Short-Term Health Effects of Air Pollution

A Case Study

N. Duan, T. Hayashi, A. H. Carlson, E. Keeler,
E. Korn, W. G. Manning

RAND

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PREFACE

This report documents the findings of a RAND Corporation study, funded through a cooperative agreement with the U.S. Environmental Protection Agency, on the health effects of air pollution. It represents the second phase of a three-phase project to evaluate the relative abilities of several analytical approaches to the measurement of pollution effects.

The data analyzed here were collected in Dayton, Ohio, in the Health Insurance Experiment (HIE) conducted by RAND for the U.S. Department of Health and Human Services. The first phase of the study applied several analytical methods to data collected from two HIE sites, Dayton and Seattle, Washington. One of the methods, the individual time series analysis based on the Whittemore-Korn model, was used in that effort with data from Seattle. In the current phase, the authors extend the individual time series analysis to HIE data from Dayton.

Reducing the adverse health effects of air pollution is an important goal of government regulations that control air pollution. By comparing the benefits with the costs of control, federal lawmakers and regulators will be in a better position to decide what level of control is most appropriate. Because the present study considers several pollutants, the results may be helpful to regulators who must choose where to assign scarce pollution control resources.

The methodology and findings should also be helpful to several other groups:

- Agencies and individuals interested in air quality and, especially, in decisions made 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 and, especially, in procedures designed to draw precise inferences from limited data.

SUMMARY

This report presents the results from the second phase of our study on the health effects of air pollution on a general population. Previously, we applied a battery of disparate analytical approaches to estimate the health consequences of air pollution, using an especially attractive set of data collected with the same data methods in two widely separated cities—Seattle and Dayton, with the results documented in Coulson et al. (1985). The analytic methods used in the first report included a simple cross-sectional analysis and three panel analyses. One of the methods, the individual time series analysis based on the Whittemore-Korn (WK) model, the individual day-to-day approach, was found to be promising in its ability to detect short-term adverse effects of air pollution. As a result of applying the method to the data from Seattle, we raised a methodological question about the WK method, namely, that the technique of individual-specific logistic regression might result in biased estimates of the health effects of interest because of the small sample sizes available for each individual. In this report, we give the estimated health effects based on applying the same WK method to Dayton, along with the results from a limited Monte Carlo study designed to address the bias question.

The data analyzed were collected during The RAND Corporation's Health Insurance Experiment. This data set was attractive for several reasons:

- It was a sample of the general population, and not of some group selected for a particular characteristic, e.g., susceptibility to air pollution.
- Data were collected in cities with levels of pollution 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 potentially confounding variables and risk factors.

RESULTS

From the first phase of our study, we found the WK or individual time series method to be promising, and recommended extending this analysis to Dayton. The results we obtained from the Dayton data during this second phase of the study are generally consistent with the results based on Seattle. Two of the criterion pollutants, SO_2 and NO_2, have significantly adverse health effects in both sites. Total suspended particulates have significantly adverse health effects in Dayton, but not in Seattle. The rather puzzling finding in the first phase of the study that ozone had a significant perverse (beneficial) health effect is not replicated in Dayton.

The results from the Monte Carlo study indicate that the bias problem does not appear to be serious for the three criterion pollutants for which we have found a significant health effect. It also indicates that the perverse ozone effect in Seattle might be the result of the bias.

LIMITATIONS

The most important limitation of our study was the exclusion of the elderly from the Health Insurance Experiment. The elderly are often regarded as being among the most susceptible to air pollution. The exclusion of the elderly also precludes an examination of the effects of air pollution on mortality.

Second, the individual time series analysis only allows us to study the short-term effects, and does not tell us about the cumulative effects of air pollution.

Finally, the method can only be applied to people who had more than a few sick episodes during the two-year period of health report data collection and therefore might not generalize to the subpopulation of very healthy individuals.

CONCLUSION

We have applied the Whittemore-Korn individual time series analysis to a second general population data set and found it to be a promising method for measuring the short-term health effects of air pollution. The results from both our Seattle and Dayton analyses (carried out in the two phases of our study) consistently identify SO_2 and NO_2 as having significant adverse health effects.

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I. INTRODUCTION

BACKGROUND

It is generally accepted that air pollution adversely affects the health of individuals exposed to it. This has been demonstrated in laboratory studies of pulmonary function for humans in closed chambers with varying levels of air pollutants. There is also support for the adverse effects from observational studies of the relationship between episodes of air pollution and acute, short-term health losses, and of the "killer fogs" of London and Donora, Pennsylvania. Some observational studies also suggest that air pollution has caused other deaths through chronic exposure (Lave and Seskin, 1977).

However, observational studies of the effects of air pollution have not yielded consistent results about the adverse effects of air pollution. This lack of consistency could be due to a variety of factors, including the use of different health measures of varying sensitivity to air pollution; the use of populations of differing susceptibilities to air pollution; the use of air pollution exposure measures with varying levels of measurement error; and the confounding of measures of air pollution exposure with other unmeasured subject or site characteristics (e.g., those susceptible to air pollution may move to less polluted locations).

Another possible reason for the lack of consistency in the results of those observational studies is the difference in the statistical methods. In particular, some studies use cross-sectional analyses, while some use panel analyses. Cross-sectional and panel analyses address different kinds of health effects.

This is the second paper in a series of reports on research by The RAND Corporation under the sponsorship of the U.S. Environmental Protection Agency to examine statistical and econometric issues in assessing the effects of air pollution on several indicators of health outcomes and health-related costs.[1] For this research, we have analyzed data from a panel study of the nonaged population in two cities with moderate levels of air pollution, Dayton, Ohio and Seattle, Washington. In this research, 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 findings from Coulson et al. (1985) suggested the individual time series analysis as a promising method to detect short-term effects of air pollution, as it was applied to data from Seattle, Washington. (See Table 4.2 for a summary of the results from that study.) In this report we apply the same analysis to the same type of data from Dayton, Ohio, to see how well the results replicate.[2] We have also conducted a Monte Carlo study to assess the potential for bias in the individual time series analysis.

ANALYTICAL APPROACH

In this report, we examine the effects of pollution exposure on time lost to illness in terms of short-term responses (daily responses to daily air quality variation). In our earlier work (Coulson et al., 1985), we examined the effects of pollution exposure on the use of health

[1]The first report in this series is Coulson et al. (1985).

[2]The primary purpose of this report is to replicate the Seattle individual time series analysis in Coulson et al. (1985). Comparisons between the individual time series analysis and cross-sectional analysis was given in the earlier report and will not be replicated here.

services and 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). We applied several analytical methods to data collected from two of the sites of the RAND Health Insurance Experiment (HIE), Dayton and Seattle. (See Sec. III below for a description of the HIE.) One of the methods, the individual time series analysis based on the Whittemore-Korn (WK) model, was applied only to the data from Seattle during the first phase. The Whittemore-Korn model yielded promising results in its ability to detect short-term adverse effects of air pollution.

In our earlier Seattle analysis, we raised a methodological question about the WK method, namely, that the use of individual-specific logistic regressions might result in biased estimates of the health effects of interest because of the small sample sizes available for each individual.

Our analysis here examines the effect of air pollution on a set of daily observations for each person individually. For each individual, we estimate a separate logistic regression for the probability that he will lose time due to illness because of air pollution.

In this panel analyses, we use the presence of repeated observations on each individual to control for unobservable individual characteristics. 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 exposure.

The Whittemore-Korn (WK) technique has been applied only twice before—in the original WK application, and by us in Coulson et al. (1985). The promise of the technique is that each individual can act as his own control. However, the technique is expensive to implement, because we must estimate a separate logistic regression for each person; in cross-sectional studies, one pooled multiple regression analysis can be estimated. By studying the behavior of this technique, by applying it to a new data set collected in a comparable manner, we can see how well the results replicate across different sites. Thus the results may help other researchers in their choice of estimation strategies.

In addition to applying the WK method to Dayton, we present the results from a limited Monte Carlo study designed to address the bias question.

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 of the adverse effects of air pollution on health outcomes. Although the study relies on data from a randomized study, the randomization was for health insurance and not for air pollution. Families with members who are susceptible to air pollution may choose to live in less polluted areas. If this happens, cross-sectional association between health and air pollution would be biased; the bias is commonly referred to as the geographical sorting bias.[3] While our panel technique can avoid geographical sorting bias, it could still be subject to other types of confounding; therefore, our conclusion does not have the unambiguous interpretation of conclusions that can be drawn from experiments.

Second, in this study, we are only examining the short-term effects of air pollution on time lost due to illness. Thus the results do not include the adverse effects of air pollution on the use of services, health status and physical well-being, or life expectancy.

[3]The geographical sorting bias would generally make air pollution appear less harmful.

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.

Fourth, this report is limited to two sites. Hence, at this point we do not know how generalizable the results are.

Finally, the sample excludes individuals who are over 65, 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 data from the Health Insurance Experiment understate the full social effects of air pollution.

RESULTS

From the first phase of our study, we found the individual time series method to be promising, and recommended extending that analysis to Dayton. The results we obtained from the Dayton data during this second phase of the study are generally consistent with the results based on Seattle data. Two of the criterion pollutants, SO_2 and NO_2, were found to have significantly adverse health effects in both sites. Total suspended particulates were found to have significantly adverse health effects in Dayton, but not in Seattle. The rather puzzling finding in the Seattle results that ozone had a significant perverse (beneficial) health effect is not replicated in Dayton.

The results from the Monte Carlo study indicate that the bias problem does not appear to be serious for the three criterion pollutants for which we have found a significant health effect. It also indicates that the perverse ozone effect in Seattle might be the result of the small sample bias.

ORGANIZATION OF THE REPORT

Section II reviews the Whittemore-Korn methodology. Section III describes the data and the sample used in this analysis. Section IV presents the results from the Dayton sample for time lost due to illness, and Sec. V reports the results of the Monte Carlo study.

II. INDIVIDUAL TIME SERIES APPROACH: METHOD

In this section, we review the individual time series analysis based on the approach proposed in Whittemore and Korn (1980). The individual time series approach is carried out in two stages. First, we estimate each individual's daily health outcome as a function of his daily aerometric exposures to assess his individual-specific response. Each individual serves as his 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, on average, do the people in the population get sick more often on polluted days than on clean days? Second, do the various individuals in the population have the same response to air pollution, or do they respond differently? Third, if the individuals respond differently, are their responses related to known characteristics about them? (For example, are children more sensitive to air pollution than adults?)

We describe the Whittemore-Korn model below. The sample and data used in this analysis are discussed in the next section. The results of the analyses applying the Whittemore-Korn technique to the HIE data are given in Sec. IV.

THE WHITTEMORE-KORN MODEL

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 hour or week; the 24-hour time 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:

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

where p_{it} is the i-th person's probability of being 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; and 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:

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

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 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 hypothesis $\gamma_j = 0$ and $\tau_j = 0$; the two hypotheses together are equivalent to the global null hypothesis $\beta_{ij} = 0$.

When there are between-individual differences it might be desirable to relate those differences to observed characteristics of the individuals. For example, it might be useful to know whether the individual's response to air pollution is related to his health status, i.e., whether a healthy person is less sensitive to air pollution than an unhealthy person. We are capable of carrying out this analysis only for dichotomous characteristics.

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 (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 be different. For example, if only smokers are 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 own control, which avoids the confounding problems that occur with cross-sectional methods (see, e.g., Coulson et al., 1985, Sec. III). 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. Furthermore, the Whittemore-Korn model allows us to estimate each person's air pollution exposure based on monitoring data obtained at close proximity to the individual's residence or worksite; such estimates are preferable to the "central site" exposure estimates used in the aggregated time series approach (Coulson et al., 1985, Sec. VI).

One limitation of the model is that it can only be applied 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 of his being sick, on a polluted day or on a clean day, is zero. 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. Thus, we restrict our analysis to people with more than a few sick days and more than a few healthy days over a period of up to two years. In the latter part of Sec. III ("Time Lost to Illness Sample") we discuss how we imposed this restriction on the HIE data.

MONTE CARLO STUDY

Another methodological issue is the possible presence of small-sample bias in the estimated logistic regression coefficients: Since logistic regression is nonlinear, the regression coefficients are asymptotically consistent, i.e., are nearly unbiased if the number of days is large, but could be biased for small sample sizes. We noted in our earlier report (Coulson et al., 1985) that there is a rather puzzling correlation between the estimated coefficients and the associated standard errors. One of the possible explanations of this correlation that was offered in the earlier report is small-sample bias. We have conducted a limited Monte Carlo study to investigate this issue; the results are given in Sec. V.

III. SAMPLE AND DATA

The data for this analysis were drawn from two sources. First, the source of data on health status and time lost due to illness was the Health Insurance Experiment (HIE). Second, the sources of data on air quality and weather were EPA's Storage and Retrieval of Aerometric Data (SAROAD), and the National Weather Service.

Here, we provide an overview of the data. For a fuller discussion, see Coulson et al. (1985). Newhouse (1974) and Brook et al. (1979), provide fuller descriptions of the HIE design.

THE HEALTH INSURANCE EXPERIMENT

The HIE was a randomized trial of the effects of different health insurance arrangements on the demand for health services and the health status of individuals. The HIE enrolled families in six sites: Dayton, Ohio; Seattle, Washington; Fitchburg, Massachusetts; Franklin County, Massachusetts; Charleston, South Carolina; and Georgetown County, South Carolina.

In the first phase of our study, we used HIE data from two of the six sites, Dayton and Seattle, for several of the analyses, except for the individual time series analysis, which was applied only to the data from Seattle because of the limited amount of time available then. Part of the task in this second phase of the study is to extend the individual time series analysis to Dayton.

Families participating in the experiment were assigned to 14 different fee-for-service or 2 prepaid group practice insurance plans. The fee-for-service insurance 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 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.[1]

To study methods effects, the HIE included 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. (The responses to those diaries provide the outcome data

[1]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.

for our analysis. See the subsection on the dependent variable for more discussion.) 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.

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.[2]

In Dayton, we found no unintended differences between the group that accepted and the group that refused the offer to participate in the study; see Newhouse et al. (1982).

THE DAYTON SAMPLE

The Dayton sample was a random sample of the general population in the Dayton metropolitan area, 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 jails and those institutionalized in long-term hospitals; (5) those in the military or their dependents; and (6) those with service-related disabilities.

The Dayton sample was reduced because of structural missing data. In this analysis we used only the individuals who were assigned (randomly) to the subexperimental treatment requiring them to file health diaries.[3] Individuals who were assigned to the alternative subexperimental treatment not to receive health diaries did not generate any data on the time lost to illness; they are excluded from this analysis.

Exposure Estimation

Assessing the relationships between health outcomes and exposure requires an estimate of the exposure of individuals to air pollution. Ideally, personal monitoring (equipping each individual with a personal monitor) would give the best measurements of individual exposures to air pollution. An alternative is to use microenvironmental monitoring, collecting air pollution concentration data in locations (microenvironments) where people spend time, such as workplace, home, etc., then combine the microenvironment concentration data to estimate exposure. Unfortunately, we could not implement either of these two methods, because this research was initiated well after the HIE data collection effort ended. Instead, we used the air quality data from EPA's database Storage and Retrieval of Aerometric Data (SAROAD) 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

[2]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.

[3]Some of the individuals might not file all the required health reports. In our analysis we also exclude those individuals who have fewer than a hundred days of usable data. See the discussion below: "Time Lost to Illness Sample."

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 used 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 [SO_2], nitrogen dioxide [NO_2], oxidants, and carbon monoxide [CO]) for the Dayton metropolitan area. 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 Health Insurance Experiment (HIE), namely, November 1974 through February 1980 in Dayton.

The number of monitoring sites for each pollutant varied 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.[4]

Missing Values. We did accept data from monitoring sites with minor breaks or gaps in their daily or hourly values, because monitoring sites break for routine maintenance. For monitoring sites with missing hourly or daily values on 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 analysis of variance (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.

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 monitoring 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 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.

For each individual, we developed a daily time series for their daytime and nighttime locations using the residence and work data described above. For the nighttime location, we used their 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.

[4]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.

We then linked day-by-day each person's day 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. Zip codes 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. Appendix Table A.4 shows the frequency count of individuals by home location on the first day of the study, and the corresponding latitude and longitude. Table A.5 shows 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. Tables 3.1 and 3.2 summarize the daily time series for two hypothetical individuals, one residing near central Dayton (zip = 45403), and one residing in the suburban community of Beavercreek (zip = 45432). The monthly averages for the aerometric variables in central Dayton is given in App. A as Figures A.1 through A.7.

Table 3.1

SUMMARY OF AEROMETRIC DATA, CENTRAL DAYTON

Variable	Mean	Standard Deviation
SO_2	0.01333535	0.01269970
TSP	96.56863049	34.60550780
Ozone	0.05816226	0.03852947
NO_2	0.04823710	0.02458808
CO	2.65585979	1.36370625
Minimum temperature	40.73461012	19.70538187
Precipitation	0.08485636	0.22749939

Table 3.2

SUMMARY OF AEROMETRIC DATA, BEAVERCREEK

Variable	Mean	Standard Deviation
SO_2	0.01333535	0.01269970
TSP	64.66313956	26.26544416
Ozone	0.05816226	0.03852947
NO_2	0.04823710	0.02458808
CO	2.65585979	1.36370625
Minimum temperature	40.73461012	19.70538187
Precipitation	0.08485636	0.22749939

TIME LOST TO ILLNESS

Unit of Analysis

The unit of analysis is a person-day for the individual time series analysis. Since in this analysis each individual is his own control, we did not use any HIE demographic or health status measures as independent variables. The only independent variables used were aerometric data, including daily air pollution measurements and meteorological variables.

Dependent Variable

In this report, we focus on one health outcome: the amount of time lost due to illness. We examine the association of air pollution (daily or annual) and the amount of time that an individual is ill. The HIE sent out diaries to the enrolled households on a regular basis, some weekly, and some biweekly. The respondents filled out the diaries and mailed them back to the HIE. They were paid for doing so. The diaries provided data on days lost from work, school, or usual activities from the health diary system.[5] For children, we know when a person was ill and whether he took time off from school or just restricted his 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 care for another family member. In the case of workers, we have data on sick-leave provisions and know if sick leave was used for a particular sick-loss day. The HIE data on time lost to illness does not contain any information on symptoms or diagnoses. Therefore, it is impossible to separate the sick-loss days related to air pollution from those that are not.

For this analysis, we used a combination of restricted activities, school loss, and work loss as given in the biweekly HIE 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 was treated as a sick day; otherwise the day was treated as a healthy day.

Because of limitations in the data, we needed to make some revisions in the Whittemore-Korn model in order to apply it appropriately. (The same revisions were made in Coulson et al., 1985.) One of the important findings in Whittemore-Korn (1980) was 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 deleted all days after a sick day and focused on the estimation for days after a healthy day. In other words, we estimated only the probability for the transition from the

[5]A random subset of the participants was required to fill out health diaries for some part of the study. Form completion rates averaged 93 percent in Seattle and 95 percent in Dayton during the fiscal year beginning July 1, 1976, and 90 and 92 percent, respectively, in the year beginnning July 1, 1977. Item nonresponse, involving 5 to 8 percent of the families, generated a call-back to the family.

For each two-week period, the diary asks whether (1) anyone telephoned a health provider (except to make an appointment); (2) anyone stayed overnight or longer in a hospital or other health facility; (3) anyone visited a health provider or purchased drugs or medical supplies; (4) any child (age 16 or under) had a fever or was sick, missed school for a half day or more due to illness or injury, or had to cut down on usual activities for a half day or more on non-school days due to illness or injury; (5) any adult had to miss work, cut down on usual activities on a nonwork day, or miss any work to visit a health provider (for oneself or to assist another). For each yes response, the family identified the person(s) and dates involved.

healthy status into a sick episode; a sick episode is dated to the first day of a series of consecutive sick days.

Time Lost to Illness Sample

The maximum number of people who could be used in this analysis is 1070—the number of HIE participants who were assigned to file health reports while in the Dayton metropolitan area. This is about one-third the sample size that was available for the Seattle sample that was analyzed in Coulson et al. (1985). On the average we have 641 daily reports per person. This is almost identical to the rate of 630 for Seattle. The maximum number of days possible for each person is 731. However, some of the participants moved out of the Dayton area before the end of the health report study period, and some failed to file all required health reports.

The HIE participants in Dayton averaged 2.60 sick episodes per person during the two-year period. This is substantially lower than the rate of 4.34 in Seattle, although the per capita amount of time at risk is about the same for the two sites. The distribution of sick episodes is fairly skewed. More than 30 percent of the HIE participants in Dayton had no sick episodes. (The rate in Seattle is just over 10 percent.) The median value was one episode. The maximum value was 57.

As was discussed towards the end of Sec. II, the logistic regression model is usually not estimable when the number of sick episodes is too low; so we needed to restrict the analysis to people with more than a few sick episodes. For the sake of comparability, we used the same restriction that was used in Seattle—we included only those people with more than three episodes; that left 260 persons in Dayton. However, those people reported 1934 sick episodes, which is almost 70 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. (In Seattle, the people with more than three episodes account for more than 80 percent of all sick episodes.)

The restricted sample of 260 persons with more than three episodes averaged 676 daily health reports per person, which is very close to the analogous rate of 684 in Seattle. The average number of days is higher than 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 could be used in the analysis. As we discussed above, because a sick episode rather than a sick day was used as the health outcome, we had to delete all days immediately following a sick day. Furthermore, some days could not be used in the analysis because of missing air pollution data. With those deletions, an average of 279 days per person remained for the 260 people with more than three sick episodes. This number was substantially lower than the corresponding rate of 425 days for Seattle, mainly because there was more missing air pollution data in Dayton than in Seattle.

There were a few people with very few days available for analysis. We made the same restriction as was used in Seattle, using only people with at least a hundred days available for analysis. Under this restriction four people were deleted leaving 256 persons in the sample. Those people averaged 283 days per person and 7.4 sick episodes each.

The SAS logistic regression algorithm failed to converge for 9 of the 256 remaining people, indicating that there were insufficient data to identify the model.[6] Restricting to the people

[6]The failure of the logistic regression algorithm to converge has two interpretations. It might indicate that there are insufficient data to estimate one or more of the coefficients. It might also indicate that there is an infinite estimated coefficient for one or more of the aerometric variables. However, we also have individuals for whom the logistic regression algorithm "barely converged," resulting in some very large estimated coefficients. Since the standard errors associated with those large coefficients are also very large (see Figs. B.8 through B.14 in App. B), we conclude that the former interpretation is preferable.

for whom the algorithm succeeds to converge, we have 247 people in the final analysis sample. They average 283 days per person and 7.5 sick episodes each. (The final analysis sample in Seattle average 429 days per person and 8.5 episodes.)

Explanatory Variables

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

The air pollution data were from SAROAD. The following daily air pollution measures were used: daily average of sulfur dioxide (SO_2); daily average of TSP; daily maximum hourly average of ozone; daily maximum hourly average of nitrogen dioxide (NO_2); and daily average of carbon monoxide (CO).[7] Air pollution at a person's residence or work location was assumed to be the same as that at the nearest monitoring site; see the discussion of exposure above and in Sec. II of Coulson et al. (1985).

Values of the various air pollution measures were distributed over days in a somewhat skewed fashion. The statistical measures of skewness are similar to those for Seattle and range 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 maintained the same specification that was used in Seattle; namely, we did not transform the air pollution variables.

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

The distribution of precipitation is very skewed, because more than 60 percent 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, and the other the amount of precipitation (or a transformed amount). However, since the effect of precipitation was not of primary interest in this study, we simply used the amount of precipitation, without a transformation.

In addition to the aerometric data, we used two calendar-related covariates to control for possible confounding effects. The first was an indicator variable for weekday versus weekend; this was 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 was an indicator variable for the first week of each two-week health report period. Because we used 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 have been 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 that there would be a substantial amount of correlation among our explanatory variables. Explanatory variables that are highly correlated with each other might be nearly collinear; i.e., one of

[7]The set of air pollution measures used in the analysis is somewhat different from that used in Seattle. The COH measurements, which were used in Seattle, were unavailable in Dayton for most of the HIE measurement period; therefore we had to drop COH as an explanatory variable. Since the effect of COH in Seattle was found to be fairly minimal and was statistically insignificant (Coulson et al., 1985), we do not see this as a major problem. We did not include CO in the analysis for Seattle because of our prior opinion that CO would not affect short-term health effects. In order to validate this prior opinion, we have included CO in the Dayton analysis. The empirical results to be presented in the next section are consistent with this prior opinion.

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 were indeed significantly correlated, but the magnitude of the correlation coefficients were all small or moderate, with the largest being the correlation between ozone and minimum temperature, about 0.6, reflecting the role of sunshine in the production of ozone (see Tables A.1 and A.2). The multiple correlations among the aerometric variables were also small, with the exception that the multiple correlation for ozone and minimum temperature were both near 0.8, as a result of the simple correlation between the two variables.

While there was a substantial amount of correlation among the aerometric variables, none of the correlation coefficients was sufficiently high to make collinearity a major concern. This was consistent with the finding in Seattle.

In summary, the descriptive nature of the data in Dayton was fairly similar with Seattle and, whenever possible, we employed the same specification for our analysis to make the two analyses comparable. The major exceptions to this general statement are the following:

- The rate of sick episodes was lower in Dayton than in Seattle.
- There were more missing air pollution data in Dayton than in Seattle.
- A few people in Dayton were deleted from the analysis because the logistic regression algorithm failed to converge.

IV. INDIVIDUAL TIME SERIES APPROACH: RESULTS

We applied individual-specific logistic regression analyses to the final analysis sample of 247 HIE participants in Dayton. We then applied the random-effects model to the estimates of the estimated responses and standard deviations of individual-specific effects. The results are given in Table 4.1. For four of the pollution measures (SO_2, TSP, NO_2, and CO), 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 three of the four, SO_2, NO_2, and TSP, the effect is statistically significant at the 1 percent level. The effect for CO is statistically insignificant. The average effect for ozone is negative, but is statistically insignificant. The average effect for precipitation is positive and statistically significant, indicating that it is more likely that a sick episode will occur on a day with precipitation. The effect for minimum temperature is negative, indicating that a sick episode is more likely on a cold day; however, the effect is statistically insignificant.

For comparison, we reproduce the analogous table from the Seattle analysis (Table 5.1 in Coulson et al., 1985) below as Table 4.2. The results in Dayton and Seattle appear to be qualitatively similar. The adverse health effects of SO_2, NO_2, and precipitation hold for both sites. The major difference is seen in ozone: in Seattle, we found the rather puzzling perverse (beneficial) effect of ozone; this result is not replicated in Dayton. On the other hand, the magnitudes of the effects are substantially different for some of the effects which are found to be qualitatively similar between the two sites. The effects of SO_2, TSP, and NO_2 in Dayton are substantially larger than in Seattle. Although some of these differences are large, none is statistically significant at the 5 percent level.

