitive results. We do not have a good explanation for the "misbehavior" of this approach. The annual cross-sectional analysis and the analysis of changes in health status over the course of the study yielded generally insignificant results for all pollutants except ozone. We recommend further application of these methods also.

While the effects measured for the pollutants generally varied with the analytical method, ozone was found to have consistently positive associations with health, most of them significant at the 10 percent

level or better. The most probable explanation is that at the low levels of ozone encountered in these two sites, ozone is correlated with something else that produces short-run beneficial effects, such as good weather.

LIMITATIONS

The most important limitation of the HIE was its exclusion of the elderly, who are often regarded as being among the most susceptible to air pollution. That exclusion also precluded an examination of the effects of air pollution on mortality. Finally, the five-year run of the HIE confined the analysis to short- and medium-term effects.

CONCLUSION

We have identified a promising method for measuring the health effects of air pollution. However, before accepting this method or discarding others, it is important that they all be tested further to determine whether the results we have derived so far are more generally applicable. We believe that the most efficient way to complete the testing would be through the further analysis of panel data.

CONTENTS

I. INTRODUCTION

BACKGROUND

It has long been accepted that air pollution degrades the health of persons exposed to it. Epidemiological studies have demonstrated quantitative relationships between episodes of air pollution and acute, short-term health losses. In laboratory studies, pulmonary function has responded negatively in humans occupying closed chambers into which air pollutants have been pumped. As for chronic, long-term effects, the Chronic Obstructive Respiratory Disease Study conducted by the University of California, Los Angeles, has shown larger annual decrements in lung function in persons living in Glendora, California, than in less polluted Lancaster, California (Detels et al., forthcoming). Air pollution caused a number of deaths in the infamous "killer fogs" of London and Donora, Pennsylvania. It is suspected that air pollution has caused other deaths more insidiously through chronic exposure (see, e.g., Lave and Seskin, 1977).

However, many observational studies of the effects of air pollution have yielded results that have been suggestive at best. They have shown that a given pollutant *may* affect a susceptible person under certain circumstances while having no effects, or even counterintuitive effects, for others. For example, Portney and Mullahy (1983) arrived at mixed results that were quite sensitive to the analytical approach employed.

There may be several reasons why these studies have not been able consistently to measure the adverse effects of air pollution. First, the measures used may not have been sensitive enough to detect an effect, or may have been applied where no effect exists. The effect may be so small that it may be obscured by random variation, or it may require carefully controlled conditions if it is to be measured. If only sensitive persons are affected and then only marginally, sample populations examined to date may have been too small. For instance, if only one percent of sensitives respond adversely, a population would have to contain several hundred sensitives to allow detection of the effect.

The second possible reason for failure to detect an effect is the difficulty of defining the effect. Air pollution is not associated with any specific disease. Respiratory effects of ambient air pollution have generally been observed only in persons with lung function already compromised by some condition such as asthma. Individuals with unimpaired respiratory systems report a variety of effects of short-term exposure, including headache, eye irritation, malaise, depression, and general irritability. Persons exercising in high levels of ozone may experience nausea. Long-term exposure may have other effects. Cigarette smoking, which is similar in some respects to breathing air pollutants, increases the long-term risk of lung and bladder cancer, ischemic heart disease, and other conditions. The sum of the effects of air pollution could be quite large, while none of the diverse individual effects may be large enough to measure. There is little basis for grouping the effects prior to analysis, given their diversity. The common association with a given pollutant that would allow grouping has not been discovered yet.

The third reason is a complex of methodological and data problems. These include the use of data aggregated over populations, unreliable estimates of pollution exposure, lack of detail on health outcomes, and incomplete data on population characteristics that may be correlated with air pollution exposure. Many studies have relied on cross-sectional data on a population's health outcomes, instead of on data recorded at more than one point in time. With cross-sectional data, pollution effects are underestimated if susceptible persons have moved to areas with better air quality.

Finally, it is possible that these methodological difficulties are not responsible for the inconsistent and negative results generated so far. It may be that air pollution has no significant negative health effect on most persons exposed to it, at least not at the levels occurring in most American cities. (Of course, we know that air pollution at high levels has serious and immediate adverse health effects.)

Obvious policy implications arise from the inability to obtain consistent and reliable measurements of the adverse health effects of air pollution, especially at moderate levels. Health officials and environmental groups have expressed concern over those effects for many years. This concern has been shared by a broad enough cross-section of the general public to lend support to the passage of numerous federal, state, and local laws regulating air quality. A growing realization of the burden that those laws place on the national economy has recently given rise to a more critical approach to the data linking air pollution to adverse health effects. Although Congress mandated clean air policy with respect to human health, regulatory agencies are increasingly concerned with comparing the costs of regulation with the benefits, monetary or otherwise, that can be realized from it. For example, if ozone had no effects at the levels usually encountered, but moderate levels of suspended particulates proved more costly in terms of health effects than the measures taken to control them, then regulators could focus on reducing particulates. With no available systematic measurements of the health effects of air pollution, however, it is not possible to estimate the benefits of air quality regulation--if indeed there are any.

OBJECTIVES AND METHODS

This report discusses work in progress conducted by The Rand Corporation under the sponsorship of the U.S. Environmental Protection Agency, the purpose being to examine the effects of air pollution on several indicators of health outcomes and health-related costs. For this research we have analyzed data from a panel study of the nonaged population in two cities with moderate levels of pollution, Dayton, Ohio, and Seattle, Washington. We have been able to examine the sensitivity of the measured effects to the use of alternative analytical approaches, in particular panel and cross-sectional techniques.

The Data

We were enjoined by EPA to use available data, rather than collect new data specific to our purpose. The principal advantages of using an existing data base are that the substantial costs of data collection have already been incurred, and the study's results will be available much sooner. There are also serious limitations, however: (1) The data may not be ideal because they were collected for another purpose. As we shall see below, a major drawback of the general population data sets is the lesser quality of the air pollution exposure measures that we can derive; the measurement error in the exposure estimates will yield biased estimates of the adverse effects of air pollution. (2) The population studied may not be fully appropriate to the analysis at hand. (3) Using existing data sets means that we are conducting an observational study, and such studies can yield estimates that are badly biased (and in some cases, the bias is of indeterminate sign, a priori). In the case of air pollution studies, the risk is that individuals who are more susceptible to air pollution will move to less polluted areas (e.g., Tucson), thus confounding the observed air pollution exposure with the unobserved sickliness of the individual. That confounding will yield underestimates of the adverse effect of air pollution.

Air quality data analyzed in this research are drawn from Storage and Retrieval of Aerometric Data (SAROAD) and some state agencies. Health outcome data are drawn from Rand's Health Insurance Experiment (HIE), conducted from 1974 to 1982 under a contract with the U.S. Department of Health and Human Services. HIE data were collected at six sites around the country. The analysis in this report is limited to data from the two largest sites, Seattle and Dayton.

We used the HIE data for several reasons. First, they include measurements of the use of medical services, and time lost due to illness (e.g., from work or school) or due to restricted activity. Thus, we can examine the effects of air pollution on several health outcomes. Second, the data are measured continuously or repetitively over time, enabling us to assess the sensitivity of the results to using both cross-sectional and panel approaches to estimating the adverse effects of air pollution. Third, it contains data on the prevalence of diseases at the

outset of the experiment and on their incidence over the course of the experiment, and on the occurrence of new episodes or exacerbations of illnesses. Information is available on measures of physiological variables over time, e.g., lung function at entry to and exit from the study. Thus, the HIE data allow the assessment of physiological changes that may be significant but too small to result in disease within the course of the experiment. The HIE also includes data on socioeconomic status, health status, health habits (e.g., smoking), and race and other demographic variables. Such variables are important because they include risk factors and confounding variables that must be controlled for if the effects of air pollution are to be properly estimated. Fourth, outcomes are recorded as they occur, allowing the elucidation of short-term effects through correlations with daily pollution and weather data.

Other data sets were evaluated for use in this study. The reasons for not accepting them are given in App. A.

Despite its advantages, the HIE does have four major limitations. First, the sample excludes individuals who are over 62, eligible for Medicare, on Medicare disability, severely handicapped, in the military, or in households in the top 3 percent of the income range. The elderly and the ill are believed to be especially susceptible to the adverse effects of air pollution. As a result, estimates based on the HIE understate the full social effects of air pollution. Conclusions about threshold concentrations required for adverse effects may also be biased. Second, HIE sites were chosen for their variation in access to health care services. This limits the validity of intersite comparisons of pollution effects, since pollution effects would be confounded with variations in site characteristics that affect the use of services (e.g., time delay in getting a doctor's appointment). Third, we had to infer pollution exposure based on available ambient air quality data, because the HIE data had already been collected. Thus, we could not obtain the more reliable estimates of individual exposure that could be derived from a microenvironmental analysis or personal monitoring. As a result, our estimates of the response to air pollution will be biased systematically toward zero (i.e., finding no effect). Fourth, this is an observational study rather than a randomized trial. To the extent that individuals may move or alter their behavior to minimize the adverse

effects of air pollution, we will systematically understate the effects of air pollution. In some of the results reported below, we have used panel data techniques to reduce this bias, but we cannot be sure that the techniques completely solve this problem.

The HIE and the variables drawn from it are discussed fully in Sec. II.

Analytical Approach

In assessing the adverse effects of air pollution on health outcomes--use of services, time lost to illness, and health status--there are two major dimensions over which we have varied our analysis. The first dimension is the choice between cross-sectional and panel approaches to the estimation. The second is the length of time over which we look for the effects of pollution.

Cross-sectional vs. Panel Approaches. In our analysis, we have used both cross-sectional and panel approaches in estimation. In the cross-sectional approach, we assign to each individual measures of his air pollution exposure based on the ambient air pollution at his work and home locations. By comparing the health outcomes of different people with different exposures, we can estimate the association of air pollution with those outcomes.

The cross-sectional approach is simple, but may lead to misestimates of the effect of air pollution for several reasons. The most important is that people may have sorted themselves out across air pollution zones based on their sickliness or other unobservable or imperfectly observable characteristics. That is, cross-sectional data may lead to biased estimates if the unobservable characteristics of the populations studied are correlated with the observed explanatory variables, including air pollution exposure.

For example, if air quality is too poor, individuals susceptible to air pollution's adverse effects may leave the area studied or die. The studies of respiratorily impaired persons in Tucson by Lebowitz, Knudson, and Burroughs (1978) includes people who moved there in part because of the perceived benefits of desert air for persons with lung problems. In our study, asthmatics and other susceptibles may move from the more to the less polluted areas of the city. In either case, areas

with different levels of pollution would have a different mix of healthy and sick individuals, with the cleaner areas having more sick people. Comparing health outcomes cross-sectionally would understate the adverse effects of air pollution, because the unobserved extra sickliness in cleaner areas would dilute the effect of air pollution on the estimates. In fact, if the geographical sorting is pronounced, we could find that higher air pollution is associated with "better" outcomes, e.g., lower use of medical services.

Cross-sectional estimates can also be biased in the other direction, that is, they can overstate the effects of air pollution. For example, if smokers are less likely to move away from smoggy areas and if smoking behavior is imperfectly controlled for in the analysis, then cross-sectional estimates would attribute part of the adverse effect of smoking behavior to air pollution.

In either case, cross-sectional data can lead to biased estimates. Without further information, the researcher cannot bound or estimate the magnitude of the bias, nor determine its direction.

Despite the problem of bias from geographical sorting, we use a cross-sectional approach as one way to analyze the effects of air pollution on each individual in the sample.[1] Each point on our regression line thus pairs one person's health outcome in a given year of the study with his or her exposure to air pollution that year. By comparing those results with our panel results, we can get some idea of the empirical value of the former, which could be useful in assessing the validity of other cross-sectional studies.

In the panel analyses, we use the presence of repeated observations on each individual to control for unobservable individual characteristics. Thus, in a panel study, we do not have to rely on the untestable cross-sectional assumption that the unobserved characteristics are uncorrelated with the observed independent variables, including air pollution exposure.

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[1]Our approach is not always a pure cross-sectional one. In the analysis of annual outcomes, we use data from the same people in different years. However, the data are analyzed using the same assumption used in cross-sectional analysis, that the error term is independent of the covariates, especially air pollution.

Panel analyses have three other major advantages over a cross-sectional study. First, our panel studies can take advantage of finer detail on timing of health events than do cross-sectional studies. The finer detail permits better estimates of the weather and air pollution exposure than is possible with data aggregated over longer periods of time. Less precise estimates of air pollution can result in underestimates of its effects on health. But, second, with a panel study, we can still check any assumptions about the differences between short- and long-term effects by examining the response in daily as well as annual data. Third, our panel analyses retain the movers and deaths occurring in the sample after baseline measurements, whereas in a pure retrospective cross-sectional design, those who moved or died are not around when the data are collected.

Nevertheless, panel studies have two major shortcomings relative to cross-sectional studies. First, due to the higher cost of collecting panel data, panel studies typically have fewer participants than can be studied in a cross-sectional analysis. This smaller sample size reduces the precision available for detecting adverse effects. Second, panel methods, such as before-and-after comparisons, are limited to detecting short- and intermediate-term effects, because the time frame for the panel is frequently only a few years.

Duration of Effect. We have used a variety of time frames for our analyses, because air pollution may result in both short-term and intermediate-term adverse effects (long-term effects cannot be analyzed using these data, which were collected over a three-to-five-year period). A concern over irreparable damage has led to some emphasis on intermediate to long-term effects in studies of susceptibles and mortality. However, we need to remember that major social costs may arise from short-term responses in a general population. The losses per individual may be small in a general population, but the large number of individuals can make for a large total loss.

In what follows, we have examined the effects of pollution exposure on use of services and time lost to illness in terms of both short-term responses (daily responses to daily air quality variation), and a somewhat longer-term annual analysis. We have also examined the effects

of pollution exposure on health status in terms of both short-term responses (air quality in the most recent month) and intermediate-term responses (average exposure over a two-and-a-half to five-year period).

Specific Approaches Employed. We have not taken all possible combinations of outcomes and time frames for both cross-sectional and panel approaches. Some combinations were precluded by data limitations. For example, we do not have daily data for our general health status and lung function measures. Some combinations were omitted because the cost would have exceeded any likely benefit. For example, a cross-sectional approach to short-term daily fluctuations in time lost to illness seemed unduly expensive.

Four sets of analyses are reported below. The first, in Sec. III, is essentially a cross-sectional approach to annual responses for use of medical services and time lost to illness.

The second set, in Sec. IV, considers the effect of recent air pollution levels on a set of daily observations on the proportion of the population ill or visiting a health provider. There is thus one data point for the whole population for each day of the study. Because the population is fixed over time and because individuals are not being compared with other individuals, we avoid the problem of there being unobserved population characteristics (e.g, susceptibility) that are correlated with pollution exposure, e.g., through geographical sorting. (The population effectively acts as its own control.)

Our third set of analyses, in Sec. V, examines the effect of air pollution on a set of daily observations for each person individually, rather than collectively. Because we follow an individual over time, we again avoid the problem of unobserved characteristics that are correlated with air pollution exposure. This analysis has the potential for improving on the second approach because it uses exposure estimates that are tailored to the individual; this should reduce any misestimate from using a single air pollution exposure value for a whole metropolitan area.

Our final approach, in Sec. VI, examines the effects of air pollution exposure (cumulative since the beginning of the study) on individual health status at the end of the study. Each individual's exit health is regressed on air pollution and entrance health. This

variant on a before-and-after comparison nets out any unobservable characteristics that may be correlated with air pollution exposure.

We apply each of these four methods to estimate the effects of air pollution on one or more of the following health variables:

- Probability of use of any outpatient health care services
- Expenditures on outpatient health care services per user
- Time lost to illness (including time lost from work, school, and other usual activity)
- A subjective measure of general health (tested for its reliability)
- Lung function

In the next section, we discuss the data we analyzed. Subsequent sections describe each of our methods and their results.

LIMITATIONS

In examining our results and conclusions, it is well to keep the limitations of our study in mind. The most important is that this is an observational and not an experimental study. Although the study relies on data from a randomized study, the randomization was for health insurance, not air pollution. Families with members who are susceptible to air pollution may choose to live in less polluted areas. As discussed above, this can lead to biased results. While our panel techniques are an improvement over cross-sectional approaches in reducing geographical sorting bias, they do not yield the kind of safe conclusions that can be drawn from experiments.

Second, in this study, we have not been able to examine the effect of air pollution on life expectancy. The sample is not large enough to look at mortality in the nonaged. The exclusion of the aged makes it doubly difficult to discern changes in survival, by reducing the sample size and by excluding the group at highest risk. In addition, the exclusion of the elderly means that our estimates of other health effects are understated. For example, the elderly are believed to be especially susceptible to air pollution. They also account for a disproportionate share of total time lost to illness, and of medical expenditures.

Third, our measure of exposure to air pollution is based on ambient monitoring sites linked to residence and work locations. The measure could be improved if we had data on housing and work characteristics (e.g. type of space heating or air conditioning), or if we knew actual individual exposures directly. The error in our measures probably biases our estimates of the effects of air pollution toward zero.

Finally, this report is limited to two sites; hence, at this point we do not know how generalizable our results are.

II. DATA AND SAMPLE

The data for this analysis are drawn from two sources. First, the source of data on sociodemographic variables, health status and habits, use of health services, and time lost due to illness is the Health Insurance Experiment (HIE). Second, the sources of data on air quality and weather are Storage and Retrieval of Aerometric Data (SAROAD), the Washington State Implementation Plan (SIP) data bases, and the National Weather Service.

THE HEALTH INSURANCE EXPERIMENT

The HIE is a randomized trial of the effects of different health insurance arrangements on the demand for health services and the health status of individuals.[1] The HIE enrolled families in six sites: Dayton, Ohio; Seattle, Washington; Fitchburg, Massachusetts; Franklin County, Massachusetts; Charleston, South Carolina; and Georgetown County, South Carolina. This analysis uses data from the Seattle and Dayton sites. In each site, families enrolled for either three or five years.

Families participating in the experiment were assigned to 14 different fee-for-service or two prepaid group practice insurance plans. The fee-for-service plans had different levels of cost sharing, which varied over two dimensions: the coinsurance rate and an upper limit on out-of-pocket expenses. The coinsurance rates (percentage paid out-of-pocket) were 0, 25, 50, or 95 percent for all health services. Each plan had an upper limit (the maximum dollar expenditure or MDE) on out-of-pocket expenses of 5, 10, or 15 percent of family income, up to a maximum of $1,000. Beyond the MDE, the insurance plan reimbursed all expenses in full. One plan had different coinsurance rates for inpatient and ambulatory medical services (25 percent) than for dental

[1] Newhouse (1974) and Brook et al. (1979), provide fuller descriptions of the design. Newhouse et al. (1979) discuss the measurement issues for the second generation of social experiments, to which the HIE belongs. Ware et al. (1980) discuss many aspects of data collection and measurement for health status.

and ambulatory mental health services (50 percent). Finally on one plan, the families faced a 95-percent coinsurance rate for outpatient services, subject to a $150 annual limit on out-of-pocket expenses per person ($450 per family). In this plan, all inpatient services were free, so that, in effect, this plan had an outpatient individual deductible. All plans covered the same wide variety of services.[2]

Two groups were enrolled in a prepaid group practice or health maintenance organization (HMO) in Seattle only. The HMO in this study is Group Health Cooperative of Puget Sound (GHC), a nonprofit organization that has been operating in the Seattle metropolitan area since 1946. The first of these two groups is the GHC experimentals, which is a random sample of the Seattle population that was not enrolled in GHC at the beginning of the experiment. This group received all services free of charge at GHC. If GHC did not provide the service, the plan fully covered services received outside GHC. The second group is the GHC controls, which is a random sample of families that had been enrolled at GHC for at least one year in 1976. The GHC control group received all care at GHC free of charge except for limited cost sharing on drugs, supplies, and outpatient mental health services.

To study methods effects, the HIE had three other randomized subexperiments. First, to increase precision in measuring changes in health status, some households were given a preexperimental physical examination; to test for a possible stimulus to utilization, the remaining households received no examination. Second, to measure sick- and work-loss days, and telephone utilization, some households filled out a diary on contacts with the health care system and on time lost to illness. To test for a stimulus of reporting on the use of services, some households filled out no forms, some filled them out weekly, and some biweekly. Third, to test for transitory aspects of the study, some households were enrolled for three years, others for five years.

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[2]See Clasquin (1973) for a discussion of the reasons for the HIE structure of benefits. Nonpreventive orthodontia and cosmetic surgery (not related to preexisting conditions) were also not covered. In the case of each exclusion, it is questionable whether anything could have been learned about steady-state demand during the three-to-five-year lifetime of the experiment. Also excluded were outpatient psychotherapy services in excess of 52 visits per year per person.

Families were enrolled as a unit with only eligible members participating. No choice of plan (or other experimental treatment) was offered; the family could either accept the experimental plan or choose not to participate. To prevent refusals, families were given a lump-sum payment equal to their worst-case financial risk associated with the plan; thus, no family was worse off financially for being in the study.[3]

In Seattle, we found no unintended differences between the group that accepted and the group that refused the offer to participate in the study; see Manning et al. (1984). A similar analysis shows no difference in Dayton; see Newhouse et al. (1982).

THE SAMPLE

The sample is a random sample of each site's population, but the following groups were not eligible: (1) those 62 years of age and older; (2) those with incomes in excess of $25,000 in 1973 dollars (or $56,000 in 1983 dollars); (3) those eligible for the Medicare disability program; (4) those in jail and those institutionalized in long-term hospitals; (5) those in the military or their dependents; and (6) those with service-related disabilities.

The sample used in this analysis includes enrollees during each full year that they participated. We excluded data on partial years of participation by newborns, adoptees, suspended participants (e.g., those who joined the military), participants who left the study before its completion, and people who moved out of the Seattle and Dayton areas.[4] A person who, for example, attrited in year 2, was included in year 1 if

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[3]Families were assigned to treatments using the Finite Selection Model (Morris, 1979). This model is designed to achieve as much balance across plans as possible while retaining randomization; that is, it reduces correlation of the experimental treatments with health, demographic, and economic covariates.

The family's nonexperimental coverage was maintained for the family by the HIE during the experimental period with the benefits of the policy assigned to the HIE. If the family had no coverage, the HIE purchased a policy on their behalf. Thus, no family could become uninsurable as a result of their participation in the study.

[4]Out-of-area moves were excluded so that we could inexpensively calculate the exposure of each participant.

he participated for all of that year. We excluded such cases because the statistical models used in this study for expenditures require equal time periods for each observation; that is, because they do not allow convolution of observations. Thus, the people who participated for only part of a year could appear to be different when their underlying behavior was in fact the same. The omission of individuals enrolled for a part year does not bias our comparisons because these individuals used health services at the same rate as full-year individuals with similar characteristics (see Manning et al., 1985, for Seattle; a similar analysis is under way for other sites).

For specific analyses, the sample was further reduced because of missing data. For example, in the analysis of time lost due to illness, we include only those individuals who filed health diaries for two years. Individuals who were randomly assigned to the no-health-diary subexperiment or who did not file the required forms were excluded.

Independent Variables

We used five groups of independent variables: insurance plan and other experimental treatments, health status measures, smoking variables, sociodemographic and economic measures, and measures of exposure to various pollutants. These variables are described below.

Insurance Plan Variables. We have used dummy variables to represent the insurance plans, one for each of the following insurance plans: the GHC controls; any fee-for-service plan with out-of-pocket cost-sharing (25-percent, 50 percent, or 95 percent) for the family; and a fee-for-service insurance plan with a family coinsurance rate of zero percent (free care). The GHC experimental plan was the omitted group in Seattle and the free fee-for-service plan was the omitted group in Dayton against which comparisons were made.

Measures of Health Status. We used four measures of health status to increase the precision of our estimates of the consumption of ambulatory medical services: (1) general health perceptions; (2) physical limitations; (3) chronic disease status; and (4) mental health status. Each of these measures is based on the self-administered Medical History Questionnaire for individuals 14 years or older. Measures for children are based on questionnaires filled out by parents.

All of the health status data used in this report were collected at the beginning of the study; a summary description of each is presented below.

The General Health Index (GHI) is a continuous score from 0 through 100 based on 22 questionnaire items for individuals aged 14 and over and 7 items for children (aged less than 14). The items measure perceptions of health at present, in the past, and in the future; the items also measure believed resistance to illness and health worry. GHI refers to health in general and does not specify a particular component of health. The construct is a subjective assessment of personal health status. The reliability and validity of GHI have been extensively studied and documented (Ware, 1976; Davies and Ware, 1981; and Eisen et al. (1980)).

One reason we chose the GHI was that the results of extensive validity testing could be used to place some perspective on observed differences resulting from air quality. For instance, the impact of chronic diseases, everything equal, is 5.6 points for hypertension and 10 points for chronic obstructive pulmonary disease or diabetes (Brook, 1983). People with FEV_1/predicted FEV_1[5] of 45 percent or less had a GHI 25 points lower on average than those with 91 percent or more. The death rate in the study was 25/1,000 for those with GHI under 63, 6/1,000 for those with GHI from 63 to 76 and 1/1,000 for those with GHI from 76 to 100.

The physical limitations measure is scored dichotomously (PHYSLM: 1 = limited, 0 otherwise) to indicate the presence of one or more limitations due to poor health. It is based on 12 questionnaire items for adults and 5 items for children measuring four categories of limitations: self-care (eating bathing, dressing); mobility (confined, or able to use public or private transportation); physical activity (walking, bending, lifting, stooping, climbing stairs, running); and usual role activities (work, home, school). The reliability and validity of these measures have been studied and documented by Stewart et al. (1977, 1978, 1981a, 1981b), and Eisen et al. (1980).

[5]Forced expiratory volume in one second is a measure of lung function.

The disease measure is a simple count of the number of diseases or problems (out of a possible 26), for individuals aged 14 or more (Manning, Newhouse, and Ware, 1982). The disease list includes kidney disease and urinary tract infections, eye problems, bronchitis, hay fever, gum problems, joint problems, diabetes, acne, anemia, heart problems, stomach problems, varicose veins, hemorrhoids, hearing problems, high blood pressure, hyperthyroidism, and ten other diseases or problems.

The Mental Health Inventory (MHI) is based on 38 questionnaire items measuring both psychological distress and psychological well-being, as reflected in anxiety, depression, behavioral and emotional control during the last month, general positive effect and interpersonal ties. The reliability and validity of this measure has been studied and documented by Veit and Ware (forthcoming); Ware, Veit, and Donald (forthcoming); Ware et al. (1979, 1980); and Williams et al (1981). We used a similar construct for children aged 5 to 13, based on 12 questionnaire items (Eisen et al., 1980).

Smoking Variables. The model used in our analysis also contained covariates for smoking status. These included dummy variables for whether an individual was a cigarette smoker, an exsmoker, a never smoker in a family of a smoker, and a never smoker in the household of an exsmoker. A never smoker in a family of never smokers is the omitted group. The categories are defined to be mutually exclusive.

Other Covariates. The model used in our analysis also included covariates for age, sex, race, family income, and family size. With the exception of family size, the data were collected before or at enrollment in the study.

Table 2.1 provides means for a number of these variables for the enrollment sample. Additional details on health status are available in Sec. VI.

Table 2.1

SAMPLE CHARACTERISTICS

Variable	Dayton N	Dayton Mean	Dayton Standard Deviation	Seattle N	Seattle Mean	Seattle Standard Deviation
Age	1139	6.069	17.141	3095	25.535	15.978
Female	1139	0.524	0.500	3095	0.512	0.500
EDUCDEC	1139	12.325	2.692	3095	13.012	2.409
Income[1]	1019	29600	13990	2986	37000	18300
AFDC	1019	0.048	0.214	2986	0.057	0.230
Black	1139	0.111	0.314	3095	0.027	0.161
Family size	1139	3.873	1.780	3095	3.395	1.578
GHINDX[2]	1139	73.183	7.818	3059	73.476	15.539
DISEA[3]	530	13.732	9.585	2178	11.900	8.626

NOTE: N indicates numbers of complete users.
[1] In June 1984 dollars.
[2] In Dayton, this is a replacement value based on responses to questions about health, pain, and worry.
[3] Count of chronic health problems, adults only.

Exposure Estimation

Assessing the relationships between health outcomes and exposure requires an estimate of the exposure of individuals to air pollution. Ideally, personal monitoring and microenvironmental analysis in workplace, home, and other places in which these individuals spend time could have provided this estimate. Unfortunately, we could not personally monitor the participants or conduct surveys to obtain these better estimates, because this research was initiated well after the HIE data collection effort ended. Instead, we used the SAROAD data base to estimate the exposure for each residence and work location based on air pollution levels at nearby local monitoring stations.

Data Sources. The HIE provided data on the residence location zip code of each participant at his entry into the study, and the date and location of each new permanent change in address thereafter. The HIE also provided data at intervals of approximately every six months on the labor force status of all adults, and the zip code for each employer on

the date surveyed. We use these two sets of data to implement a crude microenvironmental analysis.

We obtained daily data on air pollutants from SAROAD for the criteria pollutants (total suspended particulates (TSP), sulfur dioxide, nitrogen dioxide, oxidants, and carbon monoxide) for the Seattle-Everett and Dayton areas, including some outlying areas. We obtained data on the coefficient of haze and additional NO_2 data from the Department of Ecology for the state of Washington. The National Weather Service provided data on precipitation and temperature (minimum, maximum, and average daily values). In each case, the data covered the same period of time as the experimental period of the HIE, which was November 1974 through February 1980 in Dayton, and January 1976 through September 1981 in Seattle.

The number of monitoring sites for each pollutant varied by city and over time. We were able to use data from only a subset of the stations. Some stations were operational for only part of the period, and some had incomplete data when operational. To avoid possible data quality problems, we used only those stations which consistently reported air pollution levels over a sufficiently long time period. Our criteria for consistent reporting were that the monitoring site had to have at least six consecutive months of data for the pollutant of interest, and that each month had to include at least fifteen days of data. In the case of TSP, we generally accepted months with at least four 24-hour measurements, because TSP is routinely measured every six days.[6]

Missing Values. We did accept data from monitoring sites with minor breaks or gaps in their daily or hourly values, because monitoring sites are down for routine maintenance. For monitoring sites with missing hourly or daily values in a specific day, we replaced the missing values with imputed values based on the diurnal pattern of pollution levels, estimated from an additive two-way ANOVA model that identified the diurnal pattern and the effect of the day. For TSP, we used a similar model to impute missing daily values based on the day of the week pattern.

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[6]We made exceptions to the general criteria on the number of days in a month when the station was the only one reporting in that month.

Estimating Daily Exposures. The process for estimating daily exposures for each person involved three steps: calculating daily summaries for each monitoring site, creating a location history for each individual, and matching each individual's location history to monitoring sites.

For each monotoring site collecting hourly data, we calculated daily summaries of the pollutant levels. These included daytime and nighttime averages and maximums. The daytime values were based on readings from 8 AM to 6 PM and nighttime from from 6 PM to 8 AM. The analytic day was defined as the period from 6 PM on the previous calendar day to 6 PM on the day in question. This seemed to be a behaviorally more meaningful definition of a day than the usual midnight to midnight definition.

We developed a daily time series for each individual's daytime and nighttime locations, using the residence and work data described above. For the nighttime location, we used the home zip code, because our work data did not include information on which shifts were worked. For the daytime location for workers, we used the work zip code of the employer mentioned on the temporally nearest survey of work information. For children and for adults without paying jobs (e.g., housewives and the retired), we used the home zip code. We assumed that children attended neighborhood schools. For all individuals, we used the home zip code for the weekend. The HIE data on employment did not provide the information necessary to do a finer breakdown of work days and hours.

We then linked, day by day, each person's daytime and nighttime zip code to the daily summary for the geographically nearest monitoring site for each pollutant. The distance between the individual zip code and the monitoring station was measured using the latitude and longitude of the zip code's post office and the monitoring site's location. Although it would have been preferable to match the population center of mass for each zip code, we believe that the approximation error is minor in our case. Zips with high population densities have small areas, leading to only a small error in distance. Zip codes with low densities and large areas were typically in rural areas with clean air and few alternatives for matching. Tables 2.2 and 2.3 show the frequency of individuals by

Table 2.2

SEATTLE RESIDENCE ZIP CODES AND MAP COORDINATES

Zip Code	Count	Percent	Latitude	Longitude
98002	228	7.37	47.31	122.23
98003	103	3.33	47.31	122.31
98004	45	1.45	47.58	122.17
98005	9	0.29	47.61	122.15
98006	44	1.42	47.61	122.15
98007	101	3.26	47.61	122.15
98008	27	0.87	47.50	122.23
98011	78	2.52	47.72	122.22
98020	155	5.01	47.79	122.34
98022	3	0.10	47.21	121.99
98027	35	1.13	47.56	122.07
98031	124	4.01	47.40	122.25
98033	188	6.07	47.68	122.19
98036	94	3.04	47.84	122.29
98040	72	2.33	47.58	122.19
98043	56	1.81	47.80	122.30
98047	25	0.81	47.27	122.25
98052	49	1.58	47.64	122.15
98055	135	4.36	47.48	122.20
98062	4	0.13	47.47	122.36
98072	2	0.06	47.75	122.16
98100	3	0.10	47.63	122.33
98101	5	0.16	47.61	122.33
98102	29	0.94	47.63	122.31
98103	101	3.26	47.68	122.34
98104	3	0.10	47.60	122.33
98105	42	1.36	47.66	122.31
98106	63	2.04	47.52	122.35
98107	22	0.71	47.68	122.37
98108	18	0.58	47.52	122.30
98109	24	0.78	47.59	122.36
98111	3	0.10	47.63	122.33
98112	35	1.13	47.63	122.33
98115	103	3.33	47.68	122.30
98116	38	1.23	47.55	122.38
98117	46	1.49	47.63	122.33
98118	61	1.97	47.56	122.28
98119	48	1.55	47.63	122.36
98121	3	0.10	47.61	122.34
98122	56	1.81	47.61	122.31
98125	57	1.84	47.71	122.30
98126	12	0.39	47.54	122.37
98133	60	1.94	47.73	122.34

Table 2.2 (cont.)