The results shown in Table 4.1 indicate that SO_2 has a significant association with higher probabilities of sick episodes. 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 10.5 logit units in the probability of a sick episode. If the average SO_2 level in downtown Dayton almost triples from its present 0.013 ppm to the 0.030 ppm, the primary federal standard level for the annual average, the probability that the average person would experience a sick

Table 4.1

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL
SUMMARIES FOR THE AEROMETRIC EFFECTS OVER
THE DAYTON FINAL ANALYSIS SAMPLE:
AVERAGE RESPONSES
(N = 247)

Aerometric Attribute	Estimated Coefficient	z Statistic
SO_2 (ppm)	10.53	4.7
TSP ($\mu g/m^3$)	0.00247	2.3
Ozone (ppm)	−0.84	−0.7
NO_2 (ppm)	3.53	2.6
CO (ppm)	0.013	0.6
Minimum temperature (F)	−0.0032	−1.4
Precipitation (in.)	0.499	3.8

Table 4.2

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

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

episode would increase by 0.18 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 0.18 in the logarithm of the probability of having a sick episode is equivalent to multiplying the probability of a sick episode by 1.20. For the final analysis sample, on the average, this is equivalent to an increase from 0.026 sick episodes per person-day to 0.031. This should be considered a fairly major impact on health. (It should be noted, of course, that the assumption that the SO_2 level increases from its present 0.013 ppm to 0.030 ppm is also a major deterioration of air quality.)

An alternative way to interpret the health significance (as opposed to statistical significance) of the results in Table 4.1 is to calculate the change in sick episode rate when the air pollutant level increases by 10 percent. For example, a 10-percent increase in SO_2 would cause sick episodes per person-day to increase from 0.026 to 0.0264. For TSP, the same 10-percent increase would raise sick episodes to 0.0266. For ozone, sick episodes would fall to 0.0259. For NO_2, sick episodes would increase to 0.0264.

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 4.3. None of these between-individual differences is statistically significant. (This is rather different from the result in Seattle, where several of the aerometric effects had significant between-individual differences. It should be noted, though, that the final sample size in Seattle is more than five times that in Dayton.) In other words, the individuals in the Dayton sample do not respond differently to aerometric attributes to an extent that can be detected as being statistically significant. However, we need to interpret this result carefully, because the amount of information available for detecting between-individual differences is fairly limited in our data. For some of the aerometric attributes, e.g., TSP and NO_2, the between-individual standard deviation tau is substantially larger than the corresponding average effect. However, the former is found to be statistically insignificant, while the latter (of a smaller magnitude) is found to be statistically significant. This indicates that there is more information in estimating the average effect than in estimating the between-individual standard deviation tau.

The Dayton data also replicated the finding from Seattle that there is a strong negative association between the coefficients and their standard errors; see Figs. B.8 through B.14 in

Table 4.3

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE FINAL ANALYSIS SAMPLE: BETWEEN-INDIVIDUAL DIFFERENCES (N = 247)

Aerometric Attribute	Tau	z Statistic
SO_2 (ppm)	0.0	0.0
TSP ($\mu g/m^3$)	0.0055	1.3
Ozone (ppm)	0.56	0.01
NO_2 (ppm)	6.91	1.4
CO (ppm)	0.0	0.0
Minimum temperature (F)	0.0105	1.0
Precipitation (in.)	0.44	0.7

App. B. Motivated by the repeated finding of this phenomenon, we conducted a limited Monte Carlo study. The details are described in Sec. V of this report.

An alternative to the random-effects model discussed above is the analysis of individual z statistics. For each aerometric variable and each individual, we divide the estimated logistic coefficient by the estimated standard error to obtain the individual's z statistic for this aerometric variable.[1] Under the null hypothesis that there is no response to this aerometric variable for all individuals, the z statistic follows a standard normal distribution with mean zero and standard deviation one.[2] Under the alternative hypothesis that the response to this aerometric variable is nonzero, the z statistic does not follow a standard normal distribution. In particular, the mean of the z statistic is nonzero. Therefore, we can test for the presence of the response using the average of the individual-specific z statistics; if the average is significantly different from zero, we reject the null hypothesis that there is no response.[3] Further details on the z analysis and further summary measures of the individual responses are given in App. B.

The results for the average effects based on the two methods are summarized qualitatively in Table 4.4.

The two sets of results are rather dissimilar. However, 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. We base our interpretations on the random-effects model analysis, which is more efficient than the z analysis if the Whittemore-Korn model is valid.[4] The discrepancies between the two approaches, which were also seen in Seattle, warrant further investigation and should be considered an indication that the random-effects model analysis results must be interpreted carefully.

[1]Most statistical packages refer to this quantity as the t statistic; for a nonlinear regression analysis such as logistic regression, the only distribution theory available is that the t statistic is asymptotically normal; therefore we can make statistical inferences treating the t statistic as a normal score z.

[2]The statement is true only in the asymptotic sense when the number of days for each individual is large.

[3]The random-effects model and the z analysis are both based on weighted averages of the individual responses. In essense, the random-effects model weights each individual by the reciprocal of the square of the individual's standard deviation; the z analysis weights each individual by the reciprocal of the standard deviation.

[4]If the Whittemore-Korn model is valid, the reciprocal of the square of the standard deviation is the optimal weight; therefore the random-effects model analysis is more efficient than the z analysis.

Table 4.4

SIGNIFICANCE OF THE AVERAGE RESPONSES

Aerometric Attribute	Random-effects Model	Individual z Statistics
SO_2	+ *	–
TSP	+ *	+
Ozone	–	– *
NO_2	+ *	+
CO	+	– *
Minimum temperature	–	– *
Precipitation (in.)	+ *	+

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

Another caveat about the results is that the estimated logistic regression coefficients might be subject to small-sample bias because logistic regression is nonlinear. We have conducted a limited Monte Carlo study to investigate this issue (see Sec. V).

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. Part of the reason for doing this comparison is to assess the presence of differential susceptibility. Furthermore, this comparison is crucial for understanding the generalizability of our analysis. As was noted earlier, we cannot apply the individual logistic regression to individuals with very few sick episodes, and have to restrict attention to individuals with more than three sick episodes. If the comparison indicates the presence of differential susceptibility, we cannot generalize our results to the subpopulation with three or fewer sick episodes.

We use the same cutoff threshold that we used in Seattle, and compare the responses for those with seven or more sick episodes (the sick subpopulation, containing 140 individuals, 57 percent of the final analysis sample) with those with four to six sick episodes (the less sickly subpopulation, containing 107 individuals, 43 percent of the final analysis sample). The percentages are fairly close to the analogous percentages in Seattle, where 53 percent of the final analysis sample had seven or more sick episodes and 47 percent had four to six episodes.

The average responses for the two subpopulations are given in Tables 4.5 and 4.6. The column "z for the contrast" in Table 4.6 gives the z statistics for the difference between the average responses in the two subpopulations.

For all the aerometric attributes, the z's for the contrast are statistically insignificant, indicating there is no detectable difference between the responsiveness of the people with seven or more sick episodes as compared with people with four to six sick episodes. If we take this result literally, we can further infer that our results can probably be generalized to the people with three or fewer sick episodes.

The lack of statistical significance needs to be interpreted carefully, though. The magnitudes of some of the contrasts are fairly large. The effect of SO_2 for the people with four to six sick episodes is almost 50 percent higher than that for the people with seven or more sick

Table 4.5

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SICK SUBPOPULATION: AVERAGE RESPONSES (N = 140)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	Efficiency
SO_2 (ppm)	9.06	3.5	0.73
TSP ($\mu g/m^3$)	0.0026	2.0	0.67
Ozone (ppm)	−0.95	−0.7	0.73
NO_2 (ppm)	2.88	1.8	0.69
CO (ppm)	0.015	0.6	0.76
Minimum temperature (F)	−0.0031	−1.1	0.73
Precipitation (in.)	0.404	2.3	0.61

Table 4.6

META-ANALYSIS BASED ON THE RANDOM EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LESS SICKLY SUBPOPULATION: AVERAGE RESPONSES (N = 107)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	14.52	3.3	1.1
TSP ($\mu g/m^3$)	0.0020	1.0	−0.2
Ozone (ppm)	−0.72	−0.3	0.1
NO_2 (ppm)	5.66	2.2	0.9
CO (ppm)	−0.0023	−0.1	−0.4
Minimum temperature (F)	−0.0076	−1.6	−0.8
Precipitation (in.)	0.72	3.3	0.9

episodes. The effects of NO_2 and precipitation nearly doubled. Apparently, the precision in those estimated contrasts is fairly limited. In other words, the lack of statistical significance does not rule out the possibility that there might be some relevant differences which cannot be detected with the precision available in our Dayton data.

If we regard the average responses given in Table 4.5 for the sickly subpopulation and those given in Table 4.1 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, the estimates in Table 4.1 are based on 1.76 times as many people as the estimates in Table 4.5; 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 4.5. The efficiency is based on the precision of the estimated average responses. For each aerometric attribute, the efficiency is the ratio of the variance of the average coefficient in Table 4.1 to the corresponding variance of the average coefficient in Table 4.5. For all aerometric attributes, the efficiency of the sickly subpopulation is about 70 percent. In other words, the near doubling of the

Table 4.5

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SICK SUBPOPULATION: AVERAGE RESPONSES (N = 140)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	Efficiency
SO_2 (ppm)	9.06	3.5	0.73
TSP ($\mu g/m^3$)	0.0026	2.0	0.67
Ozone (ppm)	−0.95	−0.7	0.73
NO_2 (ppm)	2.88	1.8	0.69
CO (ppm)	0.015	0.6	0.76
Minimum temperature (F)	−0.0031	−1.1	0.73
Precipitation (in.)	0.404	2.3	0.61

Table 4.6

META-ANALYSIS BASED ON THE RANDOM EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LESS SICKLY SUBPOPULATION: AVERAGE RESPONSES (N = 107)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	14.52	3.3	1.1
TSP ($\mu g/m^3$)	0.0020	1.0	−0.2
Ozone (ppm)	−0.72	−0.3	0.1
NO_2 (ppm)	5.66	2.2	0.9
CO (ppm)	−0.0023	−0.1	−0.4
Minimum temperature (F)	−0.0076	−1.6	−0.8
Precipitation (in.)	0.72	3.3	0.9

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number of individuals from the 140 sickly persons to the 247 in the final analysis sample by the inclusion of the 107 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 short, the amount of information for each healthy person is only about 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 him 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, under the assumption that they have the same expected responses.

Tables 4.7 and 4.8 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 healthy subpopulation only increases the effective sample size by about 25 percent. That is, each sickly individual contributes about four times the information that a less sickly individual contributes to the estimation of tau.

Table 4.7

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SICK SUBPOPULATION: BETWEEN-INDIVIDUAL DIFFERENCES (N = 140)

Aerometric Attribute	Tau	z for the Attribute	Efficiency
SO_2 (ppm)	2.45	0.1	0.83
TSP ($\mu g/m^3$)	0.0066	1.7	0.67
Ozone (ppm)	3.09	0.4	0.77
NO_2 (ppm)	8.15	1.6	0.74
CO (ppm)	0.0	0.0	0.90
Minimum temperature (F)	0.011	1.0	0.83
Precipitation (in.)	0.49	0.6	0.62

Table 4.8

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LESS SICKLY SUBPOPULATION: BETWEEN-INDIVIDUAL DIFFERENCES (N = 107)

Aerometric Attribute	Tau	z for the Attribute	z for the Contrast
SO_2 (ppm)	0.0	0.0	−0.03
TSP ($\mu g/m^3$)	0.0	0.0	−0.8
Ozone (ppm)	0.0	0.0	−0.1
NO_2 (ppm)	0.0	0.0	−0.7
CO (ppm)	0.091	0.3	0.3
Minimum temperature (F)	0.0	0.0	−0.4
Precipitation (in.)	0.0	0.0	−0.8

For both subpopulations, none of the aerometric attributes has a statistically significant between-individual difference. For many of the effects, the maximum likelihood estimate for tau is actually zero. As was noted earlier, this lack of detectable difference should be interpreted carefully in view of the limited amount of information available from the rather small Dayton sample.

FURTHER COMPARISONS OF SUBPOPULATIONS

We have conducted several more subpopulation comparisons. These include two more contrasts based on the subjects' health status—one in which we dichotomize according to the subject's pulmonary susceptibility, and one in which we dichotomize according to the subject's lung function. We also made contrasts between adults and children. The results are given below.

Lung Function. We classify people into healthy and less healthy subpopulations based on their lung function, using the forced expiratory volume in a second (FEV_1, which was measured for a random subset of HIE participants aged 20 and over who were assigned to the subexperimental treatment receiving a preexperimental physical exam. (See Coulson et al., 1985, Sec. II, for further discussions on this measure.) 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 90 persons for whom we have FEV_1 measurements, 27 fall into this subpopulation; the other 63 are classified as low-FEV_1 persons. (We do not have the FEV_1 measurement for all HIE participants.) The results are given in Tables 4.9 and 4.10. None of the comparisons between these two subpopulations is statistically significant.

Pulmonary Susceptibility. We define a person as being susceptible to pulmonary problems if he has one of the important pulmonary diseases such as asthma, emphysema, or hay fever. We have 47 persons who fall into this category. The results are given as Tables 4.11 and 4.12. None of the comparisons between these two subpopulations is statistically significant.

Table 4.9

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE HIGH FEV_1 SUBPOPULATION: AVERAGE RESPONSES (N = 27)

AerometricAttribute	Estimated Coefficient	z for the Attribute
SO_2 (ppm)	17.37	2.5
TSP ($\mu g/m^3$)	0.0050	1.6
Ozone (ppm)	−1.17	−0.3
NO_2 (ppm)	5.32	1.2
CO (ppm)	−0.034	−0.5
Minimum temperature (F)	−0.0093	−1.3
Precipitation (in.)	0.42	1.1

Table 4.10

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE LOW FEV_1 SUBPOPULATION: AVERAGE RESPONSES (N = 63)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	5.69	1.3	−1.4
TSP ($\mu g/m^3$)	0.0016	0.7	−0.9
Ozone (ppm)	−0.36	−0.2	0.2
NO_2 (ppm)	4.92	2.0	−0.1
CO (ppm)	−0.0056	−0.1	0.2
Minimum temperature (F)	−0.0013	−0.3	1.0
Precipitation (in.)	0.30	1.2	−0.2

Table 4.11

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE SUSCEPTIBLE SUBPOPULATION: AVERAGE RESPONSES (N = 47)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
SO_2 (ppm)	8.03	1.5
TSP ($\mu g/m^3$)	0.00096	0.4
Ozone (ppm)	2.01	0.9
NO_2 (ppm)	4.74	1.7
CO (ppm)	−0.24	−0.5
Minimum temperature (F)	−0.0031	−0.6
Precipitation (in.)	0.32	1.2

Table 4.12

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE NONSUSCEPTIBLE SUBPOPULATION: AVERAGE RESPONSES (N = 200)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	11.04	4.4	0.5
TSP ($\mu g/m^3$)	0.0029	2.3	0.8
Ozone (ppm)	−1.91	−1.4	1.5
NO_2 (ppm)	3.64	2.5	−0.1
CO (ppm)	0.019	0.8	0.9
Minimum temperature (F)	−0.0045	−1.6	−0.2
Precipitation (in.)	0.60	3.8	0.9

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. Second, children are known to generally spend more time outside than adults; therefore, 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. In the final analysis sample, we have 156 adults and 91 children. The results are given in Tables 4.13 and 4.14. We found children to be significantly more "responsive" to ozone: The adults do not have a significant response to ozone, while the children have a significant perverse (beneficial) response to ozone; the difference between the two groups is statistically significant at the 5 percent level.

Table 4.13

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE ADULT SUBPOPULATION: AVERAGE RESPONSES (N = 156)

Aerometric Attribute	Estimated Coefficient	z for the Attribute
SO_2 (ppm)	8.68	3.2
TSP ($\mu g/m^3$)	0.0033	2.3
Ozone (ppm)	0.81	0.6
NO_2 (ppm)	4.08	2.6
CO (ppm)	0.015	0.6
Minimum temperature (F)	−0.0036	−1.2
Precipitation (in.)	0.49	3.0

Table 4.14

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE CHILDREN SUBPOPULATION: AVERAGE RESPONSES (N = 91)

Aerometric Attribute	Estimated Coefficient	z for the Attribute	z for the Contrast
SO_2 (ppm)	14.09	3.7	1.1
TSP ($\mu g/m^3$)	0.00043	0.2	−1.3
Ozone (ppm)	−5.21	−2.3	−2.2
NO_2 (ppm)	2.63	1.1	−0.5
CO (ppm)	0.0065	0.2	−0.2
Minimum temperature (F)	−0.0054	−1.2	−0.3
Precipitation (in.)	0.65	2.7	0.5

CONCLUSIONS

Three of the criterion pollutants, SO_2, TSP, and NO_2, exhibit statistically significant associations with the probability of becoming sick: Individuals are more likely to become sick on days with higher levels of the pollutants than on days with lower levels. This result is based on using each individual as his own control, and is also controlled for several sources of possible confounding, including meteorological conditions (daily minimum temperature and amount of precipitation) and possible calendar effects (weekday versus weekend).

To the extent of precision available in our data, different individuals appear to have the same response to the aerometric characteristics. First, we found the between-individual differences to be statistically insignificant (Table 4.3). Moreover, the subpopulation contrasts were generally found to be statistically insignificant. The only exception to the above general conclusion is the significant difference between adults' and children's response to ozone.

V. MONTE CARLO STUDY OF THE WHITTEMORE-KORN MODEL

In Dayton, we found a fairly strong correlation between the coefficients in the fitted individual-specific logistic regression models and the corresponding standard errors. This finding replicates the similar earlier Seattle finding (Coulson et al., 1985) based on the data from Seattle.

When we first discovered this phenomenon in Seattle, we offered two possible explanations. First, there may be a negative association between the true individual coefficients and their true standard deviations. We considered this explanation to be implausible 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 conjectured 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 have larger biases. Since these same individuals will tend to have larger standard errors of their coefficients, this could lead to the observed associations in Figs. B.8 through B.14 in App. B.

To check the small sample bias conjecture, we conducted a limited Monte Carlo study. We used the observed independent variables and simulated random sick episodes based on a given logistic regression model. We then ran a logistic regression on the simulated sick episode data to see whether there is a negative association between the simulated estimated coefficients and their standard errors. Because there is no association between the true individual coefficients and their standard errors, we can determine whether the small-sample bias of the estimated coefficients is the source of the observed negative association.

The verification of this type of small-sample bias would have important implications for the use of the 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 indicate the need for improvements in the methods of analysis using the Whittemore-Korn model to reduce the small-sample bias.

SPECIFICATION OF THE MONTE CARLO STUDY

In this subsection, we describe the assumed models and data used in the Monte Carlo study. Usually there are two approaches to designing a Monte Carlo study. First, one might use a fairly simple model and generate all data randomly according to the model, then study the behavior of the analysis methods applied to the Monte Carlo data. The advantages of this approach are simplicity and the ease of modifying the model. The disadvantage is that the assumed simple models might have little bearing on the empirical problem of interest.

The alternative approach is to use a model fitted on empirical data, generate new data according to this fitted model as if it were the true model, then analyze the Monte Carlo data and compare it with the fitted model which is actually the true model for the Monte Carlo data. The advantage of this approach is that the "true" model from which the Monte Carlo data were generated is based on fitting empirical data. If the fitting procedure is realistic, this model should be close to the true (but unknown) model from which the empirical data were originally generated.

We chose the second approach for our Monte Carlo study. We took the true model for the Monte Carlo study to be the following:

$$logit(p) = log(p/(1-p))$$

$$= \alpha + 10.658SO_2 + 0.0028TSP - 0.32\ OZONE + 3.14\ NO_2$$

$$+ 0.013\ CO - 0.0034\ minimum\ temperature + 0.88\ precipitation\ ,$$

where p is the probability of a sick episode occurring on a certain day; α is the intercept coefficient to be determined below, SO_2 is the sulfur dioxide level given in ppm, etc. The coefficients are based on a preliminary random-effects model that was fitted to a subset of the Dayton data. (Since we did not find any significant between-individual variation in the Dayton sample, we have used the same set of regression coefficients for all hypothetical individuals.) All aerometric variables are centered to have mean zero. The intercept determines the prevalence of sick episodes. In the different Monte Carlo trials, we used five different values of α: 3/300, 4/300, 5/300, 6/300, and 8/300.

Consider two trials with the same aerometric data and different intercepts. They can be interpreted as two different individuals subject to the same exposure but each having his own propensity to get sick. We give the results below aggregated across different *alphas*.

We take a total Monte Carlo sample of 10,000 hypothetical individuals, each with 300 days of data. We partition the total sample into ten groups, each corresponding to a specific combination of home and work zip codes.[1] Five of the ten groups are workers whose home and work zip codes are given as combinations 2, 3, 4, 8, and 9 in Table 5.1. The other five are non-workers whose daytime exposure is also based on the home zip code, and are given as combinations 1, 5, 6, 7, and 10 in Table 5.1. Each of the ten groups is assigned 1,000 individuals, and each group is further decomposed into five blocks of 200 individuals each. The five blocks correspond to the five different values of α.

Table 5.1

ZIP CODES USED IN MONTE CARLO STUDY

	Zip Code	
Group number	Home	Work
1	45403	NA
2	45403	45414
3	45403	45429
4	45405	45402
5	45407	NA
6	45418	NA
7	45424	NA
8	45424	45402
9	45424	45459
10	45432	NA

[1]The zip code zones cover both urban and suburban locales around the Dayton metropolitan area, and are chosen to be areas where a substantial number of HIE participants reside/work (see Table 2.1).

For each of the ten zip code combinations, we create the aerometric time series, using the same methods that were described in Sec. III, as if we had an HIE participant residing/working at this locale. The range of time used in this time series creation is two years; however, because of missing aerometric data, we only have 370 days available for analysis.

For each of the 10,000 hypothetical individuals, we use the model described above, with the assigned value of α and the aerometric time series corresponding to the individual's zip code combination. We then generate the individual's sick episodes in accordance with the given logistic regression model, treating days as being stochastically independent.[2]

For each hypothetical individual, we apply the same screening rule that we applied to the HIE sample: We delete the individuals with three or fewer sick episodes, and delete the individuals for whom the logistic regression failed to converge; we retain 8267 hypothetical individuals after this restriction. We then apply the same logistic regression and random-effects model analysis as we did for the HIE sample. The results are given below.

RESULTS

Table 5.2 gives the combined results based on all hypothetical individuals. The first column gives the regression coefficients based on the random-effects model applied to all hypothetical individuals; the second column gives the true regression coefficients used in the model from which the Monte Carlo data were generated. The standard errors for the estimated coefficients are given in parenthesis under the coefficients.

Table 5.2

META-ANALYSIS BASED ON THE RANDOM-EFFECTS MODEL SUMMARIES FOR THE AEROMETRIC EFFECTS OVER THE ENTIRE MONTE CARLO SAMPLE: AVERAGE RESPONSES (N = 8267)

Aerometric Attribute	Estimated Coefficient (s.e.)	True Coefficient
SO_2 (ppm)	10.92	10.66
	(0.023)	
TSP ($\mu g/m^3$)	0.0025	0.0028
	(0.000010)	
Ozone (ppm)	−0.82	−0.32
	(0.15)	
NO_2 (ppm)	3.58	3.14
	(0.16)	
CO (ppm)	0.023	0.013
	(0.0024)	
Minimum temperature (F)	−0.0043	−0.0034
	(0.000029)	
Precipitation (in.)	1.39	0.88
	(0.020)	

[2]Strictly speaking, we are generating sick days instead of sick episodes, because we have not eliminated consecutive sick days. However, since we have assumed for this analysis that the days are stochastically independent, and the probability of having consecutive sick days is very small, the difference should be small.

Based on the results in Table 5.2, there appears to be a detectable amount of finite sample bias in the Monte Carlo sample. For all aerometric attributes, the estimated coefficients are somewhat different from the true coefficients, the difference being statistically significant relative to the Monte Carlo standard errors given in the table. For example, the estimated coefficient for SO_2 differs from the true coefficient by 0.26, which is small compared with the true coefficient (10.66), but is more than ten times larger than the standard error.

On the other hand, the magnitudes of the bias are rather small. For all three air pollution variables which were found to have a significant health effect in Dayton, namely, SO_2, TSP, and NO_2, the magnitude of the bias is small or moderate, ranging from about 3 percent to about 10 percent. Apparently, the bias might not affect the qualitative nature of our earlier empirical findings.

One of the largest biases is found for ozone. The true coefficient (−0.32) indicates a small perverse effect for ozone, while the estimated coefficient indicates a much larger perverse effect. Therefore, the small-sample bias could be a possible explanation for the perverse effect of ozone that was found earlier in Seattle.

The discussion above for Table 5.2 is restricted to the overall bias averaged over all individuals. The bias for the individuals in the Monte Carlo study is given in Figs 5.1 through 5.7, in which we have plotted the estimated coefficients against the associated standard errors.

For most of the aerometric attributes, the Figures indicate that there is an association between the estimated coefficients and the corresponding standard errors. This indicates that the association found in the HIE data between coefficients and standard errors might be the result of small-sample bias; i.e., coefficients with larger standard errors are more biased. This is generally consistent with the finding in the HIE data, both in Sec. IV and in Coulson et al. (1985); however, the associations are not all negative, as are the associations found in the HIE data.

In conclusion, the average responses for SO_2, TSP, and NO_2 appear to be free from the small-sample bias, while the response to ozone might be subject to a substantial small-sample bias. The association between the individual-specific coefficients and the associated standard errors might be the result of small-sample bias; that is, the coefficients with larger standard errors might be more biased, although the Monte Carlo results are not all consistent with the results based on the HIE data.