Zip Code	Count	Percent	Latitude	Longitude
98136	51	1.65	47.53	122.39
98144	59	1.91	47.59	122.29
98146	27	0.87	47.48	122.35
98148	5	0.16	47.44	122.31
98155	37	1.20	47.75	122.29
98166	48	1.55	47.43	122.34
98168	131	4.23	47.47	122.30
98177	40	1.29	47.73	122.36
98178	35	1.13	47.50	122.25
98188	63	2.04	47.40	122.28
98199	29	0.94	47.65	122.40
98201	40	1.29	47.96	122.23
98203	56	1.81	47.97	122.20
98204	62	2.00	47.92	122.20
98206	3	0.10	47.96	122.23

Table 2.3

DAYTON RESIDENCE ZIP CODES AND MAP COORDINATES

Zip Code	Count	Percent	Latitude	Longitude
45305	44	3.86	39.64	84.07
45324	47	4.13	39.80	84.02
45342	52	4.57	39.66	84.27
45377	60	5.27	39.89	84.19
45402	2	0.18	39.76	84.19
45403	45	3.95	39.76	84.15
45404	15	1.32	39.79	84.17
45405	52	4.57	39.79	84.22
45406	15	1.32	39.79	84.24
45407	34	2.99	39.76	84.22
45408	32	2.81	39.74	84.22
45410	19	1.67	39.75	84.16
45414	44	3.86	39.82	84.21
45415	47	4.13	39.82	84.25
45417	19	1.67	39.75	84.25
45418	32	2.81	39.72	84.25
45419	78	6.85	39.71	84.16
45420	65	5.71	39.72	84.14
45424	157	13.78	39.83	84.14
45426	14	1.23	39.80	84.29
45427	15	1.32	39.75	84.28
45429	44	3.86	39.68	84.15
45431	24	2.11	39.77	84.10
45432	78	6.85	39.74	84.10
45439	9	0.79	39.69	84.22
45440	19	1.67	39.66	84.11
45449	7	0.61	39.67	84.24
45459	70	6.15	39.65	84.19

home location on the first day of the study, and the corresponding latitude and longitude. Table 2.4 and 2.5 show the monitoring sites used in our analysis for each pollutant, and their latitude and longitude.

These daily summaries for each individual provided the exposure data for the analysis of the individual daily time series of episodes of sickness (see Sec. V) and provided the basis for longer-term summaries. For each pollutant, we calculated each individual's monthly, yearly, and study-long average and maximum exposure to each pollutant. We also calculated the average of the daily maximums.

For the aggregate time series, we used a different approach to estimating air pollution exposure. For that analysis, we used only one observation for each day. In both Dayton and Seattle, we used the readings from downtown monitoring sites as our estimate of air pollution exposure. Clearly, this approach misestimates the exposure of individuals who live some distance from the central area. To use better individual estimates requires either doing a cross-sectional analysis or turning to the individual time series analysis.

Tables 2.6 and 2.7 provide summary statistics on the daily pollution levels for calendar year 1976 used in the aggregated time series. Because the values are from centrally located monitoring locations, the pollution levels present a worst-case summary for the daily levels. Tables 2.8 and 2.9 provide a summary of the annual level of exposure for the pollutants used in the annual analysis of Sec. III. The unit of observation is a person for one year. Hence, each area of the two cities is weighted by the number of people who live and work there, fully adjusted for changes in residence and employment. Tables 2.10 and 2.11 provide a summary of the cumulative exposure for each pollutant over the course of the study for the pollutants used in the before-and-after analysis of health status in Sec. VI. The unit of observation is a person. Hence each area of the two cities is weighted implicitly by the amount of time that people live and work there.

Table 2.4

DAYTON MEASURING STATIONS AND MAP COORDINATES

Station	Latitude	Longitude	CO	COH	NO2	OZONE	SO2	TSP
0800001G01	39.83	84.42						X
1100001G01	39.63	84.17						X
1260001G01	40.00	83.80				X		X
1660002G01	39.77	84.21	X					
1660003G01	39.76	84.19	X					
1660014G01	39.76	84.19						X
1660015G01	39.77	84.18						X
1660017G01	39.75	84.24						X
1660019G01	39.81	84.19		X	X	X	X	X
1660021G01	39.75	84.13	X					
1660022G01	39.70	84.31						X
1660025G01	39.76	84.20	X	X		X	X	
1660026G01	39.75	84.19	X					
1940001G01	39.74	84.63						X
2040001G01	39.79	84.03						X
2040003G01	39.83	84.00						X
2440002G01	39.63	84.37						X
2640001G01	40.10	84.63						X
2640002G01	40.10	84.61						X
2985001G01	39.87	84.14				X		
3240002G01	39.70	84.14						X
3240003G01	39.73	84.19						X
4280002G01	39.65	84.28				X		X
4500001G01	39.79	84.13		X			X	
4500002G01	39.80	84.35						X
4500003G01	39.85	84.33						X
4500004G01	39.79	84.13					X	X
4500005G05	39.64	84.22	X					
4550001G01	39.71	84.21						X
4760001G01	39.94	84.02						X
4790001G01	39.74	84.39						X
5100001G01	39.72	84.18						X
5520002G01	40.14	84.23		X		X	X	
5520003G01	40.14	84.24						X
5520004G01	40.14	84.21						X
5640001G01	39.84	84.72				X		
6380001G01	39.93	83.81						X
6380002G01	39.95	83.76						X
6380003G01	39.91	83.77						X
6380004G01	39.92	83.81		X		X	X	
6580001G01	39.96	84.17						X
6660001G01	39.80	84.30						X
6680001G01	40.04	84.20						X
6880001G01	39.90	84.21						X
6880003G01	39.89	84.20						X
7300001G01	39.96	84.33						X
7670001G01	39.81	84.03						X
7720001G01	39.70	83.93						X
7720002G01	39.71	83.93						X
7740001G01	39.80	83.89						X

Table 2.5

SEATTLE MEASURING STATIONS AND MAP COORDINATES

Station	Latitude	Longitude	COH	NO2	OZONE	SO2	TSP
0100003I01	47.31	122.23					X
0120002I01	47.61	122.20					X
0120004F01	47.61	122.20					X
0180001F01	47.57	122.62					X
0180002I01	47.58	122.61	X			X	
0640003I01	47.98	122.21	X			X	X
0960001I01	47.40	122.23	X	X	X		X
0960002I01	47.39	122.23					X
0980001I03	47.70	121.79					X
0980010F01	47.55	122.04			X		
0980013I01	47.33	122.31	X			X	
0980014I02	47.35	122.46				X	
1560002F01	47.16	122.51	X		X		X
1760002I01	47.48	122.20					X
1760003I01	47.48	122.21					X
1840001I01	47.60	122.33					X
1840001P01	47.60	122.33					X
1840007I01	47.66	122.39					X
1840009I01	47.62	122.35	X				X
1840057I02	47.56	122.27	X			X	X
1840058I01	47.45	122.28	X		X		
1840059F01	47.54	122.33	X		X		X
1840066I02	47.57	122.35					X
1840068I01	47.52	122.32					X
1840072F01	47.56	122.31					X
1840073I01	47.70	122.34	X				X
1840074F01	47.60	122.33					X
1840079F01	47.60	122.33					X
1840080F01	47.57	122.31		X			
2100001I01	47.40	122.22	X		X		
2140001I01	47.25	122.44					X
2140001P01	47.25	122.43					X
2140003I01	47.27	122.51	X			X	X
2140004I02	47.26	122.41	X				X
2140005I01	47.30	122.42	X			X	X
2140006I01	47.24	122.40					X
2140013I01	47.28	122.52				X	
2140015I01	47.23	122.43	X				X
2140017F01	47.20	122.49				X	X
2195001I01	47.46	122.25	X				
ST1776K64B	47.51	122.30	X	X			
ST2718P46B	47.09	122.62		X			
ST2718P47B	47.11	122.64		X			
ST3100S05B	48.08	122.19	X	X			

Table 2.6

1976 DAILY AIR POLLUTION LEVELS:
SEATTLE AGGREGATED TIME SERIES

	Pollutant		
Measure	Average SO_2	Average COH	Maximum Ozone
Mean	0.00977	0.788	0.0284
Std. Dev.	0.00964	0.518	0.0132
Quantiles			
100	0.0642	2.492	0.07
99	0.0454	2.368	0.07
95	0.0260	1.817	0.05
75	0.0150	1.082	0.04
50	0.0071	0.602	0.03
25	0.0021	0.388	0.02
5	0.0004	0.226	0.01
1	0	0.156	0.01
0	0	0.105	0
n	251	362	257

NOTE: Sample sizes vary due to incomplete pollutant data.

Table 2.7

1976 DAILY AIR POLLUTION LEVELS:
DAYTON AGGREGATED TIME SERIES

	Pollutant				
Measure	Average SO_2	Average COH	Average TSP	Maximum Ozone	Average NO_2
Mean	0.0152	0.273	106.44	0.0718	0.0244
Std. Dev.	0.0127	0.159	40.21	0.0396	0.0113
Quantiles					
100	0.0893	1.026	277.0	0.190	0.0648
99	0.0625	1.018	232.6	0.173	0.0592
95	0.0368	0.610	180	0.150	0.0438
75	0.0212	0.350	128	0.095	0.0315
50	0.0112	0.234	99	0.065	0.0226
25	0.0061	0.160	79	0.040	0.0158
5	0.0021	0.101	52	0.020	0.0083
1	0	0.087	33	0.010	0.0054
0	0	0.087	17	0.005	0.0035
n	310	102	366	361	332

NOTE: Sample sizes vary due to incomplete pollutant data.

Table 2.8

ANNUAL AIR POLLUTION SUMMARY: SEATTLE

	Pollutant			
Measure	Average SO_2	Average COH	Average TSP	Maximum Ozone
Mean	0.0101	0.603	60.83	0.1203
Std. Dev.	0.0025	0.117	15.02	0.0340
Quantiles				
100	0.0143	0.889	123.81	0.17
99	0.0143	0.880	112.76	0.17
95	0.0135	0.863	92.58	0.17
75	0.0122	0.662	66.62	0.16
50	0.0102	0.608	57.89	0.12
25	0.0089	0.532	51.18	0.10
5	0.0052	0.425	41.84	0.07
1	0.0047	0.320	40.13	0.06
0	0.0041	0.280	25.46	0.05
UnitS				
n	9707	7609	9707	9707

NOTE: Sample size for COH is lower due to incomplete COH data in some years.

Table 2.9

ANNUAL AIR POLLUTION SUMMARY: DAYTON

	Pollutant			
Measure	Average SO_2	Average COH	Average TSP	Maximum Ozone
Mean	0.0105	0.189	70.17	0.155
Std. Dev.	0.0039	0.053	13.01	0.033
Quantile				
100	0.0265	0.313	120.26	0.200
99	0.0160	0.284	106.81	0.200
95	0.0154	0.275	97.14	0.200
75	0.0145	0.249	76.71	0.190
50	0.0112	0.170	67.28	0.145
25	0.0073	0.146	60.66	0.127
5	0.0049	0.130	53.65	0.115
1	0.0048	0.127	47.32	0.089
0	0.0005	0.097	32.27	0.170
N	3989	2156	3992	3992

NOTE: Sample sizes vary due to incomplete COH data for some years.

Table 2.10

AVERAGE EXPOSURE OVER THE STUDY: SEATTLE
(n = 2386)

	Pollutant			
Measure	Average SO_2	Average TSP	Average Maximum Daily Ozone	Average COH
Mean	0.0101	61.02	0.0294	0.642
Std. Dev.	0.0018	10.98	0.0013	0.088
Quantiles				
100	0.0123	108.22	0.0328	1.038
99	0.0122	100.17	0.0321	0.821
95	0.0119	83.42	0.0313	0.821
75	0.0115	67.10	0.0301	0.683
50	0.0111	59.45	0.0296	0.637
25	0.0084	53.27	0.0286	0.581
5	0.0067	46.92	0.0272	0.505
1	0.0065	43.85	0.0261	0.456
0	0.0061	42.05	0.0254	0.448

Table 2.11

AVERAGE EXPOSURE OVER THE STUDY: DAYTON
(n = 956)

	Pollutant			
Measure	Average SO_2	Average TSP	Average Maximum Daily Ozone	Average COH
Mean	0.0113	70.47	0.0524	2.341
Std. Dev.	0.0021	11.04	0.0062	0.273
Quantiles				
100	0.0141	102.49	0.0695	2.723
99	0.0141	98.42	0.0659	2.722
95	0.0141	92.97	0.0637	2.711
75	0.0139	76.10	0.0585	2.617
50	0.0110	68.31	0.0501	2.431
25	0.0095	61.28	0.0479	2.025
5	0.0080	57.23	0.0447	1.949
1	0.0079	53.59	0.0437	1.931
0	0.0079	51.86	0.0431	1.855

Unit of Analysis

The unit of analysis is a person-year for the annual analysis, a day for the aggregated and individual time services analysis, and a person for the cumulative health status analysis. See the following sections for further details.

Dependent Variables

In this report, we focus on three sets of health outcomes: use of health services, time lost due to illness, and health status. Here we provide a brief overview of the outcomes. The following sections provide greater detail.

Use of Health Services. We have confined our analysis largely to medical services delivered in an ambulatory setting, excluding outpatient psychotherapy and dental services.[7] In Dayton, we use actual expenditures as a measure of the use of medical services. In Seattle, we use imputed expenditures so that we may include the GHC participants in the analysis. Excluding that group would degrade our precision substantially. For GHC participants, expenditures include both in- and out-of-plan use. Claims filled by participants provide data on the amount and type of fee-for-service use. Abstracted medical records provide data on amount and type of use at GHC (see Goldberg, 1983). We use expenditures where possible rather than visits because expenditures reflect the intensity of the service provided as well as the frequency of use. In the aggregated time series analysis, we use the probability of any use on that day.

Because GHC does not bill its patients for services rendered, there is no readily available, preexisting measure of the aggregate value of procedures provided. Instead, we have imputed a value to procedures provided by GHC based on the California Relative Value Study codes. To preserve comparability, the same imputation has been made for procedures provided in the Seattle fee-for-service sector. See Manning et al. (1984) for further details.

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[7] In Seattle we also exclude drugs and supplies, because we have not developed an imputation algorithm for drugs and supplies obtained at GHC.

Time Lost to Illness. We examine the association of air pollution (daily or annual) and the amount of time that an individual is ill. The HIE provides data on days lost from work, school, or usual activities from the health diary system. For children, we know when a child was ill or took time off from school or merely restricted his or her activities. For adults, we know when a person missed work or restricted activities because of illness. We know the dates involved if a person (e.g., a mother) missed work or school in order to visit a doctor or to care for another family member. In the case of workers, we have data on sick-leave provisions and know if the time off was used for a particular sick-loss day. The HIE data on time lost to illness do not contain any information on symptoms or diagnoses. Therefore, it is impossible to separate sick-loss days related to air pollution from those which are not.

Health Status. We also examine the association between air pollution and changes in health status between the beginning and end of the study. The HIE collected data on subjective assessments of health as well as obtaining objective measures of lung function, cholesterol, and other physiological conditions.

We use the general health index (GHI) described earlier as a single unifying measure of health status. Data for this subjective assessment were collected at entry into the study, and annually thereafter. Because data were collected for everyone, we can examine effects in a general population.

In addition to a general measure, the HIE collected data on the presence and severity of the more common chronic health diseases and problems. In this study, we use seven measures related to cardiopulmonary problems:

(1) The shortness-of-breath scale is a five-point scale ranging from no shortness of breath to severe shortness of breath. The scale is based on responses to four questions on a self-administered questionnaire (Rosenthal et al., 1981).

(2) Chronic bronchitis is based on self-reported information regarding phlegm production, prior diagnosis, and treatment by a physician (Foxman, Lohr, Brook, et al., 1982).

(3) Hay fever is a three-point scale, with separate categories for never had hay fever, had hay fever in the past but not the last 12 months, and had hay fever in the last 12 months (Beck et al., 1981, 1983).

(4) An eight-point scale for asthma for children (aged 5-13) with a value of 0 for those who did not have asthma in the last 12 months, and values 1-7 that are based on the duration of the condition in the past 12 months (Beck et al., 1981, 1983).

(5) A seven-point scale for chest pain, with categories corresponding to the frequency of chest pain from no-chest pain to chest pain almost every day (Rosenthal et al., 1981).

(6) Exercise Pain is a three-point scale with categories for never have chest pain, have pain when walking fast or uphill, and have pain when walking normally on level ground (Rosenthal et al., 1981). This measure differs from the prior one, in that it covers chest pain while exercising. Used only in Dayton because measure (5) was not available for that site.

(7) For individuals over 20, the HIE provides data on lung function from spirometry tests. We use a measure of forced expiratory volume in one second (FEV_1) as a percentage of FEV_1 predicted using age, sex, and height regressions. The coefficients for these prediction equations are from Knudson et al. (1976). This measure is available for a random 60 percent of the sample at enrollment and all of the samples at exit from the HIE.

These measures are fully documented in a series of Rand Reports under the governing title *Conceptualization and Measurement of Physiologic Health for Adults,* in the volumes for congestive heart failure, chronic obstructive airway disease, hay fever, and angina pectoris. For children aged less than four, see *Measurement of Physiologic Health for Children*.

III. INDIVIDUAL ANNUAL APPROACH: METHODS AND RESULTS

METHODS

Our first set of results is based on comparisons of the annual use of outpatient medical services and time lost to illness with an estimated annual exposure to air pollution for each individual. For this analysis, we thus have one observation per person per year, with a maximum of five years of data, i.e., five observations per person. As noted in Sec. II, estimates of each individual's exposure to air pollutants have been made by mapping residence and work locations to the nearest monitoring stations. These estimates are corrected for change of job and residence.

We use two estimation techniques. First, for expenditures on medical services, we use a two-part model. One part is a probit regression model for the probability that a person will use outpatient medical services during the course of the year. The other is a weighted least-squares equation for the logarithm of expenditures for those persons who did use outpatient medical services. (See App. B for further details.) The only difference between the Dayton and the Seattle analyses of expenditures is that we include the costs of drugs and supplies in the Dayton numbers. Other analyses have shown that the demand for these products derives largely from outpatient visits.

For time lost to illness, we use a negative-binomial regression model for the number of days with any school or work loss or restricted activity during the year; this model is similar to the one used in Hausman, Wise, and Ostro, 1983 (see App. B). data are based on biweekly reports of time lost due to illness. We use the negative binomial rather than a Poisson model because the data exhibit overdispersion.

The estimation techniques used here operate on the same assumption used in cross-sectional analyses. That assumption is that the unobserved determinants of the use of ambulatory medical services and of time lost to illness are uncorrelated with the explanatory variables. That is a reasonable assumption to make for the insurance variables, because the insurance coverage was randomly assigned to each family.

Thus, each insurance plan has the same mix of sickly and healthy individuals. However, for this analysis, it is more important that the assumption hold for the air pollution variables, and, unfortunately, it is less likely that it does. Levels of air pollution were not randomly assigned. Families with members who are susceptible to the adverse effects of air pollution may choose to live in less polluted areas. To the extent that the HIE measures of health status measure the true health status with error, the measurement error in health status may be correlated with air pollution exposure, and the estimates may be biased. Put another way, the analytical techniques do not use the repeated observations on each person to purge the estimates of any tendency for more sickly individuals (net of the HIE health status measures) to live in less polluted areas.[1]

RESULTS

Use of Medical Services

Table 3.1 presents the estimated coefficients for the two-part model for annual ambulatory expense in Seattle and Dayton.[2] The data come from claims filed at the time of service use. All pollutants were entered into the model together, so the coefficient for each pollutant represents the partial effect of that pollutant alone and excludes the effects of any correlated pollutants.

None of the effects on ambulatory expenses are significant, except for those of ozone and COH on expenditures per user in Seattle. The ozone effect, however, is in the unexpected direction: Higher levels are associated with lower expenditures on health. As a matter of fact, all effects of increased ozone and TSP appear beneficial. The effects of SO_2 vary. On the whole, the effects on expenditures appear to be more significantly counterintuitive than those on probability of use. Also, air pollution appears to be associated with beneficial results

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[1]We do adjust for intrafamily correlation and intertemporal correlations using a random-effects specification. However, this assumes that the errors are uncorrelated with the explanatory variables. If there is adverse selection into cleaner areas of each city, that assumption does not hold for air pollution.

[2]CO is excluded from Seattle, COH from Dayton, and NO_2 from both because of missing data and confounding with other variables.

Table 3.1

EFFECTS OF AIR POLLUTION ON USE OF AMBULATORY MEDICAL SERVICES
(T-STATISTICS IN PARENTHESES)

	Seattle		Dayton	
Pollutant	Probability of Any Use	(Log) Expenditures per User	Probability of Any Use	(Log) Expenditures per User
SO	+.2008 (+1.35)	-2.6246 (-0.51)	+0.0908 (+0.82)	-0.0503 (-0.67)
CO	---	---	-0.0240 (-0.115)	+0.008 (0.07)
TSP	-0.0014 (-0.55)	-0.0011 (-0.51)	-0.209 (-0.92)	-0.189 (-1.16)
Ozone	-0.1213 (-1.63)	-5.8020 (-2.71)	-0.127 (-0.75)	-1.161 (-1.29)
Coefficient of haze	+0.2036 (+0.77)	+0.1828 (+1.78)	---	---

more consistently in Dayton than in Seattle. But again, almost all of these "effects" are not significantly different from zero. If we combine the results from the two parts of the model, we find no significant effect of air quality on the use of ambulatory medical services in either city.

Because susceptibles may respond differently to air pollution than the rest of the population, we examined them in a separate analysis. We defined an individual as susceptible if he suffered from hay fever, asthma, or shortness of breath. Use of outpatient services in Dayton increased in response to greater levels of air pollution, not to an extent that could be considered significant ($\chi^2(8) = 12.14$), but to a much higher degree than one would expect at random ($p = .5$). In large part, this result is due to a greater likelihood of use of services by

susceptibles as CO and TSP levels increase (t = 1.55 and 1.98, respectively). In Seattle, we had a larger sample of susceptibles. The analysis there showed mixed results, but there were significant increases in total expenses with falling ozone (t = -2.44) and TSP (t = -1.69).

Time Lost to Illness

Table 3.2 gives elasticities of air pollution with respect to time lost to illness for Seattle and Dayton.[3] The number of days lost to illness was not significantly related to annual air pollution for most pollutants in both cities. The exceptions were TSP in Seattle and ozone in Dayton, both of which are associated with decreasing time losses to illness. In fact, almost all the insignificant effects were also in the "wrong" direction.

Table 3.2

ELASTICITIES OF AIR POLLUTION WITH RESPECT TO TIME LOST TO ILLNESS

	Seattle		Dayton	
Pollutant	Coefficient	(t-Statistic)	Coefficient	(t-Statistic)
TSP	-0.643	-2.16	-0.352	-0.81
S	-0.024	-0.73	+0.034	+0.19
Ozone	-0.034	-0.95	-0.598	-1.93
CO_2	--	--	-0.262	-0.63
COH	-0.510	-1.50	--	--

[3]The elasticities indicate the proportional change in time lost for a doubling of air pollution. For example, a 100 percent increase in ozone in Seattle would result in a 3.4 percent decrease in time lost to illness.

DISCUSSION

We find that the use of ambulatory medical services and time lost to illness generally do not increase from year to year or from place to place as air pollution levels increase. In fact, we observe that ozone is significantly, counterintuitively associated with expenditures for medical services in Seattle and with time lost to illness in Dayton. It is thus possible that, at levels encountered in those cities, increases in ozone are associated with beneficial effects on health. For example, years with high ozone may be warmer, sunnier years with less sickness. The ozone variable may be picking up these omitted weather variables. The inclusion of a dummy variable for year should reduce this bias.

There are several possible explanations for the general lack of significant findings:

- The absence of a true effect.
- The sorting out of individuals across pollution zones.
- The use of annual rather than daily data.
- Omitted weather variation from year to year.

The second and third of these might be ruled out by changing the analytical approach.

Geographical Sorting

Although we have used data from a randomized trial, this study of air pollution is observational, because we have not randomized individuals to differing levels of air pollution. If people with respiratory problems are more likely than healthy people to live in areas with better air quality, then the estimates of the adverse effects of air pollution could be biased downward to the point that the coefficients have the wrong sign.

To investigate further the potential for geographical sorting, we reestimated the effect of air pollution with a fixed-effects model for use of ambulatory medical services and for time lost to illness; there is a fixed effect for each individual. In each case, we regressed the annual ambulatory expenses, stated as a deviation from each person's

mean, on the levels of the four pollutants, each stated as a deviation from each person's mean. By taking each person's independent and dependent variables as deviations from his own mean, we allow each person to act as his own control; see Maddala (1971). In effect, we changed the individual annual analysis from a cross-sectional to a panel study. The results for expenditures in both cities and for time lost to illness in Seattle were estimated by ordinary least squares.[4] We did not attempt to estimate the parameters of a fixed-effect version of the two-part model.

Applying the fixed-effects model in Seattle reduced the beneficial effect of ozone on total ambulatory expenditures to insignificance ($t = -0.75$) and changed the beneficial effects of the other pollutants to adverse effects, though still insignificant ones; the overall test statistic was $F(4,6041) = 0.47$. Applying the fixed-effects model to expenditures in Dayton yielded insignificant and perverse effects for TSP and SO_2 (overall $F(4,3920) = 4.96$).

Using Annual Data

The lack of significant results may be attributable to the inappropriate aggregation of an individual's responses over time. Colds and other sicknesses may occur in the winter, while air pollution is highest in the summer or spring. Using air pollution values based on regional variation in spring air quality to explain behavior driven by winter-versus-spring differences is inappropriate. For instance, in Dayton, the period of highest ozone levels occurred in July 1976, but most of the use of health services in that year, especially for respiratory problems, occurred during the late fall through early spring. Thus, the highest ozone reading could not have caused the greater part of the use of services for that year. In addition, the use of annual averages for air pollution and annual expenditures largely ignores the importance of short-term fluctuations in air quality and illness. Annual values are less variable than monthly or daily values. The less variable the independent measure, the less precision for its coefficient.

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[4]We did not correct the standard errors for the negative correlations among observations induced by taking observations as deviations from individual means.

We have used two other approaches to estimate the adverse effects of air pollution on use of medical services and time lost to illness. Both take fuller advantage of the information in the time series of daily (rather than annual) values, and the second controls for geographical sorting. The next two sections describe these approaches, their limitations, and their results.

IV. AGGREGATED DAY-TO-DAY APPROACH

To take full advantage of the available day-by-day information on variations in health outcomes and air pollution, we have used two related methods. The first of these aggregates the responses across individuals so that we have one observation for each day. This method is not affected by individuals' sorting themselves out geographically on the basis of their susceptibility to pollution. We do not compare the responses of geographically separate individuals with each other, because we look at a fixed population. The second method examines each individual's daily time series separately, using the Whittemore-Korn technique; the latter approach is described in the next section.

DATA AND METHODS

The sample for the analysis of time lost to illness consists of all individuals who were assigned to file health diaries with HIE. Although the diaries ran into the third year in Seattle and the fourth year in Dayton, we eliminated the partial data from those years to ensure against seasonal imbalance in the analysis. Also, the health diary data for the first year in Dayton were not available from the HIE. Thus, we used the data from HIE years 2 and 3 in Dayton and 1 and 2 in Seattle. These two pairs of years happened to match each other very closely on the calendar.

To allow comparability with the time-lost results, the sample for analyzing the use of medical services consists of all HIE participants present in Seattle for years 1 and 2 and in Dayton for years 2 and 3.

For each day, we tallied the number of individuals in this subsample and the number reporting any physician visits, days in a hospital, sick loss, work loss, or restricted activity. We used a maximum-likelihood logistic regression model to estimate the association of each day's air quality level with the proportion of the population reporting each health outcome (any visit, any hospitalization, any sick loss, or any work loss)[1].

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[1]Because of the staggered enrollment dates in the experiment, the population at risk varied by month. The results are weighted to reflect the differences in sample size.

The analyses we report here are those with lag times between pollutant level and health effect that were found to capture most of the effect without losing information because of gaps in the pollution time series. For pollutants other than ozone, each day's health outcomes are estimated as a function of the logarithms of that day's and the preceding two days' average pollutant concentrations. Estimation of ozone is the same except that daily maximums are used instead of daily averages.

The independent variables include air quality variables, along with indicator variables for day of the week and month of the year. We included the daily and monthly variables to avoid confounding true air pollution effects with true daily and seasonal effects. Air quality varies markedly by day of the week and season of the year, often in the same direction as daily and seasonal health effects. For example, air pollution levels are lower on weekends, and so are use of services and time lost to illness. Part of the lower use of services is due to reduced availability of physician services (except for emergency departments) on weekends. Part of the lower time lost to illness is due to the fact that schools are closed and few people work weekends. As a result of including monthly and daily dummies, our estimation procedure controls for variation between days of the week and months of the year in estimating the effects of air quality.

Our data include measures of air pollution levels taken at a single point in each city.[2] In both cities, we analyze for SO_2 and ozone. TSP was included in the Dayton analysis but not in Seattle, where TSP was measured every sixth day; including TSP in Seattle would have reduced substantially the number of observations. In Seattle, COH was used as a proxy for TSP. NO_2 and CO were also included only in the Dayton analysis. Because of gaps in the daily data for individual pollutants and lack of overlap among the pollutant time series, we focus on each

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[2] Because we have only one data point for each day, we could use only one value of air pollution level for each pollutant. We used values from a centrally located, usually downtown monitor. See the "Exposure" discussion in Sec. II.

pollutant taken one at a time. Using the intersection of the six time series would have dramatically reduced our precision. Hence, the effect reported here is the marginal rather than the partial effect, because we have not controlled for other pollutants.

RESULTS

Use of Ambulatory Services

As shown in Table 4.1, only ozone has significant effects on use of ambulatory health services in Seattle (when the other two pollutants are included in the model). The signs for SO_2 and Ozone are as expected--increases in air pollution are associated with higher probability of visiting a physician. However, the magnitudes of these adverse effects of air pollution are small. For SO_2, a 100-percent degradation in air quality is associated with a 0.5-percent increase in the proportion of the population visiting a doctor. For ozone, there would be a 4.3-percent increase. A 100-percent increase in COH is associated with a 0.9-percent decrease in visits.

In Dayton, the aggregated day-to-day analysis shows a significant association at the 10 percent level or better between the likelihood of visiting a medical provider and the level of NO_2 and SO_2, and at better

Table 4.1

RESPONSE TO AIR QUALITY: EFFECTS OF A 100-PERCENT INCREASE IN AIR POLLUTION ON DAILY PROBABILITY OF A VISIT IN SEATTLE

Pollutant	% Before 100% Change	% After 100% Change	Percentage Change [a]	$x^2(3)$ for Air Pollution Parameters
COH	1.486	1.473	-0.9	38.11[b]
SO	1.475	1.482	+0.5	4.00
Ozone	1.481	1.544	+4.3	7.74[c]

NOTE: Percentage in first column differs by pollutant because of different gaps in time series.
[a] 100% x [(col 2 - col 1)/col 1].
[b] Results significant at 1 percent level.
[c] Results significant at 10 percent level.

than the 1-percent level for ozone, and TSP (see Table 4.2). However, in contrast to Seattle, the signs are not as expected for these pollutants: Increases in these pollutants are associated with a lower rather than a higher probability of a visit. Only CO exhibits an adverse effect of increased air pollution. As in Seattle, the magnitudes of all effects of air pollution are quite small, with a 100-percent degradation in air quality leading to less than a four-percent change in the number of individuals seeking medical care.

Hospital Days

The effects of air pollutant concentrations on the likelihood of being in the hospital was generally insignificant in both cities. This lack of significance is largely attributable to the rareness of hospitalization. The one exception to this pattern was TSP in Dayton ($\chi^2(3) = 21.28$). In this case, a 100-percent degradation in air quality was associated with a 33-percent increase in use of services. The

Table 4.2

RESPONSE TO AIR QUALITY: EFFECTS OF A 100-PERCENT INCREASE ON AIR POLLUTION ON DAILY PROBABILITY OF A VISIT IN DAYTON

	Proportion Visiting Physician			
Pollutant	% Before 100% Change	% After 100% Change	% Change[a]	$\chi^2(3)$ for Air Pollution Parameters
NO_2	1.251	1.223	-2.2	7.57[b]
SO_2	1.203	1.179	-2.0	7.20[b]
CO	1.255	1.289	+2.7	28.19[c]
TSP	1.236	1.189	-3.6	23.06[c]
Ozone	1.239	1.194	-3.8	12.96[c]

NOTE: Percentage in first column differs by pollutant because of different gaps in time series.
[a] 100% x [(col 2 - col 1)/col 1].
[b] Results significant at 10 percent level.
[c] Results significant at 1 percent level.

magnitude of this effect is implausibly large. In Dayton, most of the other pollutants--SO_2, CO, and ozone--did have effects in the expected direction, i.e., more pollution was associated with higher use. In Seattle, however, SO_2 and ozone exhibited beneficial but statistically insignificant effects.

Time Lost Due to Illness

Tables 4.3 and 4.4 show the effects on time lost to illness associated with each of the pollutants in Seattle and Dayton. In Seattle, lower levels of SO_2 and ozone were significantly associated with higher levels of time lost to illness, but the magnitude of the estimated effect was small. COH was not associated with significant changes in time lost due to illness. A doubling of the level of ozone would be associated with a fall in this proportion by about 10 percent. This association of air quality and time lost to illness is largely the result of sick-loss time, because the results for work loss are even less significant than one would expect from random variation.

A larger effect was found in Dayton, where a 100-percent increase in ozone concentration was associated with a 13-percent increase in time lost to illness ($\chi^2(3) = 26.15$). The effects of NO_2 and SO_2 in Dayton

Table 4.3

EFFECTS OF A 100-PERCENT INCREASE IN AIR POLLUTION ON DAILY PROBABILITY OF ANY TIME LOST TO ILLNESS IN SEATTLE

Pollutant	% Before 100% Change	% After 100% Change	Percentage Change [a]	$\chi^2(3)$ for Air Pollution Parameters
COH	2.826	2.782	-1.6	2.25
SO_2	2.668	2.589	-3.0	15.09[b]
Ozone	2.679	2.412	-10.0	12.97[c]

[a] Includes sick and work loss.
[b] 100% x [(col 2 - col 1)/col 1].
[c] Significant at 1 percent level.

Table 4.4

EFFECTS OF 100-PERCENT INCREASE IN AIR POLLUTION ON DAILY PROBABILITY OF ANY TIME LOST TO ILLNESS IN DAYTON

Pollutant	Percentage Ill [a]			$\chi^2(3)$ for Air Pollution Parameters
	% Before 100% Change	% After 100% Change	% Change[b]	
NO_2	3.042	2.902	-4.6	12.80[c]
SO_2	2.294	2.805	-4.1	15.98[c]
CO	3.095	3.031	-2.1	1.56
TSP	3.070	3.033	-1.2	1.21
Ozone	3.038	3.423	+12.7	26.15[c]

[a] Includes sick and work loss.
[b] 100% x [(col 2 - col 1)/col 1].
[c] Significant at 1 percent level.

were also significant but of the wrong sign. Both CO and TSP had insignificant effects.

DISCUSSION

The aggregated day-to-day approach displays a mixed set of associations between air quality and our health outcomes. In Seattle, increases in ozone concentration were associated with a higher probability of using ambulatory medical services but lower probability of being sick. SO_2 was negatively associated with seeing a physician, but positively associated with time lost to illness. In Dayton, higher CO has a significant adverse effect on the likelihood of visiting a medical provider, while higher TSP has an adverse effect on being hospitalized. Higher levels of ozone were associated with higher levels of time lost due to illness.

However, the results are only partially in agreement with our expectations. We also found that higher levels of NO_2, SO_2, TSP, and ozone were significantly associated with lower likelihood of visiting a provider. Higher levels of NO_2 and SO_2 were associated with lower levels of time lost to illness.

A priori, we expected that time lost to illness would be more responsive to air quality than use of services, for four reasons:

(1) One can suffer ill effects and report them as restricted-activity days without incurring the opportunity costs of not attending school or going to work, and without paying the price of a visit to see a physician.

(2) It may take some time to see a physician because of delays to appointment for nonemergency care. During that period, the adverse effects of air pollution may disappear.

(3) Individuals suffering from cardiopulmonary problems may be able to treat themselves for minor adverse effects when sick, relying on a physician for treatment of only the more serious episodes.

(4) Both cities have only moderate levels of air pollution. As a result, we might expect few episodes of illness that are severe enough to be presented to a physician.

Here, we have disaggregated the data to a behaviorally more meaningful time frame. Why do we still obtain this mixed set of results? Again, either there is no effect large enough to be detected with these data, given the pollution levels in Seattle and Dayton, or the results are biased by our methods. For example, the omission of meteorological variables could have led to an omitted variables bias; during Phase II of this project, we will add such variables to the list of explanatory variables.

Also with this method, we aggregated across individuals to avoid the potential bias that would occur if sicker individuals moved to cleaner areas; in principle, the population acts as its own control. To do that required using a single source of air quality data, which came

from a downtown monitor. Thus, the air quality data measure pollution with error for much of the sample, especially those living in cleaner areas. This measurement error could bias the estimated coefficients toward zero.

In the next section, we use a technique developed by Whittemore and Korn to avoid the statistical problems in both the individual annual and in the aggregated day-to-day approaches.

V. INDIVIDUAL DAY-TO-DAY APPROACH: METHODS AND RESULTS

In this section we discuss the individual day-to-day analysis based on the approach proposed in Whittemore and Korn (1980). This approach is carried out in two stages. First, we estimate each individual's daily health outcome as a function of his or her daily aerometric exposures so as to assess the individual-specific response. Each individual serves as his or her own control in this analysis. Then, we pool the individual-specific responses and carry out a secondary analysis, the meta-analysis, in which we assess the overall response to aerometric attributes in the population. This second stage allows us to answer three key questions: First, do the people in the population on the average fall ill more often on polluted days than on clean days? Second, do individuals in the population respond the same or differently to air pollution? Third, if they respond differently, are their responses related to their known characteristics? (For example, are children more sensitive to air pollution than adults?)

We begin by describing the Whittemore-Korn model and its application to the HIE data. We then show how we derived the sample we analyzed. Finally, we present the results of the second-stage analysis for the full sample and for sickly and healthy subsamples. Appendix C presents further results of the first-stage analysis, along with comparisons of other subsamples.

THE WHITTEMORE-KORN MODEL

Synopsis

In the Whittemore-Korn model, the unit of analysis is usually taken as a person-day. (It is possible to consider other time units such as hours or weeks, but the twenty-four-hour period is usually the most convenient to work with. The HIE data are collected in daily units.) For each individual in the target population, say, the i^{th} person, and for each day in the study period, say, the t^{th} day, the model specifies a logistic regression model for the daily probability of the person's being sick:

$$\text{logit}(p_{it}) = \beta_{i0} + \Sigma_j \, x_{ijt} * \beta_{ij} \, , \qquad (1)$$

where p_{it} is the i^{th} person's probability to be sick on the t^{th} day; x_{ijt} is the level of the j^{th} explanatory variable (e.g., aerometric value) for the i^{th} individual on the t^{th} day; β_{ij} is the i^{th} person's response to the j^{th} explanatory variable; the intercept for the i^{th} person, β_{i0}, is the logit of the probability of the i^{th} person's being sick on a day when the levels of all explanatory variables are zero.

We use a random-effects (variance components) model to specify a distribution of individual responses, β_{ij}. The model specifies a meta-distribution for the individual responses as follows:

$$\beta_{ij} \sim N(\gamma_j, \tau_j^2), \qquad (2)$$

where γ_j is the average response to the j^{th} explanatory variable. If all individuals have the same response to the j^{th} explanatory variable, all β_{ij} are identical and equal γ_j. If individuals differ in their responses to the j^{th} explanatory variable, the β_{ij}'s are different from γ_j; the differences $\beta_{ij} - \gamma_j$ are the between-individual differences. The average magnitude of the between-individual differences (in the sense of L^2 distance) is given by τ_j. (If the individuals have identical responses, the corresponding parameter τ is zero.) The model (2) given above is usually known as the random-effects or (variance components) model. We will test separately the hypotheses that $\gamma_j = 0$ and $\tau_j = 0$; the two hypotheses together are equivalent to the global null hypothesis that $\beta_{ij} = 0$.

When there are between-individual differences, it might be desirable to relate them to observed characteristics of the individuals. For example, one might be interested to know whether the individual's response to air pollution is related to smoking, i.e., whether a smoker might be more sensitive to air pollution than a nonsmoker. We are currently only capable of carrying out this analysis for dichotomous characteristics. For example, we can compare smokers with nonsmokers, but we cannot relate the individual responses to a continuous specification for smoking, such as the number of cigarettes smoked per day.

For a dichotomous characteristic, we can partition the population into two subpopulations, one corresponding to each level of the characteristic. We then apply a random effects model similar to model (2) to each subpopulation, and compare the parameters γ and τ for the two subpopulations. If the characteristic being studied is related to the individual responses, the average response γ for the two subpopulations should differ. For example, if only smokers were sensitive to air pollution, the average response γ for smokers would be nonzero, while the average response γ for the nonsmokers would be zero. If the relationship between the individual responses and the characteristic being studied explains all of the between-individual differences, the parameters τ would be zero for both subpopulations.

The main advantage of the Whittemore-Korn model is that each individual serves as his or her own control, which avoids the confounding problems with the cross-sectional methods used in Sec. V. Furthermore, since the model provides estimates of each individual's responses, it allows great flexibility in the meta-analysis on differential susceptibility. We can contrast any two subpopulations defined in terms of any observed dichotomous characteristic for the individuals. Thus, this model improves on the average-response specification in the aggregated daily approach.

The Whittemore-Korn model also allows us to calculate each person's response to a local estimate of the pollution he or she is exposed to. Again, this is an improvement over the aggregated day-to-day approach, which uses one daily pollution value for everyone, introducing measurement error into the analysis.

One limitation of the model is that it applies only to short-term effects. Another limitation is that, empirically, the model cannot be applied to people who are healthy almost all the time or to people who are sick almost all the time. The logistic regression model usually is not estimable (identifiable) for those people. For example, consider a person who is healthy all the time. The empirical probability is zero that the person will be sick on either a polluted day or a clean day. The logit of the empirical probability zero is minus infinity. The effect of air pollution for this person is therefore (minus infinity) - (minus infinity), which is indeterminate.

Toward the end of this section, we will discuss how we restrict our analysis to those people with more than a few sick days and more than a few healthy days over a period of up to two years, and discuss the implications of this restriction.

Application

For the health outcome in this analysis, we use a combination of restricted activities, school loss, and work loss as given in the biweekly health diary. For each person in the sample on each day in the study period, if the person reported either a day with restricted activities due to health reasons, school loss, or work loss, the day is treated as a sick day; otherwise the day is treated as a healthy day.

Because of limitations in the data, we need to make some revisions in the Whittemore-Korn model in order to apply it appropriately. One of the important findings in Whittemore-Korn (1980) is the autocorrelation between daily disease statuses. For the same person, the day after a sick day is more likely to be a sick day than a day after a healthy day, everything else being the same. For most people in our sample, there are too few days-after-a-sick-day to allow reasonable estimation of this effect; for example, for a person with ten sick days we have only ten opportunities to estimate the probability of being sick the day after a sick day. Therefore, for most of our analysis we delete all days after a sick day and focus on the estimation for days after a healthy day. In other words, we only estimate the probability for the transition from the healthy status into a sick episode; a sick episode is dated to the first day of a series of consecutive sick days. We do, however, take up separately the questions of the length of sick episodes and how the length of the episodes responds to air quality.

SAMPLE AND DATA

Sample and Health Outcome

The maximum number of people that could be used in this analysis is 2901--the number of HIE participants were assigned to file health reports while in the Seattle metropolitan area.[1] On the average we have

[1]The individual day-to-day approach has not yet been applied to the Dayton sample. That will be done during the second phase of the research.

630 daily reports per person. The maximum number of days possible for each person is 731. However, some of the participants moved out of the Seattle area before the end of the health report study period and some failed to file all required health reports.

The HIE participants averaged 4.34 sick episodes per person. The distribution of sick episodes was fairly skewed. More than ten percent of the HIE participants had no sick episodes. The median value was 3 episodes. The maximum value was 77.

As discussed earlier, the logistic regression model is usually not estimable when the number of sick episodes is too low, so we need to restrict the analysis to people with more than a few sick episodes. We have chosen to include only those people with more than the median number of episodes (3). This leaves us with 1249 persons. However, those people report 10,582 sick episodes, which is more than 80 percent of the total number of sick episodes. Therefore, in terms of the number of sick episodes, the loss due to this restriction is minor.

The restriction to people with more than a few sick episodes can be viewed as an optimal strategy to make the best use of analytic resources. The people with a few episodes contribute less information than the people with one episode. (As discussed above, the response of a person who is healthy throughout the study period is undefined, and thus contributes no information at all.) In the next subsection, we examine empirically the implications of this strategy.

The restricted sample of 1249 persons with more than three episodes yields an average of 684 daily health reports per person. That average exceeds that for the whole sample because people with fewer health reports are more likely to have three or fewer episodes and therefore be deleted according to the restriction rule.

Not all person-days with health reports can be used in the analysis. As discussed above, we use a sick episode instead of a sick day as the health outcome, so we have to delete all days immediately following a sick day. Furthermore, some days cannot be used in the analysis because of missing air pollution data. With those deletions, we have an average of 425 days per person.

There are a few people with very few days available for analysis. We choose to restrict to people with at least 100 days available for analysis. This restriction deletes 11 people and leaves us with 1238 persons in the final analysis sample. They average 429 days per person and 8.5 sick episodes each.

EXPLANATORY VARIABLES

For this analysis, we use three groups of explanatory variables: air pollution measures, meteorological measures, and calendar effects.

The air pollution data are from SAROAD and the Washington State Department of Ecology. The following daily air pollution measures are used: daily average of sulphur dioxide (SO_2), daily average of coefficient of haze (COH), daily average of TSP, daily maximum hourly average of ozone, and daily maximum hourly average of nitrogen dioxide (NO_2). Air pollution at a person's residence or work location is assumed to be the same as that at the nearest monitoring site (see the discussion of exposure in Sec. II).

Values of the various air pollution measures are distributed over days in a somewhat skewed fashion. The statistical measure of skewness ranges between one and two. Had the skewness been larger, the results of the analysis might have been dominated by a few outliers and would thus have been unstable. In such situations, it is necessary to transform the skewed variable to get more stable results. Given the moderate amount of skewness, we choose not to apply transformations.

We also use daily minimum temperature and daily precipitation data from the National Weather Service. Because meteorological measures are available from only one weather station, those values are assumed to apply to all residences and work locations.

The distribution of precipitation is very skewed, because more than half of the days have no precipitation. If the effect of precipitation were of primary interest in this study, one might specify the effects of precipitation as two entries in the logistic regression--one an indicator variable for a day with precipitation, the other the amount of precipitation (or a transformed amount). However, since the effect of precipitation is not of primary interest in this study, we use a simple linear specification.

In addition to the aerometric data, we use two calendar-related covariates to control for possible confounding effects. The first is an indicator variable for weekday versus weekend; this is a possible confounding factor because the levels of air pollution are usually higher on weekdays than on weekends, and people are more likely to report sickness during weekdays than during weekends. The second is an indicator variable for the first week of each two-week health report period. Because we use a self-administered diary that might not have been filled out daily, the accuracy of reporting in the earlier part, say, the first week, might be different from that in the latter part, say, the second week.

The aerometric attributes are closely interrelated, e.g., ozone is generated from a photochemical process and usually has low or null levels on rainy days. Therefore, we expected substantial correlation among our explanatory variables. Explanatory variables that are highly correlated might be nearly collinear, i.e., one of the explanatory variables might be nearly a linear combination of some of the others. In such cases, the logistic regression model might not be estimable or might be ill-conditioned, and the estimated results would be unstable. Most of the pollution measures are indeed significantly correlated, but the magnitudes of the simple and multiple correlations are all moderate; the largest ones are under 0.6 (see Tables C.1, C.2, and C.3). Thus, collinearity among the explanatory variables is not a major concern.

GENERAL RESULTS

On applying the random-effects model to estimate the average responses and standard deviations of individual differences, we obtain the results given in Table 5.1. For four of the pollution measures (SO_2, COH, TSP, and NO_2), the average effect of pollution is positive, indicating that there is a higher probability of having a sick episode on a polluted day than on a clean day. For two of the four (SO_2, NO_2), the effect is statistically significant at the one percent level. The average effect for ozone is negative and statistically significant, as is the effect for minimum temperature, which is negative and

Table 5.1

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE FINAL ANALYSIS SAMPLE (N=1238):
AVERAGE RESPONSES

Aerometric Attribute	Estimated Coefficient	z Statistic
SO_2 (ppm)	7.94	6.12
COH	0.0150	0.38
TSP ($\mu g/m^3$)	0.00061	1.50
Ozone (ppm)	-3.46	-4.46
NO_2 (ppm)	1.33	3.18
Minimum temperature (F)	-0.0132	-8.12
Precipitation (in.)	0.684	12.8

statistically significant. The average effect for precipitation is positive and statistically significant.

Table 5.2 summarizes the results for the average effects based on the random-effects model and on another approach to correcting the analysis for the instability of outliers--analyzing individual z statistics (see App. C). While the two sets of results are not

Table 5.2

SIGNIFICANCE OF THE AVERAGE RESPONSES

Aerometric Attribute	Random-Effects Model	Individual z Statistics
SO_2	+ *	-
COH	+	- *
TSP	+	- *
Ozone	- *	- *
NO_2	+ *	-
Minimum temperature	- *	- *
Precipitation	+ *	+

NOTE: +: average response is positive;
-: average response is negative;
*: effect is statistically significant at the 5-percent level.

identical, they do not contradict each other: There are no instances in which one approach gives a statistically significant positive result and the other method gives a statistically significant negative result.

The two approaches both indicate that ozone has a significant association with lower probabilities of sick episodes. The two approaches also agree that higher minimum temperature is significantly associated with lower probabilities of sick episodes, and that precipitation might be associated with a higher probability of sick episodes.

The random-effects model indicates that SO_2 has a significant association with higher probabilities of sick episodes, which is not corroborated in the individual z statistic approach. If we accept the association estimated from the random-effects model as real, the magnitude of the association can be interpreted as follows. The meta-analysis estimates that an increase of one ppm SO_2 is associated with an increase of 7.94 logit units in the probability of a sick episode. If the average SO_2 level in downtown Seattle triples from its present 0.01 ppm to 0.03 ppm, the primary federal standard level for the annual average, the probability that the average person would experience a sick episode would increase by 0.16 logit units. For most people the probability of having a sick episode is small on any day, so the logit scale is very well approximated by the logarithm scale. An increase of 0.16 in the logarithm of the probability of having a sick episode is equivalent to multiplying the probability of a sick episode by 1.17. For the final analysis sample on the average, this is equivalent to a increase from 0.020 sick episodes per person-day to 0.023.

Equivalently, a 10-percent increase in SO_2 would cause sick episodes per person-day to go from 0.02 to 0.0202. The effects for other pollutants are smaller. For COH, sick episodes would increase to 0.02002. For TSP, the same increase would raise sick episodes to 0.0201. For ozone, sick episodes would fall to 0.0188. For NO_2, sick episodes would increase to 0.0201.

As discussed above, an advantage of the random-effects model is that it allows estimation of the standard deviation for between-individual differences. These are given as the tau parameters in Table 5.3. For three of the aerometric attributes, COH, NO_2, and minimum

Table 5.3

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE FINAL ANALYSIS SAMPLE (N=1238):
BETWEEN-INDIVIDUAL DIFFERENCES

Aerometric Attribute	Tau	z Statistic
SO_2 (ppm)	0.0	0.00
COH	0.348	1.98
TSP ($\mu g/m^3$)	0.00156	0.41
Ozone (ppm)	5.54	1.38
NO_2 (ppm)	5.53	4.24
Minimum temperature (F)	0.0193	3.28
Precipitation (in.)	0.0	0.00

temperature, there is a statistically significant between-individual difference, i.e., the individuals in our sample do not respond similarly to these aerometric attributes. For two of the three, the tau parameter is much larger than the average responses given in Table 5.1. Therefore, a significant fraction of the people might have a response in the opposite direction from the one given by the average response. For example, the tau parameter for NO_2 is 5.53, while the average response is 1.33 (both given in terms of logit per ppm NO_2.) If we take those estimates as true values, we calculate that the probability of a negative response (opposite the direction given by the average response) for any given individual is 0.405. Thus, about 40 percent of the people have a negative association between NO_2 and sick episode, while about 60 percent have a positive association.

We also found a strong negative association between the coefficients and their standard errors (see Figs. C.8-C.14 in App. C). There are two possible explanations for this unexpected phenomenon. First, there may be a negative association between the true individual coefficients and their true standard deviations. We find this possibility unlikely because of the consistency of the negative associations across the different pollutant and aerometric variables. Second, the observed negative associations may be a statistical

artifact. We conjecture that the small sample bias of the maximum likelihood estimates of the logistic regression coefficients may be the cause. In particular, individuals with smaller numbers of sick episodes may tend to have larger (negative) biases. Since these same individuals will tend to have larger standard errors of their coefficients, this could lead to the observed negative associations in Figs. C.8-C.14.

Fortunately, we are in the position to be able to test this conjecture by performing some computer simulations in the second phase of this project. By using the observed independent variables and simulating random sick episodes based on the logistic regression model, we will see if there is a negative association between the simulated estimated coefficients and their standard error. Since in this simulation we will know that there is no association between the true individual coefficients and their standard deviations, we will determine if the small sample bias of the estimated coefficients is the cause of the negative association.

The verification of this type of small sample bias would have important implications for the present analyses and for other studies using the Whittemore-Korn model. First, it would suggest that the down-weighting of the coefficients with the larger standard errors is appropriate since they are likely to be more biased. If this were the case, then the random-effects analysis would be more appropriate than the z analysis. Secondly, it would suggest improvements in the methods of analysis using the Whittemore-Korn model to reduce the small sample bias.

COMPARISON OF SICKLY AND LESS SICKLY SUBPOPULATIONS

In this subsection, we contrast the responses to air pollution on the part of sickly people with those of less sickly people. The first criterion we use for sickliness is the number of sick episodes, rather than the presence or severity of disease.

We compare the responses for those with 7 or more sick episodes (the sick subpopulation, containing 655 individuals, 53 percent of the final analysis sample) with those with 4 to 6 sick episodes (the less sickly subpopulation, containing 583 individuals, 47 percent of the final analysis sample.)

The average responses for the two subpopulations are given in Tables 5.4 and 5.5. The column "z for the contrast" in Table 5.5 gives the z statistics for the difference between the average responses in the two subpopulations.

Table 5.4

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE SICK SUBPOPULATION (N=655):
AVERAGE RESPONSES

Aerometric Attribute	Estimated Coefficient	z for the Attribute	Efficiency
SO_2 (ppm)	5.12	3.33	0.711
COH	0.00811	0.18	0.722
TSP ($\mu g/m^3$)	0.00060	1.24	0.708
Ozone (ppm)	-3.60	-3.91	0.711
NO_2 (ppm)	1.11	2.23	0.704
Minimum temperature (F)	-0.0125	-6.43	0.697
Precipitation (in.)	0.576	9.37	0.751

Table 5.5

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE LESS SICKLY SUBPOPULATION (N=583):
AVERAGE RESPONSES

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	14.9	6.07	3.39
COH	0.0435	0.58	0.40
TSP ($\mu g/m^3$)	0.00043	0.54	-0.18
Ozone (ppm)	-3.45	-2.34	0.09
NO_2 (ppm)	2.20	2.89	1.21
Minimum temperature (F)	-0.0151	-5.08	-0.73
Precipitation (in.)	1.01	9.46	3.54

The only two aerometric attributes with significantly different responses are SO_2 and precipitation. The less sickly subpopulation is more responsive to SO_2--almost three times more; it is also more responsive to precipitation--almost twice more. This result is surprising because in the random-effects model for the final analysis sample as a whole (Table 5.3), we found no between-individual differences for SO_2 or precipitation. As discussed above, we expected to detect differential susceptibility only for those aerometric attributes with significant between-individual differences. For the final analysis sample, we found significant between-individual differences only for NO_2 and COH. We therefore expected that those would be the two potential candidates for subpopulation comparisons. For the other attributes, the random-effects model for the final analysis sample indicated that all individuals had the same response, so we did not expect to see any difference between subpopulations. It is especially surprising that the subpopulation difference is statistically significant only in SO_2 and precipitation, the only two aerometric attributes with zero estimates for tau in the final analysis sample. These two attributes would have been the least likely to have any between-individual differences. We do not have a good explanation for this result.

The discrepancy in the response of the less sickly and sickly to SO_2 and precipitation implies that there are some important limitations for the generalizability of the results obtained through the Whittemore-Korn method. As discussed above, we have chosen to include in the final analysis sample only those individuals with more than three sick episodes. We therefore have to question whether our results are generalizable to the "very healthy" people with three or fewer sick episodes. Where the comparisons between sickly and healthy people result in null findings, we might infer that the "very healthy" people might have the same response. However, the positive SO_2 finding indicates that people's responses to SO_2 are associated with their health. Thus, the response of the "very healthy" people to SO_2 cannot be inferred from our analysis.

If we regard the average responses given in Table 5.4 for the sickly subpopulation and those given in Table 5.1 for the final analysis sample as two unbiased sets of estimates of the same unknown true parameters, then it is of interest to know how much more information we gain from the inclusion of the less sickly subpopulation. In other words, because the estimates in Table 5.1 are based on 1.89 times as many people as the estimates in Table 5.4, do we gain almost twice the information? We would expect not, because the precision of the coefficients of the less sickly people should be less than that of the coefficients of the people with more sick episodes. The results are given as the "efficiency" column in Table 5.4. The efficiency is based on the precision of the estimated average responses. For each aerometric attribute, the efficiency is ratio of the variance of the average coefficient in Table 5.1 to the average coefficient in Table 5.4. For all aerometric attributes, the efficiency of the sickly subpopulation is about 70 percent. In other words, the near doubling of the number of individuals from the 655 sickly persons to the 1,238 in the final analysis sample, owing to the inclusion of the 583 less sickly persons, only increases the effective sample size by about 43 percent (i.e., 70 must be multiplied by 1.43 to get to 100). In other words, the amount of information for each healthy person is less than half that for each sickly person.

It appears reasonable to conclude that the more sick episodes a person has, the more information we can expect the person to contribute. This confirms our earlier conjecture that restricting the analysis to people with more than a few sick episodes is an optimal strategy to make the best use of analytic resources.

Tables 5.6 and 5.7 give the between-individual differences within each of the two subpopulations. In terms of estimating the tau parameter, the standard deviation of between-individual differences, the sickly subpopulation has efficiencies of about 80 percent. Thus, for estimating tau, the near doubling of sample size with the inclusion of the less sickly subpopulation increases the effective sample size by only about 25 percent. In other words, each sickly individual contributes

Table 5.6

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE SICK SUBPOPULATION (N=655):
BETWEEN-INDIVIDUAL DIFFERENCES

Aerometric Attribute	Tau	z for the Attribute	Efficiency
SO_2 (ppm)	3.37	0.17	0.801
COH	0.381	2.16	0.827
TSP ($\mu g/m^3$)	0.00279	1.14	0.756
Ozone (ppm)	6.48	1.68	0.788
NO_2 (ppm)	5.87	4.27	0.798
Minimum temperature (F)	0.0214	3.57	0.793
Precipitation (in.)	0.00	0.00	0.867

Table 5.7

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE LESS SICKLY SUBPOPULATION (N=583):
BETWEEN-INDIVIDUAL DIFFERENCES

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
SO_2 (ppm)	0.00	0.00	-0.07
COH	0.00	0.00	-0.82
TSP ($\mu g/m^3$)	0.00	0.00	-0.42
Ozone (ppm)	0.00	0.00	-0.68
NO_2 (ppm)	1.76	0.186	-1.68
Minimum temperature (F)	0.00	0.00	-1.47
Precipitation (in.)	0.00	0.00	0.00

about four times the information that a less sickly individual contributes to the estimation of tau.

For the less sickly subpopulation, all the aerometric attributes except NO_2 have no between-individual variation; even the tau parameter for NO_2 is statistically insignificant. Thus, although there appear to be some nontrivial differences in the tau parameters within each subpopulation, none of the differences is statistically significant (from the column "z for the contrast" in Table 5.7).

We also examined differences in individual responses to air pollution and weather for two other definitions of sickliness. First, we split the subpopulation into those with FEV_1 greater or less than that expected given the individual's sex, age, and height. Second, we split the population into those with or without symptoms of chronic obstructive pulmonary disease. For both comparisons, we found no statistically significant differences in either the average responses or between-individual responses. (See App. C for details.)

COMPARISONS OF OTHER SUBPOPULATIONS

We have also examined differences in responses between children and adults (18 and over) and between smokers and nonsmokers. For both sets of comparisons, there were no statistically significant differences in average responses to air pollution. However, there was significantly less between-individual variation in children's responses to NO_2 than in adults'. (See App. C for details.)

LENGTH OF EPISODE

We also examined how the length of the episodes varied with air quality. The dependent variable was the logarithm of the number of days in the episode. The independent variables included the same set of nonaerometric variables used above. For the air quality measures, we included the air pollution on the first day of the episode and on the prior day. The response was estimated using a fixed effects model; that is, each individual's variables were taken as a deviation from that person's mean, and OLS was used on the deviated data.

We found no statistically significant association between air pollution and the length of the episode $F(10, 10386) = 1.06$. Thus, we believe that the response of time lost to illness (in days) is largely captured by the number of episodes of illness.

VI. INDIVIDUAL STUDY-LONG APPROACH: METHODS AND RESULTS

Our final approach was to analyze the intermediate-run effects of cumulative exposure to air pollution over two-and-a-half to five years upon the change in each individual's health from the beginning to the end of the study. Each individual serves as his or her own control and provides one data point for the estimation of effects.

METHODS

Sample

The samples for the study-long, health-effects analysis consisted of the 2,386 people in Seattle and the 956 in Dayton for whom we had the following information: (1) enrollment and exit health-status data, and (2) 30 or more months of residence or work location data, so we knew the levels of pollution they were exposed to. By design, everyone who completed the study except newborns should have had medical history questionnaire enrollment data.[1] Thus, restricting the sample to those with enrollment information costs very little in precision. One of the health measures, lung function, was collected in the screening examination that was given to all adults at exit, but to a randomly selected 60 percent at entry. For this measure, we had to use an efficient statistical method for combining the 60 percent with entry values with the other 40 percent (Dagenais, 1971).

Restricting the sample to those with extensive pollution data excludes those who moved early, and also the approximately 10 percent of enrollees who did not complete the study for whom we have no exit information. Most of those people left during the first year. Since the pollution levels differ from season to season and year to year, average pollution exposure for individuals with short periods of pollution data differed systematically from that for people who stayed in the study longer, and including them would have confounded the results. Since we have exit health information on those who moved, we

[1]Form and item nonresponse were very low on enrollment data collection.

did include people who moved after staying most of the time where we could monitor pollution levels.

Dependent Variables

We assessed effects on the HIE's General Health Index, a summary integrative measure of health perceptions, and a set of respiratory health indicators. The GHI is based on answers to 22 questions for adults and seven items for children (aged less than 14) that assess health generally.[2] It is scaled from 0 (worst health) to 100 (best health). The average for our full adult sample is 71, with a standard deviation of 15.

For adults,[3] our respiratory health indicators included FEV_1/predicted FEV_1, a general measure of lung function, which should be sensitive to widespread mild effects on the order of minor changes in smoking behavior.[4] We also used self-reported hay fever, chronic bronchitis, shortness of breath, and frequency of chest pain (in Dayton, pain when exercising) as measures of self-reported illness. For children less than 14, we used two measures of illness: hay fever and asthma. Information on all these measures is displayed in Table 6.1 for Seattle; the Dayton values are similar.

The health measures were also used to define a susceptible group for separate analysis. We hoped that this would shed light on overall general results and give more precision to analyses of rarer problems. The susceptible group of adults used in these analyses were those over 18 at enrollment who reported chronic bronchitis, congestive heart failure, chest pain, or shortness of breath. These were 354 out of 1,502 adults in the Seattle sample and 120 out of 661 adults in the Dayton sample. In the Seattle sample, there were 64 children who could reasonably be deemed "susceptible"; in Dayton, there were too few to

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[2]There were actually two questionnaires, one used at entry in Dayton and the other at entry in Seattle and at exit in both cities.

[3]The cutoff age used by the HIE for adulthood varied with the measure.

[4]Lung capacity depends on height, age, and sex, as well as disease. We control these factors using the results of Kory et al. (1961) and Kory and Smith (1974). See Foxman et al. (1982) for details.

Table 6.1

EXIT VALUES OF DEPENDENT VARIABLES USED IN ANALYSIS

Variable	Mean	Standard Deviation	Range	Direction of Better Health	Time Period Considered
Adult GHI	72	15	0-100	+	--
Hay fever status	1.44	0.8	1-3	--	last year
Shortness of breath	0.17	0.5	0-4	--	last 3 months
Chronic bronchitis (phlegm)	0.12	0.4	0-3	--	last year
Chest pain[a]	0.3	1.1	0-6	--	last year
Exercise pain	0.08	0.3	0-2	--	last year
Lung function (percent pred. FEV_1)	100	18	21-164	+	--
Child GHI	77	15	30-100	+	--
Hay fever status	1.34	0.7	1-3	--	last year
Asthma status	0.43	1.7	0-8	--	last year

[a]Chest pain used in Seattle.

[b]Exercise pain used in Dayton because chest pain not available.

even attempt a statistical analysis (because hay fever was not on the Dayton enrollment child-health questionnaire.