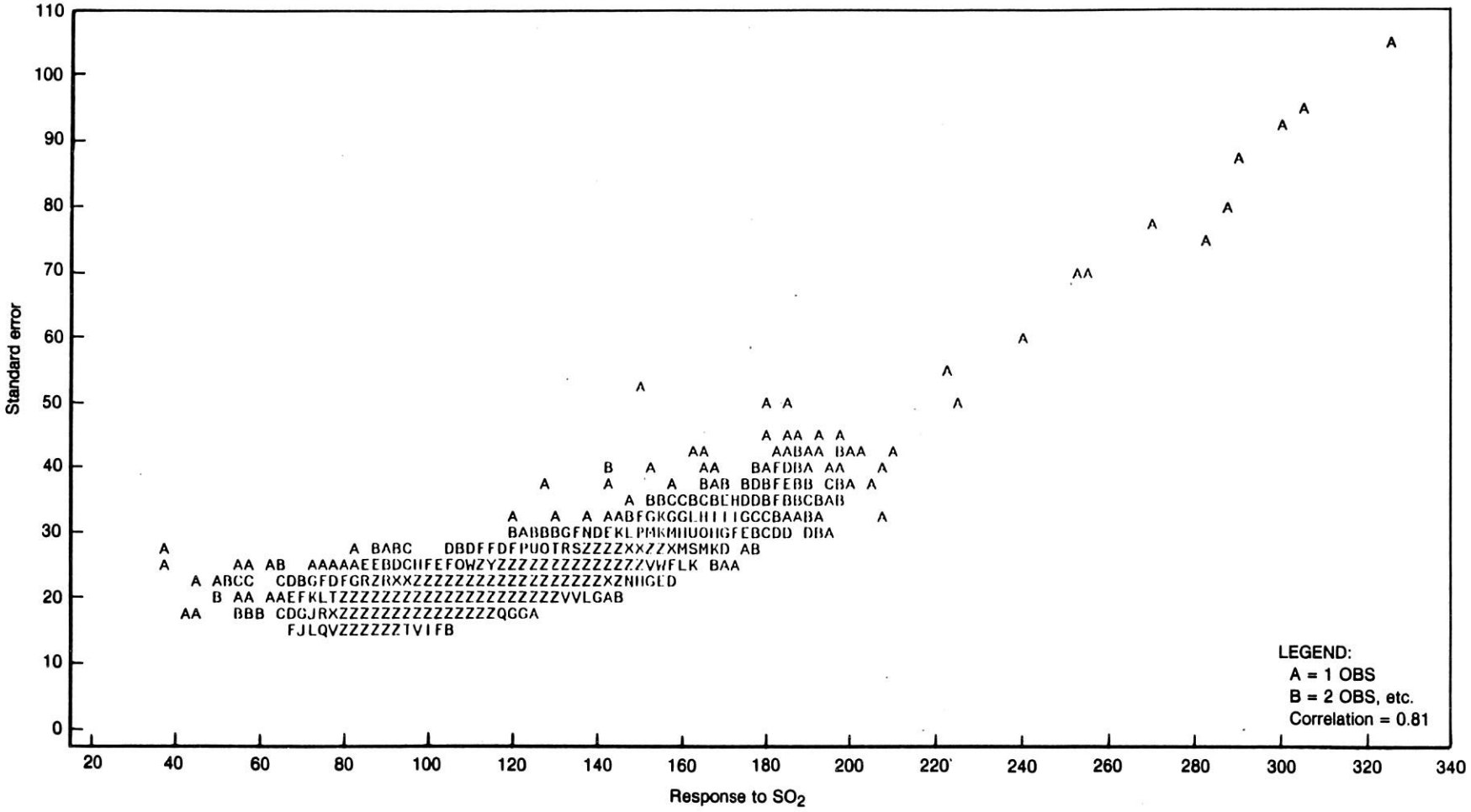

Fig. 5.1—Scatterdiagram of estimated individual responses to SO_2 by the associated standard errors, Monte Carlo Study

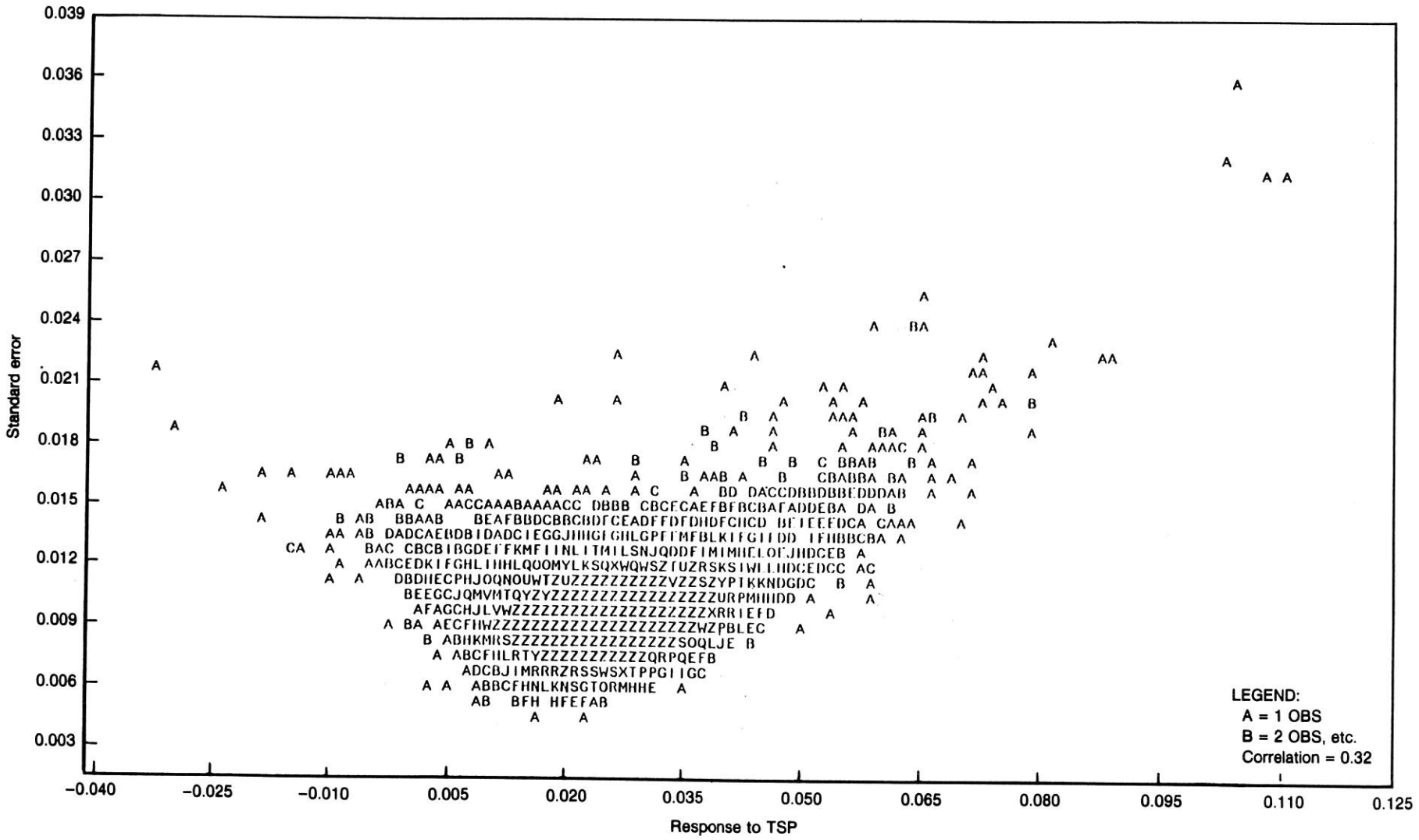

Fig. 5.2—Scatterdiagram of estimated individual responses to TSP by the associated standard errors, Monte Carlo Study

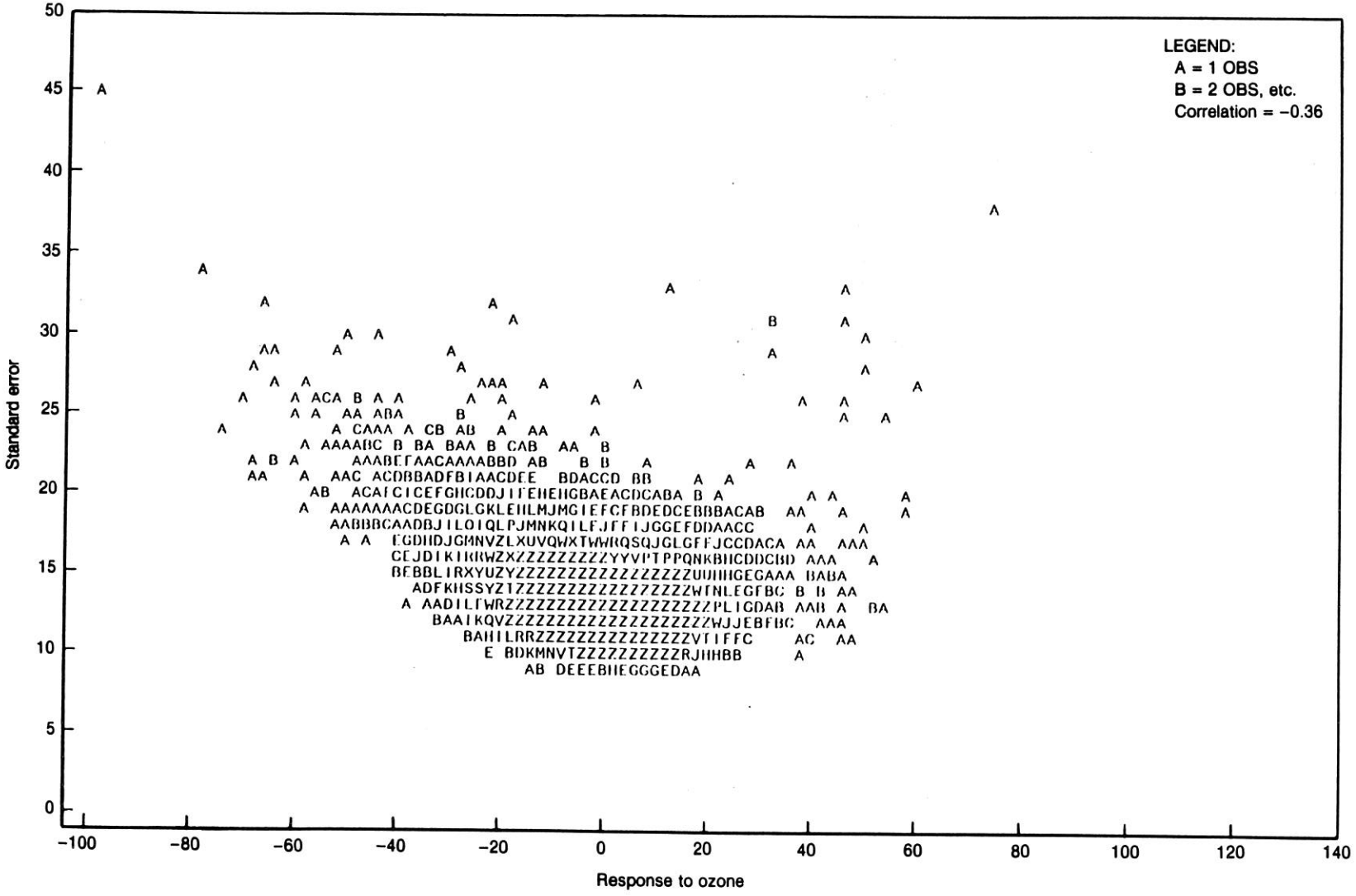

Fig. 5.3—Scatterdiagram of estimated individual responses to ozone by the associated standard errors, Monte Carlo Study

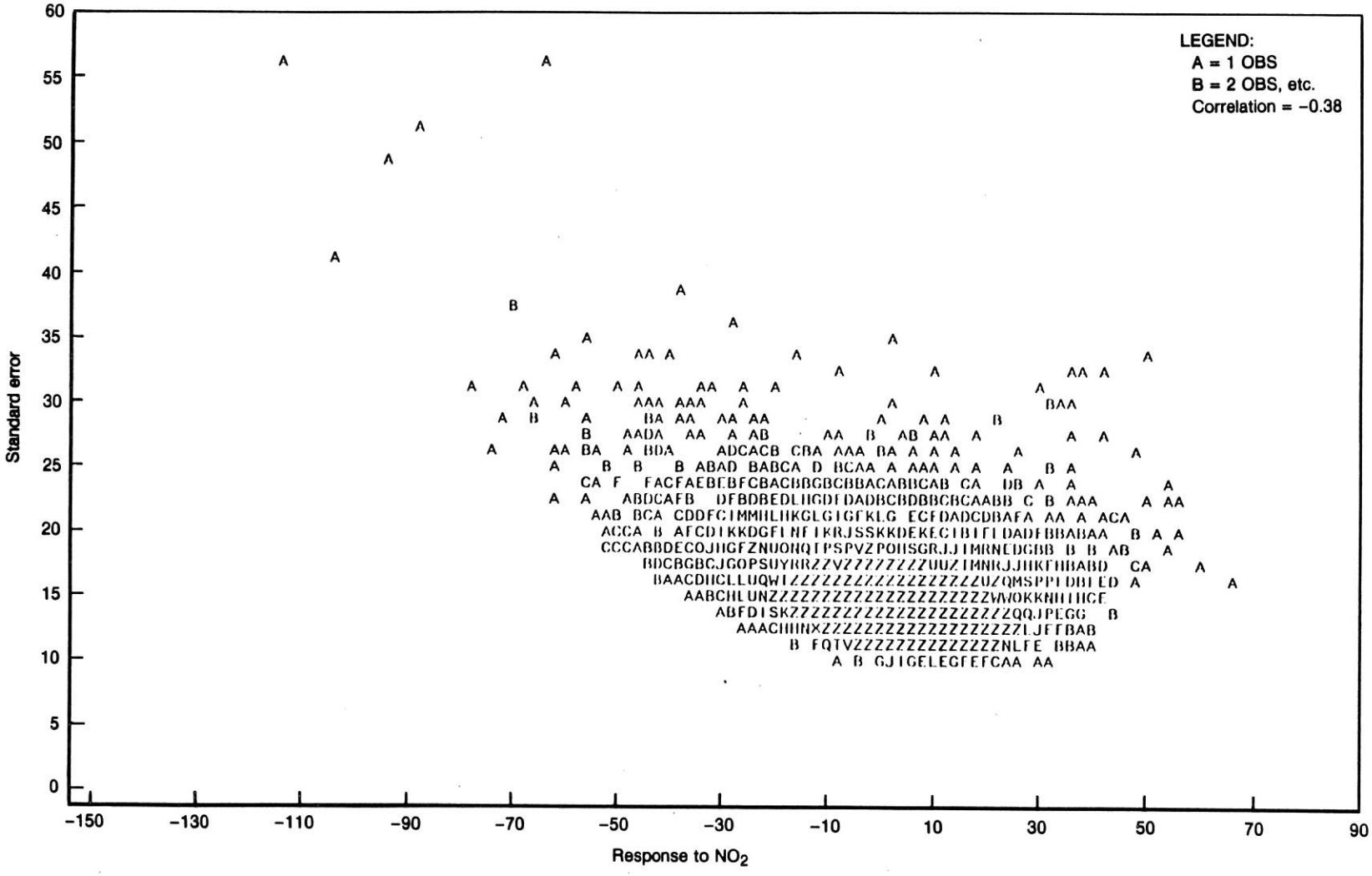

Fig. 5.4—Scatterdiagram of estimated individual responses to NO_2 by the associated standard errors, Monte Carlo Study

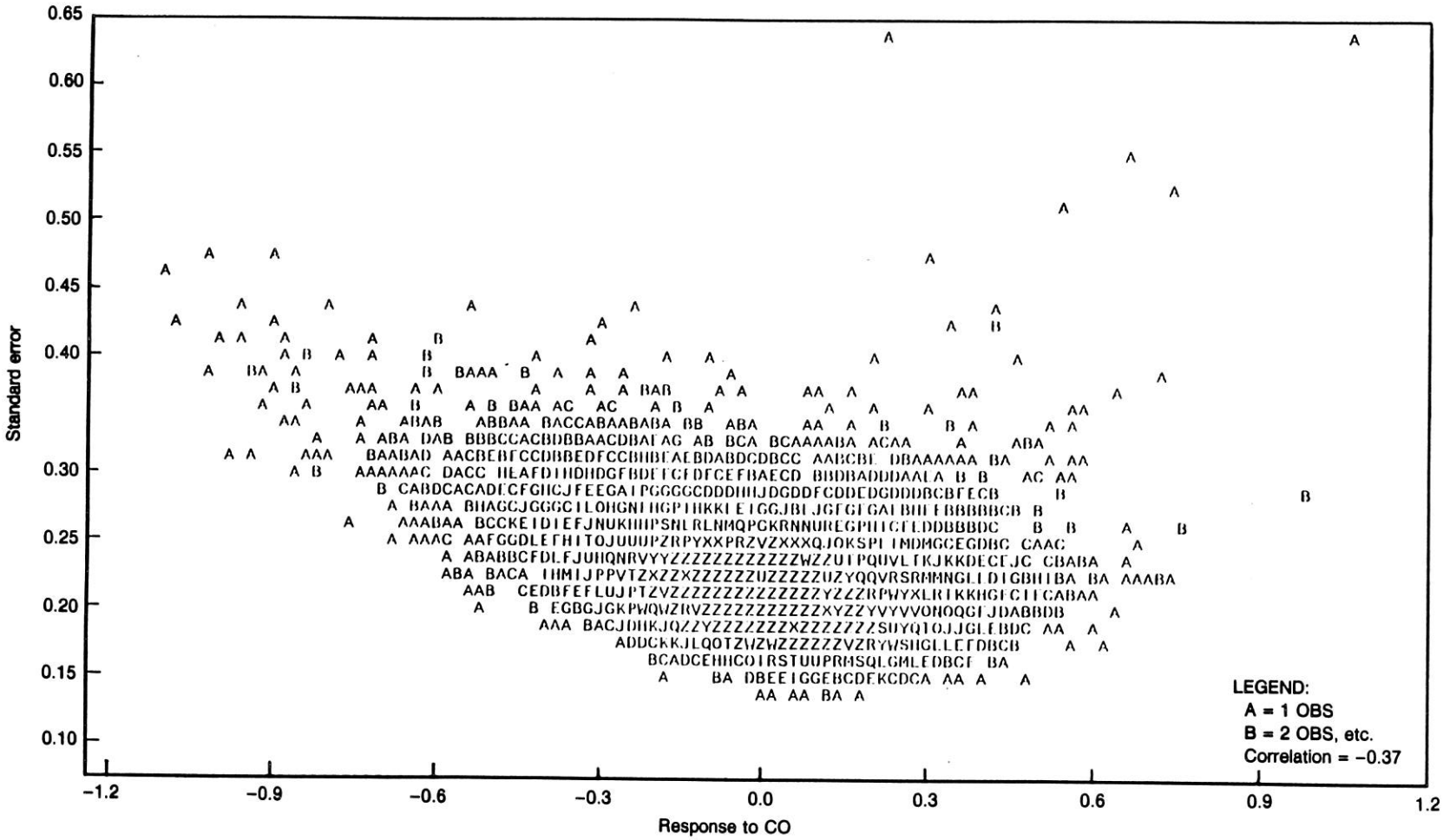

Fig. 5.5—Scatterdiagram of estimated individual responses to CO by the associated standard errors, Monte Carlo Study

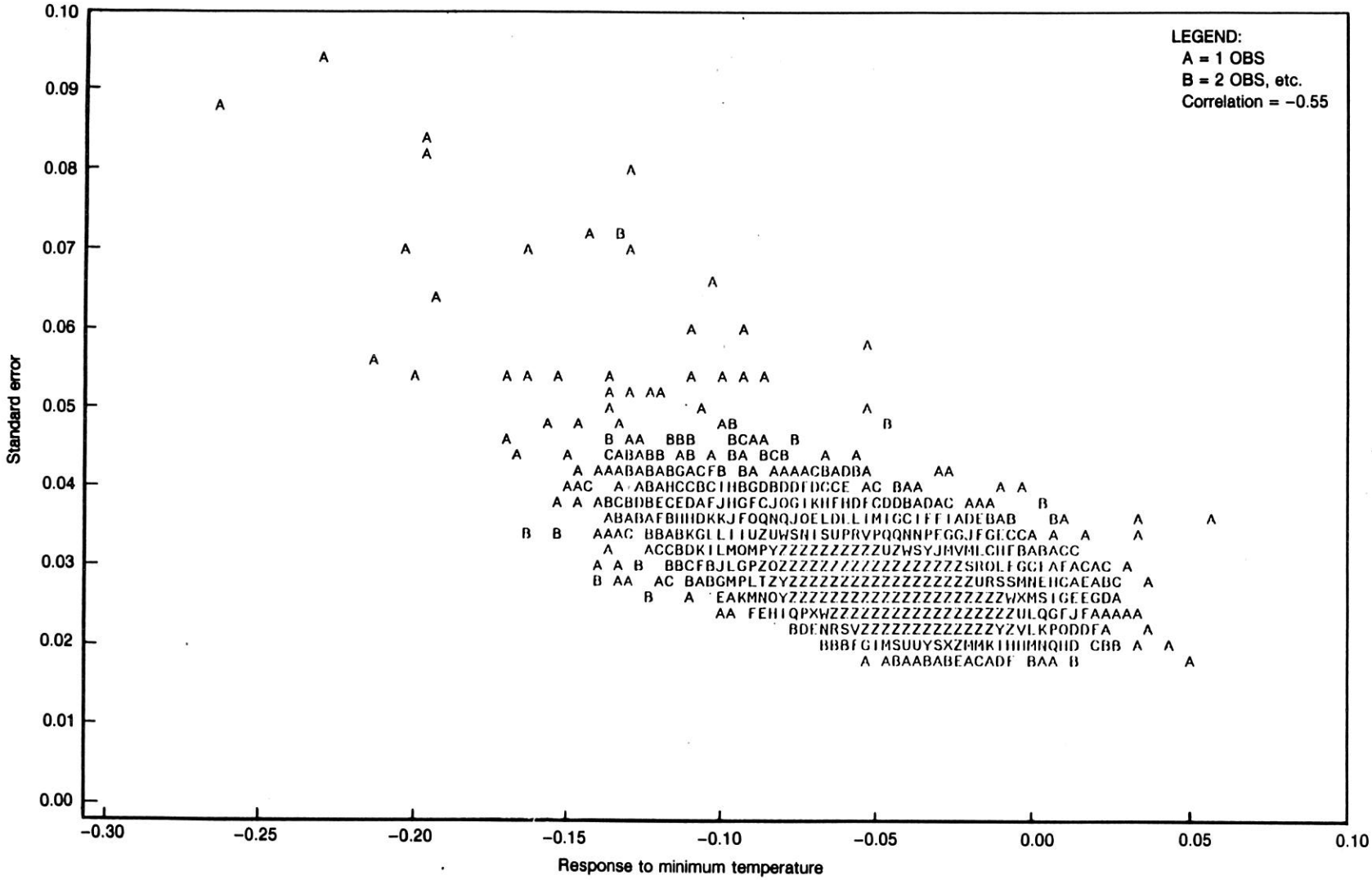

Fig. 5.6—Scatterdiagram of estimated individual responses to minimum temperature by the associated standard errors, Monte Carlo Study

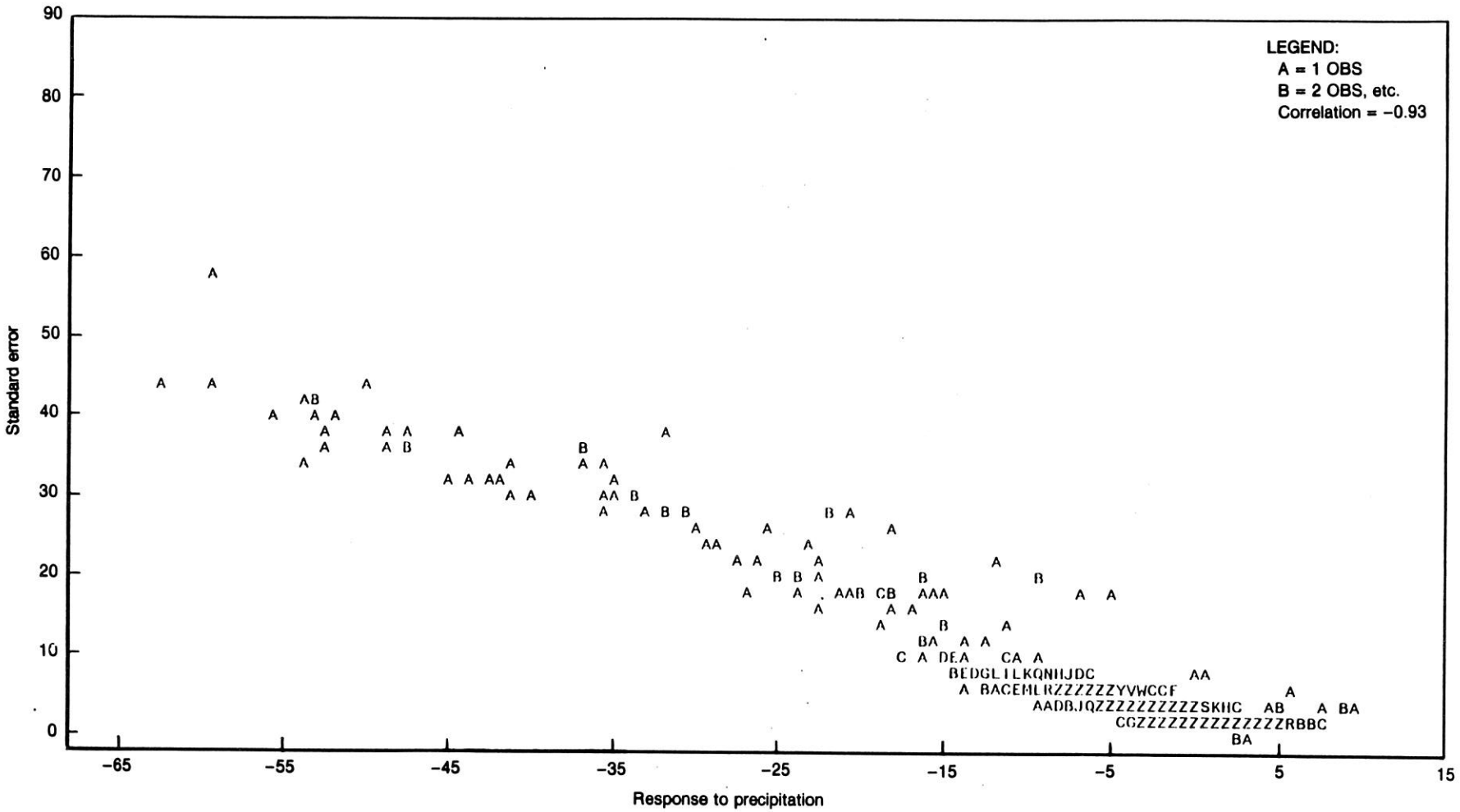

Fig. 5.7—Scatterdiagram of estimated individual responses to precipitation by the associated standard errors, Monte Carlo Study

Appendix A

CORRELATION COEFFICIENTS

Correlation coefficients were obtained for the aerometric variables for the individual day-to-day (Whittemore-Korn) analysis. These are presented in Tables A.1 and A.2 for, respectively, two of the zip code zones in the Dayton metropolitan area, 45403 and 45432. The former is near the central part of the city, the latter is in the suburb of Beavercreek. For each pair of explanatory variables, the upper entry for the item is the correlation coefficient, and the lower entry is the probability that the correlation coefficient is zero. Table A.3 gives the multiple correlation coefficient for each explanatory variable with the rest of the explanatory variables. Table A.4 gives the frequency of individuals by home zip code locations. Table A.5 gives the location of the monitoring stations. Figures A.1 through A.7 plot the monthly averages against time for the aerometric variables. Figures A.8 through A.14 plot the daily fraction of the total population[1] with any time lost as a result of illness (e.g., school or work loss) against pollution levels, temperature, and precipitation. A dot in the plot indicates one or more daily observations at that value. The curves traced by the # signs are the moving averages for the plots. For each aerometric variable, we ranked the observations by the aerometric variable, and then averaged both the sick-loss rate and the aerometric variable over groups of size 40 (or less). These averages are denoted by the # signs.