Air Quality Variables

In this analysis, we began with six measures of air quality: average TSP, SO_2, COH, and CO; average daily maximum ozone; and maximum hourly ozone over the course of the study. (See the discussion of exposure in Sec. II for more details.) The maximum hourly ozone measure was used only in Seattle, since that measure did not exhibit enough geographical variation in Dayton to make it useful. CO was used in Dayton but not Seattle. All measures except maximum hourly ozone were averaged over the full period for which we had data. Correlation analyses showed that the various measures of long-run air quality were relatively independent in Seattle, with COH and SO_2 having a pairwise correlation of 0.45, and the rest of the pairwise correlations all below 0.25. We dropped COH from the Dayton analysis because we found COH, SO_2, and ozone to be highly correlated. TSP and CO were not correlated with any of the other measures. For most analyses, we split the

population by exposure quartiles for each measure and contrasted those whose air quality was in the worst one-fourth and the second-worst one-fourth against those in the best half. Because there did not appear to be strong nonlinearities in health effects, we can use the measures directly. The results with continuous measures of air pollution are quite similar to the results with indicator variables.

Because air quality can have short- and long-run effects on health status, we also used measures of air quality in the month preceding measurement at the start and end of the study. We expected that the general health measures--General Health Index, lung function--would be most affected by immediate experience. Also, the shortness-of-breath scale was based on recall of only the most recent three months. Even the other specific disease measures, which asked for experience over the past year, could have been colored by recent experience. By taking the difference between air quality at the exit exam and at the enrollment exam, we obtain a measure that is independent of long-run average experience, and should capture short-run effects on the final outcome. In Seattle, the enrollment SO_2 values were unusual--negatively correlated with the long-run average--so we used only the exit value. In Dayton, SO_2 was measured for only half the participants at exit, so we used only the enrollment values as an independent variable.

Model

Did air quality over the course of the study affect health at exit? To answer that question, we used regression methods to estimate effects of exposure history controlling for initial value of health, age, sex, race, education, smoking history, and time in study (3 or 5 years). Because health is stable over time, the most important explanatory variable is the health measure at enrollment. This can be incorporated in three ways: First, by looking at changes over time:

$$\text{Health (exit)} - \text{health (entry)} = a + b_1 \times \text{age} + \qquad (1)$$

$$b_2 \times \text{pack years} \ldots + c_1 \times \text{ozone} + c_2 \times \text{TSP} \ldots$$

Second, by bringing health at entry to the right-hand side and not constraining its coefficient to be one:

$$\text{(2)}\quad \text{Health (exit)} = d \times \text{health (entry)} + a + b_1 \times \text{age} + \qquad (2)$$

$$b_2 \times \text{pack years} \ldots + c_1 \times \text{ozone} + c_2 \times \text{TSP} \ldots$$

Third, by omitting entry health altogether--dropping d × health (entry) from Eq. (2):

$$\text{Health (exit)} = a + b_1 \times \text{age} + b_2 \times \text{pack years} \ldots \qquad (3)$$

$$+ c_1 \times \text{ozone} + c_2 \times \text{TSP} \ldots$$

The advantage of Eq. (3) is that long-term effects on people whose air quality exposure is fairly stable over time will also be seen in health at entry, so that taking differences as in the top two equations will dilute the apparent effects of lifetime air quality. The disadvantage of the last (cross-sectional) approach is that it is very vulnerable to bias arising from selection by people of where they live and work. Equation (1) is best against bias, but is overly affected by random variation of health at entry. In Eq. (2), the regression method selects the appropriate weight to put on health at enrollment, and this middle specification is the one presented most often in this section.

RESULTS

Adults

The GHI is the most aggregate measure of health effects studied. For adults, the effect on general health status exerted by each air quality measure taken separately is shown in Table 6.2. Dashes indicate t-values less than one in absolute value, and the blanks on the best quarter indicate it was the group against which the others were compared. Maximum hourly ozone measure had significant adverse effects in Seattle. People in the worst two quarters for average daily maximum

Table 6.2

EFFECTS OF SINGLE POLLUTANTS ON ADULT GENERAL HEALTH INDEX

Variable	TSP	SO_2	COH	Ozone Average	Ozone Max.	CO
Seattle (N = 1640)						
Worst quarter	-1.0	--	-1.0	-1.9[a]	--	
Second-worst	-1.4	0.9	--	-2.3[a]	--	
Second-best	-1.4	--	--	--	--	
Best (reference group)	--	--	--	--	--	
Dayton (N = 661)						
Worst	--	--		--		--
Second-worst	-2.5	--		--		-2.2
Second-best	-2.1	--		--		-2.2
Best (reference group)	--	--		--		--

NOTE: See subsection on methods for interpretation of GHI. Numbers represent average differences in average GHI from those with least pollution exposure. Only coefficients with a t-value greater than 1 in absolute value are shown. Blanks indicate that the variable was not included.

[a]Significant at 0.05 level.

ozone had a GHI score of more than 2 points lower than those living in the best areas by that measure. This is about one-fourth the difference found between those with diabetes or chronic obstructive pulmonary disease and those nondiseased adults in the sample. In Dayton, however, there was no effect of ozone. Other air quality measures in Dayton and Seattle had less significant adverse effects, except for SO_2, which had an insignificant positive effect in Dayton. These results and further results for lung function were not sensitive to a number of minor variations: (1) whether the entry and exit exam values were used; (2) whether air quality measures were split by quartiles (as in Tables 6.2 and 6.3) or entered linearly (as in Table 6.4); (3) whether teenagers were included or not; and (4) whether air quality measures were considered one by one, as in Table 6.2, or all at once, as in the subsequent tables. Thus, in Tables 6.3 and subsequently, the results give partial effects of each pollutant controlling for the others. The initial value of GHI was by far the most important predictor of exit GHI.

Table 6.3

REGRESSION COEFFICIENTS FOR GENERAL ADULT POPULATION, SPLIT BY QUARTILES: SEATTLE

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Chest Pain
Initial measure	0.60[a]	0.44[a]	0.72[a]	0.35[a]	0.28[a]	0.21[a]
Ozone						
Max, worst quarter	--	4.7[a]	-0.12[a]	0.06	-0.04	--
Max, second worst	--	2.8[a]	--	0.09[a]	--	+0.12
Avg, worst quarter	-2.1[a]	--	--	--	0.03	--
Avg, second worst	-2.6[a]	2.4[a]	--	--		+0.11
TSP						
Worst quarter	--	2.4	-0.08	--	-0.04	-0.27[a]
Second worst	--	2.1	-0.04	--	-0.04	-0.09
COH						
Worst quarter	--	--	--	--	--	--
Second worst	-1.3	--	-0.05	-0.05	--	--
SO_2						
Worst quarter	--	--	--	0.06	0.03	--
Second worst	+1.1	1.3	--	0.04	-0.03	--
Other variables[b]						
Sample size	1,499	1,235	1,499	1,296	1,338	1,346
R^2	0.38	0.34	0.54	0.26	0.15	0.11

NOTE: Adults defined as 14 and over for GHI and hay fever, 18 and over for shortness of breath, chronic bronchitis, and chest pain. See subsection on methods for interpretation of GHI. Only coefficients with t-values greater than 1 are shown.

[a]Significant at 0.05 level.

[b]Age, female, female × age, nonwhite, four measures of smoking behavior, education, time in study (3 or 5 years).

As shown in Tables 6.3 and 6.4, better lung function was associated with poor air quality in Seattle, and the association with ozone was significant. For the other four measures, a high score indicates more disease, so a negative sign (as in hay fever) shows a positive association of hay fever with good air quality, and positive signs (as in the shortness-of-breath scale) show a positive association of shortness of breath with poor air quality. For these diseases, higher TSP is consistently associated with better health, and higher SO_2 often with worse health, in Seattle. However, virtually all of these effects are not significant at the 5 percent level. There were fewer effects in

Table 6.4

EFFECTS ON GENERAL ADULT POPULATION, ENTERED CONTINUOUSLY: SEATTLE

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Chest Pain
Initial measure	0.60	0.47	0.72	0.34	0.28	0.21
Ozone max	--	74	-2.55	0.79	-0.73	--
t	--	2.76[a]	-2.80[a]	1.02	1.14	--
Ozone average	-678	613	16	11.50	10.90	--
t	-2.54[a]	1.64	1.26	1.08	1.23	--
TSP average	--	--	-0.0044	--	-0.002	-0.006
t	--	--	-2.57[a]	--	-1.68	-1.99[a]
COH average	5.7	--	0.35	--	--	--
t	1.3	--	1.69	--	--	--
SO_2 average	--	--	--	--	--	--
t	--	--	--	--	--	--
Other variables[b]						
Sample size	1,499	1,313	1,499	1,296	1,338	1,346
R^2	0.38		0.54	0.26	0.15	0.09

NOTE: See notes for Table 6.3.

Dayton, as shown in Tables 6.5 and 6.6. Indeed, most of Table 6.5 is blank.

In Seattle, results for the susceptible population are generally less significant, because the sample is much smaller (Tables 6.7 and 6.8). The negative coefficients relating the GHI to air quality are as large as those for the general population, but they are not significant. The consistent associations between poor air quality and better lung function are not present in the susceptible group, and indeed, the effects of ozone are reversed. The associations between bad air quality and shortness of breath are much larger in the susceptible group.

Table 6.5

EFFECTS ON GENERAL ADULT POPULATION, SPLIT BY QUARTILES (DAYTON)

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Exercise Pain
Initial measure	0.68	0.68	0.60	0.56	0.39	0.30
Ozone						
Worst quarter	--	--	-0.19	--	-0.06	-0.05
Second worst	--	--	--	0.13	--	--
TSP						
Worst quarter	--	--	-0.19[a]	--	--	--
Second worst	--	--	--	--	--	--
COH						
Worst quarter	--	--	--	--	--	--
Second worst	--	2	--	0.09	--	-0.05
SO_2						
Worst quarter	--	--	--	--	--	--
Second worst	--	--	-0.19[a]	--	--	--
Other variables[b]						
Sample size	661	494	637	540	546	572
R^2	0.39	0.41	0.27	0.29	0.20	0.20

NOTE: See notes for Table 6.3.

Table 6.6

EFFECTS ON GENERAL ADULT POPULATION, ENTERED CONTINUOUSLY: DAYTON

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Exercise Pain
Initial measure	0.68	0.65	0.61	0.56	0.39	0.30
Ozone	--	--	-18	--	-7	-4.3
t	--	--	-2.39	--	-1.64	-1.13
TSP average	--	--	-0.005	--	--	--
t	--	--	-2.01	--	--	--
CO average	--	--	--	0.16	--	-0.10
t	--	--	--	1.12	--	-1.56
SO_2 average	--	--	-75	--	--	--
t	--	--	-2.80	--	--	--
Other variables[b]						
Sample size	661	523	637	540	546	572
R^2	0.38	--	0.27	0.29	0.20	0.20

NOTE: See notes for Table 6.3.

Table 6.7

EFFECTS ON SUSCEPTIBLE ADULT POPULATION, SPLIT BY QUARTILES: SEATTLE

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Chest Pain
Initial measure	0.6	0.53	0.65	0.32	0.19	0.21
Ozone						
Max, worst quarter	--	--	--	--	--	--
Max, second worst	-2.4	--	--	0.11	--	--
Avg, worst quarter	-2.0	-3.60	--	--	--	--
Avg, second worst	--	--	--	--	--	0.27
TSP						
Worst quarter	-2.1	--	-0.11	0.12	--	--
Second worst	--	--	--	--	-0.09	-0.31
COH						
Worst quarter	--	--	--	--	--	--
Second worst	--	--	0.20	-0.16	--	--
SO_2						
Worst quarter	--	--	--	0.21[a]	--	--
Middle quarter	--	2.70	--	0.15	--	0.49[a]
Other variables[b]						
Sample size	352	291	335	316	332	351
R^2	0.42	0.46	0.50	0.35	0.20	0.19

NOTE: See notes for Table 6.3.

Table 6.8

EFFECTS ON SUSCEPTIBLE ADULT POPULATION, ENTERED CONTINUOUSLY: SEATTLE

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Chest Pain
Initial measure	0.6	0.51	0.66	0.31	0.20	0.20
Ozone max	--	--	-3.5	--	--	--
t	--	--	-1.56	--	--	--
Ozone average	-850	--	--	42.5	25	--
t	-1.48	--	--	1.35	1.00	--
TSP average	-0.13	--	-0.007	--	--	--
t	-1.5	--	-1.5	--	--	--
COH average	9.8	--	0.68	--	--	-1.41
t	1.05	--	1.34	--	--	-1.39
SO_2 average	--	--	--	--	--	90
t	--	--	--	--	--	1.79
Other variables[b]						
Sample size	352	319	335	316	332	351
R^2	0.42	--	0.50	0.33	0.20	0.16

NOTE: See notes for Table 6.3.

In Dayton, the effects of air pollution on the health of susceptibles are insignificant (Table 6.9 and 6.10), as would be expected from the insignificant effects on the general population. The negative coefficients relating the GHI to air quality are larger than those for the general population, but they are not close to significant. The associations between poor air quality and better lung function, less hay fever, and less exercise pain are more consistent and stronger in the susceptible group. The results for exercise pain are the most striking. Since "never exercise because of chest pain" is scored the same as pain while exercising, this correlation is not the result of

Table 6.9

EFFECTS ON SUSCEPTIBLE ADULT POPULATION, SPLIT BY QUARTILES: DAYTON

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Chest Pain
Initial measure	0.56	0.55	0.55	0.53	0.27	0.15
Ozone						
Avg, worst quarter	--	--	-0.32	-0.51	--	-0.31
Avg, second worst	--	6	--	--	--	-0.26[a]
TSP						
Worst quarter	--	7[a]	--	--	--	-0.31[a]
Second worst	-5	4	--	--	--	--
CO						
Worst quarter	--	7	--	--	--	-0.32
Second worst	-7	5	--	--	--	--
SO_2						
Worst quarter	--	--	--	--	--	0.20
Second worst	--	--	--	--	--	--
Other variables[b]						
Sample size	122	103	121	115	115	116
R^2	0.46	0.56	0.34	0.39	0.22	0.43

NOTE: See notes for Table 6.3.

Table 6.10

EFFECTS ON SUSCEPTIBLE ADULT POPULATION, ENTERED CONTINUOUSLY: DAYTON

Variable	GHI	Lung Function	Hay Fever	Shortness of Breath	Chronic Bronchitis (Phlegm)	Exercise Pain
Initial measure	0.64	0.40	0.47	0.54	0.21	0.08
Ozone average	--	378	-26	-45	-25	-36
t	--	1.29	-1.25	-1.53	-1.56	-2.41
TSP average	--	0.13	-0.008	--	--	-0.012
t	--	1.05	-1.17	--	--	-2.48
CO average	-7.5	--	--	--	--	-0.58
t	-1.04	--	--	--	--	-2.29
SO_2 worst	--	1,776	--		-61	--
t	--	2.18	--	--	-1.16	--
Other variables						
Sample size	116	108	115	115	115	116
R^2	0.43	--	0.29	0.34	0.21	0.37

NOTE: See notes for Table 6.3.

people staying inside. Perhaps it reflects selection out of low-air-quality areas by people with angina.

In the model that produced the results in Tables 6.3 through 6.10, exit health was a linear function of entry health and other variables. We also looked at two other specifications. First, we used change in general adult GHI and lung function, entry to exit, as the dependent variable, i.e., Model (1) above. The results for effects of air pollution on changes in health were similar to results for exit health values regressed on initial. This is not surprising since both the GHI and lung function are quite stable over time. Thus, the coefficients of the initial measure shown in the tables on general adult health are not too different from +1 (the value implicitly assumed by studying changes).

Second, we considered the purely cross-sectional results of regressing exit GHI and lung function values on cumulative exposure to air pollution during the study period. These results are shown in Tables 6.11 and 6.12. Since there is no adjustment for the stable differences between people, much less of the variation in the health measures is explained (R^2 is much smaller).

In Seattle, the associations with air quality for both the GHI and lung function stay the same. Since both the change in health and exit health are similarly correlated with air quality, it seems that the

Table 6.11

EFFECTS OF POLLUTANTS ON HEALTH IN SEATTLE:
A CROSS-SECTIONAL ANALYSIS

Variable	General Health Index (End)	Lung Function (End)	General Health Index (Start)	Lung Function (Start)
Ozone				
Max, worst quarter	--	4.9[a]	--	--
Max, second worst	-1.1	3.5[a]	-1.4	3.1
Avg, worst quarter	-1.6	1.5	--	--
Avg, second worst	-3.0[a]	2.5	--	--
TSP				
Worst quarter	--	--	--	-6.1[a]
Second worst	--	--	--	-2.4
COH				
Worst quarter	--	--	--	--
Second worst	--	--	--	--
SO_2				
Worst quarter	--	--	--	--
Second worst	--	--	-1.5	-4.2[a]
Other variables[b]				
Sample size	1,642	1,235	1,649	667
R^2	0.08	0.04	0.17	0.13

NOTE: See notes for Table 6.3.

Table 6.12

EFFECTS OF POLLUTANTS ON HEALTH IN DAYTON:
A CROSS-SECTIONAL ANALYSIS

Variable	General Health Index (End) Susceptibles	Lung Function (End)	General Health Index (Start) Susceptibles	Lung Function (Start)
Ozone				
Worst quarter	-8	-8[a]	-11[a]	-6
Second worst	--	-6[a]	-4	-5
TSP				
Worst quarter	-5	-5	-5	-3
Second worst	-6	-3	--	-4
CO				
Worst quarter	--	--	--	--
Second worst	-9[a]	--	-4	-5
SO_2				
Worst quarter	--	--	--	--
Second worst	-7	-4	--	--
Other variables[b]				
Sample size	116	265	116	265
R^2	0.32	0.23	0.39	0.23

NOTE: See notes for Table 6.3.

initial value, the variable used to adjust for stable differences, must not be highly related to subsequent air quality. This conjecture is somewhat borne out by the last two columns of Table 6.11. General health is not greatly associated with subsequent ozone, and poorer lung function is associated with higher TSP and SO_2 levels. How can health change and exit health be associated with pollution levels during the study while initial health is not? One possible explanation is geographical sorting prior to the study. Another is that air quality before the study started is not highly correlated with subsequent air quality, and that there are noticeable medium-run responses to the change. Another explanation is that the increased precision of the

before-and-after technique reveals something missed by the cross-sectional approach.

In Dayton, on the other hand, the cross-sectional analysis yields consistent positive associations between low air quality and both the GHI and lung function. Contrasting the cross-sectional with the longitudinal analyses, we see that poor health may be related to low air quality cross-sectionally, but that the relationship does not increase over time. The cross-sectional relationship may be due to selection or previous long-term exposure, but exposure over the three years of the study does not seem to have had many effects.

We incorporated variables representing air quality for the month before the initial and final exams. These short-term effects on physiological measures were generally weak, but in the right direction (adverse). Because short-term air quality was not highly correlated with long-term air quality, inclusion or exclusion of short-term measures had little effect on the estimated effects of long-term air quality.

Children

The GHI for children under 14 was associated with air quality in Seattle in a peculiar way (Tables 6.13 and 6.14). There were strong associations of better health with higher levels of TSP and lower levels of ozone. Hay fever was not associated with air quality, but higher levels of ozone were related to more asthma.

The Dayton results were more consistent, if unexpected. Higher levels of all air pollutants were associated with better general health and less hay fever (Table 6.15). Several of these effects were significant. Asthma was not associated with air quality.

The sample of susceptible children in Dayton was too small to be analyzed. Even in Seattle, the sample of susceptible children with complete information was so small that only very large effects would have been significant. No such effects were found, but the right halves of Tables 6.13 and 6.14 show some marginal effects. The (positive) effect of TSP on general health disappears, but otherwise the results for sickly children are similar to the results for all children.

Table 6.13

EFFECT OF AIR QUALITY ON HEALTH IN CHILDREN UNDER 14, SPLIT BY QUARTILES: SEATTLE

Variable	All Children			Susceptible Children		
	General Health	Hay Fever	Asthma	General Health	Hay Fever	Asthma
Initial value	0.42[a]	0.63[a]	0.52[a]	0.45[a]	0.08	0.54[a]
Ozone						
Max, worst quarter	--	--	--	--	--	-1.5
Max, second worst	3.1	--	0.8[a]	--	--	--
Avg, worst quarter	-3.8[a]	--	0.9[a]	-5	--	1.6
Avg, second worst	-2.6	--	0.4	--	--	--
TSP						
Worst quarter	6.4[a]	--	--	--	--	--
Second worst	8.6[a]	--	--	--	--	--
COH						
Worst quarter	--	--	--	-6	--	--
Second worst	--	-0.17	--	--	--	--
SO_2						
Worst quarter	--	0.14	-0.4	--	--	-1.0
Middle quarter	--	--	--	--	--	--
Other variables[b]						
Sample size	630	423	251	0.41	0.19	0.51
R^2	0.25	0.34	0.27	64	63	37

[a]Significant at 0.05 level.

[b]Age, female, female × age, nonwhite, two measures of parental smoking behavior, parents' education, time in study (3 or 5 years).

Table 6.14

EFFECTS OF AIR POLLUTION ON HEALTH IN CHILDREN UNDER 14, ENTERED CONTINUOUSLY: SEATTLE

	All Children			Susceptible Children		
Variable	General Health	Hay Fever	Asthma	General Health	Hay Fever	Asthma
Initial value	0.43	0.64	0.53	0.43	0.12	0.51
Ozone max	-0.49	--	--	187	-9.8	--
t	-1.10	--	--	1.12	-1.06	--
Ozone average	-1,175	--	340	--	--	1,123
t	-1.73	--	2.69	--	--	2.02
TSP average	--	--	--	--	--	--
t	--	--	--	--	--	--
COH average	--	0.89	1.88	--	4.0	7.8
t	--	1.62	1.12	--	2.39	1.40
SO_2 average	--	--	-130	--	-82	-419
t	--	--	-1.55	--	-1.01	-1.41
Other variables[b]						
Sample size	629	423	251	64	63	37
R^2	0.23	0.34	0.25	0.39	0.24	0.50

NOTE: See notes for Table 6.13.

Table 6.15

EFFECT OF AIR QUALITY ON HEALTH IN CHILDREN UNDER 14, SPLIT BY QUARTILES: DAYTON

Variable	General Health	Hay Fever	Asthma
Initial Value	1.04	--	0.41
Ozone			
Avg, worst quarter	7.3[a]	--	--
Avg, second worst	6.5[a]	--	--
TSP			
Worst quarter	--	-0.21	--
Second worst	--	-0.22	0.49
CO			
Worst quarter	4.6	-0.46[a]	--
Second worst	--	-0.17	--
SO_2			
Worst quarter	--	0.29	--
Second worst	--	--	--
Other variables[b]			
Sample size	283	269	109
R^2	0.19	0.10	0.37

NOTE: See notes for Table 6.13.

CONCLUSIONS

In the general Seattle sample, GHI seemed adversely affected by ozone, and better lung function was associated with most of the measures of air quality. The picture for specific diseases is less clear. Among susceptibles, both the negative effects on general health and the positive effects on lung function are less significant; indeed, ozone may have a negative effect on lung function, but the results are not significant enough for clear interpretation. Either the effects are weak, or there are simply not enough susceptibles here to show much. For children, most of the results are in the "right" direction, but few are significant.

Effects in Dayton were even less significant than those in Seattle, and many showed an unexpected association of better health with poorer air quality (Table 6.16). In fact, we did not find a single significant effect of lower air quality in Dayton. The general health of adults was unrelated to air quality, but hay fever and lung function in susceptible

Table 6.16

EFFECT OF AIR QUALITY ON HEALTH IN CHILDREN UNDER 14, ENTERED CONTINUOUSLY: DAYTON

Average	General Health	Hay Fever	Asthma
Initial Value	1.12	--	0.39
Ozone average	1,071	- 20	--
t	3.26	-1.07	--
TSP average	0.10	-0.006	--
t	1.26	-1.42	--
CO average	6.4	-0.51	--
t	1.46	-2.07	--
SO_2 average	2,026	--	--
t	1.90	--	--
Other variables[b]			
Sample size	283	269	109
R^2	0.19	0.08	0.35

NOTE: See notes for Table 6.13.

groups and hay fever and general health in children were associated in the "wrong" way. Ozone, in particular, was generally related to better health. Specific health problems showed even more insignificant results than the general health measures. The main reason for the lack of significant effects in Dayton is probably sample size.

This study of intermediate range effects used a before-and-after method instead of the cross-sectional approach most commonly seen. Using initial status as a control reduces the problem of selection bias, but allows us to look only at changes over the course of observation (here 3 or 5 years). The cross-sectional approach will be relatively better if people do not move much and select work and home locations independently of pollution levels, and if air pollution effects on health are gradual. If people do move in such a way that health is correlated with pollution, then cross-sectional studies can be quite misleading. As it turned out, the cross-sectional results differed between Seattle and Dayton, evidence that selection may be more responsible for these observed results than accrued damage to health. Ideally, one would like a before-and-after design with a very long period for health effects to appear, but follow-up in such studies is difficult and expensive.

VII. DISCUSSION

The results in the preceding sections exhibit a mixed set of associations between air pollution and the three classes of health outcomes: use of outpatient medical services, time lost due to illness, and health status. They also exhibit a mixed set of results depending on the method used to estimate the adverse effect of air pollution. In many cases, higher levels of air pollution are associated with better health outcomes.

Closer examination reveals several patterns. Two of those are of special note because they dominate the overall pattern of "perverse" results, where higher levels of air pollution are associated with better health outcomes. First and most striking is the large number of significant results for ozone. Ozone was responsible for many of the significant effects--largely positive (beneficial) in the case of outpatient health expenditures and time lost to illness, and both positive and negative (adverse) in the case of the health status measures. There were positive estimates for ozone from both the annual (cross-sectional) and panel results and from both the short-term and intermediate-term results.

The second trend is the large number of significant results obtained in the aggregated day-to-day approach, which is a panel analysis for a fixed population. Three quarters of the estimated effects are significant at the 10 percent level, and half are significant at the 1 percent level. Of the significant results, half are positive (beneficial). All of the pollutants except CO have at least one significant and "perverse" positive effect.

In contrast, the individual daily analysis in Seattle (based on the random-effects model) yields negative (adverse) estimates for all of the pollutants except ozone. SO_2 and NO_2 are statistically significant for the average person at the 1 percent level.

The other panel analysis was the examination of air pollution effects on health status. Except for ozone, we observed few statistically significant effects of air pollution on health status.

This was was true for both sites, for children and adults, for the general population, and for the susceptible (i.e., sickly) population. We did observe some significant results for some of the scales for specific diseases. The significant results were of mixed sign. Of the 18 non-ozone results, only one was significant at the 10 percent level. If there were no true effect, we would expect to see about two significant findings just at random at the 10 percent level.

The results for the annual analyses are mixed. Those results are obtained by allowing for correlated responses in a manner that ignores the possibility that individuals may geographically sort themselves out in response to air pollution. Hence, the method has embedded in it the same potential for bias that exists in pure cross-sectional approaches. Except for ozone, only 2 out of 18 results are significant at the 10 percent level. One is negative (adverse) and the other positive (beneficial). If there were no true air pollution effect, we would expect to get one positive and one negative significant result at random.

By and large, the results for the pollutants other than ozone are not statistically significantly different from zero, aside from those obtained from the aggregated daily approach. For the other three methods, no pollutant showed more than two significant effects out of an average of eight possibilities each.

The effects of pollution on health did not vary in any easily generalizable way between Seattle and Dayton. The overall effects of pollution were the same for each city as they were for both taken together, i.e., generally mixed and insignificant with a tilt toward the positive. Breaking it down by pollutant, there were many instances of varying results, but the general summary given above for both cities together could apply almost as well to each considered separately.

Our several analyses of susceptibles provided results that were consistent with those of the population as a whole. However, the failure to find a greater sensitivity to pollution among susceptibles probably reflects our lack of precision. For example, in the before-and-after comparisons of the intermediate term effects of air pollution on health status, we generally found larger effects for susceptibles. These larger effects were not large enough to compensate for the

reduction in sample size. In a general population study of the nonaged, there are few very susceptible individuals.

This summary of our findings should not be construed to mean that air pollution has no appreciable or significant adverse effect on health outcomes. There are a number of methodological reasons why we could have obtained these largely null findings, or in the case of ozone, a counterintuitive result. In the following, we discuss these findings with special attention to the methodological lessons to be drawn.

OZONE

It is so commonly assumed that air pollution is bad for health under all circumstances that our finding that ozone is frequently associated with significant beneficial short-run effects seems puzzling. There are a number of methodological or threshold explanations for why a pollutant would show no ill effect on health in a given study. It is more difficult to understand how an air pollutant could be consistently found to have a significant association with improved health. We suspect that the ozone results are due to a confounding of ozone with meteorology or some other omitted variables that have an independent and beneficial effect on health outcomes. The levels of ozone in these two cities may be low enough that the adverse effects of ozone are outweighed by any beneficial effects of the omitted but correlated explanatory variables.

Our ozone findings are not inconsistent with chamber studies and prior observational analyses. Chamber studies have indicated that ozone exposure at levels as high as 0.3 to 0.4 ppm can be tolerated without adverse effects by individuals sitting quietly in the chamber (Adams et al., 1981). Exercising individuals demonstrate acute effects at much lower levels, usually starting around 0.20 to 0.24 ppm (Avol et al., 1983; Brookshire et al., 1982; Delucia and Adams, 1977; Evans et al., 1976). Experts in chamber studies indicate that susceptible individuals under exercise conditions may respond adversely at 0.12 ppm, the federal standard (Adams et al., 1981). Only a small number of the chamber studies have used levels below 0.12 ppm. Few have recorded effects below about 0.2 ppm, even for exercising individuals (Folinsbee et al., 1978; Javitz et al., 1983).

Thus, there are no chamber data to suggest that ozone concentrations well below the federal standard produce measurable or appreciable short-term adverse effects. The effects threshold appears to fall somewhere between 0.12 and 0.4 ppm, depending on the susceptibility and activity of the exposed person.

A number of studies have suggested no ozone effect or even a positive effect on free-living populations, especially where ozone levels studied have not been high. For instance, reanalysis of data from Houston by Javitz et al. (1983) at SRI International indicates a small, consistent drop in the probability of symptoms as the ozone level increased from zero to concentrations of 0.03 to 0.09 ppm. Once ozone levels reached 0.12 ppm, the probabilities of many symptoms began to increase and continued to do so as ozone levels rose further, thus replicating the findings of the chamber studies. It should be noted that these are symptoms and not lung function measures.

The levels of ozone to which our sample was exposed fell well below the range at which the chamber and Javitz studies showed adverse effects. Seattle experiences very little ozone exposure over the one-hour federal standard. The exposure in Dayton is substantially greater, but even the highest concentrations did not exceed 0.2 ppm--below the ozone level at which most chamber studies have shown effects for exercising individuals.

The ozone results may reflect the confounding of ozone with some omitted but beneficial variable. For example, the absence of variables on cloud cover may impart a small but statistically significant bias to the results from the individual daily time series if individuals in Seattle are less likely to be ill or feel blue when it is sunny.[1] A similar bias may have been introduced into the aggregated daily time series by the omission of meteorological variables.

Alternatively, the ozone effect may be related to the short-term positive reaction that many people have on being exposed to light negative ions. Negative ions are produced by electrical equipment (including ionizers, of course), thunderstorms, sunshine, and wind.

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[1]Our analyses adjust for precipitation and temperature, but not sunshine.

Regardless of the source, production of negative ions is almost invariably accompanied by the production of low levels of ozone. The positive reaction to negative ions may reduce symptoms, the recognition of symptoms, or complaints of symptoms. In our study, these would translate into fewer visits to physicians and less sick loss.

Despite the short-run positive association of ozone with healthier outcomes at low levels of pollution, there may be underlying and ongoing damage associated with this low-level ozone exposure which would only be expressed in long-term effects. The health status findings for ozone in Seattle are consistent with a longer term adverse effect, when the short term use of services and sick-loss exhibit a positive association. Detels et al. (forthcoming) report possible long-term effects of oxidant exposure, including possible cumulative damage associated with ozone concentrations below 0.10 ppm. Obviously, further research into this entire problem would be very helpful.

METHODS EFFECTS

Aggregated Daily Time Series

Our results are sensitive to the methods used to do the analysis. In particular, the aggregated day-to-day approach yields many significant findings, most of which are of the wrong sign, i.e., they indicate a beneficial association of air pollution with health outcomes. For a given pollutant, the effects do not even have the same sign for the two different sites and the two outcomes analyzed. Despite the significance of the results, the magnitude of the effect is typically quite small. A doubling of the air pollution level is usually associated with a change in the health indicator of about 2 to 4 percent.

Why the aggregated daily approach "misbehaves" is an open question at this point. Given the size of the inference statistics and the small effects of a doubling in the level of air pollution, it is clear that this method has the precision to pick up small effects, including those where a doubling of the air pollution level would be associated with changes in visit rates and sick-loss of only 2 percent. We suspect that the aggregated time series may be picking up the beneficial aspects of other factors correlated with higher levels of air pollution. A likely

suspect is the day-to-day variation in meteorological conditions; our specification controlled for monthly and day of the week effects, but not cloud cover, temperature, or precipitation. Whatever the explanation, this easy-to-use method clearly requires more scrutiny before a decision is made as to whether it should be applied routinely in measuring air pollution effects.