Table A.1

CORRELATION COEFFICIENTS FOR AEROMETRIC VARIABLES, CENTRAL DAYTON

	TSPAV	MINTEMP	PRECIP	SO_2AV	COAV	OZOMX	NO_2MX
TSPAV	1.00000	0.27196	−0.02628	0.10543	0.05862	0.37112	0.15634
	0.0000	0.0001	0.4781	0.0290	0.1256	0.0001	0.0001
MINTEMP	0.27196	1.00000	0.11009	0.01192	−0.09321	0.57364	0.29293
	0.0001	0.0000	0.0029	0.8056	0.0147	0.0001	0.0001
PRECIP	−0.02628	0.11009	1.00000	0.00158	−0.13281	−0.10080	0.07569
	0.4781	0.0029	0.0000	0.9740	0.3915	0.0064	0.0500
SO_2AV	0.10543	0.01192	0.00158	1.00000	0.16928	0.02253	0.15884
	0.0290	0.8056	0.9740	0.0000	0.0006	0.6417	0.0017
COAV	0.05862	−0.09321	−0.03281	0.16928	1.00000	−0.16621	0.13428
	0.1256	0.0147	0.3915	0.0006	0.0000	0.0001	0.0008
OZOMX	0.37112	0.57364	−0.10080	0.02253	−0.16621	1.00000	0.34993
	0.0001	0.0001	0.0064	0.6417	0.0001	0.0000	0.0001
NO_2MX	0.15634	0.29293	0.07569	0.15884	0.13428	0.34993	1.00000
	0.0001	0.0001	0.0500	0.0017	0.0008	0.0001	0.0000

AV = Average
MX = Maximum

[1]This includes all individuals with diary data. Some of these individuals are not in the individual time series file.

Appendix A

CORRELATION COEFFICIENTS

Correlation coefficients were obtained for the aerometric variables for the individual day-to-day (Whittemore-Korn) analysis. These are presented in Tables A.1 and A.2 for, respectively, two of the zip code zones in the Dayton metropolitan area, 45403 and 45432. The former is near the central part of the city, the latter is in the suburb of Beavercreek. For each pair of explanatory variables, the upper entry for the item is the correlation coefficient, and the lower entry is the probability that the correlation coefficient is zero. Table A.3 gives the multiple correlation coefficient for each explanatory variable with the rest of the explanatory variables. Table A.4 gives the frequency of individuals by home zip code locations. Table A.5 gives the location of the monitoring stations. Figures A.1 through A.7 plot the monthly averages against time for the aerometric variables. Figures A.8 through A.14 plot the daily fraction of the total population[1] with any time lost as a result of illness (e.g., school or work loss) against pollution levels, temperature, and precipitation. A dot in the plot indicates one or more daily observations at that value. The curves traced by the # signs are the moving averages for the plots. For each aerometric variable, we ranked the observations by the aerometric variable, and then averaged both the sick-loss rate and the aerometric variable over groups of size 40 (or less). These averages are denoted by the # signs.

Table A.1

CORRELATION COEFFICIENTS FOR AEROMETRIC VARIABLES, CENTRAL DAYTON

	TSPAV	MINTEMP	PRECIP	SO_2AV	COAV	OZOMX	NO_2MX
TSPAV	1.00000	0.27196	−0.02628	0.10543	0.05862	0.37112	0.15634
	0.0000	0.0001	0.4781	0.0290	0.1256	0.0001	0.0001
MINTEMP	0.27196	1.00000	0.11009	0.01192	−0.09321	0.57364	0.29293
	0.0001	0.0000	0.0029	0.8056	0.0147	0.0001	0.0001
PRECIP	−0.02628	0.11009	1.00000	0.00158	−0.13281	−0.10080	0.07569
	0.4781	0.0029	0.0000	0.9740	0.3915	0.0064	0.0500
SO_2AV	0.10543	0.01192	0.00158	1.00000	0.16928	0.02253	0.15884
	0.0290	0.8056	0.9740	0.0000	0.0006	0.6417	0.0017
COAV	0.05862	−0.09321	−0.03281	0.16928	1.00000	−0.16621	0.13428
	0.1256	0.0147	0.3915	0.0006	0.0000	0.0001	0.0008
OZOMX	0.37112	0.57364	−0.10080	0.02253	−0.16621	1.00000	0.34993
	0.0001	0.0001	0.0064	0.6417	0.0001	0.0000	0.0001
NO_2MX	0.15634	0.29293	0.07569	0.15884	0.13428	0.34993	1.00000
	0.0001	0.0001	0.0500	0.0017	0.0008	0.0001	0.0000

AV = Average
MX = Maximum

[1]This includes all individuals with diary data. Some of these individuals are not in the individual time series file.

Table A.2

CORRELATION COEFFICIENTS FOR AEROMETRIC VARIABLES, BEAVERCREEK

	TSPAV	MINTEMP	PRECIP	SO_2AV	COAV	OZOMX	NO_2MX
TSPAV	1.00000	0.32030	−0.00194	−0.00818	−0.00266	0.32207	0.12476
	0.0000	0.0001	0.9582	0.8658	0.9447	0.0001	0.0012
MINTEMP	0.32030	1.00000	0.11009	0.01192	−0.09321	0.57364	0.29293
	0.0001	0.0000	0.0029	0.8056	0.0147	0.0001	0.0001
PRECIP	−0.00194	0.11009	1.00000	0.00158	−0.03281	−0.10080	0.07569
	0.9582	0.0029	0.0000	0.9740	0.3915	0.0064	0.0500
SO_2AV	0.00818	0.01192	0.00158	1.00000	0.16928	0.02253	0.15884
	0.8658	0.8056	0.9740	0.0000	0.0006	0.6417	0.0017
COAV	−0.00266	−0.09321	−0.03281	0.16928	1.00000	−0.16621	0.13428
	0.9447	0.0147	0.3915	0.0006	0.0000	0.0001	0.0008
OZOMX	0.32207	0.57364	−0.10080	0.02253	−0.16621	1.00000	0.34993
	0.0001	0.0001	0.0064	0.6417	0.0001	0.0000	0.0001
NO_2MX	0.12476	0.29293	0.07569	0.15884	0.13428	0.34993	1.00000
	0.0012	0.0001	0.0500	0.0017	0.0008	0.0001	0.0000

AV = Average
MX = Maximum

Table A.3

MULTIPLE CORRELATION COEFFICIENTS FOR THE AEROMETRIC DATA

Attribute	Central Dayton	Beavercreek
SO_2	0.27	0.25
TSP	0.38	0.30
Ozone	0.79	0.79
NO_2	0.52	0.53
CO	0.24	0.28
Precipitation	0.29	0.29
Minimum temperature	0.77	0.76

Table A.2

CORRELATION COEFFICIENTS FOR AEROMETRIC VARIABLES, BEAVERCREEK

	TSPAV	MINTEMP	PRECIP	SO_2AV	COAV	OZOMX	NO_2MX
TSPAV	1.00000	0.32030	−0.00194	−0.00818	−0.00266	0.32207	0.12476
	0.0000	0.0001	0.9582	0.8658	0.9447	0.0001	0.0012
MINTEMP	0.32030	1.00000	0.11009	0.01192	−0.09321	0.57364	0.29293
	0.0001	0.0000	0.0029	0.8056	0.0147	0.0001	0.0001
PRECIP	−0.00194	0.11009	1.00000	0.00158	−0.03281	−0.10080	0.07569
	0.9582	0.0029	0.0000	0.9740	0.3915	0.0064	0.0500
SO_2AV	0.00818	0.01192	0.00158	1.00000	0.16928	0.02253	0.15884
	0.8658	0.8056	0.9740	0.0000	0.0006	0.6417	0.0017
COAV	−0.00266	−0.09321	−0.03281	0.16928	1.00000	−0.16621	0.13428
	0.9447	0.0147	0.3915	0.0006	0.0000	0.0001	0.0008
OZOMX	0.32207	0.57364	−0.10080	0.02253	−0.16621	1.00000	0.34993
	0.0001	0.0001	0.0064	0.6417	0.0001	0.0000	0.0001
NO_2MX	0.12476	0.29293	0.07569	0.15884	0.13428	0.34993	1.00000
	0.0012	0.0001	0.0500	0.0017	0.0008	0.0001	0.0000

AV = Average
MX = Maximum

Table A.3

MULTIPLE CORRELATION COEFFICIENTS FOR THE AEROMETRIC DATA

Attribute	Central Dayton	Beavercreek
SO_2	0.27	0.25
TSP	0.38	0.30
Ozone	0.79	0.79
NO_2	0.52	0.53
CO	0.24	0.28
Precipitation	0.29	0.29
Minimum temperature	0.77	0.76

Table A.4

DAYTON RESIDENCE ZIP CODES AND COORDINATES
(in degrees)

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

NOTE: Residence at start of the HIE.

Table A.5

DAYTON MONITORING STATIONS AND COORDINATES
(in degrees)

Station	Latitude	Longitude	CO	COH	NO_2	Ozone	SO_2	TSP
0800001G01	39.83	84.42						•
1100001G01	39.63	84.17						•
1260001G01	40.00	83.80				•		•
1660002G01	39.77	84.21	•					
1660003G01	39.76	84.19	•					
1660014G01	39.76	84.19						•
1660015G01	39.77	84.18						•
1660017G01	39.75	84.24						•
1660019G01	39.81	84.19		•	•	•	•	•
1660021G01	39.75	84.13	•					
1660022G01	39.70	84.31						•
1660025G01	39.76	84.20	•	•		•	•	
1660026G01	39.75	84.19	•					
1940001G01	39.74	84.63						•
2040001G01	39.79	84.03						•
2040003G01	39.83	84.00						•
2440002G01	39.63	84.37						•
2640001G01	40.10	84.63						•
2640002G01	40.10	84.61						•
2985001G01	39.87	84.14						•
3240002G01	39.70	84.14				•		
3240003G01	39.73	84.19						•
4280002G01	39.65	84.28						•
4500001G01	39.79	84.13				•		•
4500002G01	39.80	84.35		•			•	
4500003G01	39.85	84.33						•
4500004G01	39.79	84.13						•
4500005G05	39.64	84.22					•	•
4550001G01	39.71	84.21	•					
4760001G01	39.94	84.02						•
4790001G01	39.74	84.39						•
5100001G01	39.72	84.18						•
5520002G01	40.14	84.23						•
5520003G01	40.14	84.24		•		•	•	
5520004G01	40.14	84.21						•
5640001G01	39.84	84.72						•
6380001G01	39.93	83.81				•		
6380002G01	39.95	83.76						•
6380003G01	39.91	83.77						•
6380004G01	39.92	83.81						•
6580001G01	39.96	84.17		•		•	•	
6660001G01	39.80	84.30						•
6680001G01	40.04	84.20						•
6880001G01	39.90	84.21						•
6880003G01	39.89	84.20						•
7300001G01	39.96	84.33						•
7670001G01	39.81	84.03						•
7720001G01	39.70	83.93						•
7720002G01	39.71	83.93						•
7740001G01	39.80	83.89						•