Individual Daily Time Series

In contrast, the individual daily approach consistently yielded estimates of adverse effects of air pollution on time lost to illness--with the exception of ozone. On that basis alone, this technique may be the more promising for valuing the benefits of regulating air quality than the aggregated daily time series approach. In addition, there are two methodological rationales for favoring the individual time series approach. First, in this approach, we use the individual as his or her own control and estimate the response to air quality and weather, rather than trying to get some sort of average response over a population that is quite heterogeneous in the response. Thus, we can tell whether susceptibles or smokers or children are more or less responsive than the rest of the population by doing a meta-analysis on the estimated individual responses. Second, allowing each person to act as his or her own control reduces certain exposure and data problems. If a person lives in a dusty or poorly ventilated house, and dust affects the person's behavior, it will be captured in his or her coefficients. Third, we can do a much better job of estimating individual exposures in the individual daily approach than in the aggregated approach. In the former, we can use our estimate of the person's exposure, based on work and home locations. In the latter, we can use only one value of each pollutant for everyone, so we measure the individual's exposure with more error. That measurement error yields biased estimates of the true response to air pollution.

This study was the first to apply this technique to a general population. The pattern of adverse effects of air pollution detected in a moderately polluted city is evidence that the technique can detect the adverse effects of air pollution when other techniques (e.g., cross-sectional or aggregated time series) fail to give meaningful results.

Before we make too much of this finding, however, we should remember three things: First, this is in essence a case study of the technique and is subject to all the limitations of a case study. Second, the method yielded an unexpected negative correlation between the parameter estimates and their standard errors. And third, the individual daily approach is a costly one. Nevertheless, we find the results interesting and the theoretical arguments convincing enough to warrant further study of this technique. In the second phase of this research for EPA, we will examine this technique more closely. That work will include (1) simulation analysis to study the question of correlation between the coefficients and their standard errors; and (2) applications of this technique to other outcomes and to data from the Dayton site.

Annual ("Cross-Sectional") Analysis

For the reasons stated in the Introduction, we had expected the annual ("cross-sectional") approach to have the greatest likelihood of producing "perverse" results. Instead, except for ozone, the annual cross-sectional analysis basically showed no effects for air pollution on the use of health services or time lost to illness. There are five possible explanations for this. First, the absence of a result is consistent with there being a true adverse effect that is wiped out by the sorting phenomenon. If individuals who are more susceptible to the adverse effects of air pollution move to less polluted areas of the city, then the estimated effects of air pollution will be biased toward zero or could have the "wrong" sign. Although we used very good measures of health status, relative to those available on most general population data sets, the measures are not perfect and our estimates could be biased. Second, there may be no true effect in the range we are observing and sorting may not be a problem. Third, the absence of an effect could be due to aggregating over a year. The largest illness effects may occur in the winter and the highest pollution levels in the summer. The annual analysis should not "see" that time difference, and thus would find no association. Fourth, the use of an estimate of air pollution exposure based on ambient air at monitoring sites will contain a substantial amount of measurement error. All other things equal, this measurement error will bias the estimates of air pollution toward zero

in proportion to the ratio of the measurement error to the true error in the equation (see Theil, 1971, pp. 607-615). Thus, there could be a small but important true adverse effect of air pollution that would not be detected because we relied on a proxy for true air pollution exposure. Fifth, we may not have detected an adverse effect of air pollution because of lack of variation in the exposure measure. As the tables in the appendix on exposure indicate, there is much less variation in average exposure over a period of a year, than there is over a period of a day. Most of what little variation we do have at the annual level is due to geographical differences in pollution levels. The smaller the variance in the explanatory variable (e.g., SO_2), the larger the standard error of its coefficient.

Intermediate-Term Health Effects

We had expected that the use of a variant of the before-and-after comparison would allow us to detect some adverse intermediate-term effects of air pollution. The technique has the advantages that: (1) the individual acts as his or her own control, thus reducing any bias from geographical sorting; and (2) the inclusion of entry health status as a covariate should reduce the error variance substantially and increase the precision of the regression, because health status is fairly stable over time. However, we found that of the non-ozone findings for general health status and adult lung function, there were about as many significant results as one would expect at random.

Does this mean that there are no true health status effects in our two cities at these moderate levels of pollution? We think such a conclusion would be improper. The absence of a significant effect may be attributable to lack of precision. Given the measures we had of general health status, lung function, and the variation in air pollution across individuals, we had the precision to detect an effect of air pollution if it were as large as the adverse effects of smoking one-half to one pack of cigarettes a day over the same period. That is a very large effect, and most people would be concerned if air pollution had a substantially smaller effect than that. The culprit for our lack of precision is again the lack of variation in air pollution over periods of time longer than a few days. (See the discussion of exposure

in Sec. II for the variation in exposure across individuals.) Low variation in the pollutant measure implies a large standard error on the estimated effect of air pollution.

To get away from this problem of low precision, one needs to have a data set with several characteristics. First, the number of participants must be substantially larger. A tenfold increase in our sample size would reduce the detective effect to that of five to seven cigarettes a day. Second, there should be a wider range of variation in the air pollution exposure of individuals. All other things equal, the standard error of the estimate coefficient will go down as the square root of the variance of the exposure measure. Third, an increase in the number of susceptibles, who appear to have larger effects from air pollution, would make it easier to detect an effect. Finally, better measures of actual exposure (via personal monitoring or micro-environmental analysis) would reduce the bias in the estimate coefficient and enhance our ability to detect meaningful adverse effects of air pollution.

Unfortunately, data sets with these characteristics and a comprehensive set of measures on health outcomes (use of medical services, time lost due to illness, and health status) collected on a panel basis are expensive and time-consuming to generate. In the short run, it will be important to see what we can learn from existing general population data sets, despite their important flaws. One very promising avenue of research is the further application of the Whittemore-Korn technique to time series data on time lost due to illness and to the use of medical services. Our work clearly suggests that this technique can be useful in the assessment of short-term effects of air pollution.

Before embracing the individual daily approach or discarding the others, however, it is important to realize that our findings are based on only two sites. In fact, the individual daily approach was applied to only one health outcome in one site. In addition, this is the first time that this technique has been employed on a general population. The only way to be sure that the patterns we have found are "real" is to do further research in the same vein: including additional meteorological data to control for factors which may explain the positive association between ozone and health outcomes, and by applying a similar set of

approaches to several cities or data sets. Ideally, future analysis should include data from a long enough time series on people in a general population to employ the individual daily time series approach. Further research along those lines should allow us to reach a conclusion as to which approach is the most effective for valuing the regulation of air quality.

Appendix A

SELECTION OF DATA

The first analytic choice we had to make was to select a data set for the analysis. We looked for a data set with information on health status, sick-loss days, and use of health services for a general population (e.g., more than merely a subpopulation susceptible to cardiopulmonary problems). Below we briefly describe the criteria that we used in evaluating data sets, the advantages and disadvantages of each data set, and our reasons for the final selection.

For the evaluation of effects of air pollution on health outcomes, we examined the following data sets:

1. Health and Nutrition Examination Survey (HANES) I and II
2. Health Insurance Experiment (HIE)
3. Health Interview Survey (HIS)
4. National Medical Care Expenditures Survey (MNCES)
5. National Medical Care Utilization and Expenditure Survey (MNCUES)

CRITERIA FOR EVALUATION

In evaluating the alternative data sets, we used seven criteria:

1. A preference for panel over cross-sectional data.
2. The ability to create good synthetic (proxy) measures of air pollution exposure.
3. The ability to create good synthetic measures of weather.
4. Comprehensiveness of a single data set.
5. Presence of valid and reliable measures of health status.
6. Adequate within-site data.
7. Variation across sites in levels and types of air pollution.

Panel Versus Cross-Sectional Data

We would prefer a panel (cohort) data set over a cross-sectional one. In a panel data set, the longitudinal measurements on each individual allow us to control for unobservable characteristics of each individual. Thus, we do not have to rely on the untestable cross-sectional assumption that the unobserved characteristics are uncorrelated with the observed independent variables (including air pollution). If this assumption does not hold, cross-sectional data can yield biased estimates of the effects of air pollution. The direction and magnitude of the bias cannot be determined *a priori*. For example, if smokers are less likely to move away from smoggy areas (and if the smoking measure has measurement error), then cross-sectional data will overstate the effect of air pollution on cardiopulmonary problems. If individuals who are susceptible to cardiopulmonary complaints move from smoggy areas to less smoggy areas (and if the health status measure has measurement error), then cross-sectional data understate the effects of air pollution.

In contrast, with a panel data set these unobserved effects can be netted out. For the ANOCOVA case, see the fixed effects model (Maddala, 1971; Searle, 1971). For our proposed methods for the analysis of short-term health effects, see Whittemore and Korn (1980).

A panel study has three other major advantages over a cross-sectional study. First, it usually provides finer detail on timing. The finer detail on timing of health events allows us to create better weather and air pollution exposure measures than is possible with data aggregated over several months. The better the weather and exposure measures, the lower the bias in the air pollution variable coefficients. Second, panel data sets keep the movers and deaths in the sample, whereas retrospective surveys frequently lose data on movers and deaths. To the extent that air pollution may cause moves or earlier death, cross-sectional data sets will tend to have sites with samples with different unobserved characteristics, which will yield a biased set of estimates. Third, with a panel data set, we can check any assumptions about aggregation over time by examining the response in disaggregated as well as aggregated form.

Quality of Air Pollution Exposure Measure

Ideally, we would like to know each individual's history of exposure to air pollution. This would include data on levels and timing of all pollutants from any source--smoking, other indoor pollution sources, and ambient air sources. Unfortunately, no existing data sets continuously monitored each person's exposure to air pollution, with one exception.[1] Instead, we must create synthetic measures based on available data. These synthetic exposure estimates necessarily measure exposure with substantial error. But that measurement error can be reduced by selecting data sets that provide finer detail on geographic location and the timing of health events.

This may be viewed as a classic errors-in-variable problem. Air pollution exposure is measured with error because we do not have continuous monitoring for each person. Instead, we use a proxy variable such as a weighted average of surrounding monitoring stations. The finer the level of data on work and home location, the closer the synthetic measure will be to the person's true ambient air exposure. The closer the measure to the true value, the smaller the variance in measurement error and the smaller the bias in the estimated coefficient for the air pollution variable (Maddala, 1977, pp. 292-294). Other variables will also be affected by measurement error in the exposure variable because the measurement error in one variable transmits bias to all correlated independent variables. The finer the level of detail on location, the less the transmitted bias will be.

The same argument holds for the quality of the temporal match of health outcomes and air pollution exposure. Some data sets ask how much time the respondent has lost from work or school due to illness during the last several months. Without knowledge of the dates of illness, we cannot create an accurate measure of air pollution that the respondent was exposed to immediately *before* the illness. Similarly, we run the risk that we will inappropriately estimate sickness from five months ago as a function of last month's air pollution if we use a data set that has information based on questions of the form: Have you ever . . .?

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[1]The only data set available to date that continuously monitored each person's air pollution exposure was collected in EPA's recent Urban

The quality of the match is especially important for transient conditions. Some respiratory responses to air pollution are short-term and not cumulative. The poorer the temporal match and the more variable the exposure, the more likely we are to misestimate the effects of air pollution on flare-ups of chronic respiratory diseases.

Quality of Weather Data Match

Ideally, we would like to control for the weather that an individual is exposed to in order to avoid attributing to air pollution the adverse health effects associated with bad weather. For example, Denver tends to have its worst air pollution in the winter. But winter is also the season with the highest rate of cardiopulmonary problems. Failure to control for weather would overstate the adverse effects of air pollution if bad weather and air pollution were positively correlated, and understate the effects of air pollution if the two were negatively correlated. A data set that fails to provide sufficient information on location and timing, to allow matching with weather data, is therefore undesirable.

Comprehensiveness of Data Set

Ideally, we would use the same sample and variable specifications for all health outcome measures. For example, we should avoid taking health status outcomes from one sample and sick loss from another. By using the same sample, we have the same target population and the same meaning for each independent variable. Thus, when we say the effect of a variable is such and such, it means the same thing for each health outcome.

By using the same data file, we can also measure the degree to which the outcomes are correlated, that is, the extent to which large changes in health status are associated with large expenditures and sick-loss time. Knowing this correlation allows us to determine the pattern of incidence of adverse effects. Are they limited to few people or to many? If the responses are highly correlated, then we may be able to

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Scale Study in Washington, D.C., and Denver, Colorado. However, that data set does not contain any information on health outcomes.

use a simple outcome measure as a good proxy for all the dimensions of health.

Valid and Reliable Health Measures

For this analysis, we need valid and reliable measures of general health status as well as the presence and severity of certain specific health complaints (e.g., chronic bronchitis). We will need a general health status measure if we are to detect the effect of air pollution on health status in a population not suffering from chronic cardiopulmonary problems. In such a population, the effect of air pollution may be headaches, general malaise, and other "diffuse" problems.

We also need to measure the presence and severity of specific cardiopulmonary problems. The data on presence of a condition will allow us to identify the population that is most susceptible to air pollution and to measure that susceptibility. The data on severity of a condition is important because we expect that the major effect of air pollution is to worsen existing conditions instead of cause them in the first place.

For both general and specific complaints, we would prefer objective continuous measures (e.g., lung functions from a spirometric examination) or scales based on multiple items over the commonly available single-item response (e.g., How would you rate your health--excellent, good, fair, poor?). The coarser measures have suppressed a good deal of information about health status in their simplification. That additional information would make it easier to detect smaller adverse effects of air pollution.

Adequate Within-Site Data

In our original proposal, we suggested that all of the analysis should be done within a site, with separate results for separate sites. There were two major reasons for that suggestion. First, one unit of an air pollution measure is not the same thing in two different sites because different sites use different equipment and maintenance schedules, and set different internal standards. Second, any omitted variables correlated with site (and hence air pollution) can lead to biased estimates of the response surface. Third, the response of those

accustomed to air pollution exposure may be different for a given level of pollutant from that of persons not so accustomed. Thus, while there may be a dose-related response at each site, the response to a given level may be quite different across sites.

The choice of panel versus cross-sectional data and a desire for finer geographical and temporal detail are related to this point. First, in a cross-sectional data set, all of the within-person variation in air pollution exposure has been lost. If there is no within-person variation for an individual in air pollution, we cannot identify the individual component in the error term. To the extent that this individual component may be correlated with health status or air pollution exposure, the parameter estimates may be biased. Second, in a cross-sectional data set, most if not all of the within-site variation in air pollution has been lost if we cannot identify PSUs smaller than SMSAs; staggered surveys such as the HIS are an exception. If there is little or no within-site variation in air pollution, we cannot identify the site-specific component in the error term; as Hausman, Ostro, and Wise have shown, this can be important. Again, to the extent that omitted site effects are correlated with unobserved variables (including pollution mix and level), the parameter estimates will be biased. In either case, whether there is bias or not, failure to account for correlation among observations yields inefficient parameter estimates and incorrect (biased upward) inference statistics.

In addition to possible bias and efficiency concerns, the suppression of intrasite and intertemporal variation can cause a major loss in precision. There is substantial intertemporal and intrasite variation in air pollution, which is lost in cross-sectional data, especially if the geographic detail is of low quality. The standard deviation of a variable's estimated coefficient is inversely related to the variance of the variable. Thus, eliminating intrasite and intertemporal variation in air pollution reduces the variance in the exposure measure, and increases the standard deviation of the estimated coefficient.

Even if we pool all of the sites in the estimation phase, we would like to have enough respondents in each site so that we could find susceptibles in the heavily as well as lightly polluted sites. If this

condition holds, we will be able to contrast the response of susceptibles and nonsusceptibles to air pollution.

Variance in Air Pollution Across Sites

There should be substantial variation in the level and mix of air pollutants. We need variation in the levels of air pollutants in order to detect the response to air pollution. We need variation in the mix to determine the different effects of each pollutant. For example, TSP may be more cumulative while oxidants may be transitory in their impact.

ALTERNATIVE DATA SETS

Using the criteria just described, we examined five data sources: HANES I and II, the HIE, the HIS, NMCES, and NMCUES.

HANES I and II

The HANES surveys by the National Center for Health Statistics (NCHS) provide cross-sectional data on health and nutrition. Conducted in 1971-1974 and 1976-1980, they provide data on some 28,00 and 21,000 individuals in national probability samples, respectively. HANES determined the prevalence of a number of chronic conditions including coughing, asthma, hay fever, and other cardiopulmonary conditions. HANES I provides no self-reported measure of the severity of each respiratory complaint, but HANES II provides data on total work or sick-loss days during the past 12 months that were attributable to respiratory problems (other than flu and colds). There are no data on dates of sick loss. Both versions have spirometry measurements of lung function for some subset of the respondents. The only general measure of health status is the question of whether health is excellent, good, fair, or poor.

The HANES data have extremely limited information on use of health services. Most questions are of the form: Have you ever seen a doctor or been hospitalized for condition X? There is no information on how much the person has spent on the condition, or when and how often he spent it.

The ability to create synthetic measures of air pollution exposure is limited. The available geographic detail specifies the respondent's SMSA only, and then only for the largest SMSAs. Both HANES data sets contain information on smoking. Neither contains information on other indoor air pollution sources.

HIE

The Health Insurance Experiment is a randomized trial, designed to study the effects of cost sharing in HMOs on the health status, health-care use, and sick-loss of the nonaged population. The HIE enrolled some 7,770 individuals in six sites (Dayton, Ohio; Seattle, Washington; Fitchburg, Massachusetts; Franklin County, Massachusetts; Charleston, South Carolina; and Georgetown County, South Carolina). While none of these sites were extremely polluted during the mid- and late 1970s, when the HIE data were collected, each had substantial air pollution. None of them met federal ambient air quality standards during that period.

In addition to the exclusions common to all the other data sets (e.g., the military and the institutionalized), the HIE excludes the aged (62 and over) and the top seven percent of the income distribution. These twin exclusions (especially the exclusion of the elderly), the smaller sample size, the small number of sites, and the absence of a severely polluted site are the HIE's major limitations.

The HIE is a panel study. It contains repeated measurements of health status (general and condition specific), as well as dated information on health-service use and sick-loss days. The use-data include information on diagnoses, procedures, and medication prescribed and purchased. The general health status measure is a Likert-type summated rating scale based on 22 questions. Thus, the construct is a subjective assessment of personal health status. Its reliability and validity have been extensively studied (Ware, 1976; Davies and Ware, 1981). Manning, Newhouse, and Ware (1982) have shown that this measure performs significantly better than Excellent/Good/Fair/Poor in a study of health-care utilization. The study contains several measures of chronic and role limitations, the presence or absence of 26 chronic conditions, and severity measures. For several of the chronic

conditions, there exist multiple measures of prevalence and severity based upon both self-report and physical examination (with lung function measurement) by an M.D. The physical exam was administered to a random subsample at enrollment in the study and to everyone at exit. The report by Foxman et al. (1982) describes the measures for chronic obstructive airway disease.

We can build better synthetic measures for the HIE than for any of the other data sets. The HIE contains information on home zip code, dates of moves, work zip code and hours worked, and (own and family members') smoking status and history. Thus, we can build exposure measures that incorporate data from the monitoring stations not only nearest to the respondent's home but also to his work location. This definitely has less measurement error than an SMSA average variable. Also, it allows us increased precision through capturing the within-site variance in the air pollution measure.

The HIE has one further advantage over all other data sets. The HIE randomly assigned insurance plans of varying levels of generosity to enrollees. In other data sets, families can choose their own coverage by buying individual policies or by selecting which work-related policy should be used for dependents. In the HIE, random assignment breaks that correlation so that we can determine what is sickliness (here in the cardiopulmonary sense) and what is insurance coverage.

HIS

The Health Interview Survey conducted by HCHS is a continuing survey of health-related problems in the United States. Although there are repeated waves of the survey in each site, the survey does not resample the same set of individuals, except by accident. As a result, the HIS has to be considered a cross-sectional survey if the unit of analysis is to be an individual. Although similar in content, there are differences among the waves in the specific information elicited from respondents. About 120,000 individuals are sampled each year.

HIS determined the prevalence and severity of several chronic complaints including cardiopulmonary conditions (e.g., asthma, bronchitis). The severity questions ask (1) whether the individual is bothered by the condition all the time, often, once in a while, or

never, and (2) when it does bother him, whether he is bothered a great deal, some, or very little. Because HIS is a survey, there are no data on lung function. The HIS data lack a general health measure but do contain information on chronic and role limitations due to ill health, and which chronic condition is the main source of that limitation.

The HIS data contain fairly detailed information on health-care utilization and sick-loss days during the past two weeks. The survey contains questions on medical and dental visits, hospitalizations, work or school-loss days, bed disability days, and restricted activity days. In some waves of the HIS, the date of the visit or sick-loss day is provided.

The ability to create synthetic measures of air pollution is limited. The available geographic detail specifies SMSA only, and that only for the largest SMSAs. Because the responses on health-care use and sick-loss are dated or limited to a specified two-week period, the quality of the temporal match of health events and air pollution is good. The quality of the temporal match is probably exceeded only by that for the HIE data. The HIS contains information on smoking, but not on indoor air pollution sources.

NMCES and NMCUES

These two surveys are very similar in content and construction. NMCES was conducted by NCHSR on a national probability sample of 40,320 people. NMCUES was conducted by NCHS on a national probability sample of 17,000 people plus 24,000 people from the Medicaid population of four states. Each survey conducted repeated interviews for the same sample of individuals, with all responses and health events dated. Thus, the primary data are in panel form. Unfortunately, the public use files released from these two surveys have aggregated the responses into a cross-sectional data set.

The information on health status is more limited than on the other data sets. The two surveys contain data on general health status (the excellent/good/fair/poor question) as well as responses to questions on chronic and role limitations. Information on the existence of a chronic condition was collected only if there was a medical visit, sick-loss day, or limitation due to that condition. Thus, an individual with a

chronic condition in good control (i.e., no flare up) cannot be distinguished from someone without the condition. To the extent that air pollution aggravates a health condition, we cannot distinguish prevalence from severity in these two surveys. The information on conditions is not available in the cross-sectional version of the files.

Both NMCES and NMCUES have detailed information on health-care utilization and sick-loss. These include visits, hospitalization, expenditures, bed, disability and restricted-activity days, and the condition for each. There is detailed information on insurance coverage and reimbursement. Except for the HIE, this data set has the most complete insurance information of the files considered.

The severest drawback of these data sets is their inability to produce more than crude synthetic measures of air-pollution exposure. The NMCUES is expected to identify the respondent by SMSA only if the respondent is in one of the largest SMSAs. NMCES identifies census region and SMSA size but does not name the SMSA. The quality of the temporal match with the underlying file could be quite good because health events are dated. For the cross-sectional versions, the temporal match will be poor because we do not know when the event occurred over the several months period surveyed. Neither data set has smoking data.

CHOICE OF DATA SETS

After reviewing the characteristics of the available data sets for analyzing the adverse effects of air pollution on health outcomes, we have decided to use the HIE. However, because the HIE has some limitations, and other data sets some advantages, we propose that the second phase of the RAND cooperative agreement with EPA use augmented versions of some of the other candidate data files, *if certain of their shortcomings can be overcome*.

We prefer to use the HIE for our initial study because:

1. The HIE is a panel study while the others (in their present form) are cross-sectional. As mentioned earlier, with panel data on individuals, we can avoid the potential selection bias in cross-sectional data sets.

2. The HIE data can be augmented with both weather and air pollution data to provide a better geographic and temporal match than is possible with other data sets. This reduces the measurement error in generating an estimate for air pollution and weather exposure. The lower the measurement error, the lower the errors-in-variable bias in the estimates of the air pollution and weather coefficients.
3. The HIE has the most comprehensive set of health outcome measures: health-care utilization (diagnosis, procedure, and medications), sick-loss days, self-perceived health status (general and condition specific), prevalence and severity of chronic complaints, and lung function. Other data sets provide only a subset of these data.
4. The health status measures in the HIE have been validated and shown to be as reliable as or more reliable than those on other data files.

The HIE has the following disadvantages:

1. The HIE's exclusion of the elderly is an important limitation because they are a susceptible population that may behave differently from the nonaged. The effects of this limitation can only be studied by checking HIE results with other data sets.
2. The HIE has a smaller sample size than the other data sets. Nevertheless, given the lesser precision and the bias associated with cross-sectional data with limited geographic detail, the HIE's finer geographic and temporal detail will partially offset this limitation.
3. The HIE sites cover a more limited range of air pollution. The effect of this limitation can only be studied by checking HIE results with other data sets that have a wider range of air pollution levels.

We believe that the advantages of the HIE outweigh its disadvantages.

The discussion above centered on measuring (dose) responses in terms of health outcomes--utilization, health status, or sick loss--as they relate to air pollution. One of the major purposes of our work is to estimate how people value changes in levels of air pollution. To do so, we will require measures of the value of changes in health-care use, health status, and sick-loss induced by changes in air pollution. We will need data on the cost of services, the value of health status, and the opportunity cost of time. Of the data sets considered, only the HIE, NMCES, and NMCUES provide data on the cost of health services. For the cross-sectional versions of NMCES and NMCUES, the data have been aggregated so that we cannot separate cardiopulmonary from other health services (e.g., mental health treatment or maternity care). Of the data sets considered, only the HIE has sufficient labor market data to determine the opportunity cost of time. Work on this issue is now being done as part of the HIE's research for the Department of Health and Human Services. We hope to use these results in our evaluation of sick-loss.

Two evaluation problems remain. First, it is necessary to evaluate changes in health status. Second, none of the data sets available has adequate sample or data to estimate the effects of air pollution on mortality. Such an analysis will require the use of other files, or novel ways of using existing files.

Appendix B

STATISTICAL METHODS

To study the effect of air pollution on annual rates of illness and use of services, we will examine the response of participants in terms of their annual number of days lost due to illness and annual expenditures for ambulatory medical care. Rather than rely on the more common analysis of variance (ANOVA) or analysis of covariance techniques (ANOCOVA), we have used a two-part model for ambulatory medical expenditures and a negative binomial regression model for days of illness. These choices were dictated by two characteristics of these two health outcomes. First, a large proportion of the HIE participants use no medical services or have no time lost due to illness. Second, the distribution of expenses and days of illness is very skewed.

These characteristics imply that ANOVA and ANOCOVA techniques will yield imprecise (though unbiased) estimates of the effects of air pollution, even for a fairly large sample size such as that in the HIE. As Duan, Manning, Morris, and Newhouse (1983) have shown for use of medical services, a model that exploits the characteristics of the distribution of utilization can provide more precise estimates.

In this appendix, we describe our statistical methods. The topics include: the two-part model for estimating outpatient expenditures, and the negative binomial regression model for estimating days lost to illness.

TWO-PART MODEL FOR AMBULATORY MEDICAL EXPENSES

We use two equations to model the distribution of ambulatory medical expenses. The first is a probit equation for the probability that a person will receive any outpatient service during a year. This equation separates users from nonusers and therefore addresses the first characteristic described above. The second equation is a linear regression for the logarithm of total annual outpatient medical expenses of users. The log transformation of annual expenses for the group of users reduces dramatically the undesirable skewness in the distribution of expenses among users described as the second characteristic earlier.

We therefore expect the estimates from this model to be more robust than those that might be obtained from ANOVA and ANOCOVA models on untransformed expenses.

More formally, the first equation is a probit equation for the dichotomous event of zero versus positive ambulatory expense:

$$I_{1i} = x_i\beta_1 + \varepsilon_{1i},$$

$$\varepsilon_{1i} \sim N(0, 1),$$

$$E(\varepsilon_{1i} \mid x_i) = 0,$$

where ambulatory expense is positive if $I_{1i} \geq 0$, 0 otherwise; and x_i is a row vector of given individual characteristics (e.g., air pollution and age).

The second equation is a linear model on the log scale for positive ambulatory medical expenses *if* the person receives any services:

$$\ell n(AMB\$_i \mid I_{1i} \geq 0) = x_i\beta_2 + \varepsilon_{2i},$$

where $E(\varepsilon_{2i} \mid x_i, I_{1i} \geq 0) = 0$, x_i is a row vector of given individual characteristics and ε_{2i} is i.i.d. For the last equation, the error is not assumed to be normally distributed.

The likelihood function for this model is multiplicatively separable because of the way the conditional densities are calculated. The separability does *not* depend on any assumption of independence between errors in the two equations. In fact, the errors may be correlated. Separability implies that estimating the two equations by maximum likelihood *separately* provides the global full information maximum-likelihood estimates; see Manning et al. (1981), and Duan et al. (1983, 1984). We therefore estimate the two equations separately.

If the error term ε_2 in the (log) expense equation were normally distributed, then the expected ambulatory medical expense would be

$$E(AMB\$_i) = p_i \exp(x_i\beta_2 + \sigma^2_{\varepsilon 2}/2)$$

where

$$p_i = \text{Prob.}_i(AMB\$_i > 0) = \Phi(x_i\beta_1),$$

Φ = normal c.d.f.,

and where the factor $\exp(\sigma^2/2)$ is the adjustment in the mean for retransformation in the second (or conditional) equation if ε_2 were normally distributed. However, the normal assumption for ε_2 is not satisfied for the ambulatory expense data, because the residual distribution is not normally distributed. As a result of this nonnormality, the factor $[\exp(\sigma^2/2)]$ is not the correct adjustment in the mean for the retransformation from the logarithmic scale to the untransformed dollar scale and would lead to statistically inconsistent predictions of the mean expenditure.

Instead of the normal retransformation, we use the smearing estimates developed by Duan (1983). The smearing estimate, a nonparametric estimate of the retransformation factor $\phi = E(\exp(\varepsilon_2))$, is the sample average of the exponentiated least squares residuals. The smearing estimate is statistically consistent for the retransformation factor if the error distribution does not depend on the characteristics x.

A consistent estimate of the expected expense for ambulatory medical services is therefore provided by

$$E(AMB\$_i) = p_i \exp(x_i\beta_2)\phi$$

where

$$\phi = \sum \exp(\ell n\ Y_i - x_i\beta_2)/n$$

where

β_2 is a consistent estimate of β_2.

Correlation in the Error Terms in the Two-Part Model

Although we have observations for several thousand person-years of data, we do not have that number of *independent* observations. The error terms in our equations exhibit substantial positive correlations among family members and over time for individuals. These correlations exist in both equations. Failure to account for these correlations in the analysis would yield inefficient estimates of the coefficients and statistically inconsistent estimates of the standard errors. As a result, the inference statistics (t, F, and χ^2) calculated in the usual way (without adjusting for these correlations) would be too large.

All inference statistics (t, F, χ^2) reported in this report have been corrected for correlation, using a nonparametric correction similar to the random effects or intracluster model. The correction method is fully described by Rogers (1983), based on prior work by Huber (1967).

NEGATIVE BINOMIAL MODEL FOR TIME LOST TO ILLNESS

We used a negative binomial regression model to estimate the response of time lost to illness to air pollution. The negative binomial distribution is an appealing model because it can yield a large proportion of zero days and a skewed distribution of positive days; thus, the model can address the two characteristics of time lost due to illness mentioned earlier. The negative binomial model is more appealing for days than a two-part model because the negative binomial model has discrete outcomes while the two-part model has continuous outcomes. The negative binomial regression model is more appealing than a Poisson regression because the variance of days exceeds the mean; data from a Poisson distribution should have equal mean and variance.

The negative binomial model can be generated from an underlying Poisson model. Let each individual's (i's) days be drawn independently from a Poisson distribution. If different individuals have different rates that are sampled from a gamma distribution, then the observed number of days follows a negative binomial distribution where

$$\text{prob(days = n)} = \frac{\alpha+n-1}{\alpha-1} \quad \frac{\beta^{\alpha}}{1+\beta} \quad \frac{1^{n}}{1+\beta}$$

The expected values for the sample mean and variance of annual days are

$$E(\text{days}) = \alpha/\beta$$

$$\text{Var(days)} = \alpha(1+\beta)/\beta^{2}.$$

As long as β is positive, the variance exceeds the mean.

In the results below, we assume that the parameter β can be expressed as a linear combination of observed individual characteristics:

$$\ell n\beta = - x_i \delta_i,$$

where x_i is row vector of given individual characteristics, including an intercept. We assume that α is a constant.

As noted earlier, there is a substantial positive correlation among family members in their number of days of illness. In the days results, we have corrected the inference statistics for this positive correlation. This correction is similar to that of the random effects least-squares model or, equivalently, the intracluster correlation model. The correction method is fully described by Rogers (1983), based on prior work by Huber (1965).

Appendix C

INDIVIDUAL DAY-TO-DAY APPROACH: ADDITIONAL METHODS AND RESULTS

CORRELATION COEFFICIENTS

Tables C.1 and C.2 give the correlation coefficients for the explanatory variables for the individual day-to-day (Whittemore-Korn) analysis. For each pair of explanatory variables, the upper entry in the exhibit is the correlation coefficient, and the lower entry is the probability that the correlation coefficient is zero. Table C.3 gives the multiple correlation coefficient for each explanatory variable with the rest of the explanatory variables. The conclusions we draw from this analysis are given in Sec. VII.

DESCRIPTIVE SUMMARIES OF INDIVIDUAL RESPONSES

Tables C.4 and C.5 and Figs. C.1 through C.21 summarize the responses of the 1,238 persons who have more than three sick episodes and at least one hundred days available for our analysis. Table- C.4 gives the unweighted major summary statistics for the estimated individual responses; the results in the text section of Sec. V are for a weighted analysis. The top part of the exhibit gives the average of the individual responses along with other univariate summary statistics.[1] The bottom part of the exhibit gives the correlation coefficients and p values.' Further summary statistics and graphical

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[1] The units are as follows: response to SO_2 is in terms of logit per ppm SO_2, response to COH is in logit per COH, response to TSP is in terms of logit per $\mu g/m^3$, response to NO_2 is in logit per ppm NO_2, response to minimum temperature is in logit per degree Fahrenheit, response to precipitation is in logit per inch of precipitation.

[2] The number of properly estimated response coefficients to precipitation is 1185 instead of 1238, the total number of persons in the final analysis sample. There are 53 persons in the final analysis sample who never had a sick episode on a wet day, so their response to precipitation on the logit scale is minus infinity.

Table C.1

CORRELATION COEFFICIENTS FOR THE EXPLANATORY VARIABLES IN BELLEVUE

	S02AV	COHAV	TSPAV	OZOMX	N02MX	MINTEMP	PRECIP	WKDAY	FIRSWEEK
S02AV	1.00000 0.0000	0.49997 0.0001	0.18866 0.0001	0.10526 0.0044	0.41334 0.0001	-0.08572 0.0206	-0.12815 0.0005	0.10834 0.0034	0.04797 0.1957
COHAV	0.49997 0.0001	1.00000 0.0000	0.39398 0.0001	-0.35062 0.0001	0.56904 0.0001	-0.41058 0.0001	-0.03169 0.3923	0.19711 0.0001	-0.03011 0.4163
TSPAV	0.18866 0.0001	0.39398 0.0001	1.00000 0.0000	-0.07207 0.0514	0.21604 0.0001	-0.17676 0.0001	-0.12084 0.0011	0.15048 0.0001	0.03795 0.3056
OZOMX	0.10526 0.0044	-0.35062 0.0001	-0.35062 0.0514	1.00000 0.0000	-0.29838 0.0001	0.42488 0.0001	-0.012404 0.0008	-0.02514 0.4974	-0.00253 0.9455
N02MX	0.41334 0.0001	0.56904 0.001	0.21604 0.0001	-0.29838 0.0001	1.000000 0. 0000	-0.19875 0.0001	-0.13663 0.0009	0.15546 0.0001	0.02246 0.5861
MINTEMP	-0.08572 0.0206	-0.41058 0.0001	-0.17676 0.0001	0.42488 0.0001	-0.19875 0.0001	1.00000 0.0000	-0.01918 0.6047	-0.02091 0.5725	0.02936 0.4280
PREC I P	-0.12815 0.0005	-0.03196 0.3923	-0.12084 0.0011	-0.12404 0.0008	-0.13663 0.0009	-0.01918 0.6047	1.00000 0.0000	0.03979 0.2827	0.02902 0.4333
WKDAY	0.10834 0.0034	0.19711 0.0001	0.15048 0.0001	-0.02514 0.4974	0.15546 0.0001	-0.02091 0.5725	0.03979 0.2827	1.00000 0.0000	-0.00043 0.9907
FIRSWEEK	0.04797 0.1957	-0.03011 0.4163	0.03795 0.3056	-0.00253 0.9455	0.02246 0.5861	0.02936 0.4280	0.02902 0.4333	-0.00043 0.9907	1.00000 0.0000

Table C.2

CORRELATION COEFFICIENTS FOR THE EXPLANATORY VARIABLES IN DOWNTOWN SEATTLE

	S02AV	COHAV	TSPAV	OZOMX	NO2MZ	MINTEMP	PRECIP	WKDAY	FIRSWEEK
S02AV	1.00000 0.0000	0.49388 0.0001	0.30115 0.0001	0.09722 0.0086	0.41303 0.0001	-0.08544; 0.0210	0.12846 0.0005	0.10761 0.0036	0.05276 0.1547
COHAV	0.49388 0.0001	1.00000 0.0000	0.40775 0.0001	-0.38643 0.0001	0.56779 0.0001	-0..9935 0.0001	-0.02913 0.4316	0.20941 0.0001	-0.03211 0.3860
TSPAV	0.30115 0.0001	0.40775 0.0001	1.00000 0.0000	0.02248 0.5439	0.29975 0.0001	-0.10357 0.0051	-0.11558 0.0017	0.24736 0.0001	0.00372 0.9199
OZOMX	0.09722 0.0086	0.09722 0.0001	-0.38643 0.5439	1.00000 0.0000	-0.28469 0.0001	0.40125 0.0001	-0.09348 0.0114	-0.04019 0.2779	0.00674 0.8557
N02MX	0.41303 0.0001	0.56779 0.0001	0.29975 0.0001	-0.28469 0.0001	1.00000 0.0000	-0.19875 0.0001	-0.13663 0.0009	0.15546 0.0001	0.02246 0.5861
MINTEMP	-0.08544 0.0210	-0.39935 0.0001	0.10357 0.0051	0.40125 0.0001	-0.19875 0.0001	1.00000 0.0000	-0.01918 0.6047	-0.02091 0.5725	0.02936 0.4280
PREC I P	-0.12846 0.0005	-0.02913 0.4316	-0.11558 0.0017	-0.09348 0.0114	-0.13663 0.0009	-0.01918 0.6047	1.00000 0.0000	0.03979 0.2827	0.02902 0.4333
WKDAY	0.10761 0.00036	0.20941 0.0001	0.24736 0.0001	-0.04019 0.2779	0.15546 0.0001	-0.02091 0.5725	0.03979 0.2827	1.00000 0.0000	-0.00043 0.9907
FIRSWEEK	0.05276 0.1547	-0.03211 0.3860	0.00372 0.9199	0.00674 0.8557	0.02246 0.5861	0.02936 0.4280	0.02902 0.4333	-0.00043 0.9907	1. 00000 0.0000

summaries for the individual responses are given in Figs. C.l through C.7.

Generally speaking, the estimated individual responses have long-tailed distributions characterized by a few outliers. For example, for almost all people, the response to sulfur dioxide ranges between plus and minus six hundred. However, there is one person whose response (i.e., coefficient) to $S0_2$ is -1927, and another person response is 717.

The estimated individual responses are very heterogeneous. Some individuals have a large number of episodes, so we have more information on their responses. The standard errors for those individuals estimated from the logistic regression would be likely to be small. For individuals with the fewest episodes, the logistic regression model might be ill-conditioned and the estimates might be unstable. For those individuals, the standard error might be very large. Those individuals are also likely to have large estimated responses. Figures C.8 through C.14 give the scatterdiagrams of the estimated individual responses by the corresponding estimated standard errors. It can be seen that practically all estimated individual responses which are utliers are associated with large tandard errors.

Table C.3

MULTIPLE CORRELATION COEFFICIENTS
FOR THE AEROMETRIC DATA

Attribute	Bellevue	Downtown Seattle
SO2	0.40	0.42
COH	0.57	0.59
TSP	0.25	0.24
Ozone	0.26	0.35
NO2	0.32	0.29
Precipitation	0.25	0.25
Minimum temp.	0.11	0.08

Table C.4

MAJOR SUMMARIES OF THE INDIVIDUAL RESPONSES

VARIABLE	N	MEAN	STD DEV	SUM	MINIMUM	MAXIMU
S02AV	1238	-21.31056558	101.27916599	-26382.48018961	-1927.37167794	717.3526014
COHAV	1238	-0.48026882	2.24799933	-594.57280312	-26.81823368	7.5552934
TSPAV	1238	-0.00454673	0.02400547	-5.62885480	-0.21143901	0.1089210
OZOMX	1238	-18.36721365	42.72269690	-22738.61050183	-312.54080879	137.4384898
N02MX	1238	-5.79224831	31.01832979	-7170.80340487	-515.04540605	96.0278276
MINTEMP	1238	-0.00849611	0.09201381	-10.51819023	-0.90863745	0.6902243
PRECIP	1185	-2.36195872	7.13577729	-2798.92108612	-72.18282141	5.6012901

CORRELATION COEFFICIENTS / PROB > IRI UNDER HO:RHO=0 / NUMBER OF OBSERVATIONS

	S02AV	COHAV	TSPAV	OZOMX	N02MX	MINTEMP	PRECIP
SO2AV	1.00000 0.0000 1238	-0.08239 0.0037 1238	0.02421 0.3946 1238	-0.02554 0.3693 1238	-0.18341 0.0001 1230	0.02005 0.4809 1238	0.02522 0.3858 1185
COHAV	-0.08239 0.0037 1238	1.00000 0.0000 1238	-0.13746 0.0001 1238	0.21437 0.0001 1238	-0.16125 0.0001 1238	0.23503 0.0001 1238	0.05996 0.0390 1185
TSPAV	0.02421 0.3946 1238	-0.13746 0.0001 1238	1.00000 0.0000 1238	-0.02679 0.3462 1238	0.02237 0.4316 1238	-0.03233 0.2557 1238	0.04775 0.1004 1185
OZOMX	-0.02554 0.3693 1238	0.21437 0.0001 1238	-0.02679 0.3462 1238	1.00000 0.0000 1238	0.06755 0.0174 1238	-0.20027 0.0001 1238	0.17078 0.0001 1185
N02MX	-0.18341 0.0001 1238	-016125 0.0001 1238	0.02237 0.4316 1238	0.06755 0.0174 1238	1.00000 0.0000 1238	0.10795 0.0001 1238	0.07133 0.0140 1185
MINTEMP	0.02005 0.4809 1238	0.23503 0.0001 1230	-0.03233 0.2557 1238	-0.20027 0.0001 1238	0.10795 0.0001 1238	1.0000 0.0000 1238	-0.14170 0.0001 1185
PRECIP	0.02522 0.3858 1185	0.05996 0.0390 1185	0.04775 0.1004 1185	0.17078 0.0001 1185	0.07133 0.0140 1185	-0.14170 0.0001 1185	1.00000 0.0000 1185

Table C.5

MAJOR SUMMARIES OF THE INDIVIDUAL z STATISTICS FOR THE INDIVIDUAL RESPONSES

VARIABLE	N	MEAN	STD DEV	SUM	MINIMUM	MAX t MU
T1	1238	-0.04484522	1.02337494	-55.51838334	-4.57415889	3.4111633
T2	1238	-0.10446429	1.04585081	-129.32678990	-3.10154452	3.4335113
T3	1238	-0.07602341	0.99911932	-94.11698568	-2.81939992	3.3619862
T4	1238	-0.32343950	0.99B55742	-400.41810600	-3.32241690	2.8717092
T5	1238	-0.04400085	1.14199810	-54.47305559	-3.43162632	3.7636566
T6	1238	-0.21302980	1.12947762	-263.73089191	-4.13131252	3.6067947
T7	1185	0.02755357	0.88106644	32.65097658	-2.06813777	3.2595051

CORRELATION COEFFICIENTS / PROB > IRI UNDER HO:RHO=0 / NUMBER OF OBSERVATIONS

	T1	T2	T3	T4	T5	T6	T7
T1	1.00000 0.0000 1238	-0.23740 0.0001 1238	0.03032 0.2865 1238	-0.12479 0.0001 1238	-0.18948 0.0001 1238	0.05386 0.0582 1238	0.07760 0.0075 ‘1185
T2	-0.23740 0.0001 1238	1.00000 0.0000 1238	-0.18316 0.0001 1238	0.20820 0.0001 1238	-0.30861 0.0001 1238	0.25993 0.0001 1238	0.05296 0.0684 1185
T3	0.03032 0.2865 1238	-0.18316 0.0001 1238	1.00000 0.0000 1238	-0.00876 0.7581 1238	0.01239 0.6633 1238	0.04067 0.1526 1238	0.04694 0.1063 1185
T4	-0.12479 0.0001 1238	0.20820 0.0001 1238	-0.00876 0.7581 1238	1.00000 0.0000 1238	0.06902 0.0151 1238	-0.25948 0.0001 1238	0.0001 1185
T5	-0.18948 0.0001 1238	-0.30861 0.0001 1238	0.01239 0.6633 1238	0.06902 0.0151 1238	1.00000 0.0000 1238	-0.05179 0.0685 1238	0.05934 0.0411 1185
T6	0.05386 0.0582 1238	0.25993 0.0001 1238	0.04067 0.1526 1238	-0.25948 0.0001 1238	-0.05179 0.0685 1238	1.00000 0.0000 1238	-0.12379 0.0001 1185
T7	0.07760 0.0075 1185	0.05296 0.0684 1185	0.04694 0.1063 1185	.18405 0.0001 1185	0.05934 0.0411 1185	-0.12379 0.0001 1185	1.00000 0.0000 1185

UNIVARIATE

VARIABLE=SO2AV

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-21.3106	SUM	-26382.5
STD DEV	101.279	VARIANCE	10257.5
SKEWNESS	-6.34703	KURTOSIS	106.822
USS	13250715	CSS	12688490
CV	-475.253	STD MEAN	2.87846
T:MEAN=0	-7.40347	PROB>|T|	0.0001
SGN RANK	-71568.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100%	MAX	717.353	99%	112.935	
75%	Q3	26.3113	95%	76.1036	
50%	MED	-4.76002	90%	54.2619	
25%	Q1	-46.423	10%	-109.544	
0%	MIN	-1927.37	5%	-156.522	
			1%	-380.825	
	RANGE	2644.72			
	Q3-Q1	72.7343			

EXTREMES

LOWEST	HIGHES
-1927.37	159.6
-593.428	178.35
-552.25	194.46
-530.778	203.76
-509.253	717.35

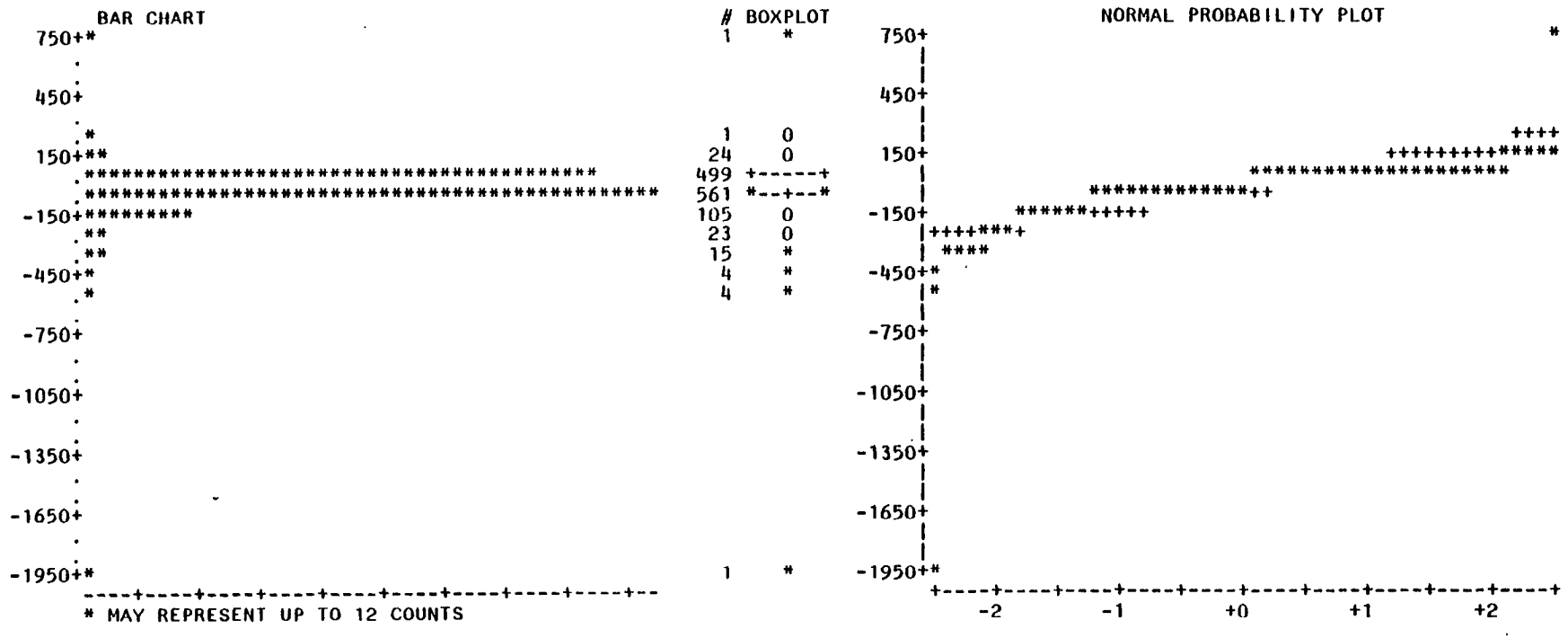


Fig. C.1--Further summaries of the individual responses to SO_2

UNIVARIATE

VARIABLE=COHAV

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-0.480269	SUM	-594.573
STD DEV	2.248	VARIANCE	5.0535
SKEWNESS	-2.42794	KURTOSIS	20.0433
USS	6536.74	CSS	6251.18
CV	-468.071	STD MEAN	0.0638904
T:MEAN=0	-7.51707	PROB>|T|	0.0001
SGN RANK	-78759.5	PROB>|S|	0.0001

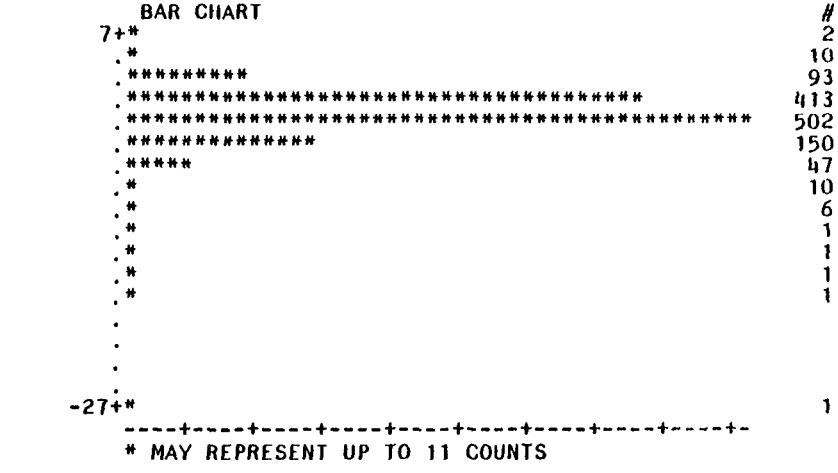
QUANTILES(DEF=4)

100%	MAX	7.55529	
75%	Q3	0.805572	
50%	MED	-0.271872	
25%	Q1	-1.39697	
0%	MIN	-26.8182	
	RANGE	34.3735	
	Q3-Q1	2.20254	

99%	4.01823
95%	2.41469
90%	1.80624
10%	-3.01064
5%	-4.13154
1%	-7.57121

EXTREMES

LOWEST	HIGHES
-26.8182	4.7786
-17.7454	4.8626
-14.1523	5.401
-12.2459	6.1885
-9.99829	7.5552



#	BOXPLOT
2	*
10	0
93	|
413	+-----+
502	*--+--*
150	|
47	0
10	0
6	*
1	
1	*
1	*
1	*
1	*

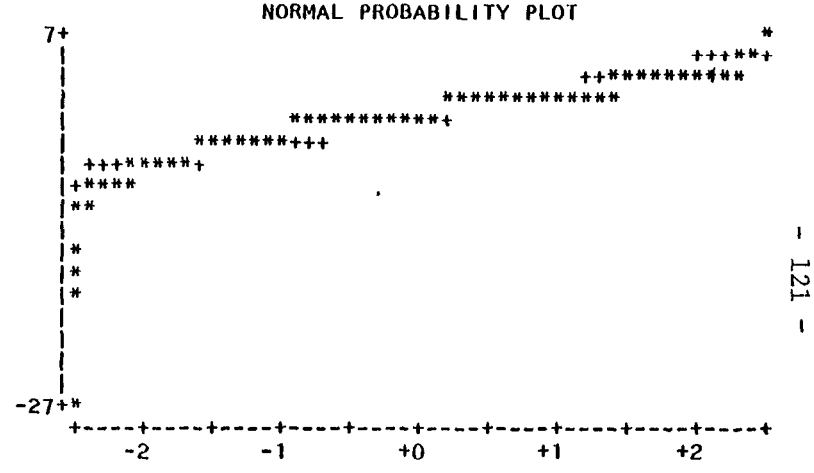


Fig. C.2--Further summaries of the individual responses to COH

UNIVARIATE

VARIABLE=TSPAV

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-.00454673	SUM	-5.62885
STD DEV	0.0240055	VARIANCE	.000576263
SKEWNESS	-1.85532	KURTOSIS	11.8126
USS	0.73843	CSS	0.712837
CV	-527.972	STD MEAN	0.00068226
T:MEAN=0	-6.66422	PROB>|T|	0.0001
SGN RANK	-71555.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100% MAX	0.108921	99%	0.0452668
75% Q3	0.00814413	95%	0.0270386
50% MED	-.00189206	90%	0.0185103
25% Q1	-0.0147184	10%	-0.0287881
0% MIN	-0.211439	5%	-0.0417656
		1%	-0.0870325
RANGE	0.32036		
Q3-Q1	0.0228625		

EXTREMES

LOWEST	HIGHES
-0.211439	0.054557
-0.17474	0.063480
-0.167015	0.078133
-0.149292	0.085432
-0.145252	0.10892

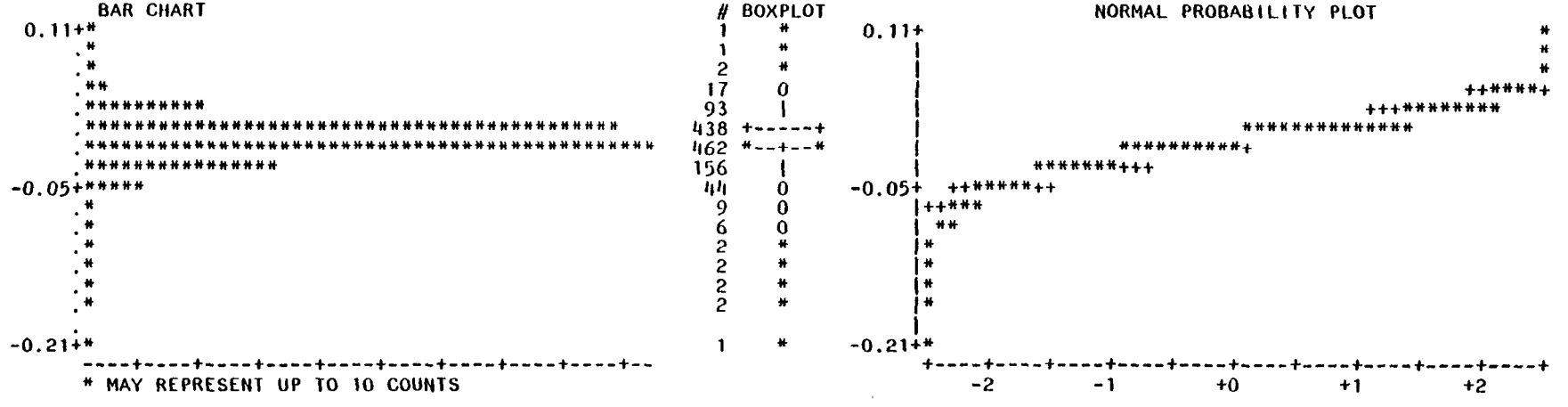


Fig. C.3--Further summaries of the individual responses to TSP

UNIVARIATE

VARIABLE=OZOMX

MOMENTS

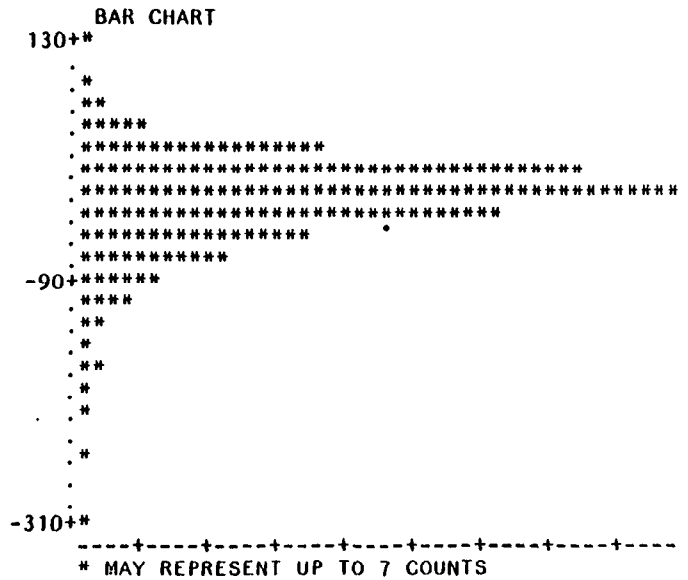
N	1238	SUM WGTS	1238
MEAN	-18.3672	SUM	-22738.6
STD DEV	42.7227	VARIANCE	1825.23
SKEWNESS	-1.3923	KURTOSIS	4.83137
USS	2675453	CSS	2257808
CV	-232.603	STD MEAN	1.21422
T:MEAN=0	-15.1267	PROB>|T|	0.0001
SGN RAND	-173278	PROB>|S|	0.0001

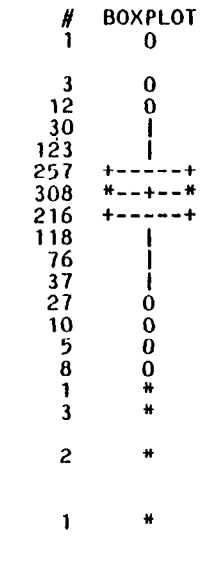
QUANTILES(DEF=4)

100% MAX	137.438	99%	67.0917
75% Q3	8.17751	95%	34.5356
50% MED	-11.2775	90%	24.9338
25% Q1	-37.2712	10%	-68.9922
0% MIN	-312.541	5%	-95.9529
		1%	-169.929
RANGE	449.979		
Q3-Q1	45.4487		

EXTREMES

LOWEST	HIGHES
-312.541	78.567
-253.312	82.017
-250.404	83.310
-216.287	87.097
-207.779	137.43





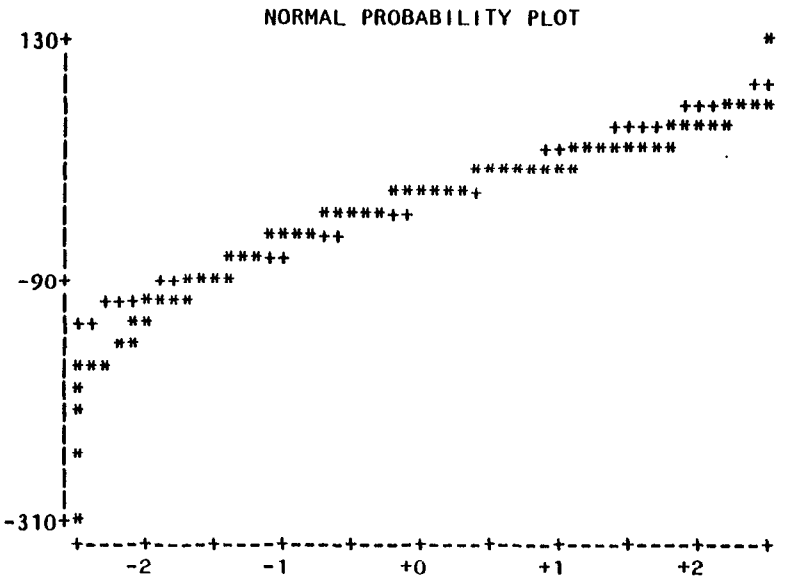


Fig. C.4--Further summaries of the individual responses to ozone

UNIVARIATE

VARIABLE=NO2MX

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-5.79225	SUM	-7170.8
STD DEV	31.0183	VARIANCE	962.137
SKEWNESS	-6.56881	KURTOSIS	87.4405
USS	1231698	CSS	1190163
CV	-535.515	STD MEAN	0.881573
T:MEAN=0	-6.57036	PROB>|T|	0.0001
SGN RANK	-63356.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100%	MAX	96.0278	99%	40.0155	
75%	Q3	9.50577	95%	25.6301	
50%	MED	-1.80924	90%	18.5293	
25%	Q1	-14.7215	10%	-33.2001	
0%	MIN	-515.045	5%	-47.8865	
			1%	-94.5008	
	RANGE	611.073			
	Q3-Q1	24.2272			

EXTREMES

LOWEST	HIGHES
-515.045	52.775
-392.219	58.785
-325.81	65.515
-178.084	73.263
-130.306	96.027

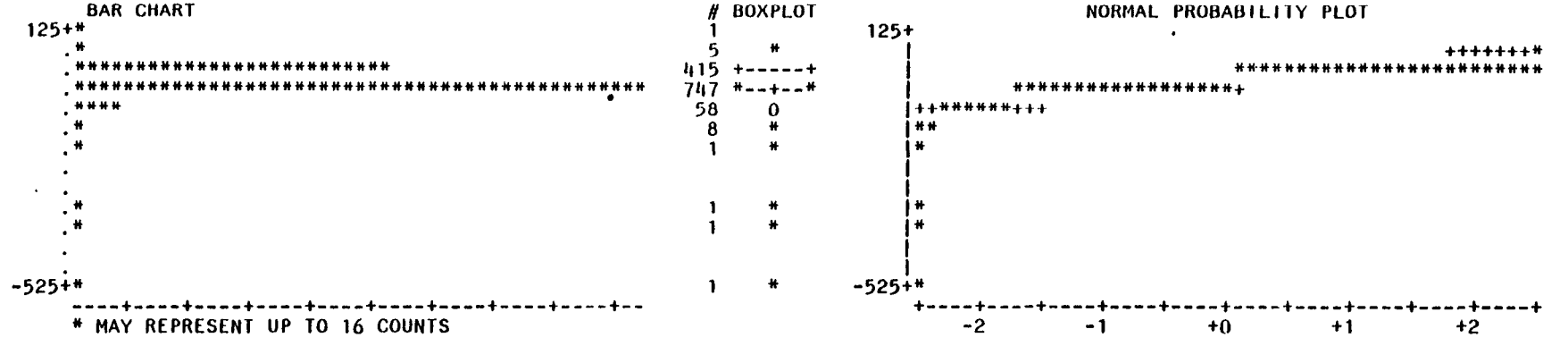


Fig. C.5--Further summaries of the individual responses to NO_2

UNIVARIATE

VARIABLE=MINTEMP

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-.00849611	SUM	-10.5182
STD DEV	0.0920138	VARIANCE	0.00846654
SKEWNESS	0.2042	KURTOSIS	17.3102
USS	10.5625	CSS	10.4731
CV	-1083.01	STD MEAN	0.00261513
T:MEAN=0	-3.24884	PROB>|T|	0.00118993
SGN RANK	-72796.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100%	MAX	0.690224	
75%	Q3	0.0317862	
50%	MED	-0.0126698	
25%	Q1	-0.0549098	
0%	MIN	-0.988637	
	RANGE	1.67886	
	Q3-Q1	0.086696	

99%	0.260319		
95%	0.130782		
90%	0.079235		
10%	-0.0987047		
5%	-0.132096		
1%	-0.20627		

EXTREMES

LOWEST	HIGHES
-0.988637	0.43042
-0.386448	0.43113
-0.357694	0.4998
-0.347597	0.65408
-0.308119	0.69022

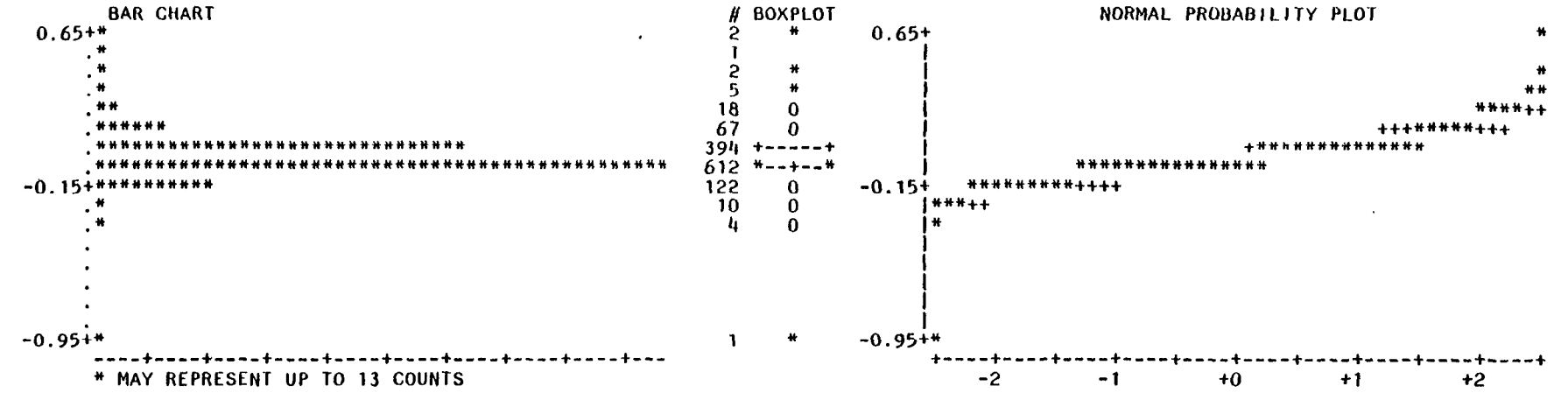


Fig. C.6--Further summaries of the individual responses to minimum temperature

UNIVARIATE

VARIABLE=PRECIP

MOMENTS

N	1185	SUM WGTS	1185
MEAN	-2.36196	SUM	-2798.92
STD DEV	7.13578	VARIANCE	50.9193
SKEWNESS	-4.53062	KURTOSIS	27.996
USS	66899.4	CSS	60288.5
CV	-302.113	STD MEAN	0.207292
T:MEAN=0	-11.3944	PROB>|T|	0.0001
SGN RANK	-115168	PROB>|S|	0.0001