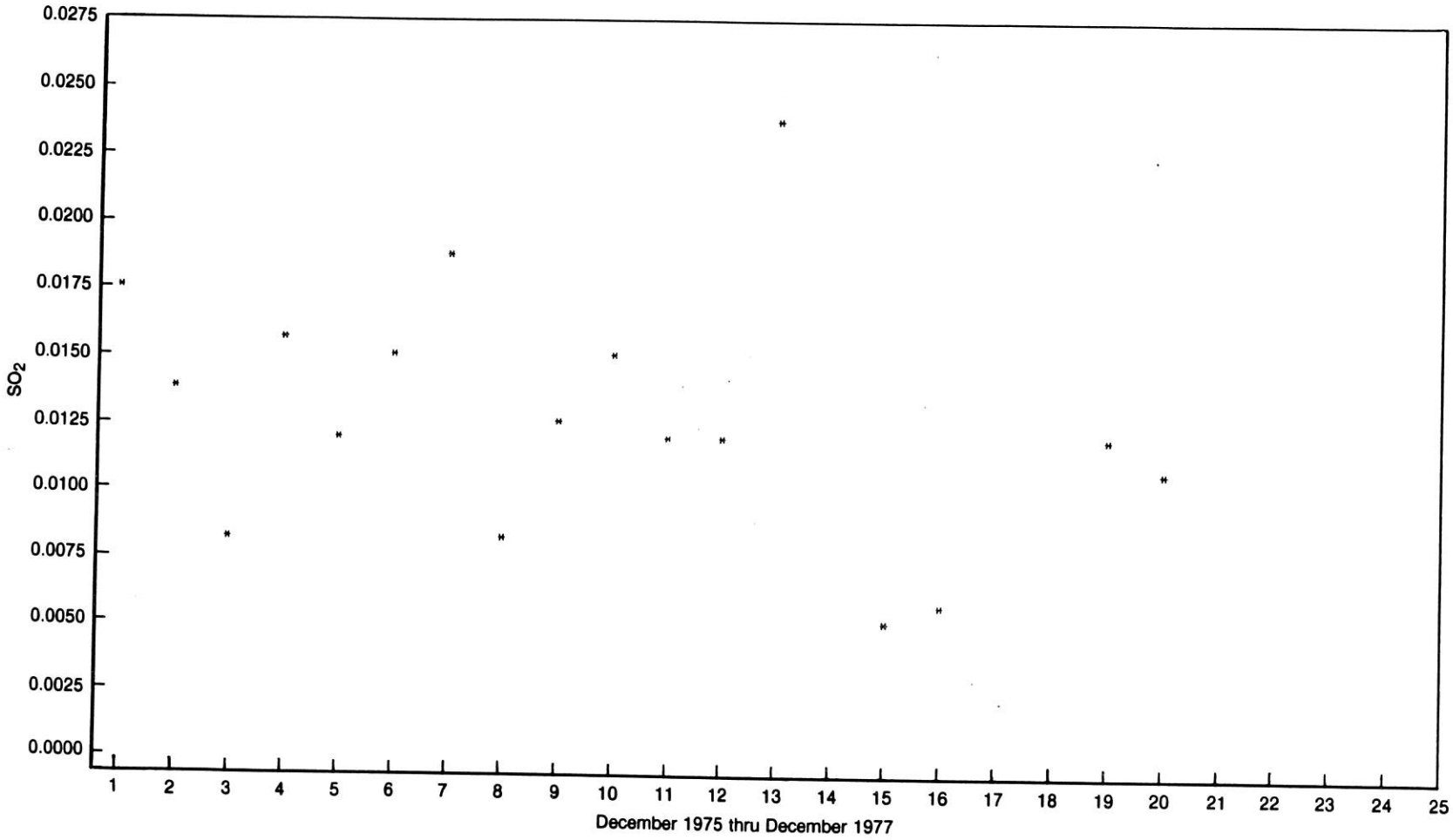

Fig. A.1—Monthly average SO_2 plotted against time, central Dayton

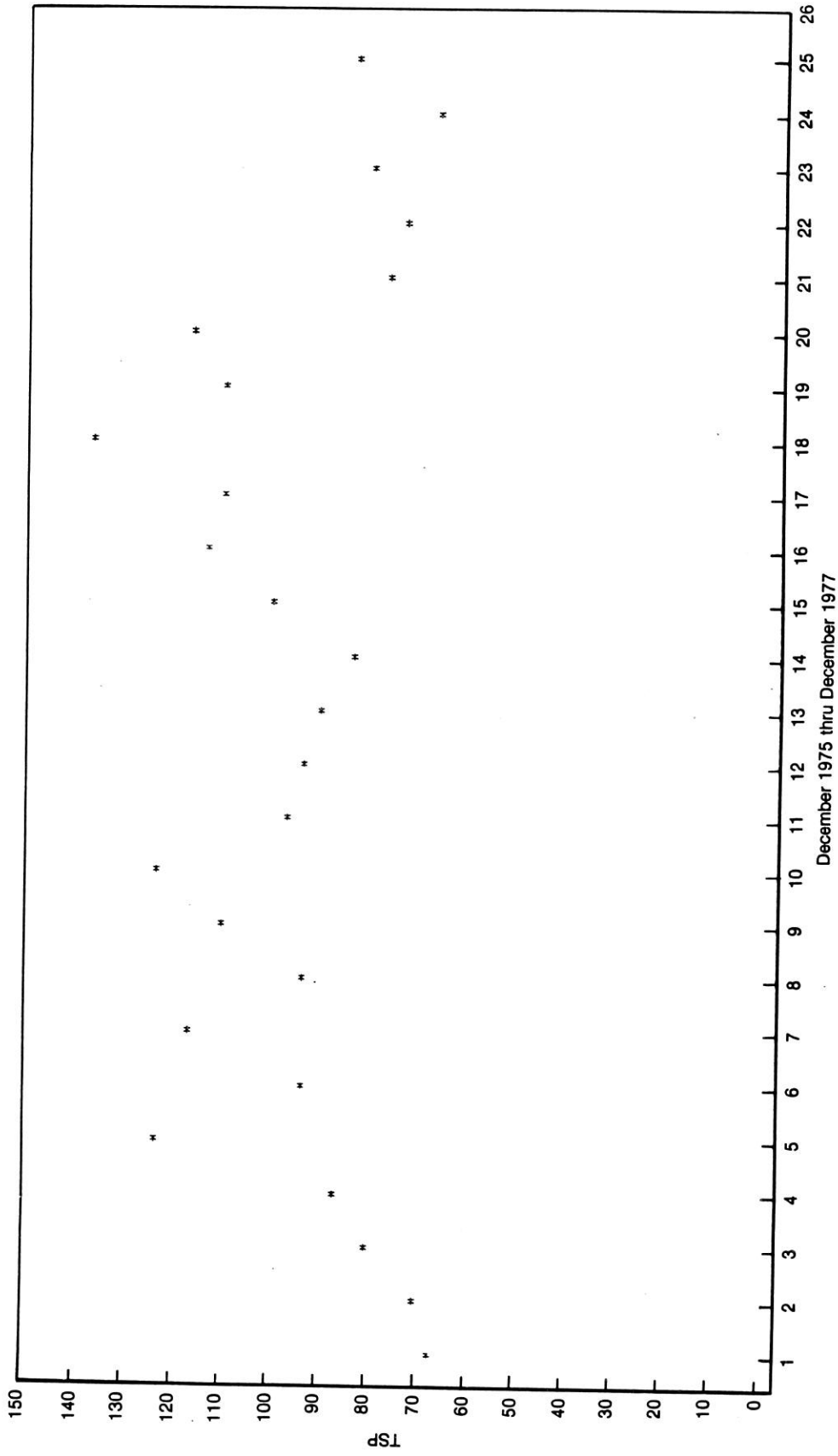

Fig. A.2—Monthly average TSP plotted against time, central Dayton

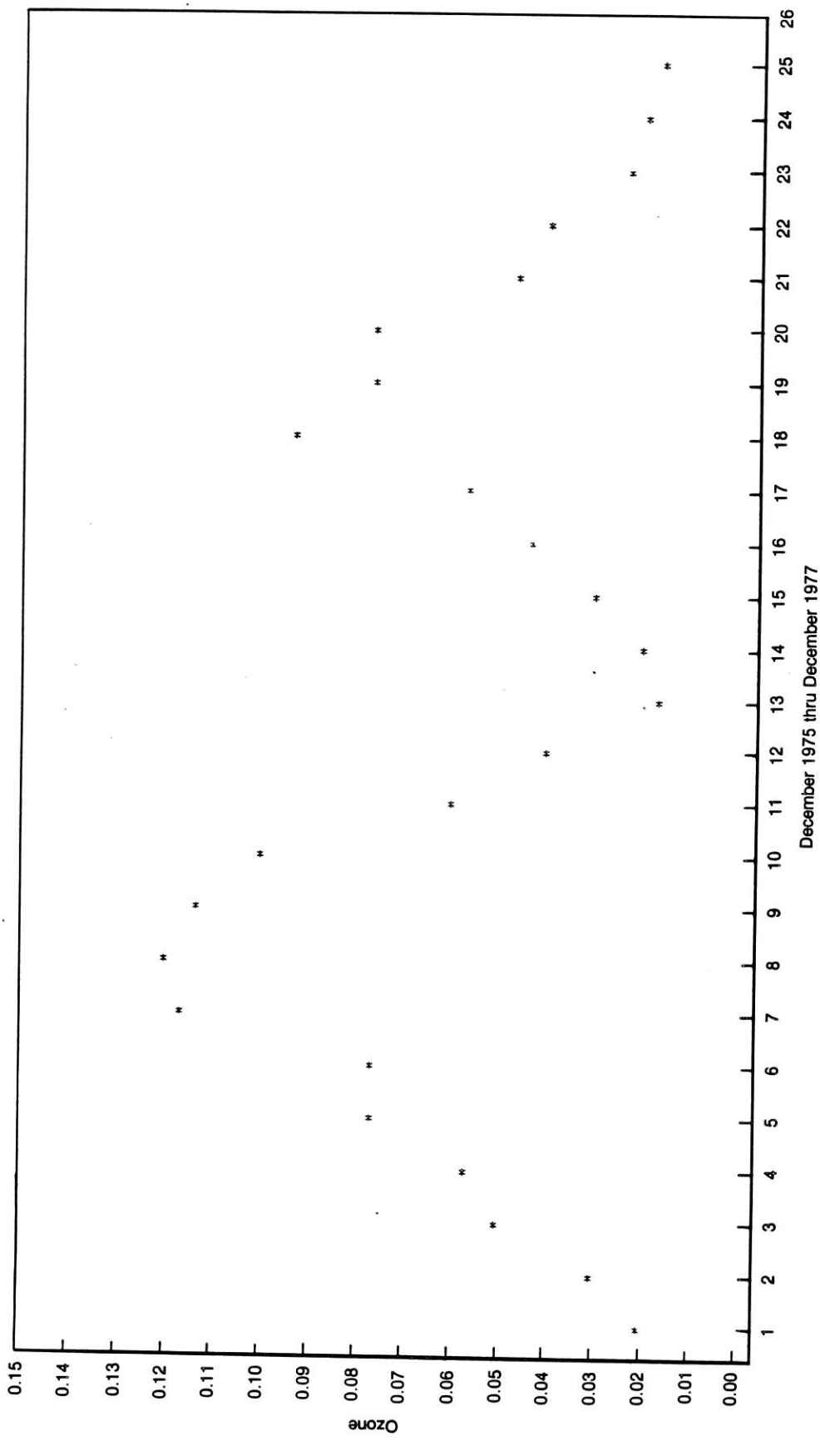

Fig. A.3—Monthly average ozone plotted against time, central Dayton

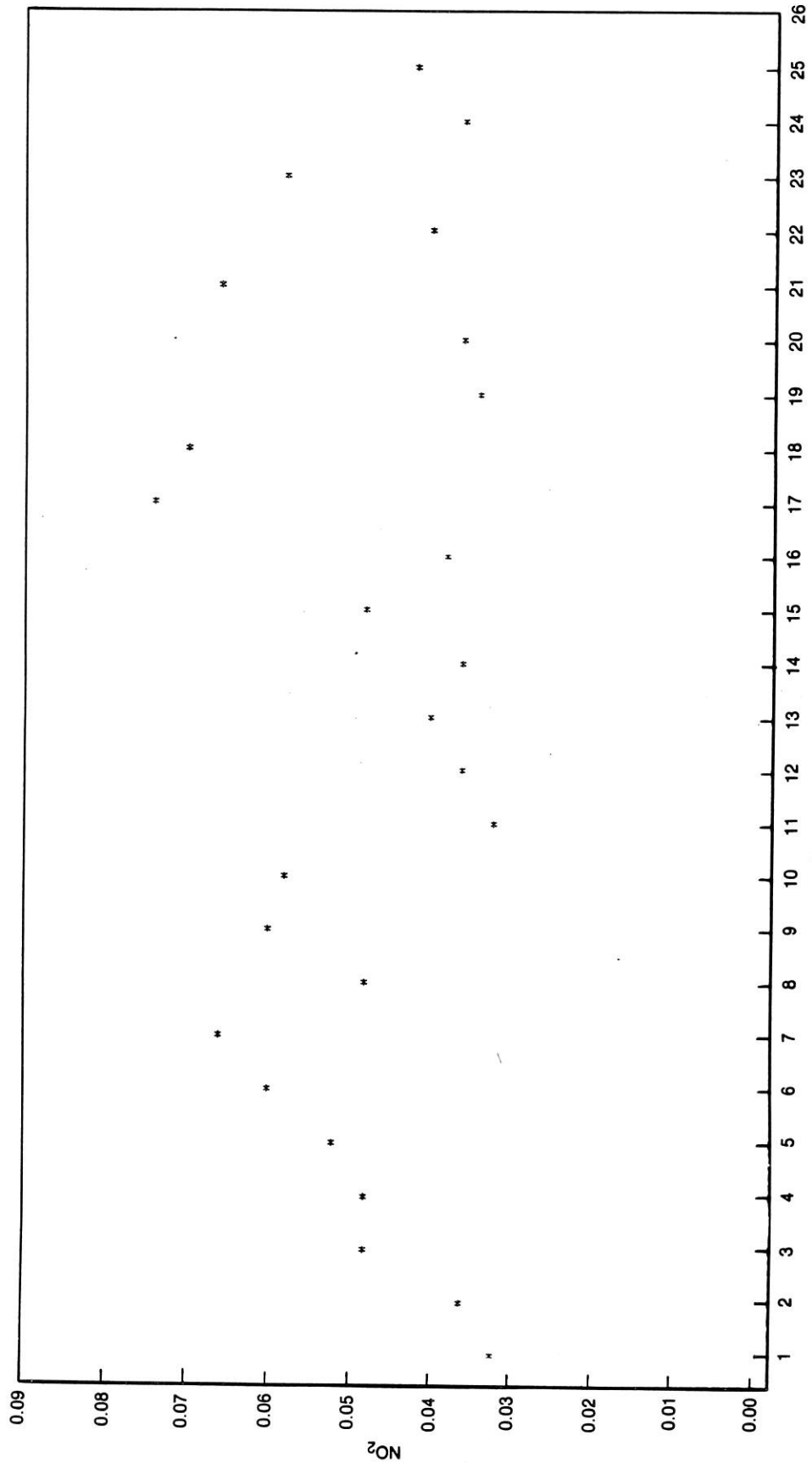

Fig. A.4—Monthly average NO_2 plotted against time, central Dayton

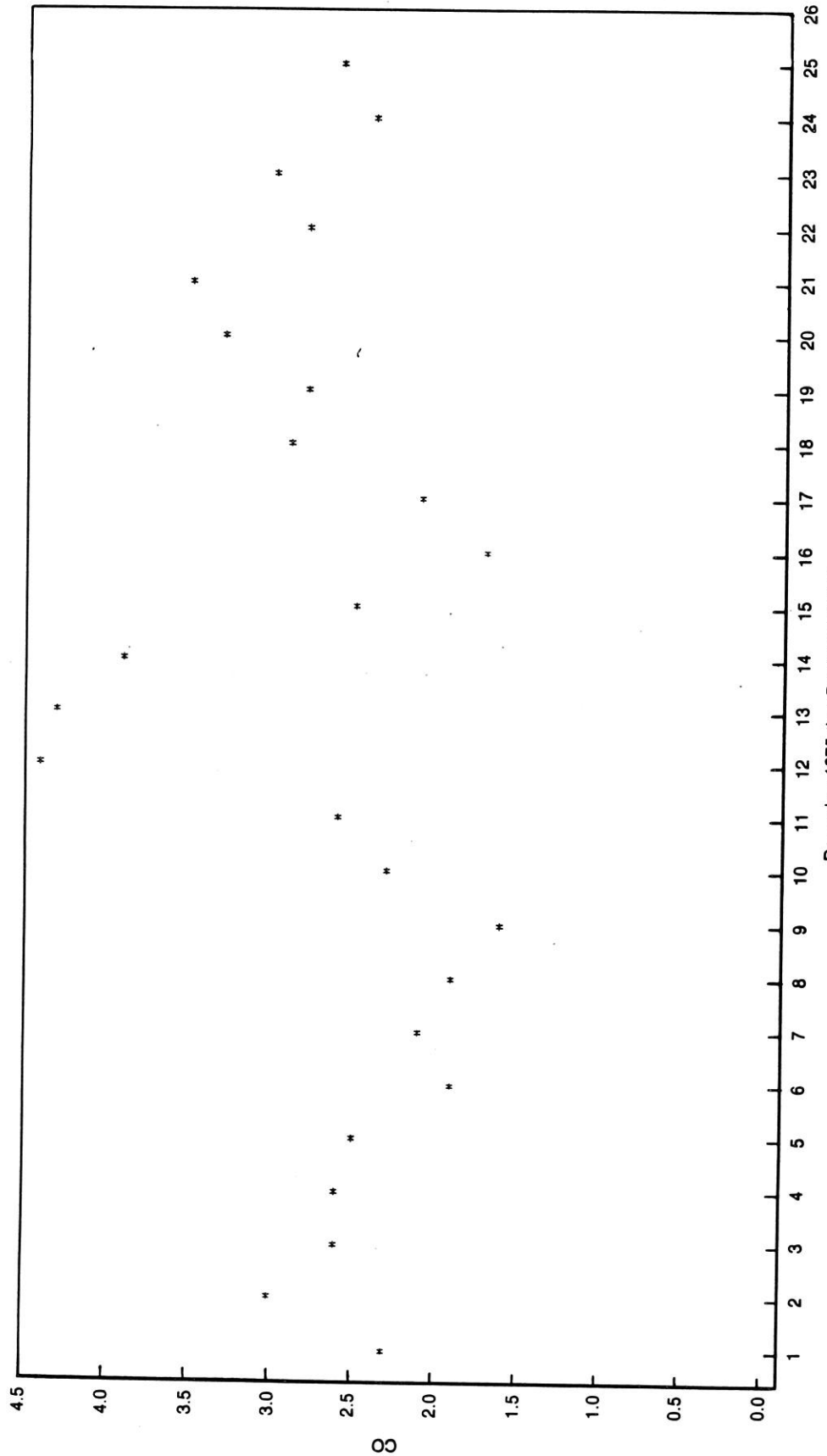

Fig. A.5—Monthly average CO plotted against time, central Dayton

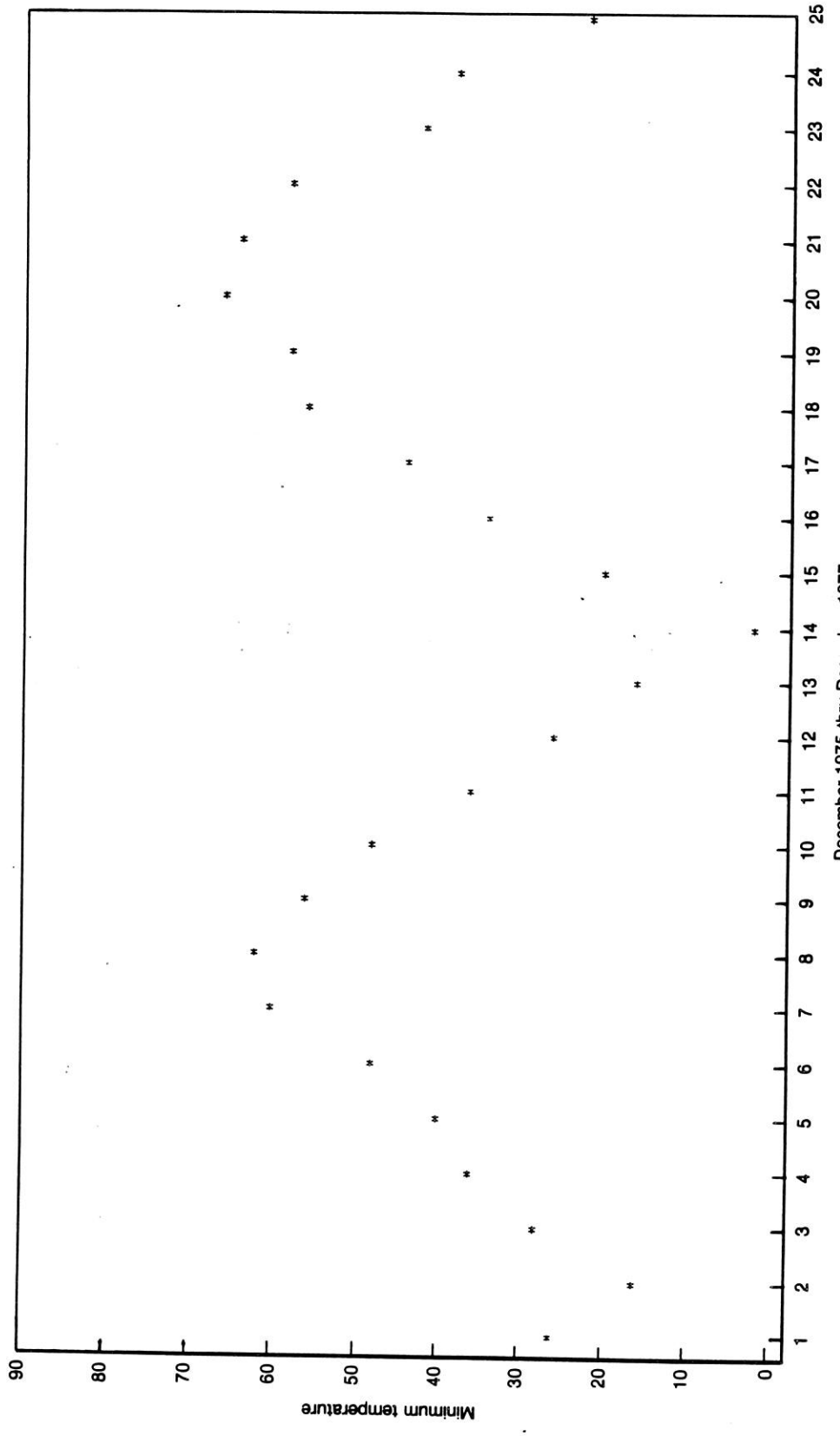

Fig. A.6—Monthly average minimum temperature plotted against time, central Dayton