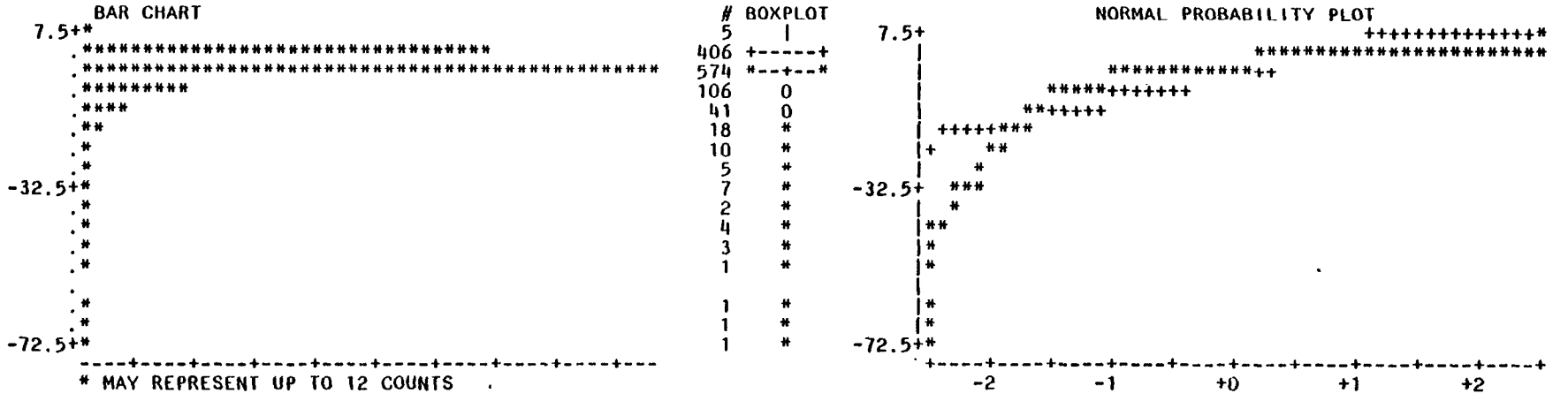
QUANTILES(DEF=4)

100%	MAX	5.60129	99%	4.16602	
75%	Q3	0.929045	95%	2.1894	
50%	MED	-0.422678	90%	1.72042	
25%	Q1	-2.72283	10%	-7.67185	
0%	MIN	-72.1828	5%	-13.7778	
			1%	-38.508	
	RANGE	77.7841			
	Q3-Q1	3.65188			

EXTREMES

LOWEST	HIGHES
-72.1828	4.5210
-68.3629	4.6455
-60.3907	4.7127
-52.7428	5.021
-47.3732	5.6012

MISSING VALUE .
COUNT 53
% COUNT/NOBS 4.28



* MAY REPRESENT UP TO 12 COUNTS

Fig. C.7--Further summaries of the individual responses to precipitation

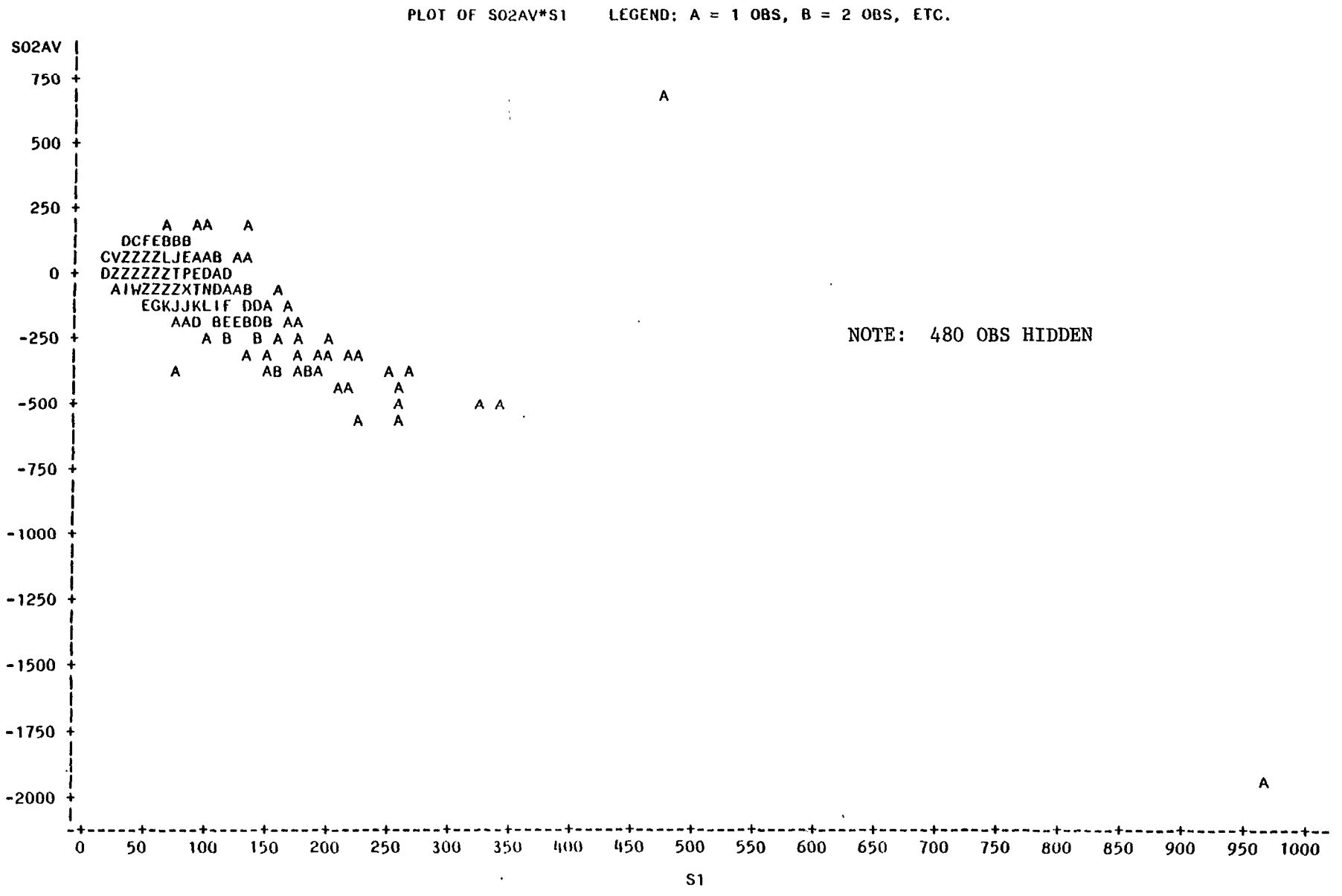


Fig. C.8--Scatterdiagram of estimated individual responses to SO_2 by the associated standard errors

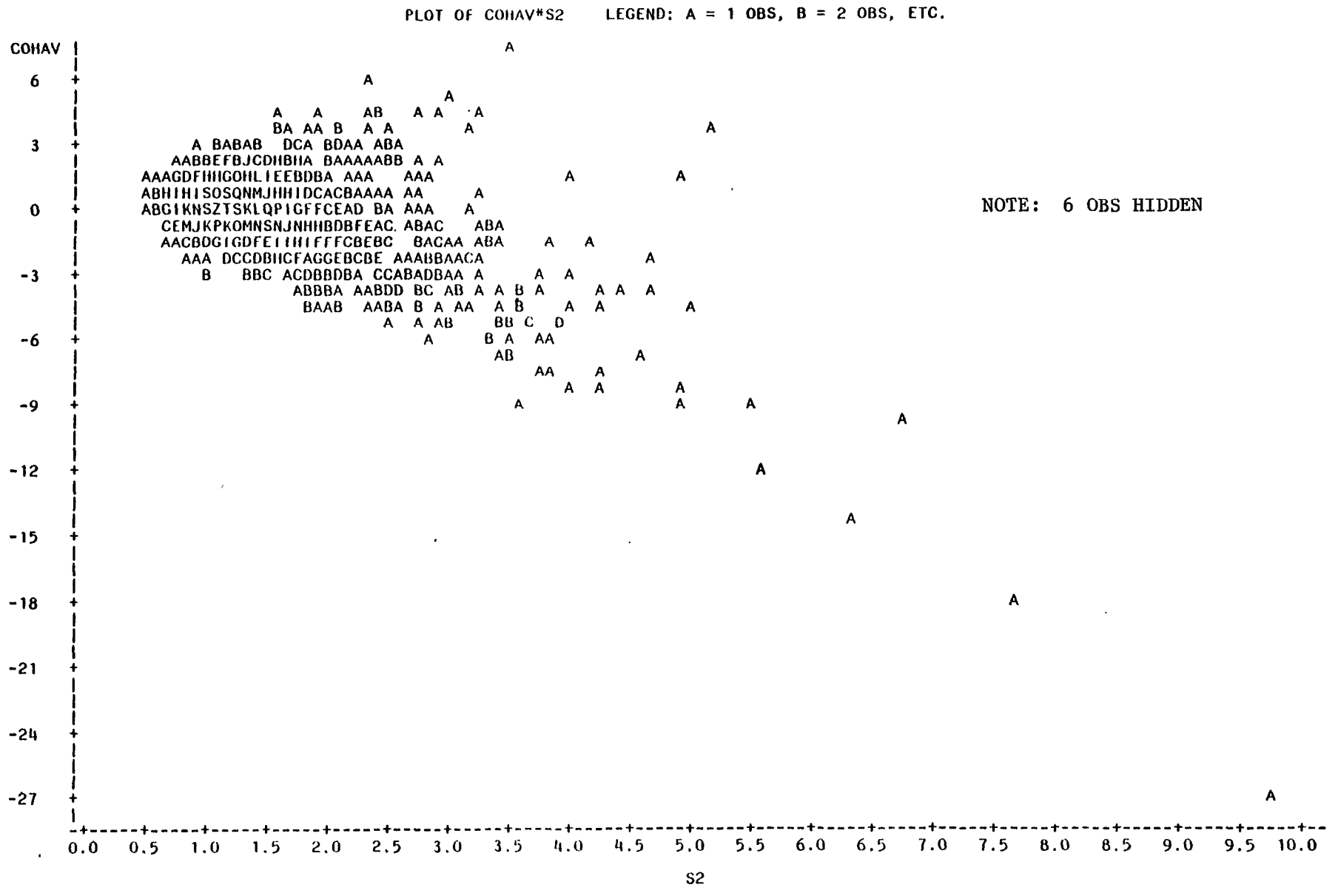


Fig. C.9--Scatterdiagram of the estimated individual responses to COH by the associated standard errors

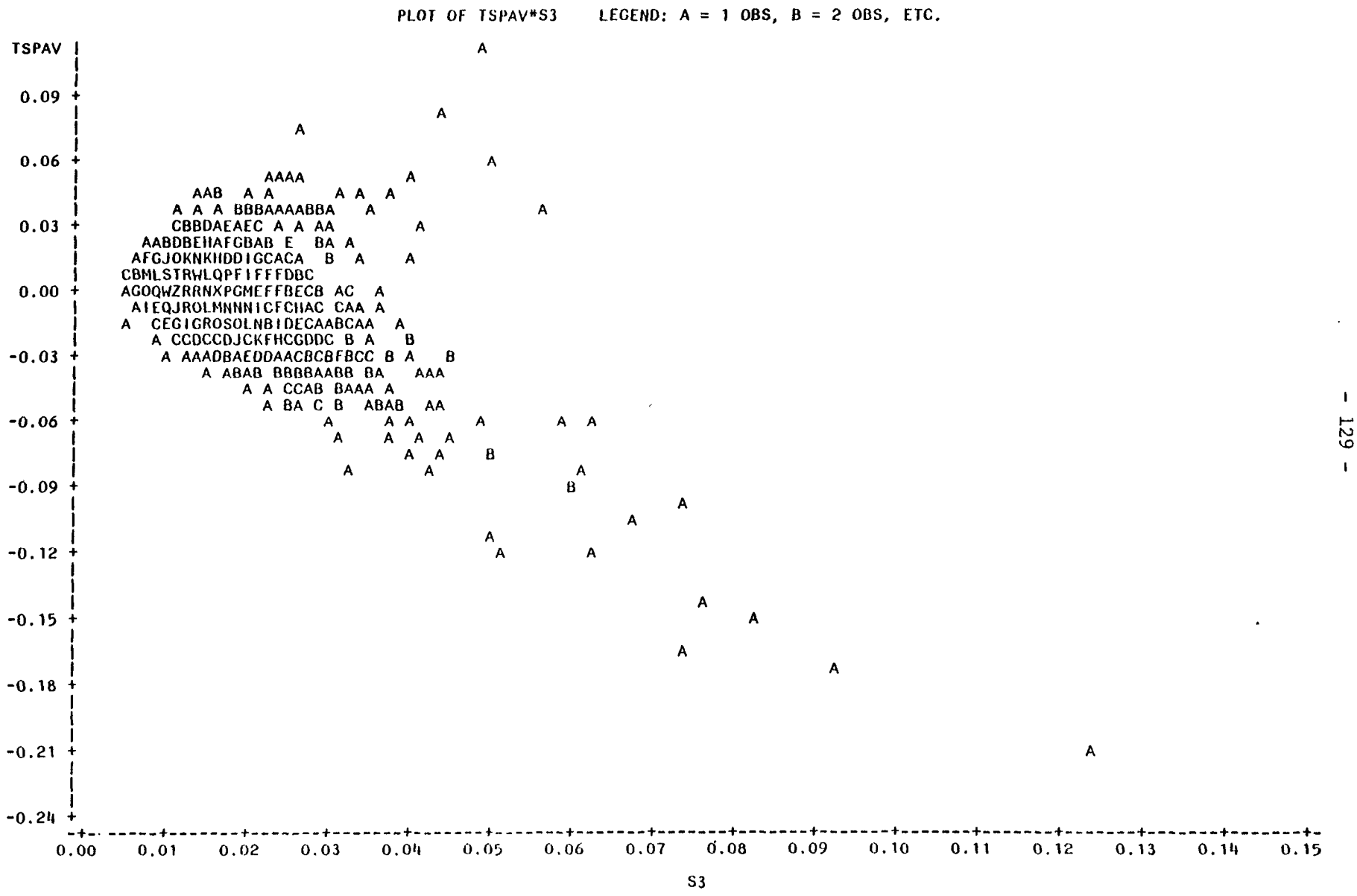


Fig. C.10--Scatterdiagram of the estimated individual responses to TSP by the associated standard errors

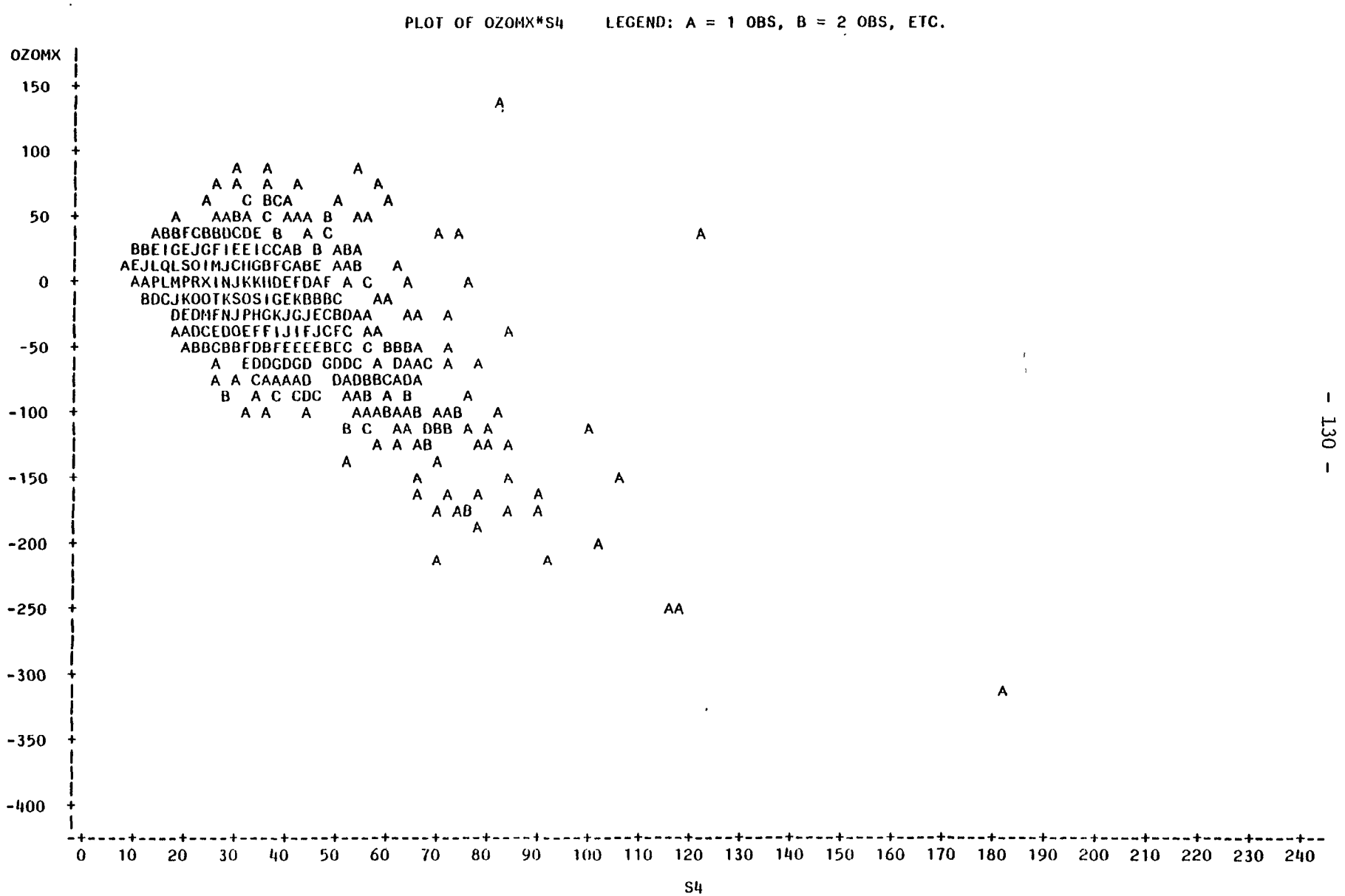


Fig. C. 11--Scatterdiagram of the estimated individual responses to ozone by the associated standard errors

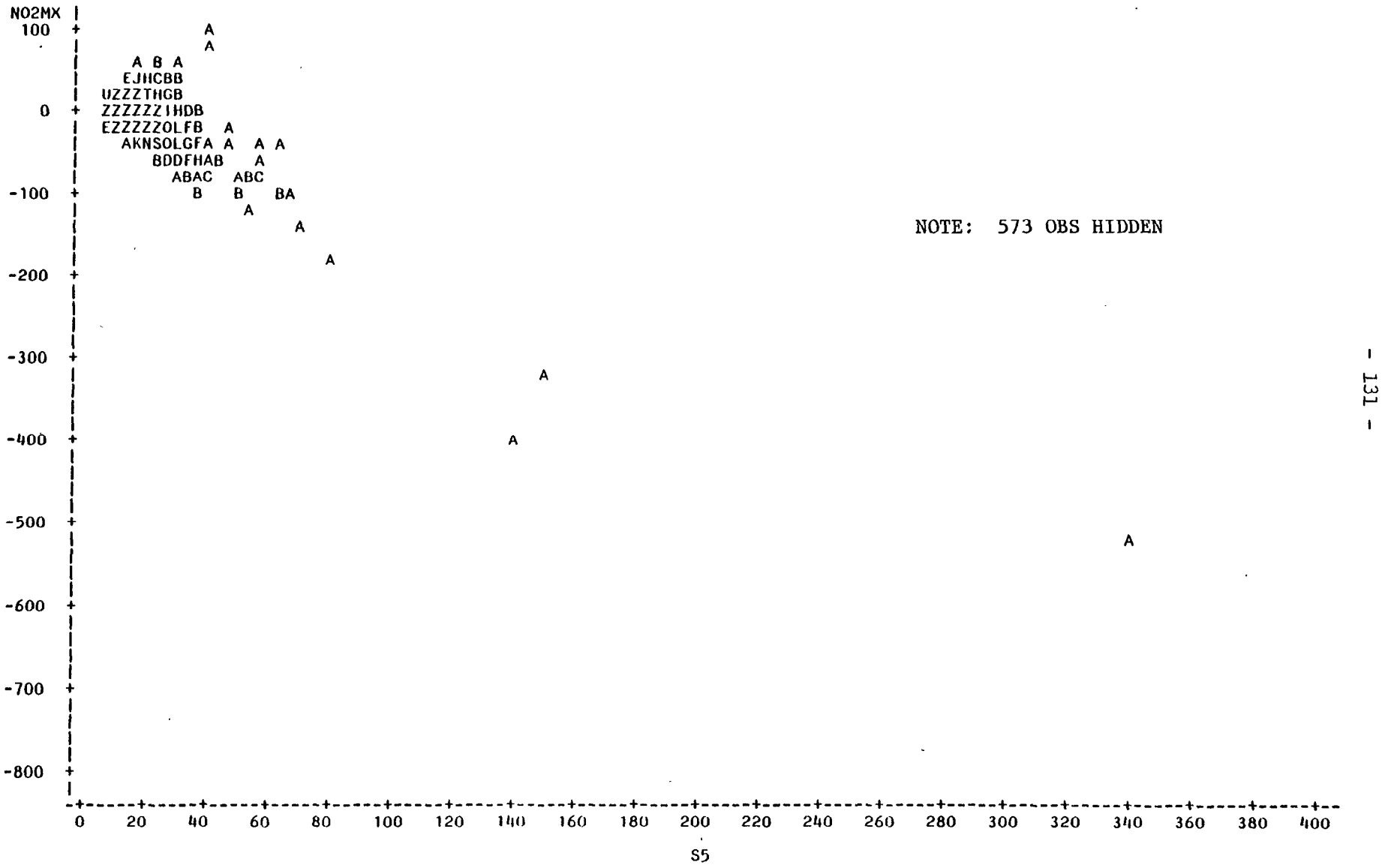


Fig. C.12--Scatterdiagram of the estimated individual responses to NO_2 by the associated standard errors

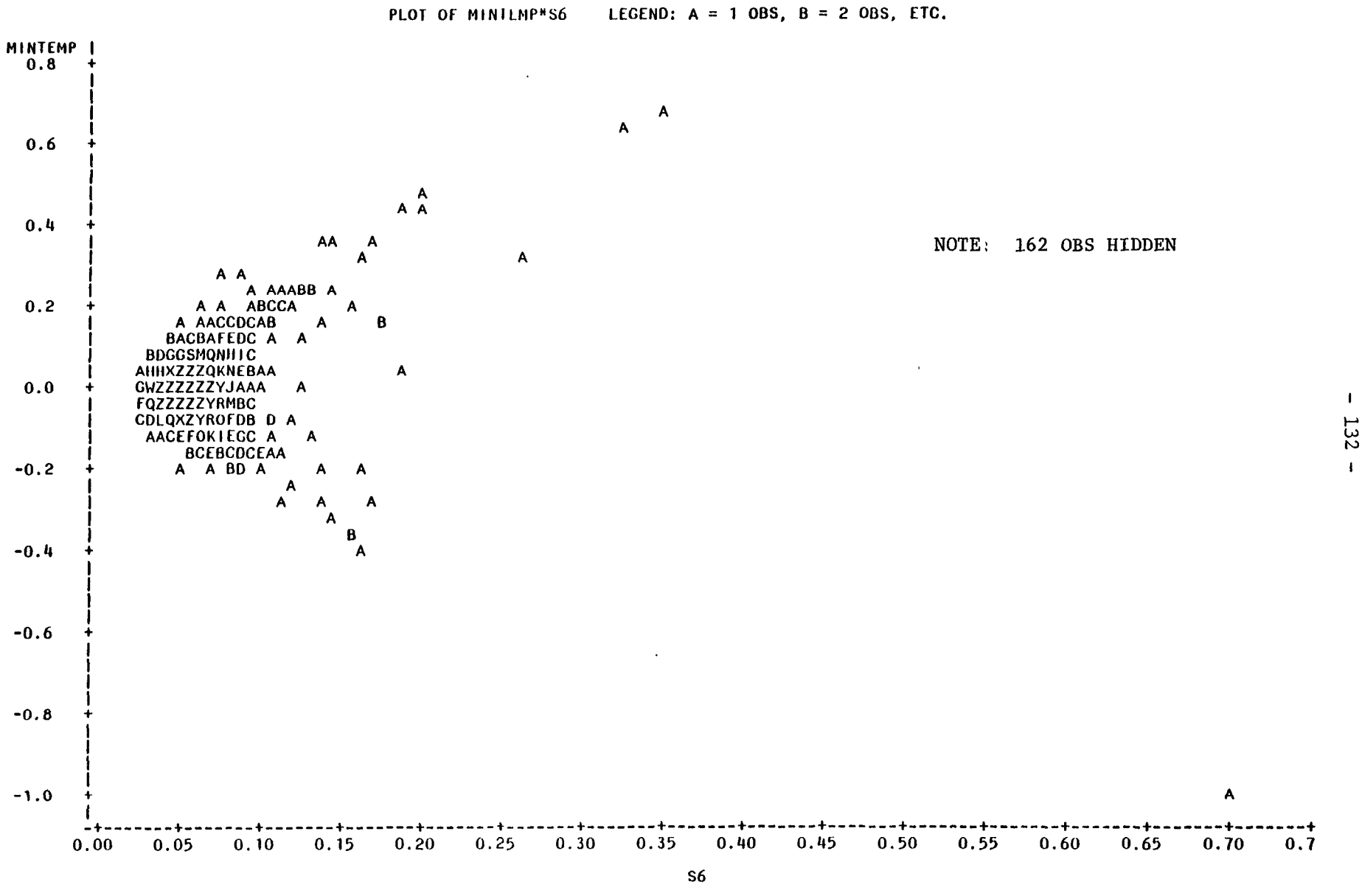


Fig. C.13--Scatterdiagram of the estimated individual responses to min. temp. by the associated standard error

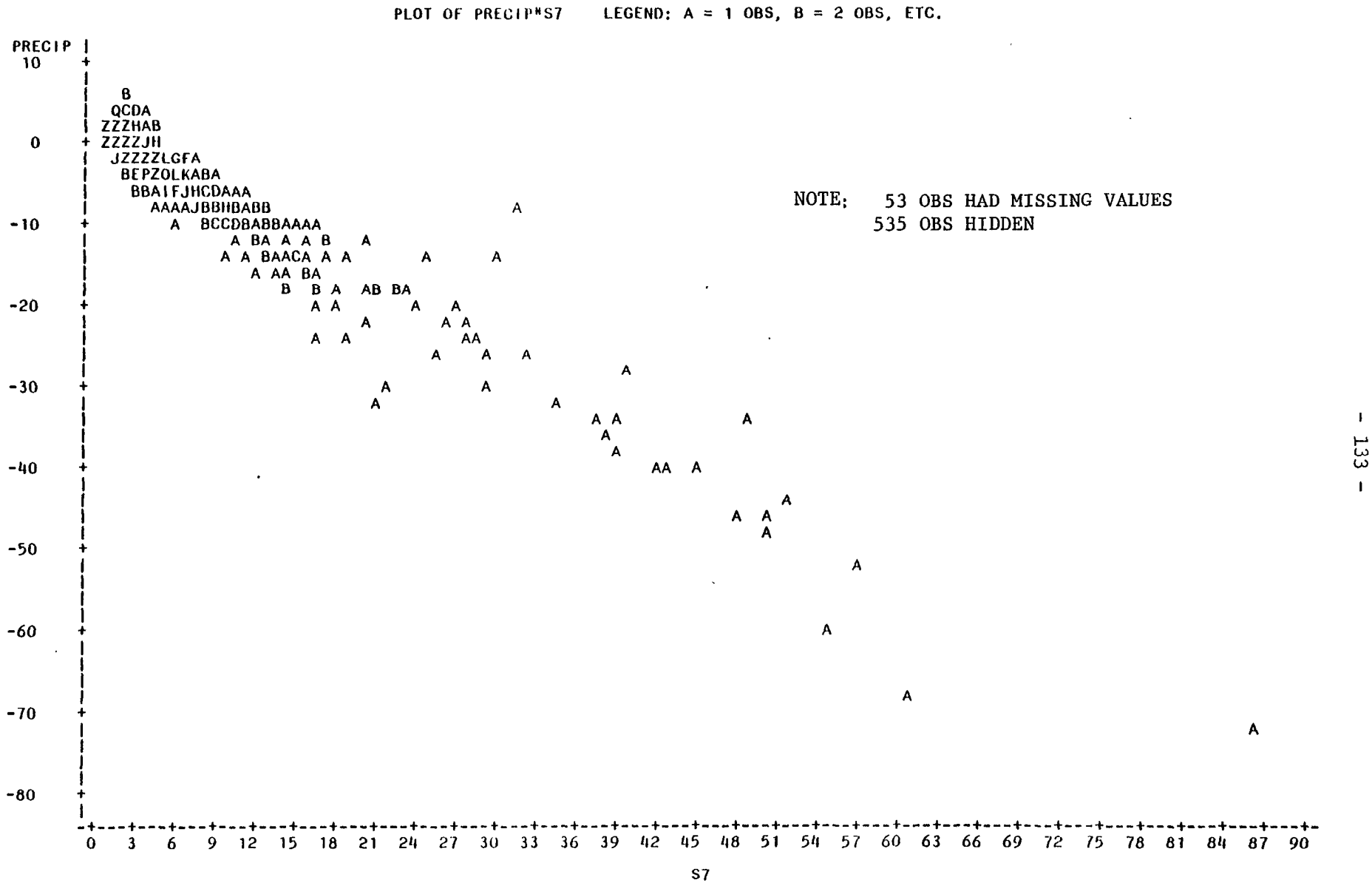


Fig. C.14--Scatterdiagram of estimated individual responses to precipitation by the associated standard errors

VARIABLE=T1

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-0.0448452	SUM	-55.5184
STD DEV	1.02337	VARIANCE	1.0473
SKEWNESS	0.171828	KURTOSIS	0.13466
USS	1298	CSS	1295.51
CV	-2282.02	STD MEAN	0.0290854
T:MEAN=0	-1.54185	PROB>|T|	0.123366
SGN RANK	-27696.5	PROB>|S|	0.0277204

QUANTILES(DEF=4)

100%	MAX	3.41116	99% 2.5619
75%	Q3	0.604064	95% 1.7311
50%	MED	-0.0942933	90% 1.30524
25%	Q1	-0.750014	10% -1.31339
0%	MIN	-4.57416	5% -1.62417
			1% -2.28655
RANGE		7.98532	
Q3-Q1		1.35408	

EXTREMES

LOWEST	HIGHES
-4.57416	2.8250
-2.76497	2.8267
-2.70562	2.861
-2.5485	3.0252
-2.50495	3.4111

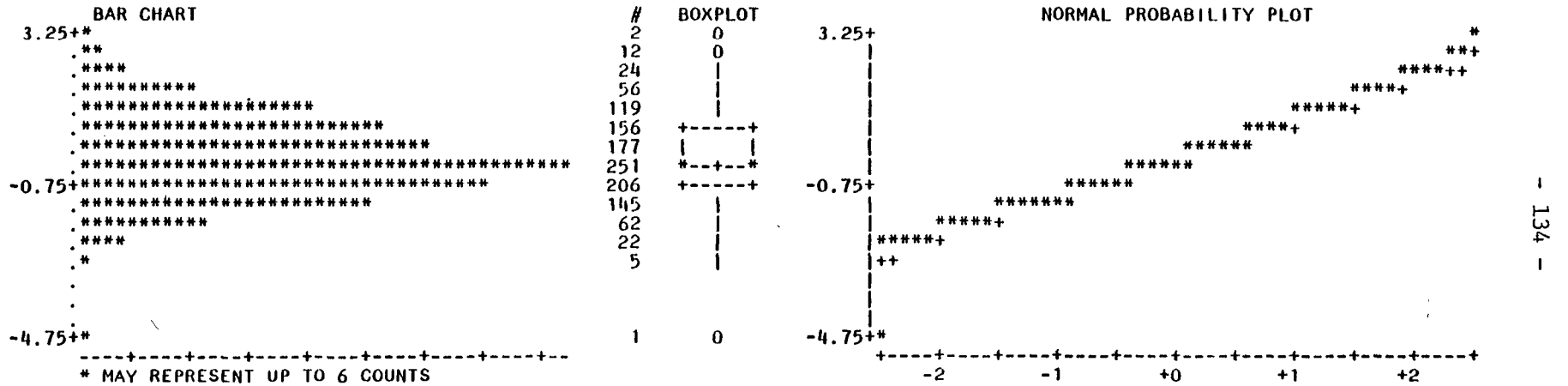


Fig. C.15--Further summaries of individual z statistics for SO_2

UNIVARIATE

VARIABLE=T2

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-0.104464	SUM	-129.327
STD DEV	1.04585	VARIANCE	1.0938
SKEWNESS	0.238488	KURTOSIS	-0.276969
USS	1366.55	CSS	1353.04
CV	-1001.16	STD MEAN	0.0297241
T:MEAN=0	-3.51446	PROB>|T|	.000456564
SGN RANK	-51227.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100%	MAX	3.43351	99%	2.31659
75%	Q3	0.626578	95%	1.7017
50%	MED	-0.20448	90%	1.31475
25%	Q1	-0.848248	10%	-1.42527
0%	MIN	-3.10154	5%	-1.72497
			1%	-2.28221
	RANGE	6.53506		
	Q3-Q1	1.47483		