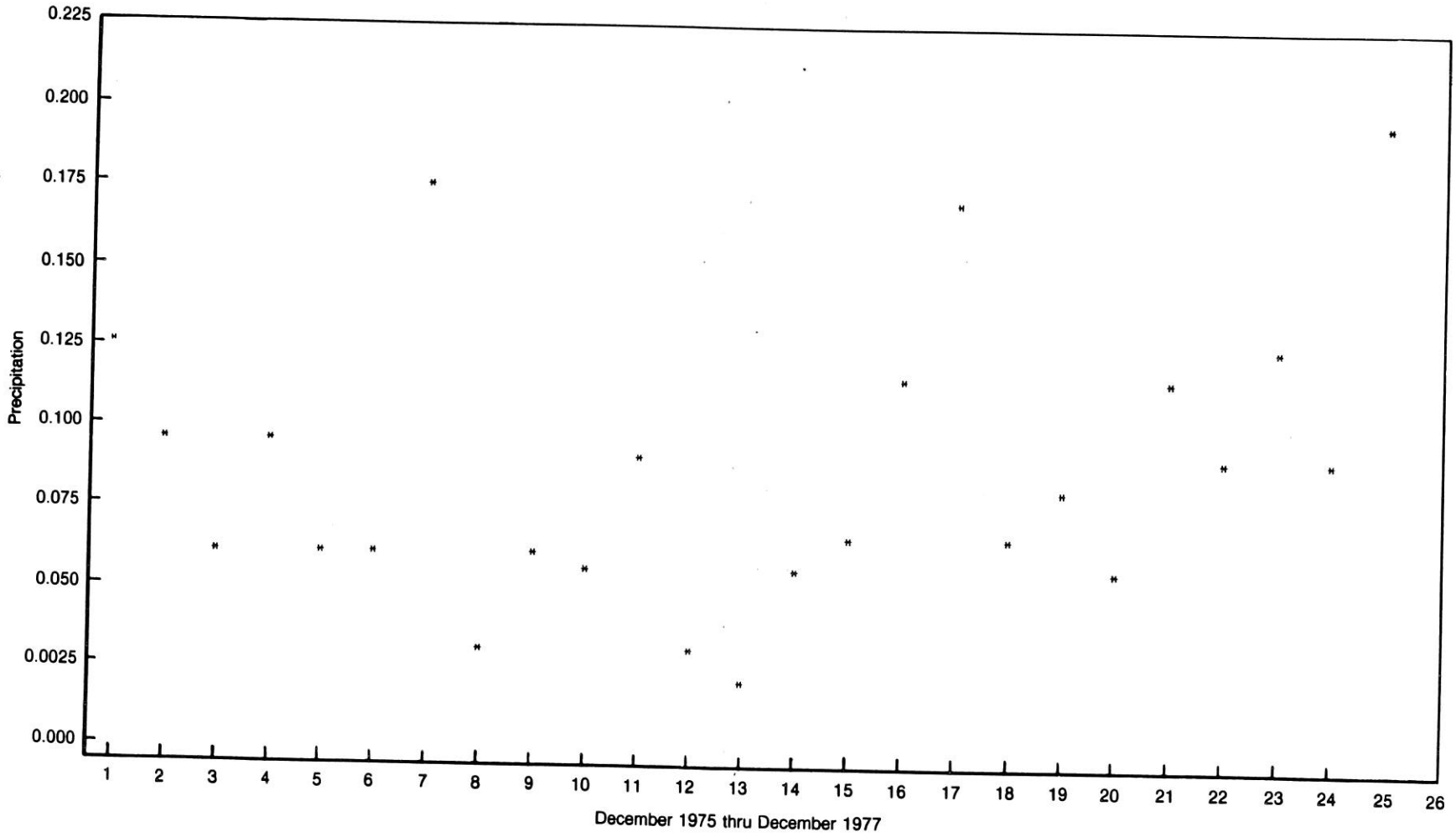

Fig. A.7—Monthly average preciptitation plotted against time, central Dayton

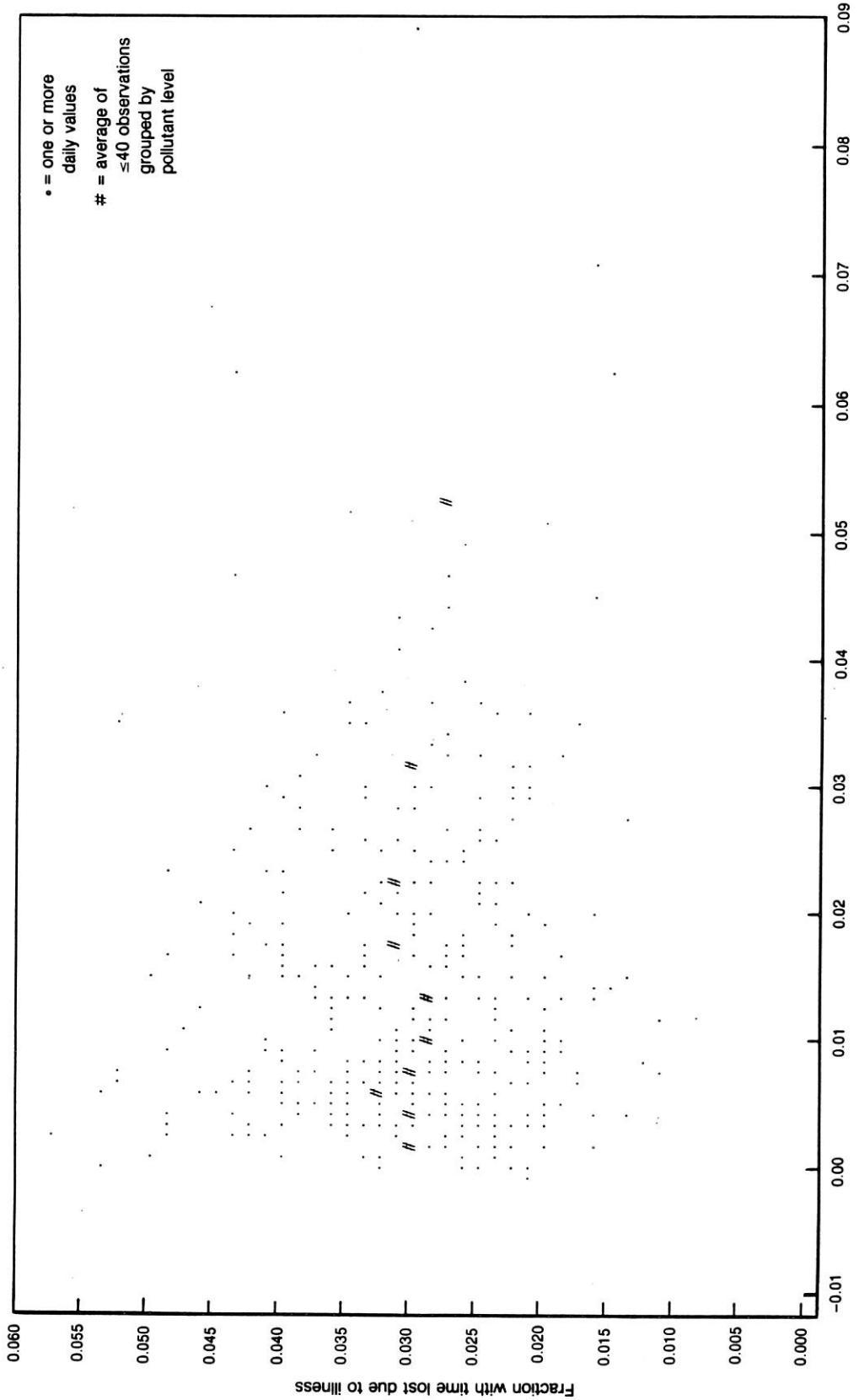

Fig. A.8—Dayton time lost due to illness: SO_2

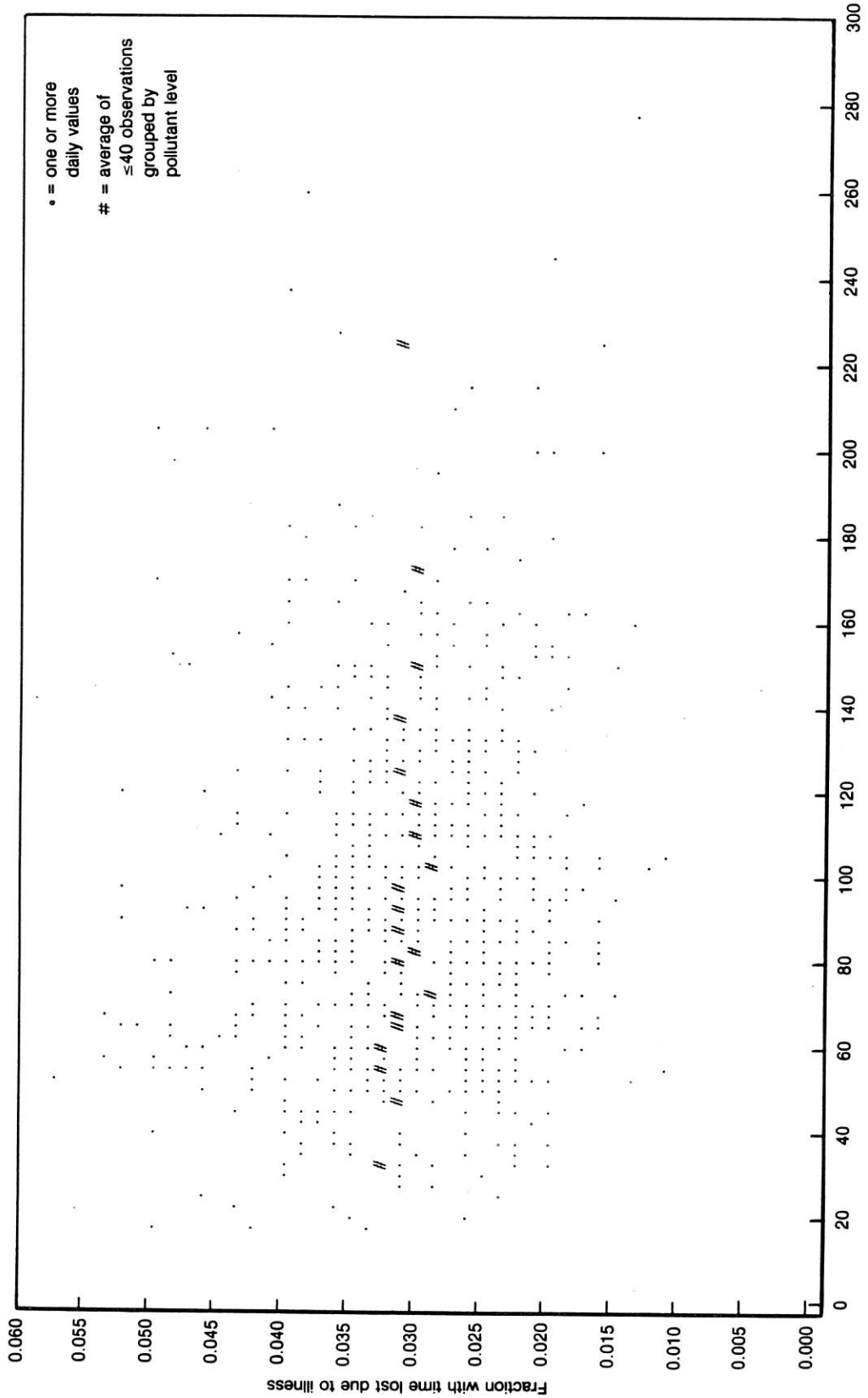

Fig. A.9—Dayton time lost due to illness: TSP

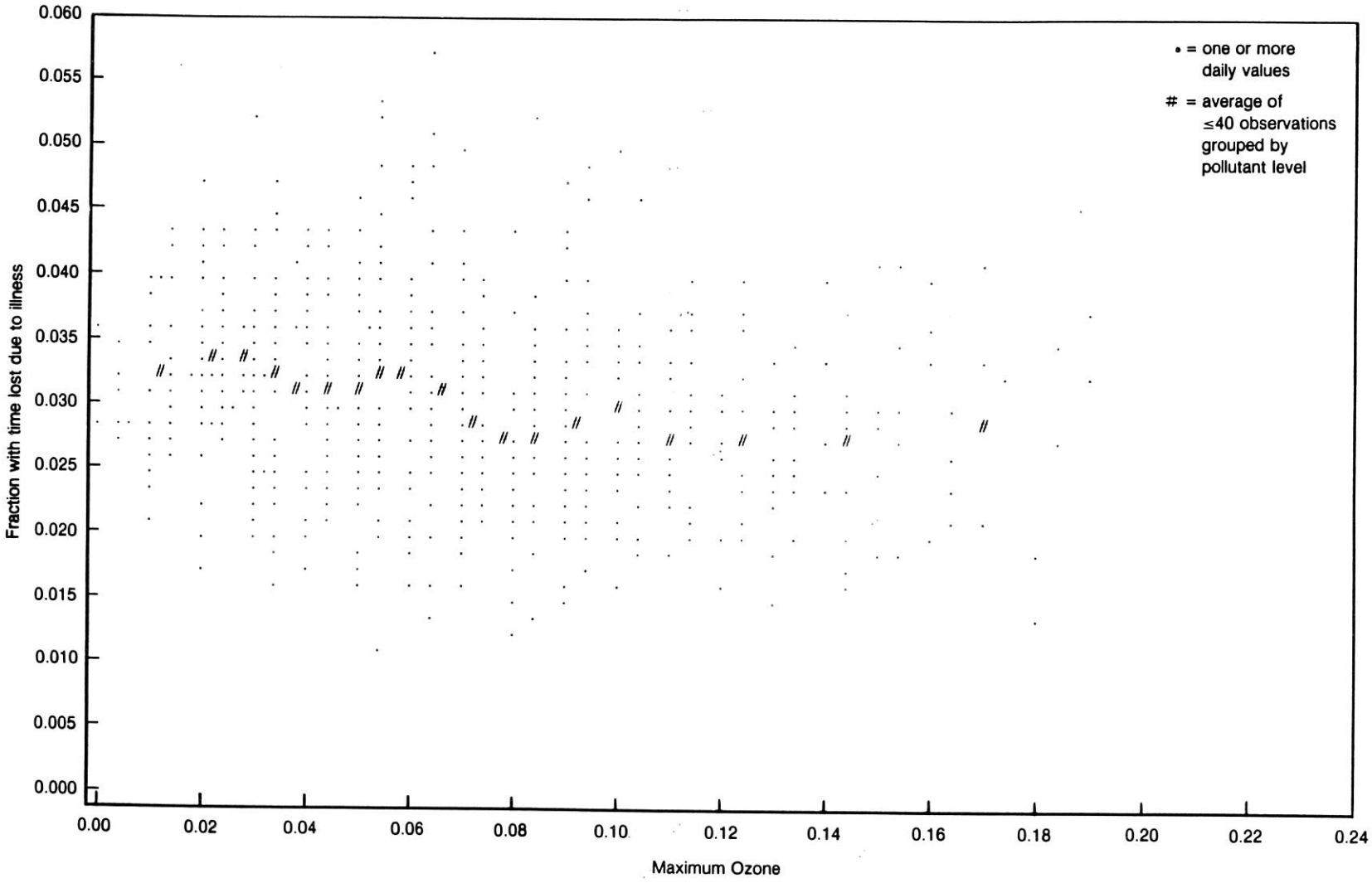

Fig. A.10—Dayton time lost due to illness: Ozone

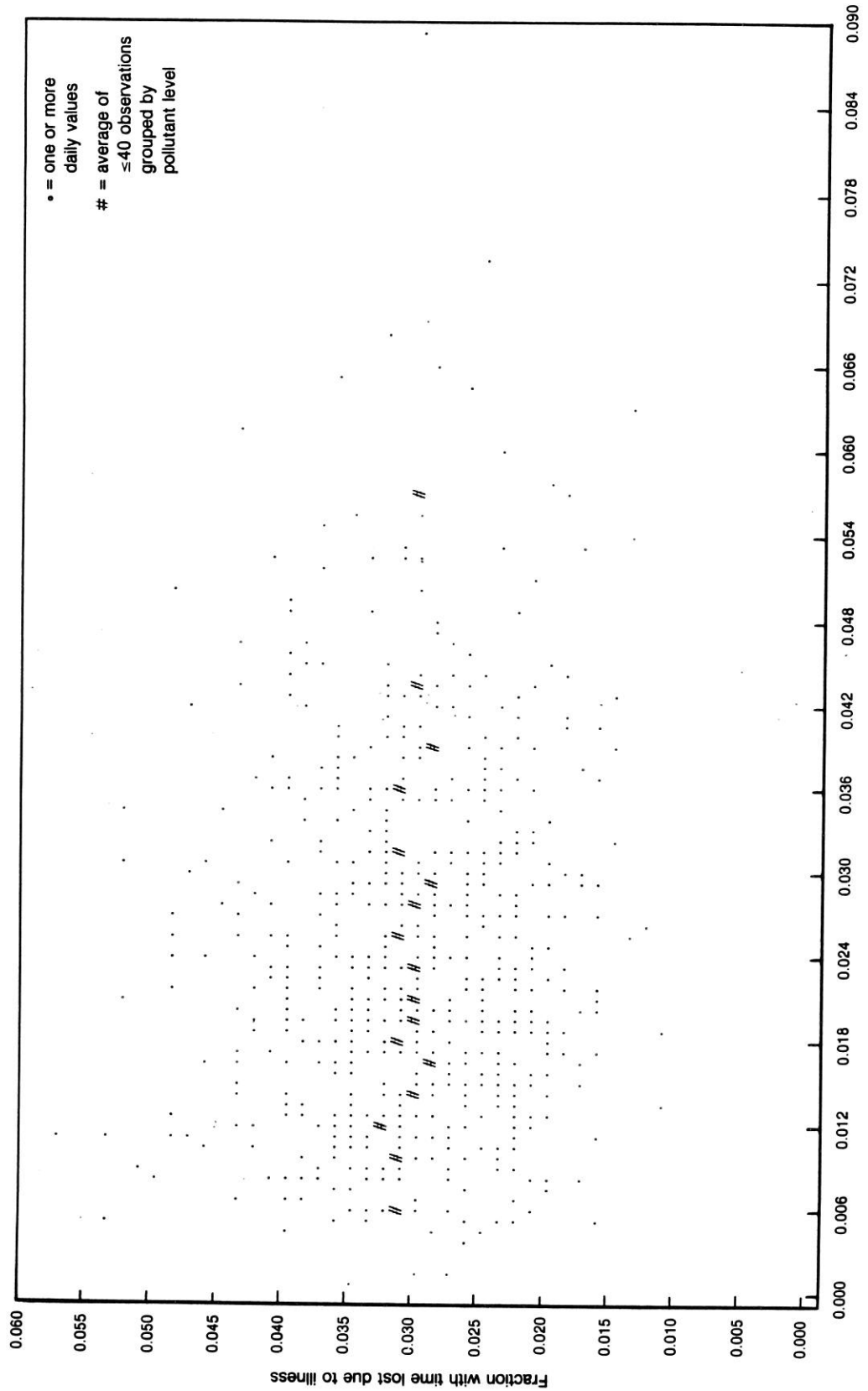

Fig. A.11—Dayton time lost due to illness: NO_2

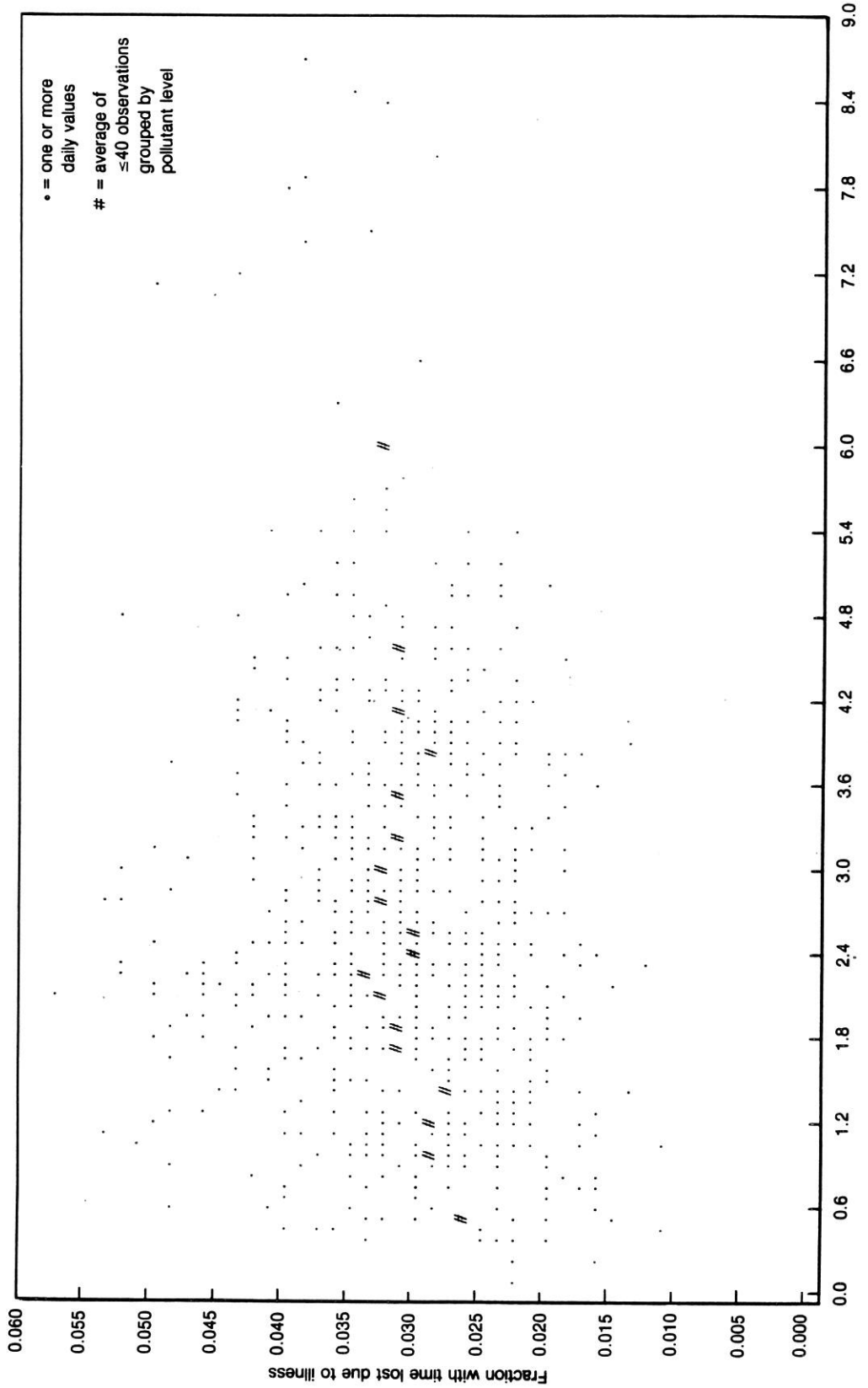

Fig. A.12—Dayton time lost due to illness: CO

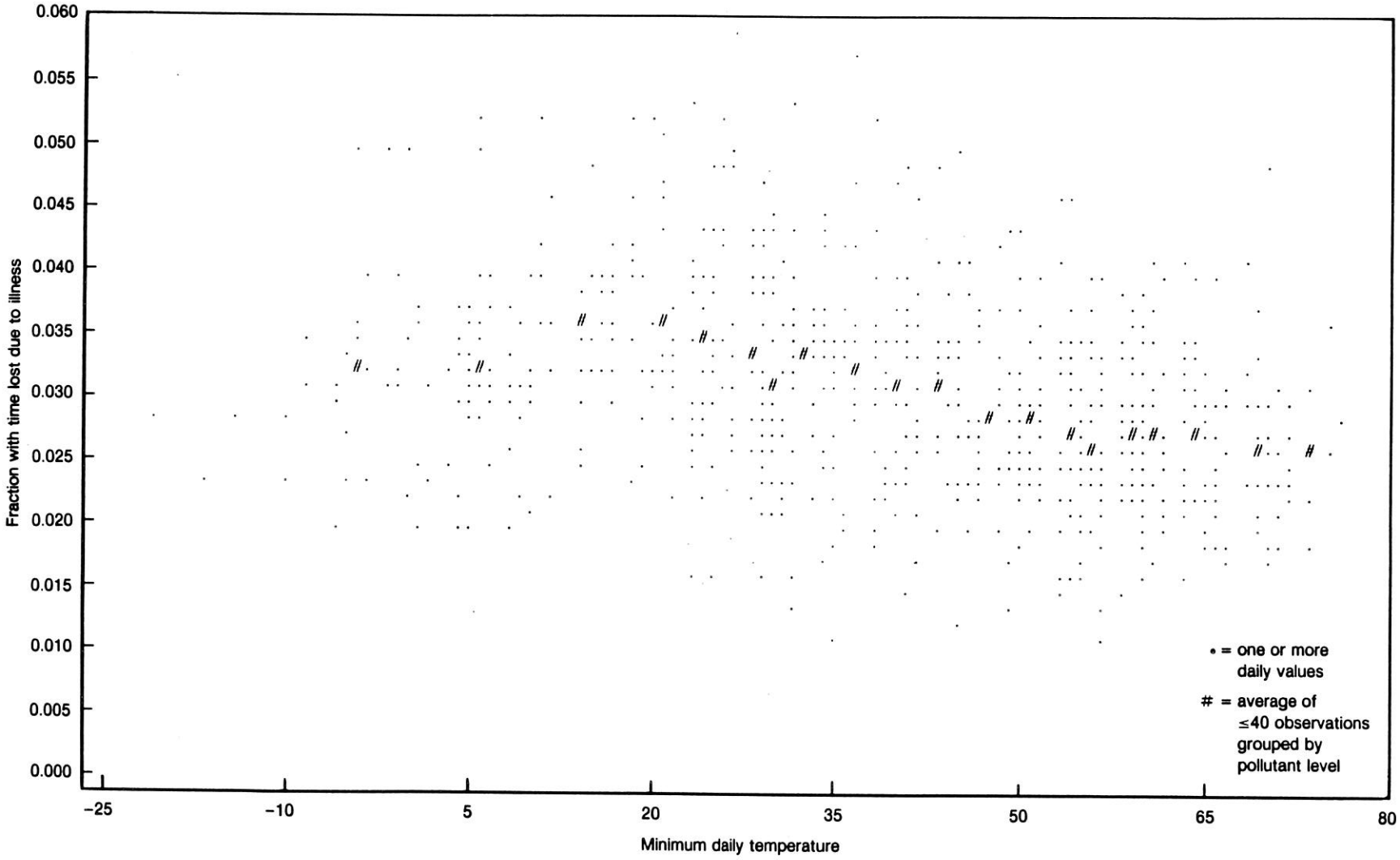

Fig. A.13—Dayton time lost due to illness: Minimum daily temperature

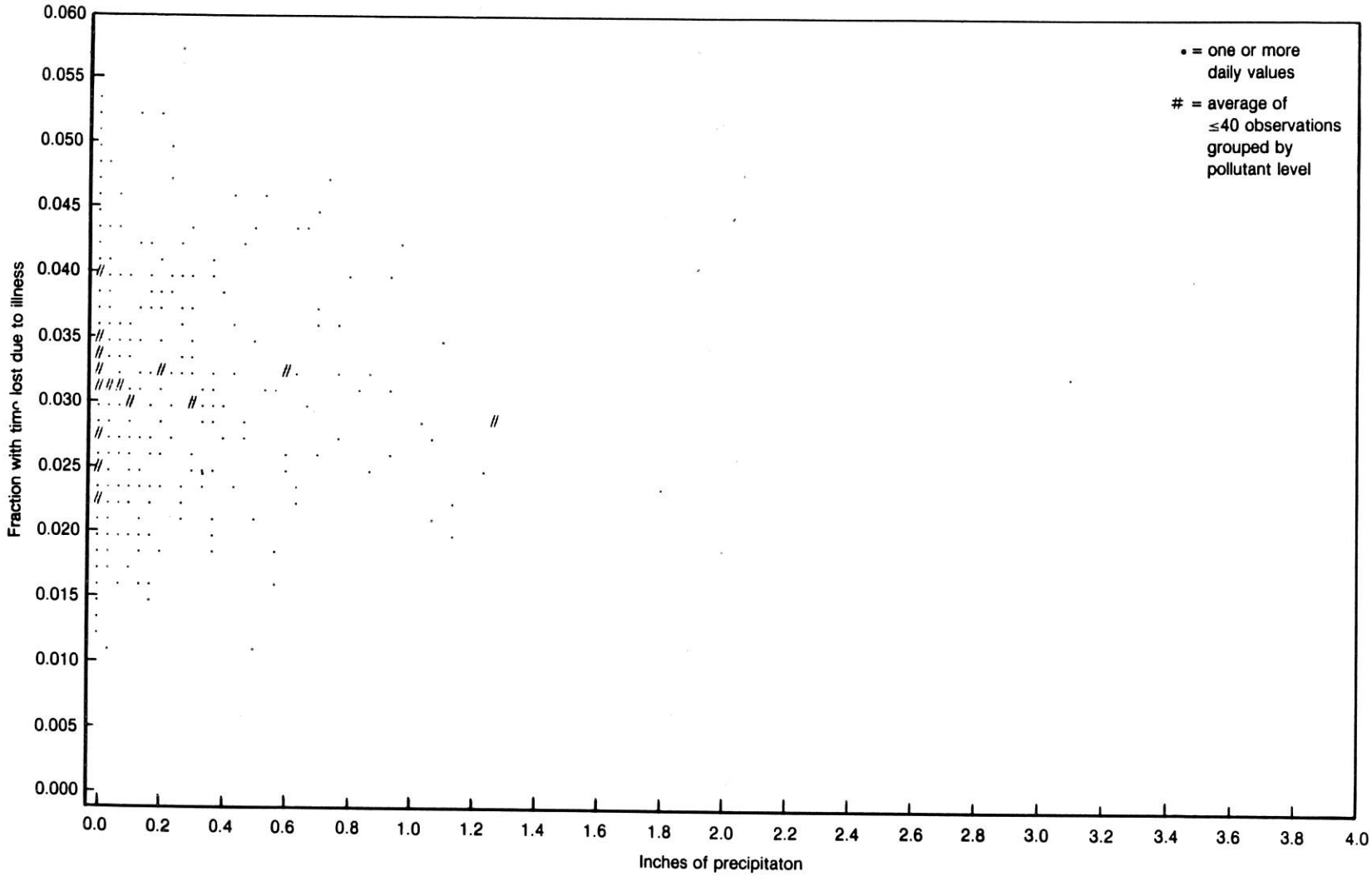

Fig. A.14—Dayton time lost due to illness: Inches of precipitation

Appendix B

DESCRIPTIVE SUMMARIES OF INDIVIDUAL RESPONSES

Tables B.1 and B.2 and Figs. B.1 through B.21 summarize the responses of the 247 persons in the final Dayton analysis sample. Table B.1 gives the unweighted major summary statistics for the estimated individual responses; the random-effects model analysis results given in the text in Sec. IV give analogous summary statistics for a weighted analysis. The top part of the table gives the average of the individual responses along with other univariate summary statistics.[1] The bottom part of the table gives the correlation coefficients and p values. Table B.2 summarizes the z statistics for the estimated individual responses; the table is formatted in the same way as Table B.1. The z analysis is discussed further in the latter part of this appendix. Further summary statistics and graphical summaries for the individual responses are given in Figs. B.1 through B.7.

Generally speaking, the estimated individual responses are somewhat skewed; compared with the comparable distributions for Seattle, the Dayton coefficients are less affected by outliers.

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 likely 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 B.8 through B.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 outliers are associated with large standard errors.

Given the heterogeneity of the estimated individual responses, the unweighted summaries given in Table B.1 and Figs. B.1 through B.7 are not very informative. The unweighted summaries given in these exhibits do not account for the heterogeneity. 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. IV, is similar to that approach (it also allows the estimation of between-individual differences).

Another way to account for heterogeneity in the estimated individual responses is to examine the individual z statistics, which rescale the estimated individual responses by their estimated precision. Table B.2 gives the major summaries for the individual z statistics. Figs. B.15 through B.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 SO_2; T2 refers to TSP; T3 to ozone; T4 to NO_2; T5 to CO; T6 to minimum temperature; and T7 to precipitation.

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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 TSP is in terms of logit per $\mu g/m^3$; response to NO_2 is in logit per ppm NO_2; response to CO is in logit per ppm CO; response to minimum temperature is in logit per degree Fahrenheit; response to precipitation is in logit per inch of precipitation.

Table B.1

MAJOR SUMMARIES OF THE INDIVIDUAL RESPONSES

Variable	N	Mean	Std Dev	Sum	Minimum	Maximum
SO_2	247	−26.655441057	80.81085188	−6583.88640983	−471.23918162	101.8552494
TSP	247	−0.00281540	0.02498728	−0.69540366	−0.15801947	0.0821089
OZO	247	−5.26193202	28.01016236	−1299.69720804	−94.23481798	97.0841922
NO_2	247	−3.83388238	30.17311604	−946.96894901	−136.54366269	74.4918969
CO	247	−0.16053787	0.52615916	−39.65285313	−2.53071610	0.9707184
Minimum temperature	247	−0.01150379	0.05181024	−2.84143736	−0.26719575	0.1743278
Precipitation	247	−1.12280032	4.55825393	−277.33167975	−29.31242365	6.2664486

CORRELATION COEFFICIENTS / PROB > |R| UNDER HO:RHO=0

	SO_2AV	TSPAV	OZOMX	NO_2MX	COAV	MINTEMP	PRECIP
SO_2AV	1.00000	−0.11777	0.14217	−0.06024	−0.01616	−0.05664	−0.00533
	0.0000	0.0646	0.0255	0.3458	0.8005	0.3755	0.9336
TSPAV	−0.11777	1.00000	0.00354	−0.11546	0.00986	−0.36036	0.04424
	0.0646	0.0000	0.9559	0.0701	0.8775	0.0001	0.4889
OZONX	0.14217	0.00354	1.00000	−0.27904	0.08894	−0.55935	0.11966
	0.0255	0.9559	0.0000	0.0001	0.1635	0.0001	0.0604
NO_2MX	−0.06024	−0.11546	−0.27904	1.00000	0.02495	0.17120	−0.16016
	0.3458	0.0701	0.0001	0.0000	0.6964	0.0070	0.0117
COAV	−0.01616	0.00986	0.08894	0.02495	1.00000	0.14229	−0.04501
	0.8005	0.8775	0.1635	0.6964	0.0000	0.0253	0.4813
MINTEMP	−0.05664	−0.36036	−0.55935	0.17120	0.14229	1.00000	−0.21525
	0.3755	0.0001	0.0001	0.0070	0.0253	0.0000	0.0007
PRECIP	−0.00533	0.04424	0.11966	−0.16016	−0.04501	−0.21525	1.00000
	0.9336	0.4889	0.0604	0.0117	0.4813	0.0007	0.0000

AV = Average
MX = Maximum

Table B.2

MAJOR SUMMARIES OF THE INDIVIDUAL Z STATISTICS FOR THE INDIVIDUAL RESPONSES

Variable	N	Mean	Std Dev	Sum	Minimum	Maximum
T1	247	−0.00593408	1.02613915	−1.46571833	−1.70943631	2.6627686
T2	247	0.03644081	1.11519976	9.00087905	−2.59248701	3.3353641
T3	247	−0.13435982	1.07211348	−33.18687524	−2.47799944	2.6264322
T4	247	0.05311599	1.10444315	13.11964839	−2.71121042	3.1131776
T5	247	−0.16001833	1.07480488	−39.52452800	−2.66010369	2.0751730
T6	247	−0.18891585	1.08346625	−46.66221521	−2.76060004	2.8530968
T7	247	0.10655116	0.90593309	26.31813536	−3.00629615	2.4354865

CORRELATION COEFFICIENTS / PROB > |R| UNDER HO:RHO=0

	T1	T2	T3	T4	T5	T6	T7
T1	1.00000	−0.11384	0.04097	−0.17107	−0.13406	−0.04495	0.02352
	0.0000	0.0741	0.5216	0.0070	0.0352	0.4820	0.7130
T2	−0.11384	1.00000	−0.01710	−0.10268	−0.01688	−0.26077	−0.00419
	0.0741	0.0000	0.7891	0.1074	0.7919	0.0001	0.9478
T3	0.04097	−0.01710	1.00000	−0.26189	0.11595	−0.59408	0.21340
	0.5216	0.7891	0.0000	0.0001	0.0689	0.0001	0.0007
T4	−0.17107	−0.10268	−0.26189	1.00000	−0.06661	−0.01106	−0.17825
	0.0070	0.1074	0.0001	0.0000	0.2970	0.8627	0.0050
T5	−0.13406	−0.01688	0.11595	−0.06661	1.00000	0.16678	−0.04561
	0.0352	0.7919	0.0689	0.2970	0.0000	0.0086	0.4755
T6	−0.04495	−0.26077	−0.59408	−0.01106	0.16678	1.00000	−0.30065
	0.4820	0.0001	0.0001	0.8627	0.0086	0.0000	0.0001
T7	0.02352	−0.00419	0.21340	−0.17825	−0.04561	−0.30065	1.00000
	0.7130	0.9478	0.0007	0.0050	0.4755	0.0001	0.0000

AV = Average
MX = Maximum

For all the aerometric attributes, 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 B.2 are close to one, and the skewness and kurtosis given in the "moments" sections of Figs. B.15 through B.21 are both small. The normal plots given in Figs. B.15 through B.21 are reasonably close to straight lines, as they should be if the distributions are close to a normal distribution.

For all pollution measures, the average z statistics given under the column "MEAN" in Table B.2 are close to zero. The effect is statistically significant at the 5 percent level for ozone, CO, and minimum temperature. (See the entries "T:MEAN=0" and "PROB>|T|" in the "moments" sections of Figs. B.15 through B.21.) Those results are also given in Table 4.4 as the second column "Individual z Statistics."

The results of the z analysis vary somewhat from those of the random-effects model. (See Tables B.1 and B.2, and also Table 4.4 in the text.) As was noted in the text, the random-effects model would give more efficient results for the Whittemore-Korn model, although the discrepancy indicates that further investigation is warranted.

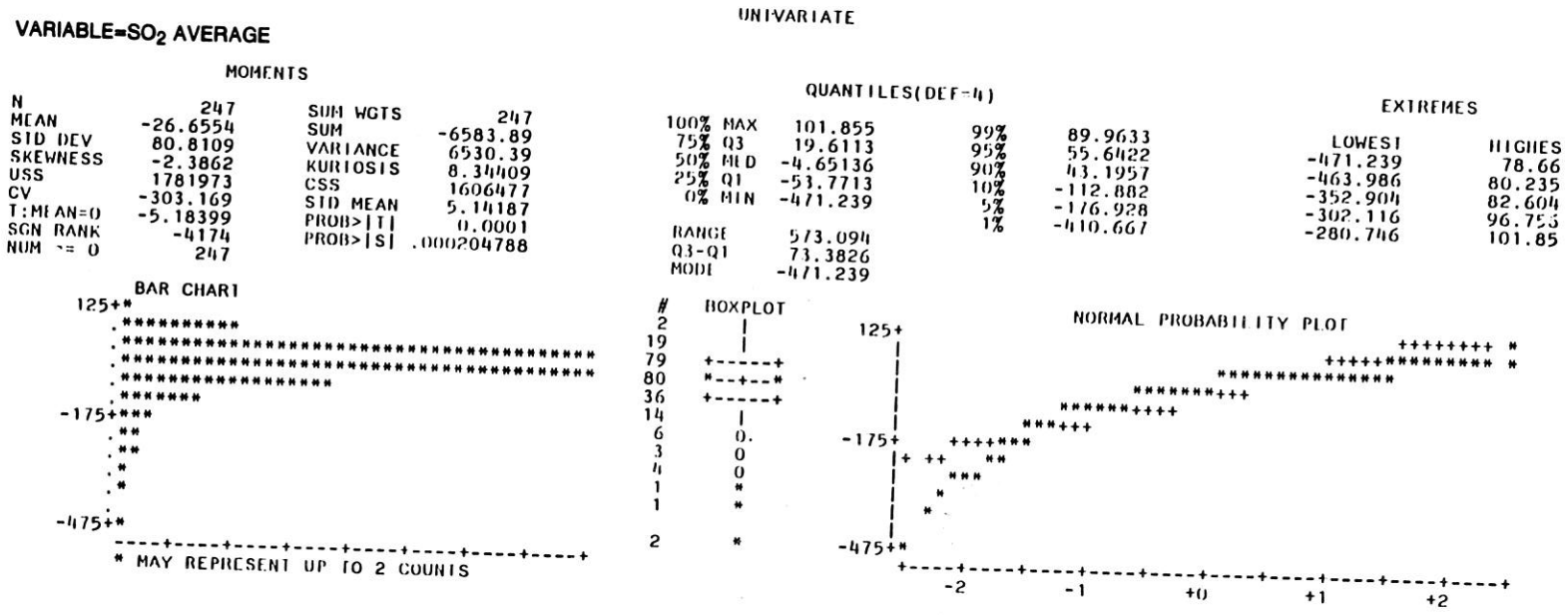


Fig. B.1—Further summaries of the individual responses to SO_2

UNIVARIATE

VARIABLE=TSP AVERAGE

MOMENTS

N	247	SUM WGTS	247
MEAN	-0.0028154	SUM	-0.695404
STD DEV	0.0249873	VARIANCE	.000624364
SKEWNESS	-1.31881	KURTOSIS	6.66923
USS	0.155551	CSS	0.153594
CV	-887.522	STD MEAN	0.0015899
T:MEAN=0	-1.7708	PROB>|T|	0.0778323
SGN RANK	-1197	PROB>|S|	0.287107
NUM ^= 0	247		

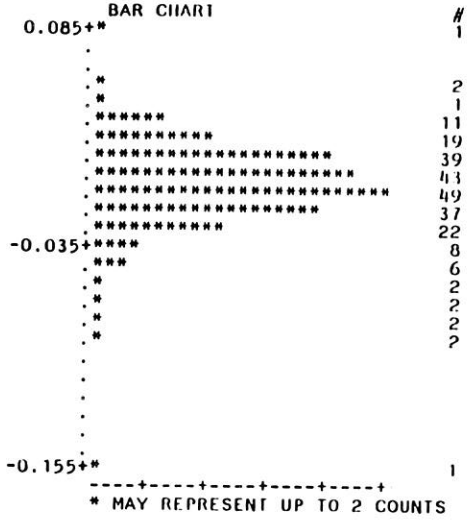
QUANTILES(DEF=4)

100%	MAX 0.0821089	99%	0.0528669
75%	Q3 0.0111012	95%	0.0313298
50%	MED-.00118258	90%	0.0251517
25%	Q1 -0.0146898	10%	-0.0274818
0%	MIN -0.158019	5%	-0.0454582
		1%	-0.0835764
RANGE	0.240128		
Q3-Q1	0.0257909		
MODE	-0.158019		

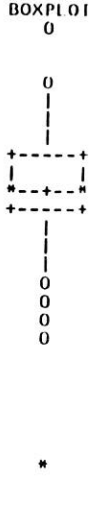
EXTREMES

LOWEST	HIGHES
-0.158019	0.039101
-0.0849502	0.044489
-0.0820881	0.051686
-0.0785857	0.053955
-0.0716583	0.082108

BAR CHART



BOXPLOT



NORMAL PROBABILITY PLOT

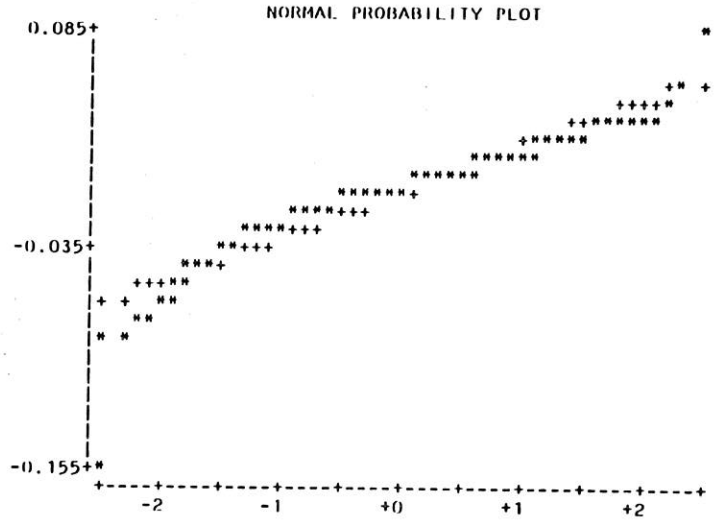


Fig. B.2—Further summaries of the individual responses to TSP

UNIVARIATE

VARIABLE=OZONE MAXIMUM AVERAGE

MOMENTS

N	247	SUM WGTS	247
MEAN	-5.26193	SUM	-1299.7
STD DEV	28.0102	VARIANCE	784.569
SKEWNESS	-0.262925	KURTOSIS	1.68017
USS	199843	CSS	193004
CV	-532.317	STD MEAN	1.78224
T:MEAN=0	-2.95242	PROB>|T|	0.00345796
SGN RANK	-2610	PROB>|S|	0.0202549
NUM ^= 0	247		

QUANTILES(DEF=4)

100% MAX	97.0842	99%	85.8282
75% Q3	10.7771	95%	33.7864
50% MED	-2.18135	90%	22.3786
25% Q1	-18.9461	10%	-42.6462
0% MIN	-94.2348	5%	-58.4236
		1%	-86.2285
RANGE	191.319		
Q3-Q1	29.7232		
MODE	-94.2348		

EXTREMES

LOWEST	HIGHES
-94.2348	49.287
-87.2395	52.220
-85.1334	80.765
-78.0548	90.50
-75.6608	97.084

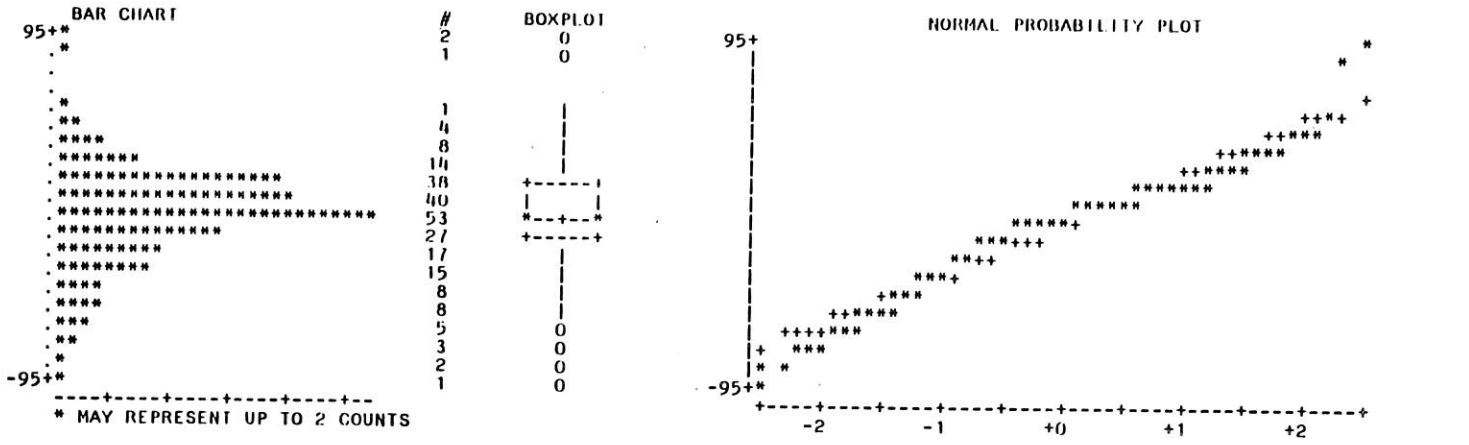


Fig. B.3—Further summaries of the individual responses to ozone

VARIABLE=NO_2 MAXIMUM AVERAGE

UNIVARIATE

MOMENTS

N	247	SUM WGTS	247
MEAN	-3.83388	SUM	-946.969
STD DEV	30.1731	VARIANCE	910.417
SKEWNESS	-1.13866	KURTOSIS	2.66586
USS	227593	CSS	223963
CV	-787.012	STD MEAN	1.91987
T:MEAN=0	-1.99695	PROB>|T|	0.0469324
SGN RANK	-681	PROB>|S|	0.5449
NUM ^= 0	247		

QUANTILES(DEF=4)

100% MAX	74.4919	99%	58.7237
75% Q3	16.1415	95%	33.5656
50% MED	-0.556027	90%	28.2203
25% Q1	-16.3527	10%	-42.2458
0% MIN	-136.544	5%	-62.8001
		1%	-116.313
RANGE	211.036		
Q3-Q1	32.4942		
MODE	-136.544		

EXTREMES

LOWEST	HIGHES
-136.544	46.690
-119.242	50.199
-113.141	56.629
-100.892	60.65
-86.5676	74.491

```
  STEM LEAF
     7 4
     6 1
     5 07
     4 2467
     3 000012234578
     2 001122233444455666788899
     1 000001111222233334445555666666677777888999999
     0 11122222333344455666777777888999
    -0 99988777766555554433333332222111111111000
    -1 9998876655544444332222111110000000
    -2 988777655532210 00
    -3 97654321100
    -4 8543221
    -5 875432
    -6 975410
    -7 640
    -8 7
    -9
   -10 1
   -11 93
   -12
   -13 7
       ----+----+----+----+----+----+----+----+----+----
       MULTIPLY STEM.LEAF BY 10**+01
```

```
#   BOXPLOT
1      0
1      |
2      |
4      |
12     |
25     |
44     +-----+
31     |     |
39     *--+--*
33     +-----+
17     |
11     |
7      |
6      |
6      0
3      0
1      0

1      0
2      0

1      *
```

NORMAL PROBABILITY PLOT

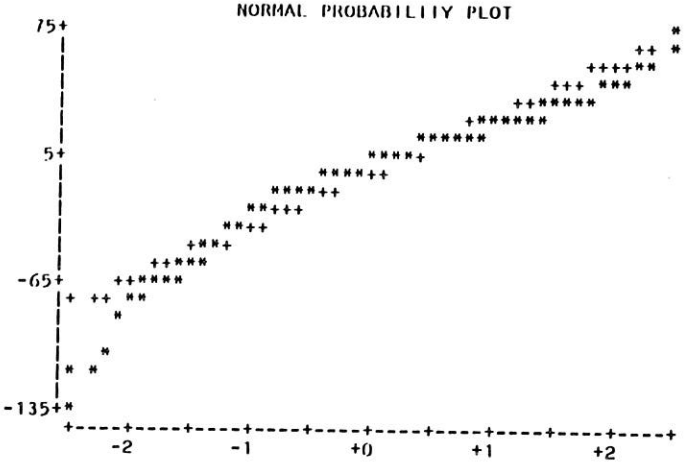


Fig. B.4—Further summaries of the individual responses to NO_2

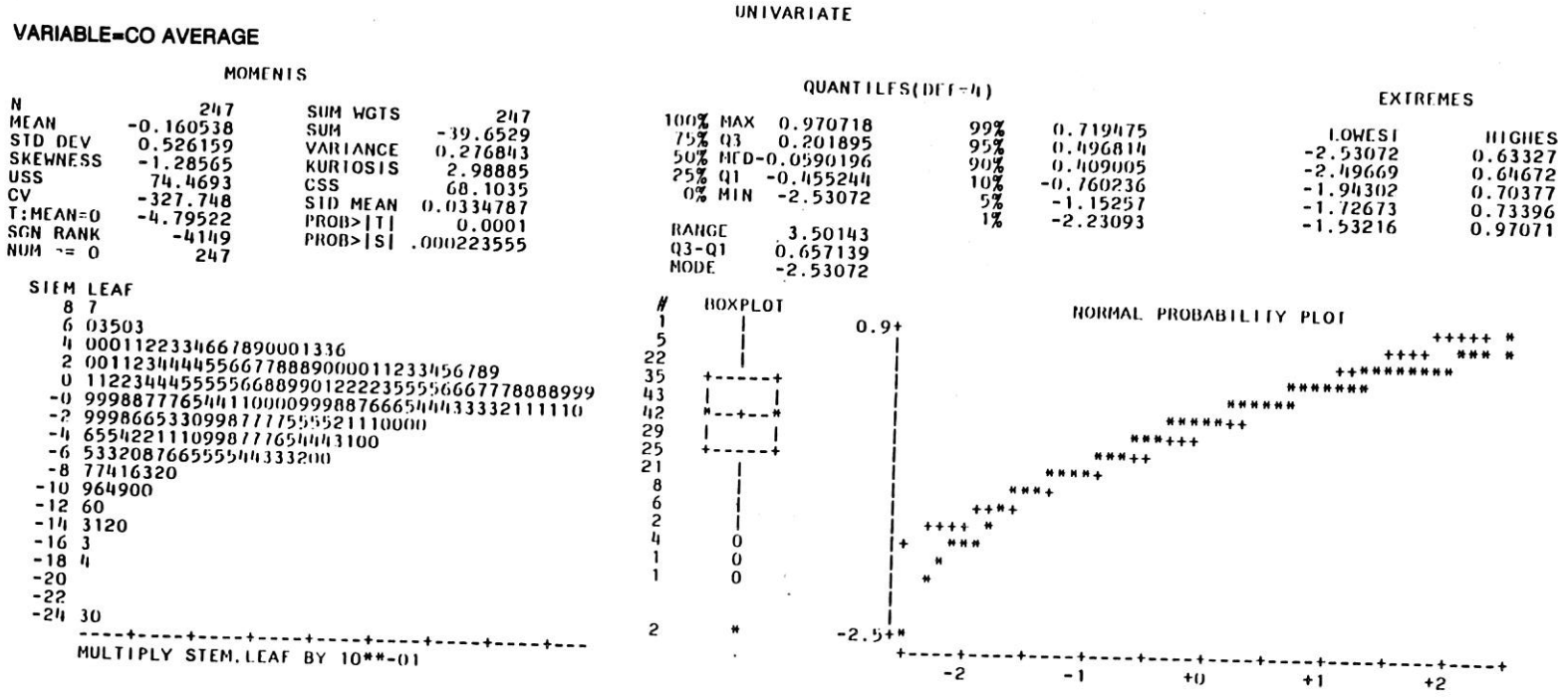


Fig. B.5—Further summaries of the individual responses to CO

UNIVARIATE

VARIABLE=MINTEMP

MOMENTS

N	247	SUM WGTS	247
MEAN	-0.0115038	SUM	-2.84144
STD DEV	0.0518102	VARIANCE	0.0026843
SKEWNESS	-0.616811	KURTOSIS	3.59973
USS	0.693025	CSS	0.660338
CV	-450.375	STD MEAN	0.00329661
T:MEAN=0	-3.48959	PROB>|T|	.000573102
SGN RANK	-3829	PROB>|S|	.000658981
NUM ^= 0	247		

QUANTILES(DEF=4)

100% MAX	0.174328	99%	0.111046
75% Q3	0.0190187	95%	0.0680851
50% MED	-.00861197	90%	0.047195
25% Q1	-0.0428927	10%	-0.0697501
0% MIN	-0.267196	5%	-0.0860393
		1%	-0.206016
RANGE	0.441524		
Q3-Q1	0.0619114		
MODE	-0.267196		

EXTREMES

LOWEST	HIGHES
-0.267196	0.096188
-0.235817	0.10712
-0.173731	0.10741
-0.126123	0.114
-0.11612	0.17432

```
STEM LEAF
  16 4
  14
  12
  10 774
   8 2626
   6 11179379
   4 011266789900156
   2 00112334449901122333444456777
   0 22334445556666677880001112222334445555577888899
  -0 998888776544443220099777766555421110000
  -2 98776554443222100099888877743221 10
  -4 9988766555532210099888865443111 0000
  -6 777776100998655100
  -8 54366521
 -10 6550
 -12 6
 -14
 -16 4
 -18
 -20
 -22 6
 -24
 -26 7
     ----+----+----+----+----+----+----+----+----+----+-
     MULTIPLY STEM.LEAF BY 10**-02
```

```
#  BOXPLOT
1     0
3     0
4     |
8     |
15    |
29    |
46  +-----+
38  *--+--*
34  |     |
35  +-----+
18    |
8     |
4     |
1     |
1     .0
1     *
1     *
```

NORMAL PROBABILITY PLOT

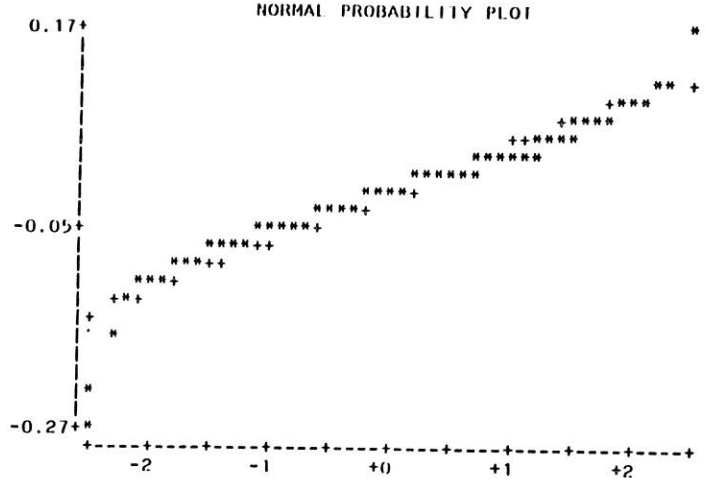


Fig. B.6—Further summaries of the individual responses to minimum temperature

UNIVARIATE

VARIABLE=PRECIP

MOMENTS

N	247	SUM WGTS	247
MEAN	-1.1228	SUM	-277.332
STD DEV	4.55825	VARIANCE	20.7777
SKEWNESS	-2.63479	KURTOSIS	10.4807
USS	5422.7	CSS	5111.31
CV	-405.972	STD MEAN	0.290035
T:MEAN=0	-3.87126	PROB>|T|	.0001138791
SGN RANK	-2740	PROB>|S|	0.0147995
NUM ^= 0	247		

QUANTILES(DEF=4)

100% MAX	6.26645	99%	5.69644
75% Q3	1.36976	95%	3.59337
50% MED	-0.0430625	90%	2.82834
25% Q1	-2.48367	10%	-5.32039
0% MIN	-29.3124	5%	-9.50147
		1%	-22.2029
RANGE	35.5789		
Q3-Q1	3.85343		
MODE	-29.3124		

EXTREMES

LOWEST	HIGHES
-29.3124	4.9548
-24.6031	5.189
-19.6028	5.4069
-17.8524	5.9636
-17.072	6.2664

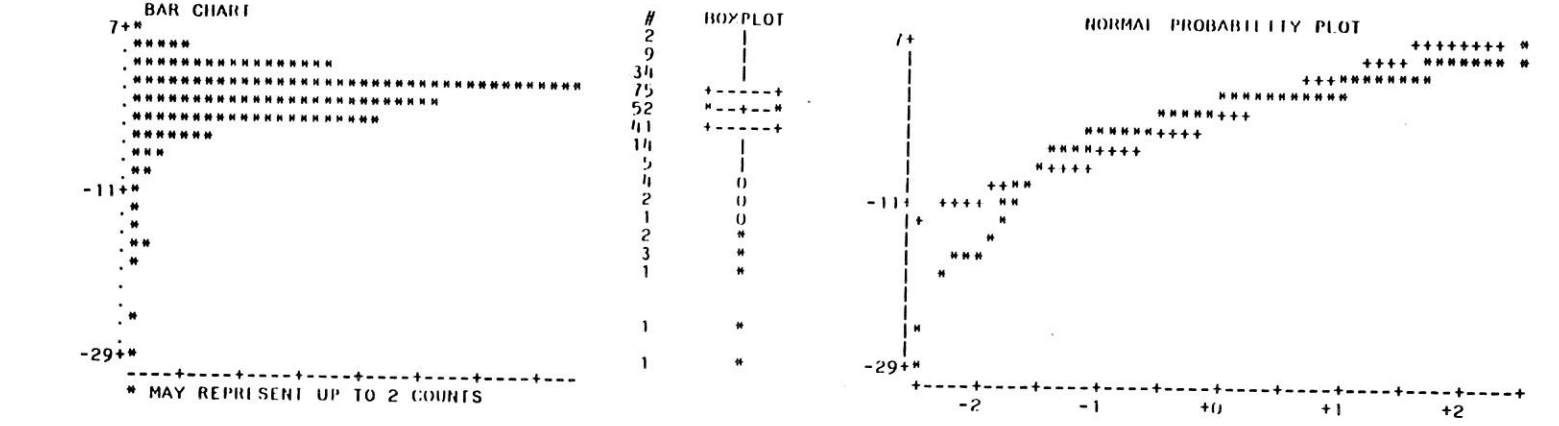


Fig. B.7—Further summaries of the individual responses to precipitation

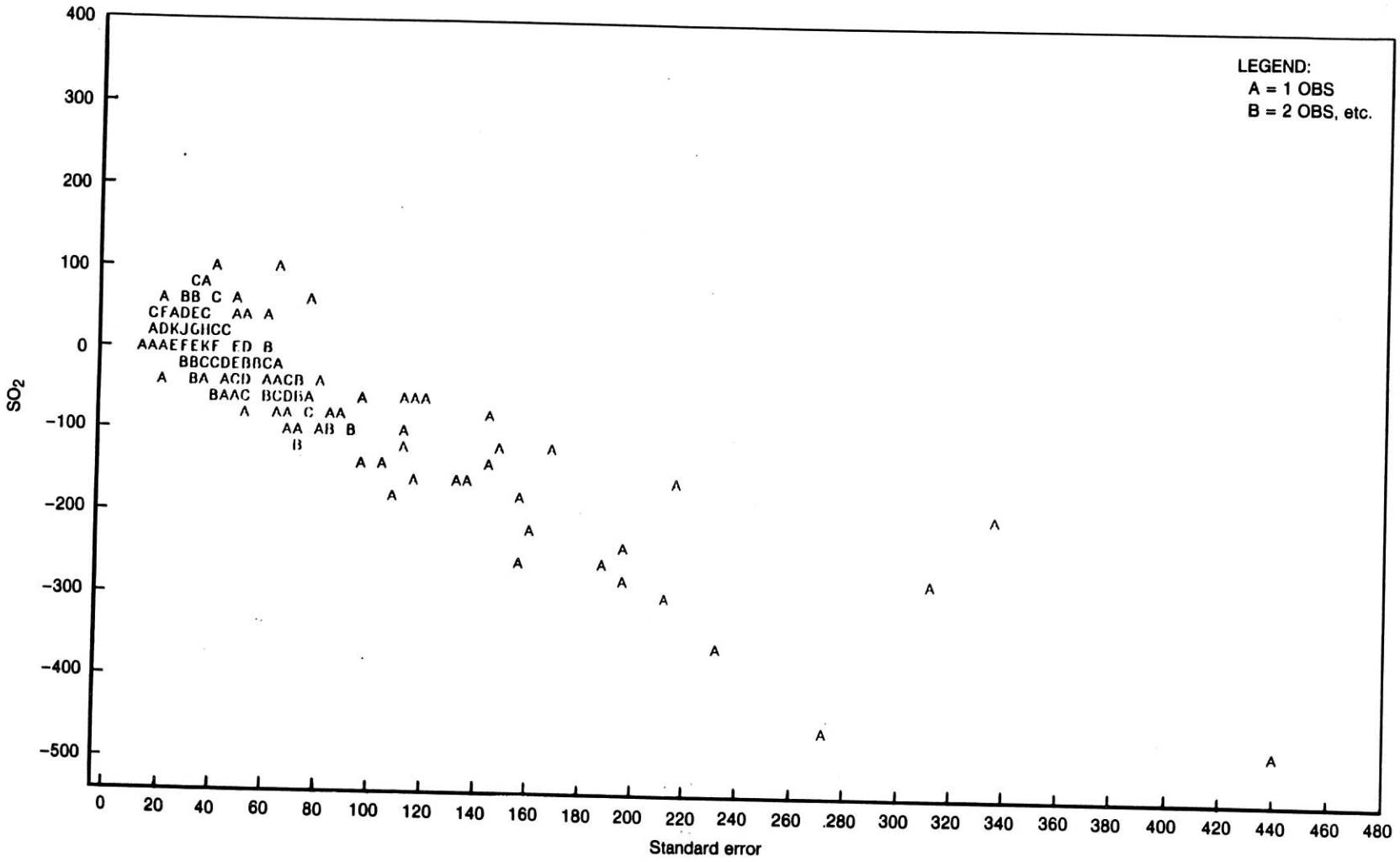


Fig. B.8—Scatterdiagram of estimated individual responses to SO_2 by the associated standard errors, Dayton

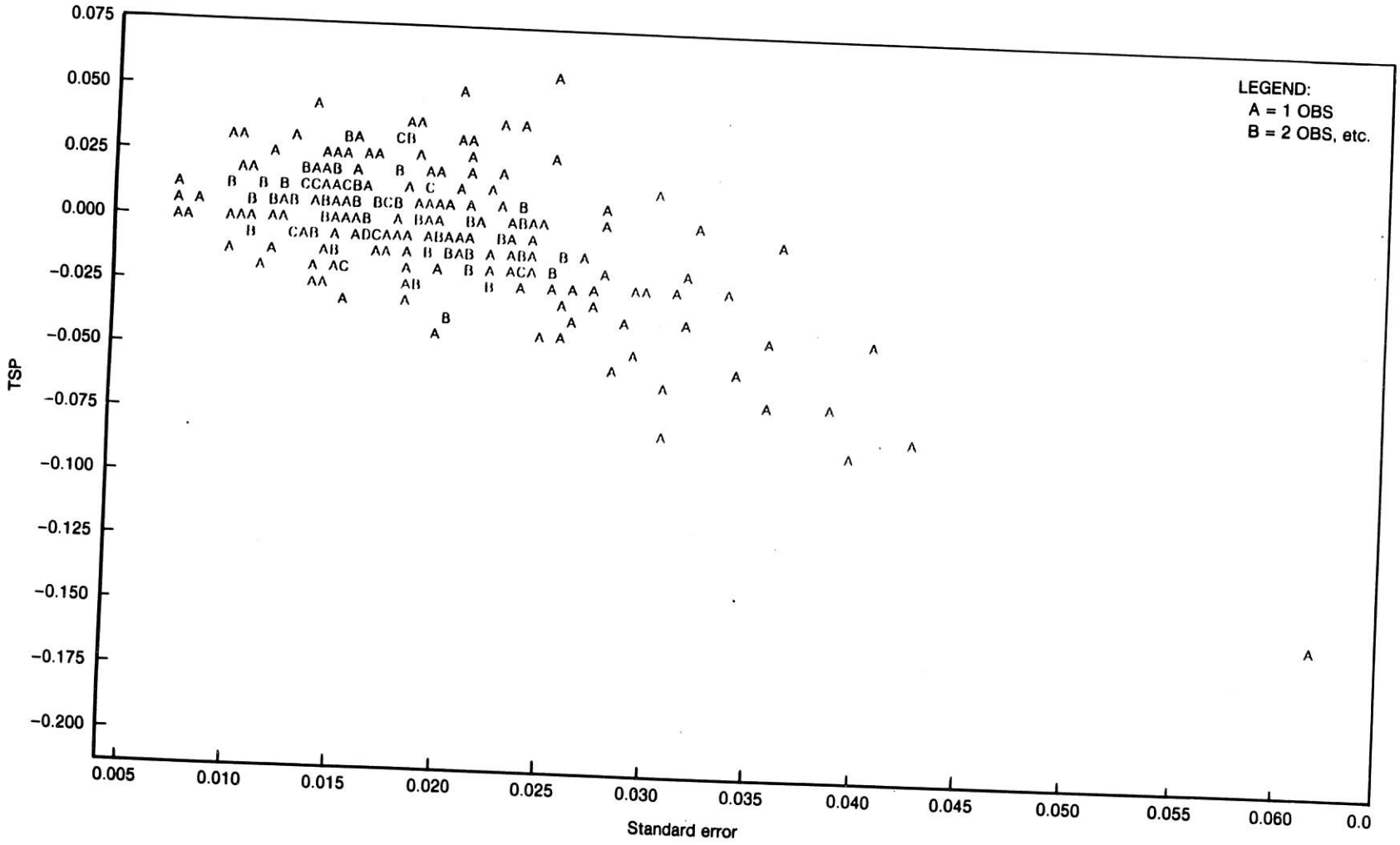


Fig. B.9—Scatterdiagram of estimated individual responses to TSP by the associated standard errors, Dayton

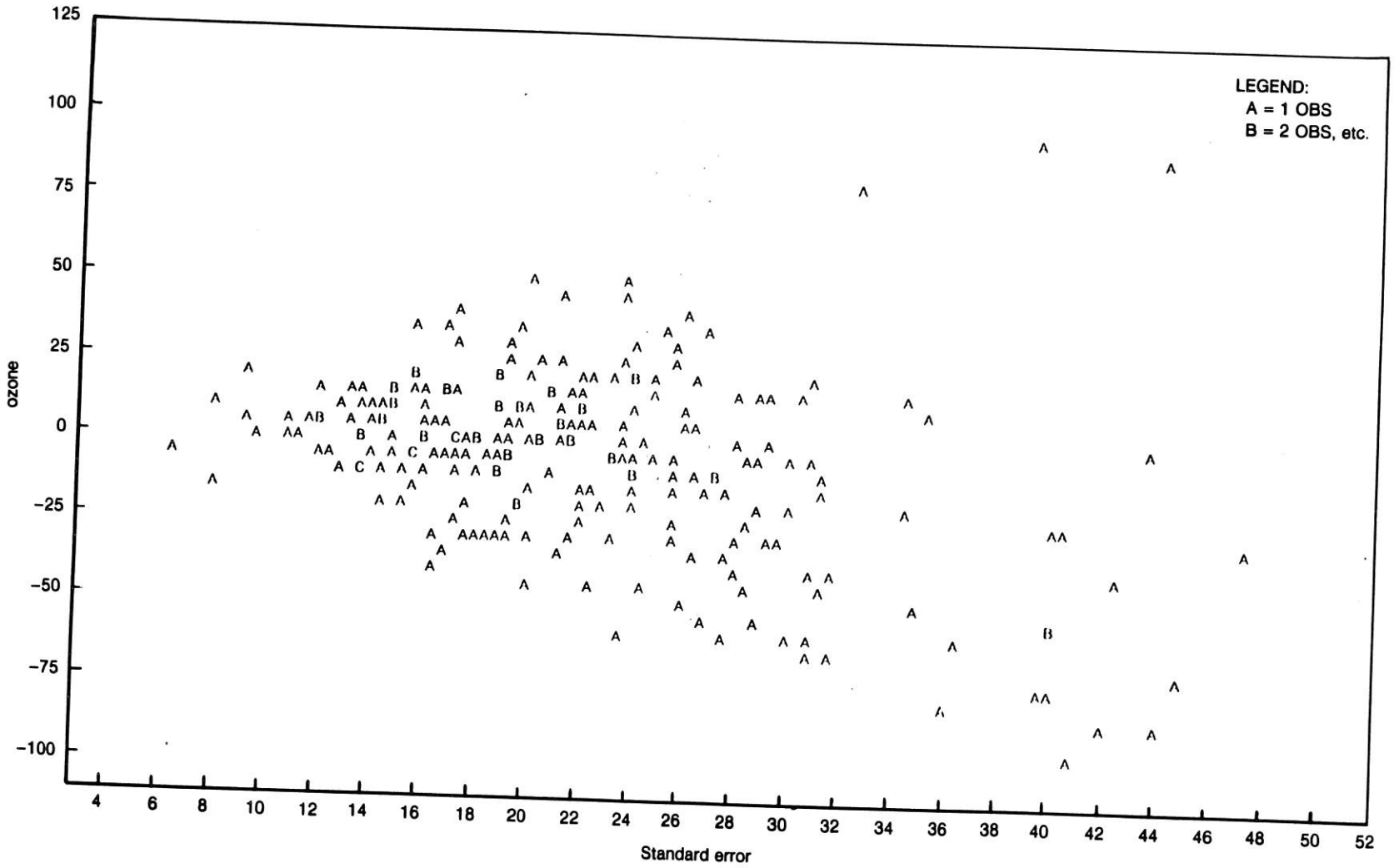


Fig. B.10—Scatterdiagram of estimated individual responses to ozone by the associated standard errors, Dayton