EXTREMES

LOWEST	HIGHES
-3.10154	2.6769
-2.75071	2.6914
-2.64821	2.8258
-2.64238	3.2119
-2.53325	3.4335

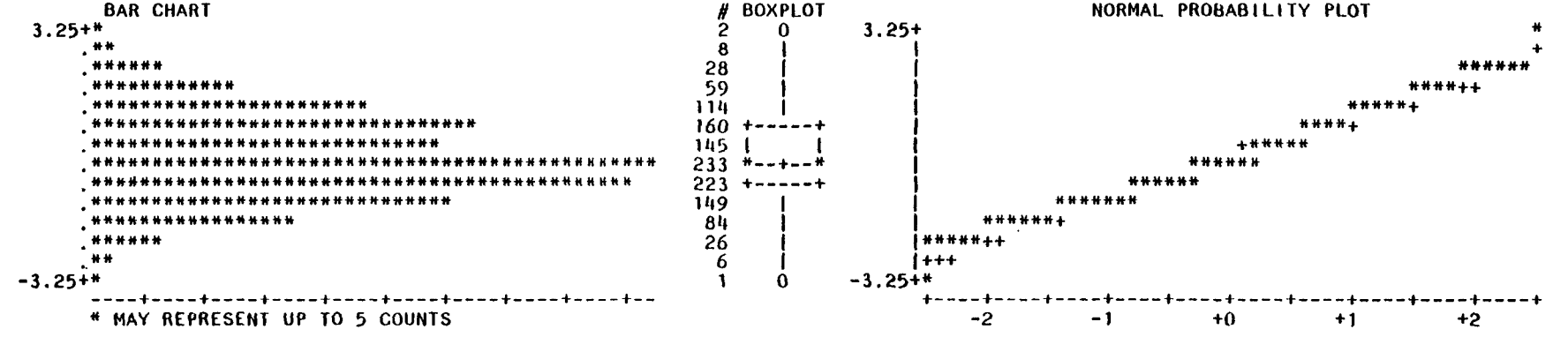


Fig. C.16--Further summaries of individual z statistics for COH

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                                    UNIVARIATE

VARIABLE=T3

                MOMENTS                                  QUANTILES(DEF=4)                    EXTREMES

N              1238      SUM WGTS       1238      100% MAX    3.36199      99%    2.36667      LOWEST      HIGHES
MEAN      -0.0760234     SUM          -94.117      75% Q3     0.607013     95%    1.65063     -2.8194     2.8220
STD DEV    0.999119     VARIANCE     0.998239      50% MED  -0.135138     90%    1.27865     -2.78312    2.8425
SKEWNESS   0.291162     KURTOSIS    -0.134518      25% Q1   -0.801785     10%   -1.30756     -2.62205     2.933
USS         1241.98     CSS           1234.82       0% MIN    -2.8194      5%   -1.59603     -2.61552    2.9738
CV         -1314.23     STD MEAN     0.028396                             1%   -2.19979     -2.37723    3.3619
T:MEAN=0   -2.67726     PROB>|T|  0.00752093      RANGE       6.18139
SGN RANK   -43402.5     PROB>|S|  .000561622      Q3-Q1         1.4088

          BAR CHART                                #    BOXPLOT                NORMAL PROBABILITY PLOT
     3.25+*                                        2       0                3.25+                                  *
         .**                                       8       0                   |                                 **
         .****                                    23       |                   |                              *****++
         .**********                              58       |                   |                          ******+
         .********************                   111       |                   |                      ******+
         .**************************             155  +-----+                  |                   ****+
     0.25+*******************************        182  |     |             0.25+                ******
         .************************************   213  *--+--*                   |             +******
         .*****************************************242  +-----+                  |          *******
         .**************************             150       |                   |       *******
         .**********                              75       |                   |  ********+
         .***                                     15       |                   |***+++
    -2.75+*                                        4       |              -2.75++
         ----+----+----+----+----+----+----+----+-                              +----+----+----+----+----+----+----+----+----+----+
         * MAY REPRESENT UP TO 6 COUNTS                                          -2        -1        +0        +1        +2
```

Fig. C.17--Further summaries of individual z statistics for TSP

UNIVARIATE

VARIABLE=T4

MOMENTS

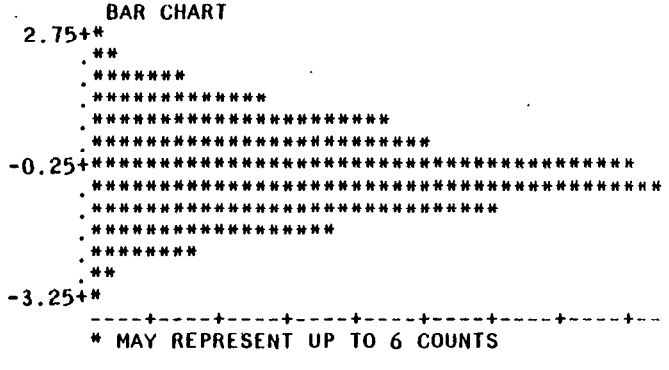
N	1238	SUM WGTS	1238
MEAN	-0.32344	SUM	-400.418
STD DEV	0.998557	VARIANCE	0.997117
SKEWNESS	0.24067	KURTOSIS	-0.0898195
USS	1362.94	CSS	1233.43
CV	-308.731	STD MEAN	0.02838
T:MEAN=0	-11.3967	PROB>|T|	0.0001
SGN RANK	-141181	PROB>|S|	0.0001

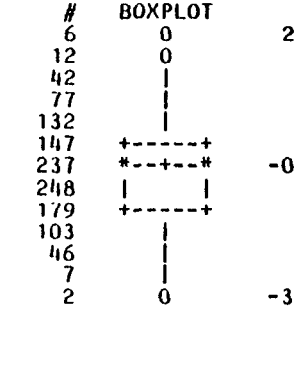
QUANTILES(DEF=4)

100%	MAX	2.87171	99%	2.06793
75%	Q3	0.323096	95%	1.43917
50%	MED	-0.3736	90%	1.00705
25%	Q1	-1.01777	10%	-1.57545
0%	MIN	-3.32242	5%	-1.91041
			1%	-2.3709
RANGE		6.19413		
Q3-Q1		1.34087		

EXTREMES

LOWEST	HIGHES
-3.32242	2.5094
-3.02209	2.52
-2.93424	2.7652
-2.88965	2.8029
-2.85837	2.8717





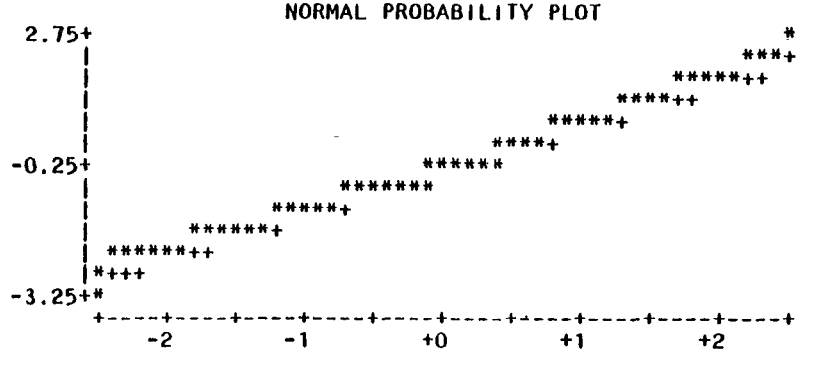


Fig. C.18--Further summaries of individual z statistics for ozone

UNIVARIATE

VARIABLE=T5

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-0.0440009	SUM	-54.4731
STD DEV	1.142	VARIANCE	1.30416
SKEWNESS	0.206539	KURTOSIS	-0.298141
USS	1615.64	CSS	1613.25
CV	-2595.4	STD MEAN	0.0324568
T:MEAN=0	-1.35568	PROB>|T|	0.17545
SGN RANK	-24696.5	PROB>|S|	0.0496709

QUANTILES(DEF=4)

100%	MAX	3.76366	99%	2.60549
75%	Q3	0.760721	95%	1.87898
50%	MED	-0.120368	90%	1.52481
25%	Q1	-0.885686	10%	-1.51569
0%	MIN	-3.43163	5%	-1.78383
			1%	-2.41731
RANGE		7.19528		
Q3-Q1		1.64641		

EXTREMES

LOWEST	HIGHES
-3.43163	3.1233
-3.15714	3.1280
-2.79287	3.170
-2.72649	3.3231
-2.70739	3.7636

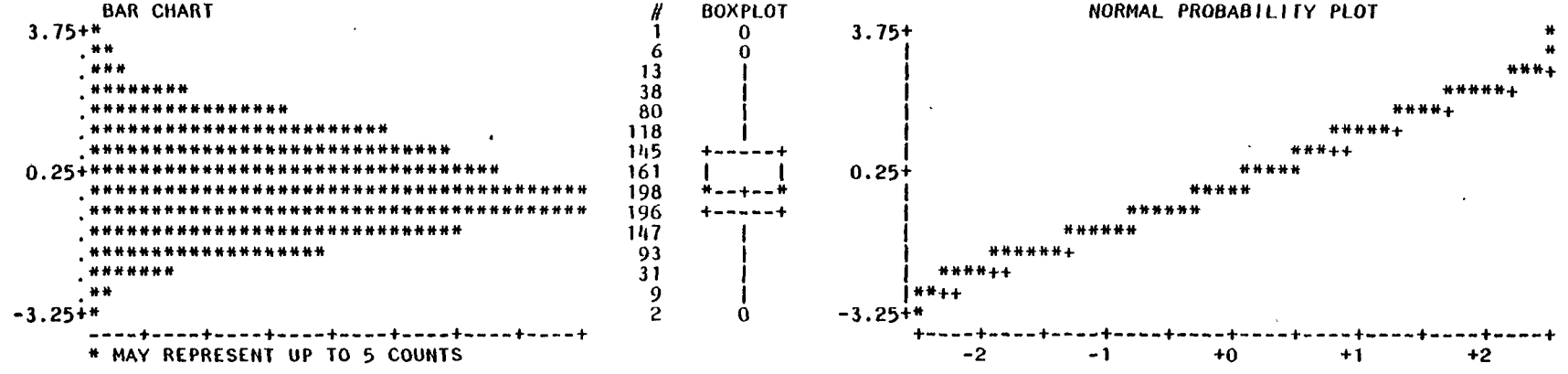


Fig. C.19--Further summaries of individual z statistics for NO_2

UNIVARIATE

VARIABLE=T6

MOMENTS

N	1238	SUM WGTS	1238
MEAN	-0.21303	SUM	-263.731
STD DEV	1.12948	VARIANCE	1.27572
SKEWNESS	0.122812	KURTOSIS	-0.189431
USS	1634.25	CSS	1578.07
CV	-530.197	STD MEAN	0.0321009
T:MEAN=0	-6.63625	PROB>|T|	0.0001
SGN RANK	-83895.5	PROB>|S|	0.0001

QUANTILES(DEF=4)

100% MAX	3.60679	99%	2.33365
75% Q3	0.554708	95%	1.77997
50% MED	-0.249678	90%	1.23396
25% Q1	-1.0153	10%	-1.66942
0% MIN	-4.13131	5%	-2.01478
		1%	-2.64554
RANGE	7.73811		
Q3-Q1	1.57001		

EXTREMES

LOWEST	HIGHES
-4.13131	2.8386
-3.41677	2.8757
-3.34178	3.099
-2.99609	3.1792
-2.84225	3.6067

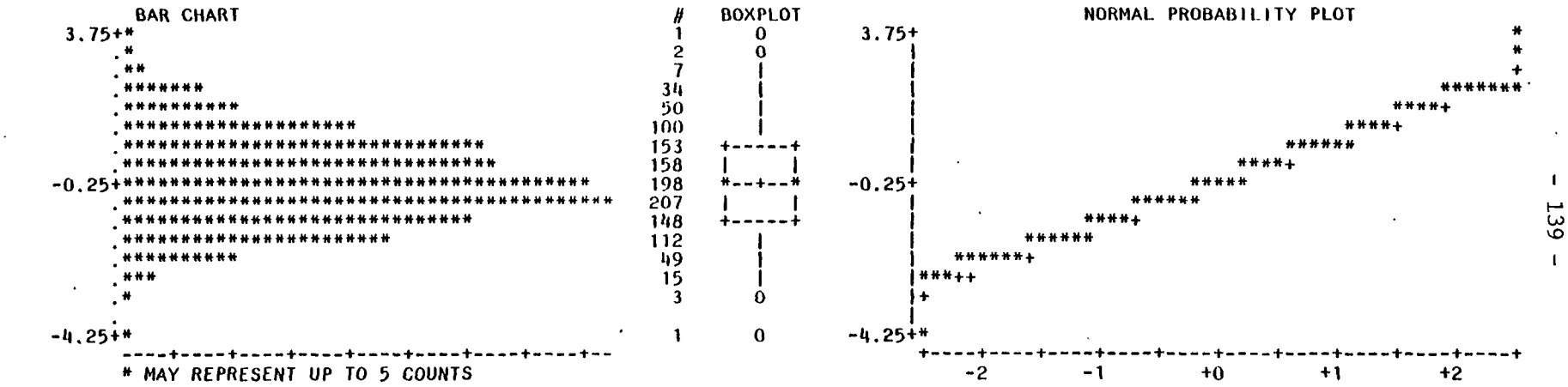


Fig. C.20--Further summaries of individual z statistics for minimum temperature

UNIVARIATE

VARIABLE=T7

MOMENTS

N	1185	SUM WGTS	1185
MEAN	0.0275536	SUM	32.651
STD DEV	0.881066	VARIANCE	0.776278
SKEWNESS	0.762011	KURTOSIS	0.0551939
USS	920.013	CSS	919.113
CV	3197.65	STD MEAN	0.0255947
T:MEAN=0	1.07654	PROB>|T|	0.281907
SGN RANK	-16054.5	PROB>|S|	0.173055

QUANTILES(DEF=4)

100%	MAX	3.25951	99%	2.42128	
75%	Q3	0.578213	95%	1.77432	
50%	MED	-0.184006	90%	1.28507	
25%	Q1	-0.673102	10%	-0.940373	
0%	MIN	-2.06814	5%	-1.06018	
			1%	-1.3541	
RANGE		5.32764			
Q3-Q1		1.25132			

EXTREMES

LOWEST	HIGHES
-2.06814	2.5942
-1.68022	2.6305
-1.59674	2.6779
-1.59628	2.7668
-1.56933	3.259

MISSING VALUE .
COUNT 53
% COUNT/NOBS 4.28

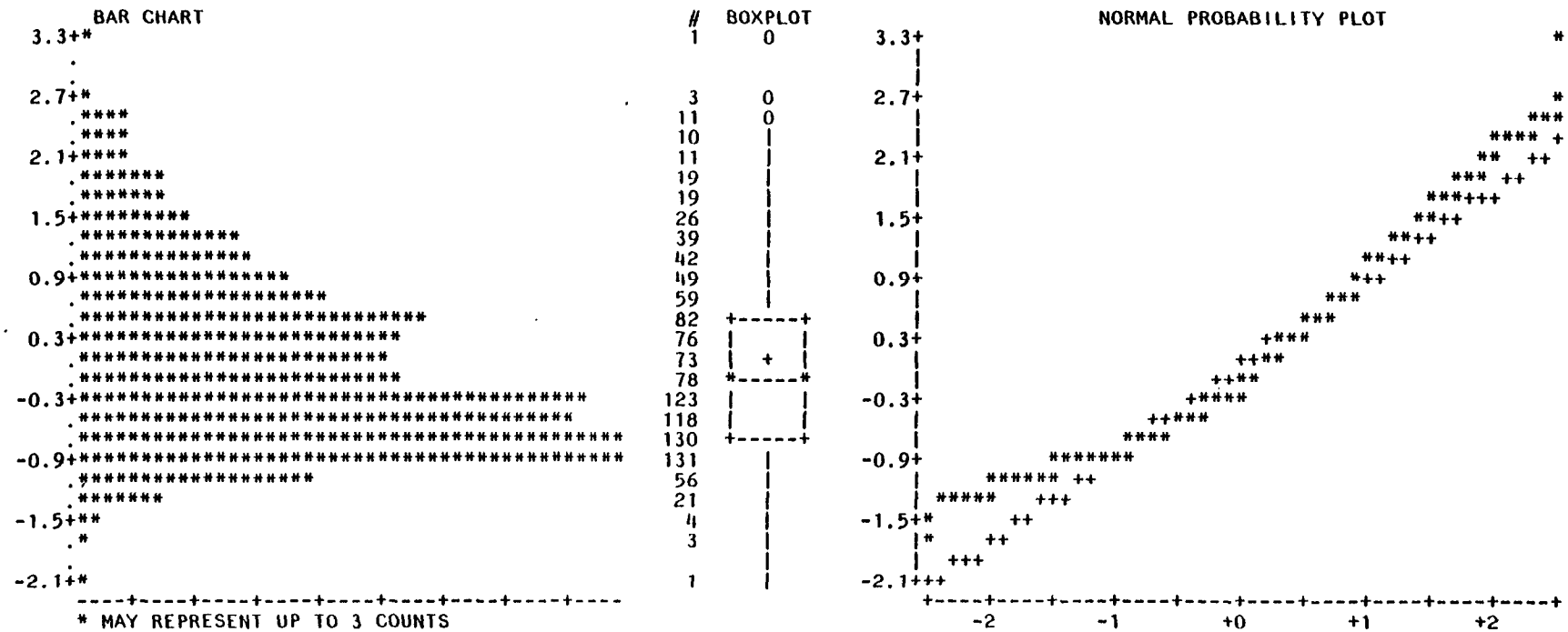


Fig. C.21--Further summaries of individual z statistics for precipitation

Given the heterogeneity of the estimated individual responses, the unweighted summaries given in Table C.4 and Figs. C.1 through C.7 are not very informative. The unweighted summaries given in these exhibits do not account for the heterogeneity. Those summaries may be dominated by outliers that are highly imprecisely estimated. One way to get around this problem would be to carry out weighted summaries of the estimated individual responses,. with weights determined from the estimated standard errors. The random-effects model, whose results are discussed in Sec. VII, is similar to that approach (it also allows the estimation of between-individual differences).

Another way to account for account for heterogeneity in summarizing is to examine the individual z statistics, which rescale the estimated individual responses by precision. Table C.5 gives the major summaries for the individual z statistics.. Figs. C.15 through C.21 give further summaries of the individual z statistics for each aerometric attribute. The variable name T1 refers to the z statistic for the individual response to $S0_2$, T2 refers to COH, T3 to TSP, T4 to ozone, T5 to $N0_2$, T6 to minimum temperature, and T7 to precipitation.

The results of the z analysis vary somewhat from those of the random-effects model. For all the aerometric attributes except precipitation, the distributions of the individual z statistics are reasonably close to a standard normal distribution: The standard deviations for the individual z statistics given under the column "STD DEV" in Table C.5 are close to one, and the skewness and kurtosis given in the "moments" sections of Figs. C.15 through C.20 are both small, ranging between 0.1 and 0.3 . The normal plots given in Figs. C.15 through C.20 are reasonably close to straight lines, as they should be if the distributions are close to a normal distribution. For precipitation, the individual z statistics are somewhat skewed.

For all pollution measures, the average z statistics given under the column "MEAN" in Table C.5 are negative, indicating that there is a lower probability for a sick episode on a polluted day than on a clean day. The effect is statistically significant at the five percent level for COH, TSP, and ozone. (See the entries "T:MEAN=0" and "PROB>|T|" in the "moments" sections of Figs. C.15 through C.21.) The average z

statistic for minimum temperature is also negative, indicating that when the minimum temperature is higher, a sick episode is less probable. The effect is statistically significant at the 5 percent level. The average z statistic for precipitation is positive, indicating a higher probability to have a sick episode on a wet day, but the effect is not statistically significant at the 5 percent level.

COMPARISON OF SUBPOPULATIONS

In the following subsections, we expand on the discussion of subpopulations in Sec. VII. We begin by taking up two alternative criteria for defining the sickly subpopulation, then proceed to contrasts between adults and children and between smokers and nonsmokers.

Sickly vs. Healthy

Lung Function. Another way we can classify people into healthy and less healthy subpopulations is to use FEV_1, as measured during the HIE. We define a person to be a high-FEV_1 person if his FEV_1 is higher than that expected based on his sex, age, height and weight. Among 383 persons for whom we have FEV_1 measurements, 282 fall into this subpopulation; the other 101 are classified as low-FEV_1 persons. For both average responses and between-individual differences, none of the comparisons between these two subpopulations is statistically significant. (See Tables C.6 through C.9).

Pulmonary Susceptibility. We define a person to be susceptible to pulmonary problems if he has one of the important pulmonary diseases such as asthma, emphysema, or hay fever. We have 422 persons who fall into this category. For both average responses and between-individual differences, none of the comparisons between these two subpopulations is statistically significant. (See Tables C.10 through C.13.)

Adults Versus Children

The comparison between adults and children is of interest for several reasons. First, adults are usually more mobile than children because of work and other activities. Therefore our measure of air pollution exposure is less accurate for adults than for children.

Table C.6

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE HIGH FEV1 PERSONS: AVERAGE RESPONSES (n = 282)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
S02 (ppm>	0.109E+02	0.404E+01
COH	-0.417E-01	-0.529E+00
TSP ($\mu g/m^3$)	0.152E-02	0.186E+01
Ozone (ppm)	-0.378E+01	-0.242E+01
N02 (ppm)	0.113E+01	0.140E+01
Min. temp. (F)	-0.625E-02	-0.196E+01
Precip. (inch)	0.668E+00	0.624E+01

Table C.7

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LOW FEV1 PERSONS: AVERAGE RESPONSES (n = 101)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
S02 (ppm)	0.116E+02	0.254E+01	0.13
COH	0.153E+00	0.104E+01	1.17
TSP ($\mu g/m^3$	0.834E-03	0.557E+00	-0.40
Ozone (ppm)	-0.865E+00	-0.311E+00	0.92
N02 (ppm)	0.126E+00	0.718E-01	-0.52
Min. temp. (F)	-0.127E-01	-0.195E+01	-0.89
Precip. (inch)	0.888E+00	0.509E+01	1.08

Table C.8

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE HIGH FEV_1 PERSONS: BETWEEN INDIVIDUAL DIFFERENCES (n = 282)

Aerometric Attribute	Tau	z for the Attribute
S02 (ppm)	0.651E+01	0.320E+00
COH	0.269E+00	0.622E+00
TSP ($\mu g/m^3$)	0.000E+00	0.000E+00
Ozone (ppm)	0.466E+01	0.496E+00
N02 (ppm)	0.434E+01	0.149E+01
Min. temp. (F)	0.168E-01	0.138E+01
Precip. (inch)	0.000E+00	0.000E+00

Table C.9

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LOW FEV_1 PERSONS: BETWEEN INDIVIDUAL DIFFERENCES (n = 101)

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
S02 (ppm)	0.000E+00	0.000E+00	-0.16
COH	0.527E+00	0.117E+01	0.78
TSP ($\mu g/m^3$)	0.520E-02	0.112E+01	1.01
Ozone (ppm)	0.714E+01	0.646E+00	0.32
N02 (ppm)	0.973E+01	0.246E+01	1.88
Min. temp. (F)	0.348E-01	0.223E+01	1.60
Precip. (inch)	0.000E+00	0.000E+00	0.00

Table C.10

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SUSCEPTIBLES: AVERAGE RESPONSES (n = 422)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
S02 (ppm)	0.780E+01	0.366E+01
COH	-0.754E-01	-0.118E+01
TSP ($\mu g/m^3$)	0.115E-02	0.168E+01
Ozone (ppm)	-0.300E+01	-0.235E+01
N02 (ppm)	0.132E+01	0.182E+01
Min. temp. (F)	-0.128E-01	-0.471E+01
Precip. (inch)	0.672E+00	0.763E+01

Table C.11

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONSUSCEPTIBLES: AVERAGE RESPONSES (n = 816)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
S02 (ppm)	0.800E+01	0.489E+01	0.07
COH	0.700E-01	0.142E+01	1.81
TSP ($\mu g/m^3$)	0.264E-03	0.521E+00	-1.04
Ozone (ppm)	-0.372E+01	-0.381E+01	-0.45
N02 (ppm)	0.138E+01	0.274E+01	0.07
Min. temp. (F)	-0.133E-01	-0.660E+01	-0.15
Precip. (inch)	0.691E+00	0.103E+02	0.17

Table C.12

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SUSCEPTIBLES: BETWEEN INDIVIDUAL DIFFERENCES (n = 422)

Aerometric Attribute	TAU	z for the Attribute
S02 (ppm)	0.189E+01	0.369E-01
COH	0.270E+00	0.779E+00
TSP ($\mu g/m^3$)	0.342E-02	0.112E+01
Ozone (ppm)	0.619E+01	0.107E+01
N02 (ppm)	0.693E+01	0.363E+01
Min. temp. (F)	0.216E-01	0.246E+01
Precip. (inch)	0.000E+00	0.000E+00

Table C.13

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONSUSCEPTIBLES: BETWEEN INDIVIDUAL DIFFERENCES (n = 816)

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
S02 (ppm)	0.000E+00	0.000E+00	-0.03
COH	0.349E+00	0.157E+01	0.40
TSP ($\mu g/m^3$)	0.000E+00	0.000E+00	-0.91
Ozone (ppm)	0.492E+01	0.866E+00	-0.31
N02 (ppm)	0.439E+01	0.231E+01	-1.84
Min. temp. (F)	0.177E-01	0.221E+01	-0.65
Precip. (inch)	0.000E+00	0.000E+00	0.00

Second, because children spend more time outside than adults, our measures of air pollution exposure based on ambient monitoring are more accurate for children than for adults. Third, adults encounter or engage in more activities that give them nonambient exposures, such as smoking and occupational exposures. Furthermore, it is conceivable that adults and children might have intrinsically different responses to air pollution.

We distinguish adults and children at age 18. Thus, in the final analysis sample we have 780 adults and 458 children. We found children to be significantly more responsive to minimum temperature; the average response for children is more than twice the average response for adults. There is also significantly less between-individual variation in children's responses to minimum temperature and $N0_2$ (See Tables C.14 through C.17.)

Smoking

A major source of nonambient exposure is smoking. Among the 780 adults in the final analysis sample, we have 276 smokers and 504 nonsmokers. For both average responses and between-individual differences, none of the comparisons between these two subpopulations are statistically significant. (See Tables C.18 through C.21.)

Among the 458 children in the final analysis sample, we have 208 who live in a household with smokers and 250 who do not. For both average responses and between-individual differences, none of the comparisons between these two subpopulations is statistically significant (see Tables C.22 through C.25.).

Table C.14

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE ADULTS: AVERAGE RESPONSES (n = 780)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
S02 (ppm)	0.850E+01	0.534E+01
COH	0.271E-02	0.568E-01
TSP ($\mu g/m^3$)	0.560E-03	0.109E+01
Ozone (ppm)	-0.257E+01	-0.268E+01
N02 (ppm)	0.166E+01	0.319E+01
Min. temp. (F)	-0.957E-02	-0.467E+01
Precip. (inch)	0.636E+00	0.9686+01

Table C.15

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE CHILDREN: AVERAGE RESPONSES (n = 458)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
S02 (ppm)	0.636E+01	0.279E+01	-0.77
COH	0.388E-01	0.568E+00	0.43
TSP ($\mu g/m^3$ >	0.4936-03	0.716E+00	-0.08
Ozone (ppm)	-0.543E+01	-0.412E+01	-1.76
N02 (ppm)	0.7696+00	0.112E+01	-1.04
Min. temp. (F)	-0.205E-01	-0.787E+01	-3.31
Precip. (inch)	0.776E+00	0.853E+01	1.25

Table C.16

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES
FOR THE AEROMETRIC EFFECTS OVER THE ADULTS
BETWEEN INDIVIDUAL DIFFERENCES
(n = 780)

Aerometric Attribute	Tau	z for the Attribute
S02 (ppm)	0.000E+00	0.000E+00
COH	0.328E+00	0.151E+01
TSP ($\mu g/m^3$)	0.312E-02	0.125E+01
Ozone (ppm)	0.650E+01	0.156E+01
N02 (ppm)	0.635E+01	0.442E+01
Min. temp. (F)	0.222E-01	0.343E+01
Precip. (inch)	0.000E+00	0.000E+00

Table C.17

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES
FOR THE AEROMETRIC EFFECTS OVER THE CHILDREN
BETWEEN INDIVIDUAL DIFFERENCES
(n = 458)

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
S02 (ppm)	0.613E+01	0.342E+00	0.28
COH	0.385E+00	0.128E+01	0.30
TSP ($\mu g/m^3$)	0.000E+00	0.000E+00	-0.73
Ozone (ppm)	0.000E+00	0.000E+00	-0.88
N02 (ppm)	0.184E+01	0.305E+00	-2.57
Min. temp. (F)	0.000E+00	0.000E+00	-2.16
Precip. (inch)	0.000E+00	0.000E+00	0.00

Table C.18

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER SMOKING ADULTS: AVERAGE RESPONSES (n = 276)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
S02 (ppm)	0.960E+01	0.351E+01
COH .	-0.166E-02	-0.205E-01
TSP ($\mu g/m^3$)	0.771E-03	0.902E+00
Ozone (ppm)	-0.424E+01	-0.252E+01
N02 (ppm)	0.115E+01	0.122E+01
Min. temp. (F)	-0.103E-01	-0.298E+01
Precip. (inch)	0.609E+00	0.545E+01

Table C.19

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONSMOKING ADULTS: AVERAGE RESPONSES (n = 504)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
S02 (ppm)	0.761E+01	0.382E+01	-0.59
COH	0.827E-02	0.142E+00	0.10
TSP ($\mu g/m^3$ >	0.449E-03	0.701E+00	-0.30
Ozone (ppm)	-0.172E+01	-0.147E+01	1.23
N02 (ppm)	0.189E+01	0.303E+01	0.65
Min. temp. (F)	-0.910E-02	-0.374E+01	0.28
Precip. (inch)	0.651E+00	0.800E+01	0.31

Table C.20

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER SMOKING ADULTS: BETWEEN INDIVIDUAL DIFFERENCES (n = 276)

Aerometric Attribute	Tau	z for the Attribute
S02 (ppm)	0.872E+01	0.551E+00
COH	0.401E+00	0.129E+01
TSP ($\mu g/m^3$)	0.345E-02	0.939E+00
Ozone (ppm)	0.679E+01	0.922E+00
N02 (ppm)	0.809E+01	0.358E+01
Min. temp. (F)	0.233E-01	0.221E+01
Precip. (inch)	0.000E+00	0.000E+00

Table C.21

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONSMOKING ADULTS: BETWEEN INDIVIDUAL DIFFERENCES (n = 504)

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
S02 (ppm)	0.000E+00	0.000E+00	-0.45
COH	0.199E+00	0.476E+00	-0.81
TSP ($\mu g/m^3$)	0.281E-02	0.799E+00	-0.25
Ozone (ppm)	0.620E+01	0.121E+01	-0.13
N02 (ppm)	0.529E+01	0.273E+01	-1.80
Min. temp. (F)	0.162E-01	0.165E+01	-0.96
Precip. (inch)	0.000E+00	0.000E+00	0.00

Table C.22

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER EXPOSED CHILDREN: AVERAGE RESPONSES (n = 208)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
S02 (ppm)	0.790E+01	0.207E+01
COH	-0.678E-01	-0.617E+00
TSP ($\mu g/m^3$)	0.377E-03	0.349E+00
Ozone (ppm)	-0.728E+01	-0.339E+01
N02 (ppm)	0.334E+00	0.293E+00
Min. temp. (F)	-0.241E-01	-0.552E+01
Precip. (inch)	0.739E+00	0.500E+01

Table C.23

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONEXPOSED CHILDREN: AVERAGE RESPONSES (n = 816)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
S02 (ppm)	0.491E+01	0.170E+01	-0.63
COH	0.120E+00	0.144E+01	1.37
TSP ($\mu g/m^3$)	0.571E-03	0.641E+00	0.14
Ozone (ppm)	-0.431E+01	-0.258E+01	1.10
N02 (ppm)	0.898E+00	0.103E+01	0.39
Min. temp. (F)	-0.178E-01	-0.526E+01	1.14
Precip. (inch)	0.798E+00	0.692E+01	0.32

Table C.24

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE EXPOSED CHILDREN: BETWEEN INDIVIDUAL DIFFERENCES (n = 208)

Aerometric Attribute	Tau	z for the Attribute
S02 (ppm)	0.146E+02	0.912E+00
COH	0.462E+00	0.106E+01
TSP ($\mu g/m^3$)	0.000E+00	0.000E+00
Ozone (ppm)	0.000E+00	0.000E+00
N02 (ppm)	0.470E+01	0.102E+01
Min. temp. (F)	0.193E-01	0.108E+01
Precip. (inch)	0.000E+00	0.000E+00

Table C.25

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONEXPOSED CHILDREN: BETWEEN INDIVIDUAL DIFFERENCES (n = 250)

Aerometric Attribute	Tau	z for the 'Attribute	z for the Contrast
S02 (ppm)	0.344E+01	0.955E-01	-0.76
COH	0.000E+00	0.000E+00	-0.91
TSP ($\mu g/m^3$)	0.000E+00	0.000E+00	0.00
Ozone (ppm)	0.000E+00	0.000E+00	0.00
N02 (ppm)	0.000E+00	0.000E+00	-0.87
Min. temp. (F)	0.000E+00	0.000E+00	-0.91
Precip. (inch)	0.000E+00	0.000E+00	0.00

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