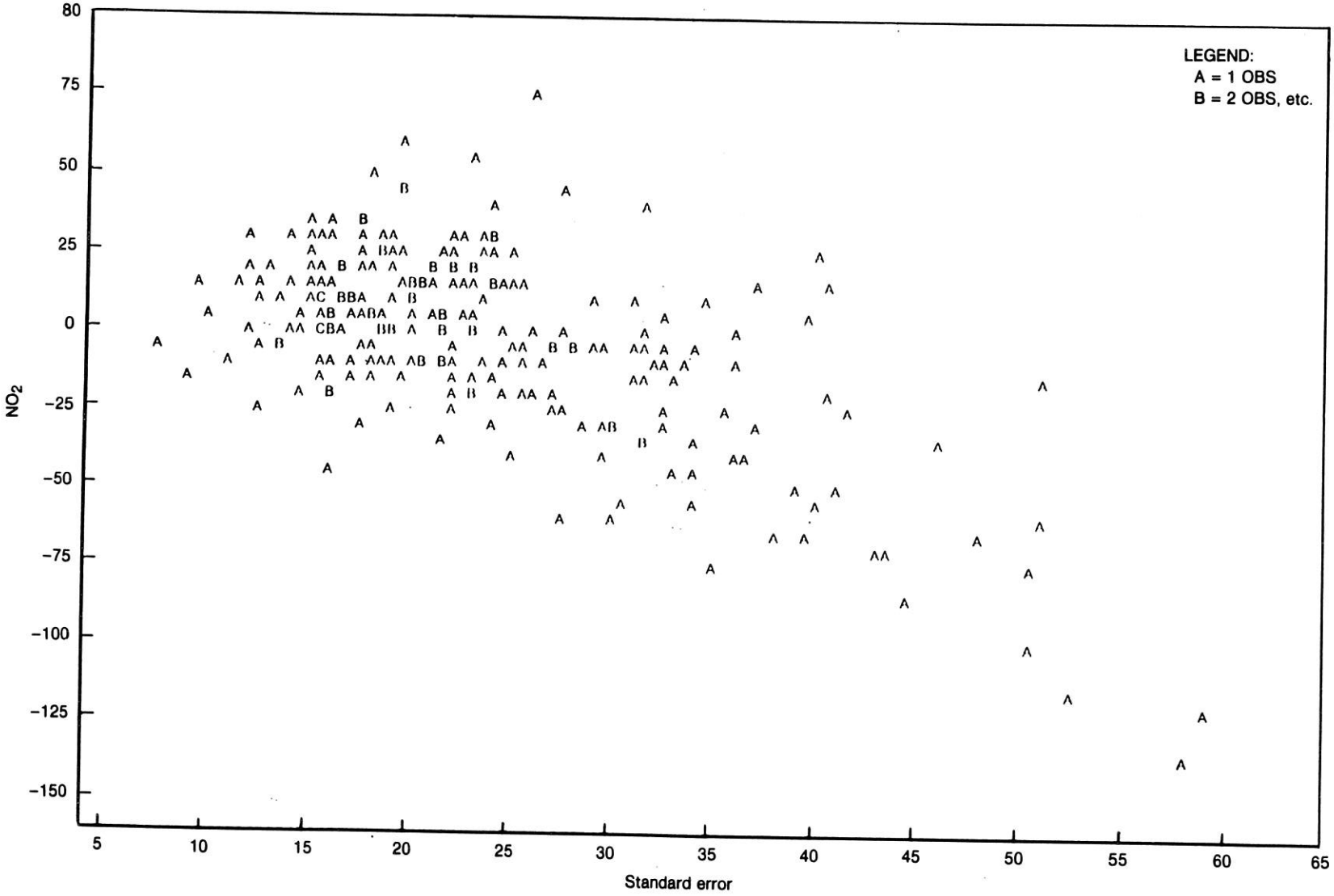


Fig. B.11—Scatterdiagram of estimated individual responses to NO_2 by the associated standard errors, Dayton

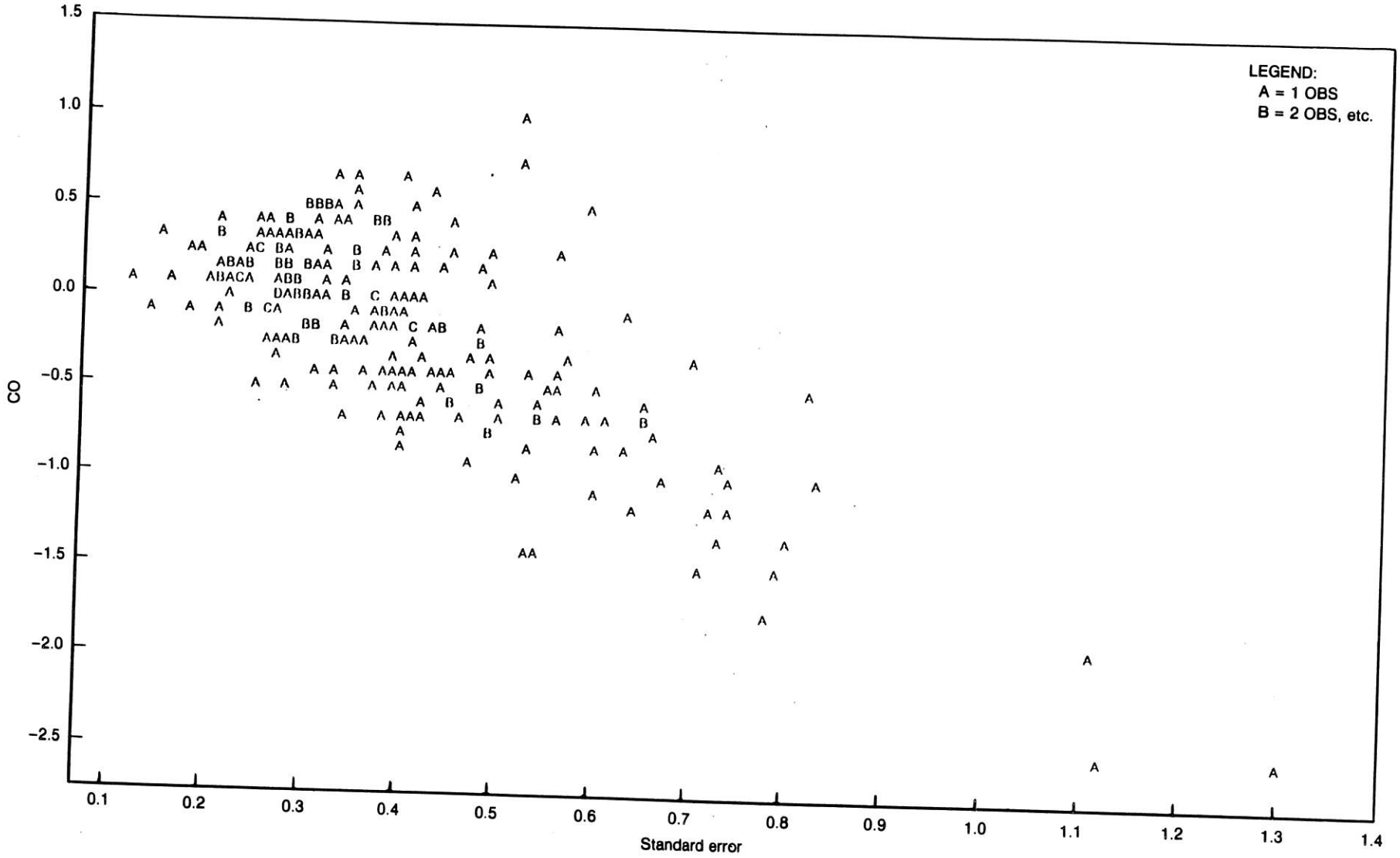


Fig. B.12—Scatterdiagram of estimated individual responses to CO by the associated standard errors, Dayton

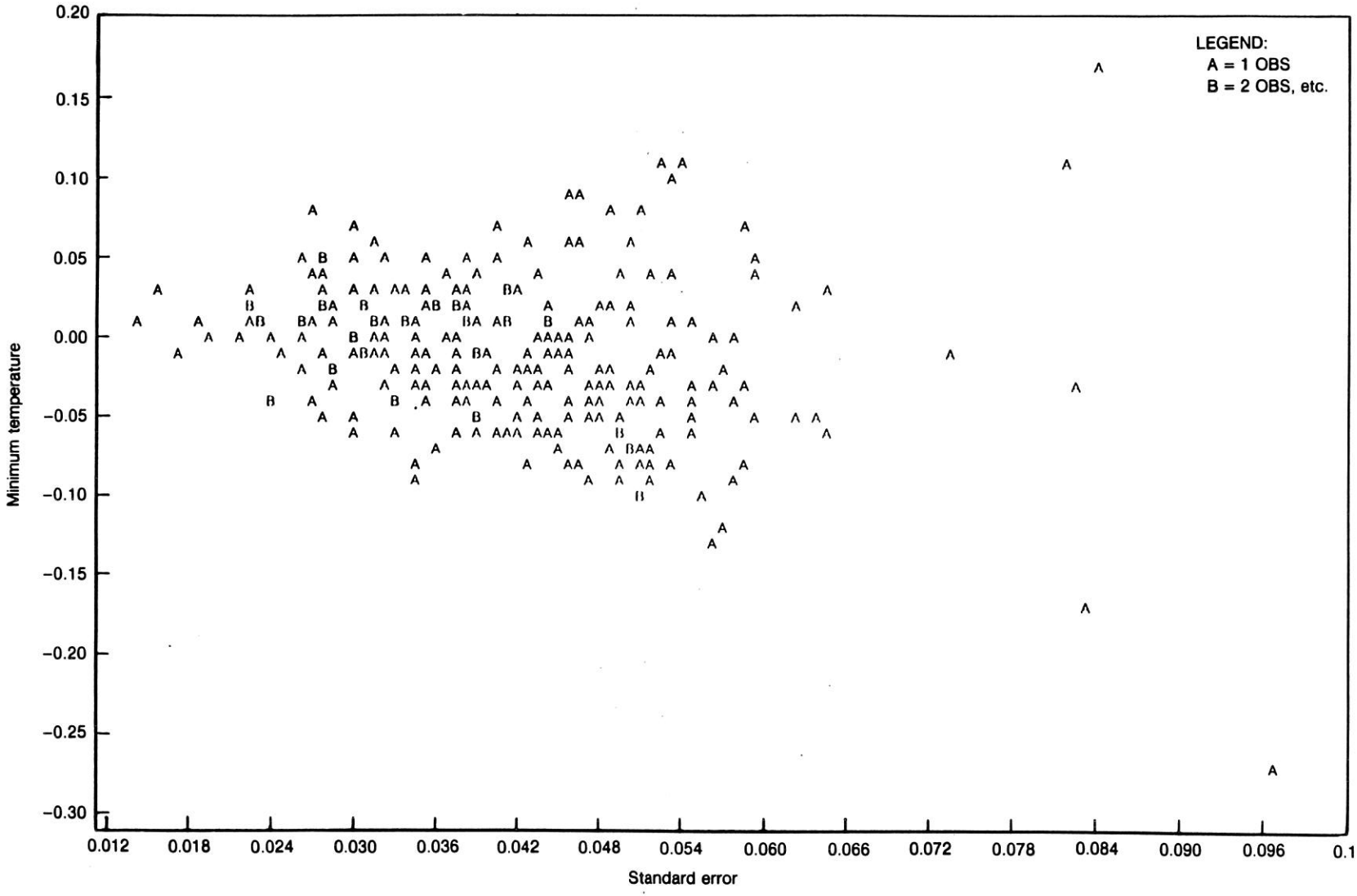


Fig. B.13—Scatterdiagram of estimated individual responses to minimum temperataure by the associated standard errors, Dayton

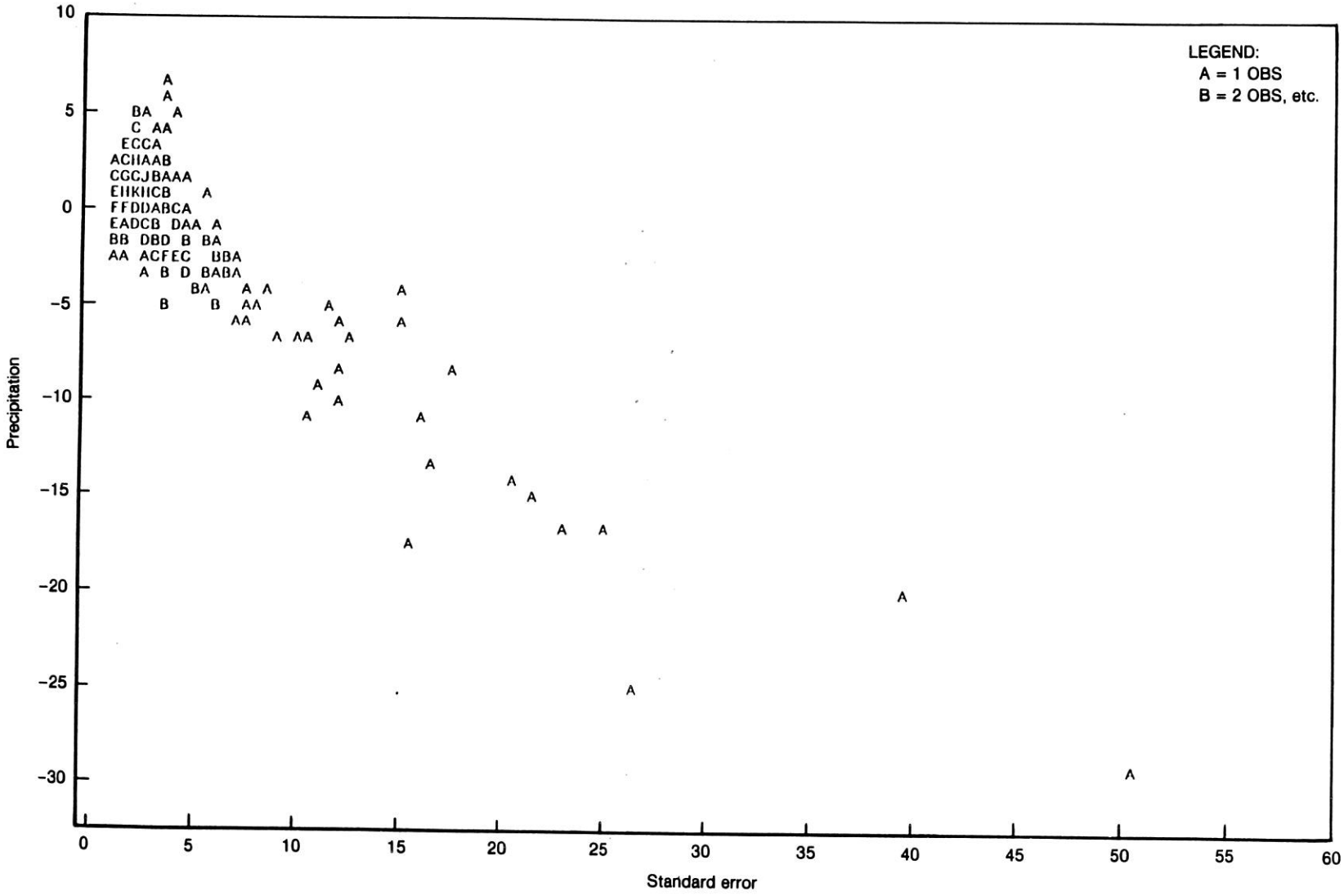


Fig. B.14—Scatterdiagram of estimated individual responses to precipitation by the associated standard errors, Dayton

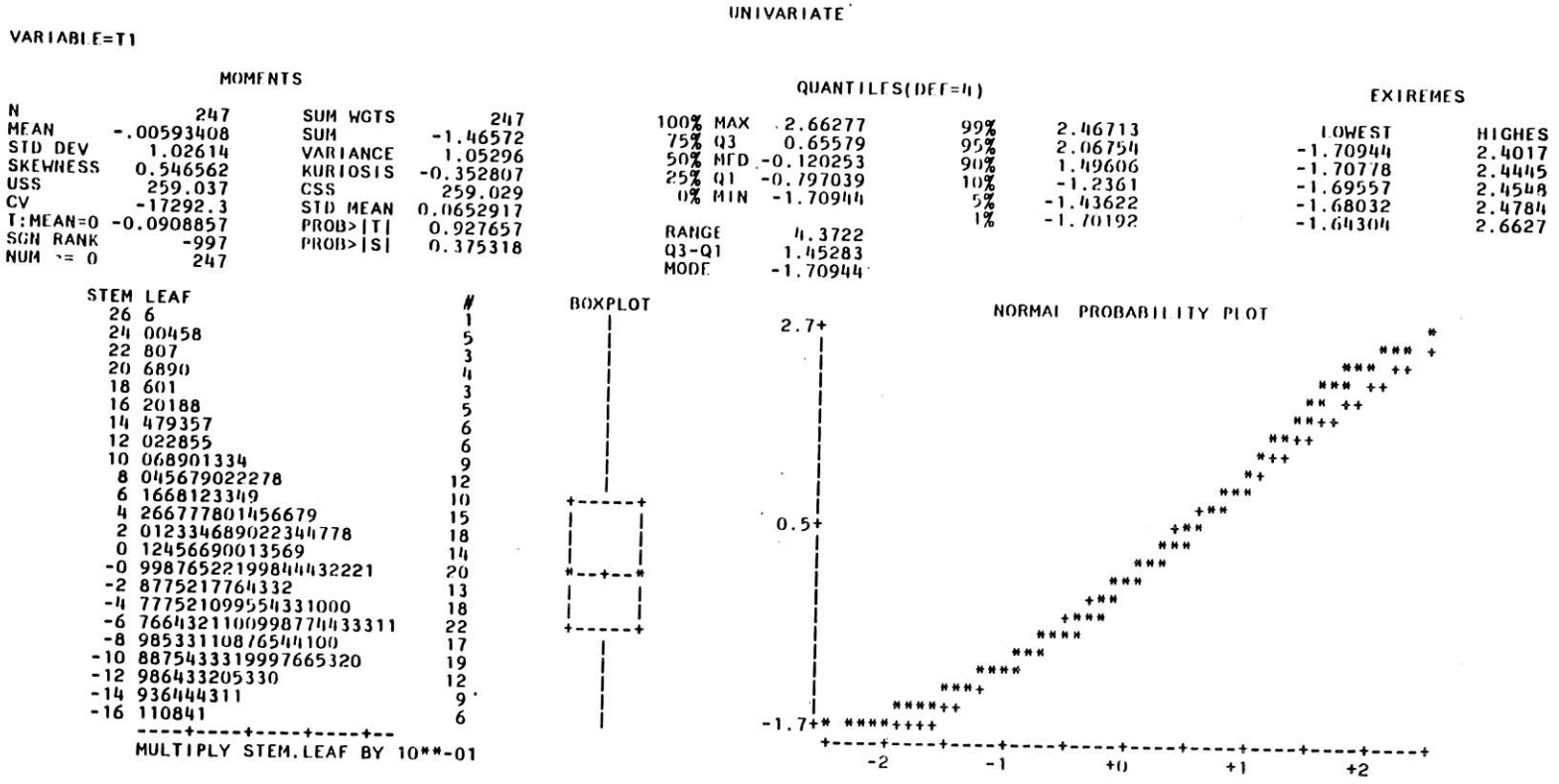


Fig. B.15—Further summaries of individual z statistics for SO_2

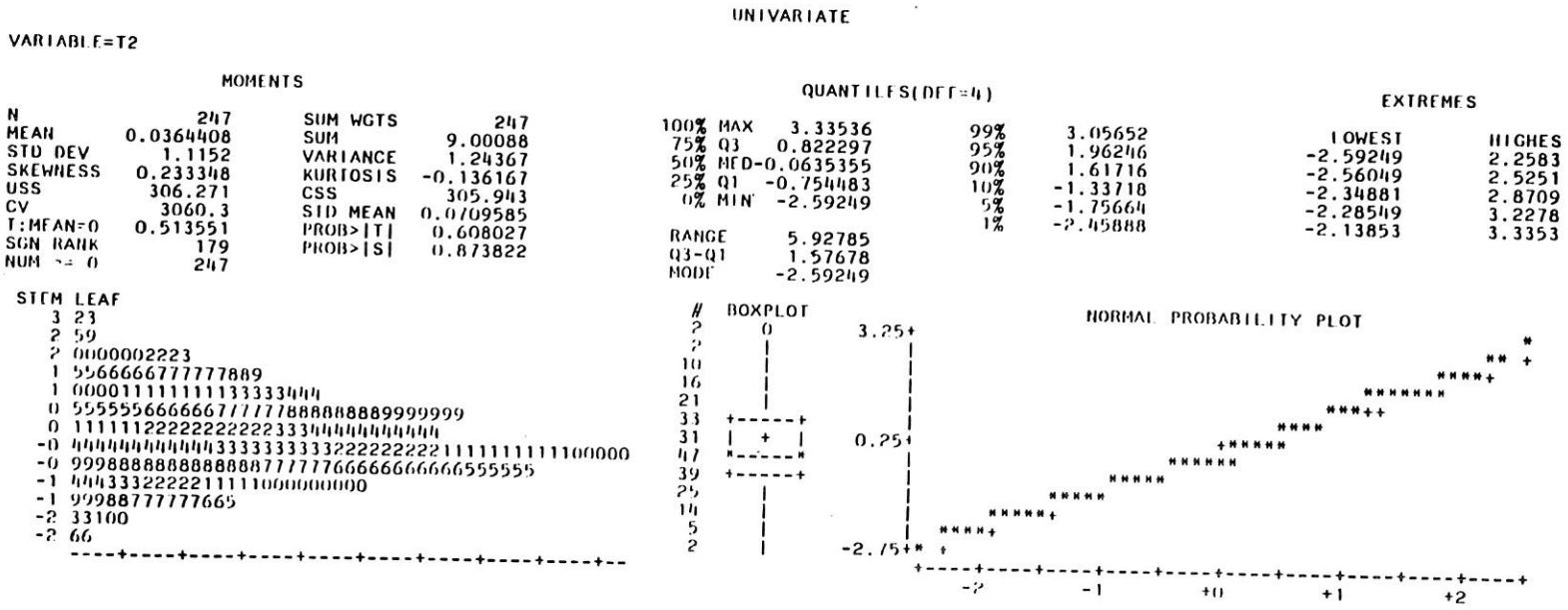


Fig. B.16—Further summaries of individual z statistics for TSP

UNIVARIATE

VARIABLE=T3

MOMENTS

N	247	SUM WGTS	247
MEAN	-0.13436	SUM	-33.1869
STD DEV	1.07211	VARIANCE	1.14943
SKEWNESS	0.0627827	KURTOSIS	-0.288167
USS	287.218	CSS	282.759
CV	-797.942	STD MEAN	0.0682169
T:MEAN=0	-1.9696	PROB>|T|	0.0500067
SGN RANK	-2104	PROB>|S|	0.0612869
NUM ^= 0	247		

QUANTILES(DEF=4)

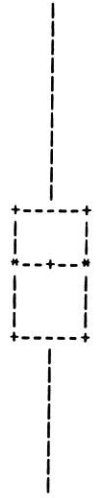
100% MAX	2.62643	99%	2.48672
75% Q3	0.593993	95%	1.71756
50% MED	-0.106814	90%	1.1788
25% Q1	-0.871228	10%	-1.62492
0% MIN	-2.478	5%	-2.0125
		1%	-2.29069
RANGE	5.10443		
Q3-Q1	1.46522		
MODE	-2.478		

EXTREMES

LOWEST	HIGHES
-2.478	2.2944
-2.30285	2.3488
-2.27751	2.4653
-2.20355	2.5064
-2.17049	2.6264

```
STEM LEAF                            #
  26 3                               1
  24 71                              2
  22 95                              2
  20 5685                            4
  18 26                              2
  16 713                             3
  14 27                              2
  12 0126782                         7
  10 0371466788                     10
   8 0355555812233899               15
   6 0022356811888                  13
   4 1123566880133599               16
   2 2444555601234566899            19
   0 122336789122 4899              16
  -0 99976322108876433311           20
  -2 9998777665210966543            19
  -4 99998866444337653222 0         21
  -6 98776542210                    11
  -8 533108730                       9
 -10 941106532                       9
 -12 887771998710                   12
 -14 8872888840                      9
 -16 849971                          6
 -18 830096                          6
 -20 762210520                       9
 -22 080                             3
 -24 8                               1
     ----+----+----+----+----+-
     MULTIPLY STEM.LEAF BY 10**-01
```

BOXPLOT



NORMAL PROBABILITY PLOT

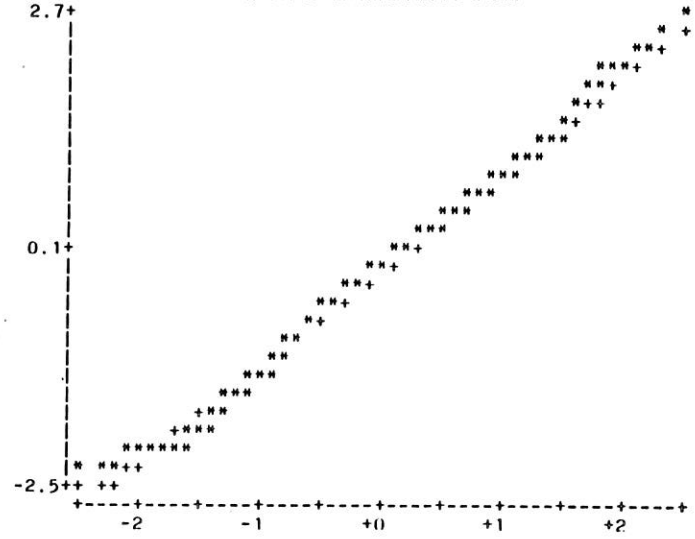


Fig. B.17—Further summaries of individual z statistics for ozone

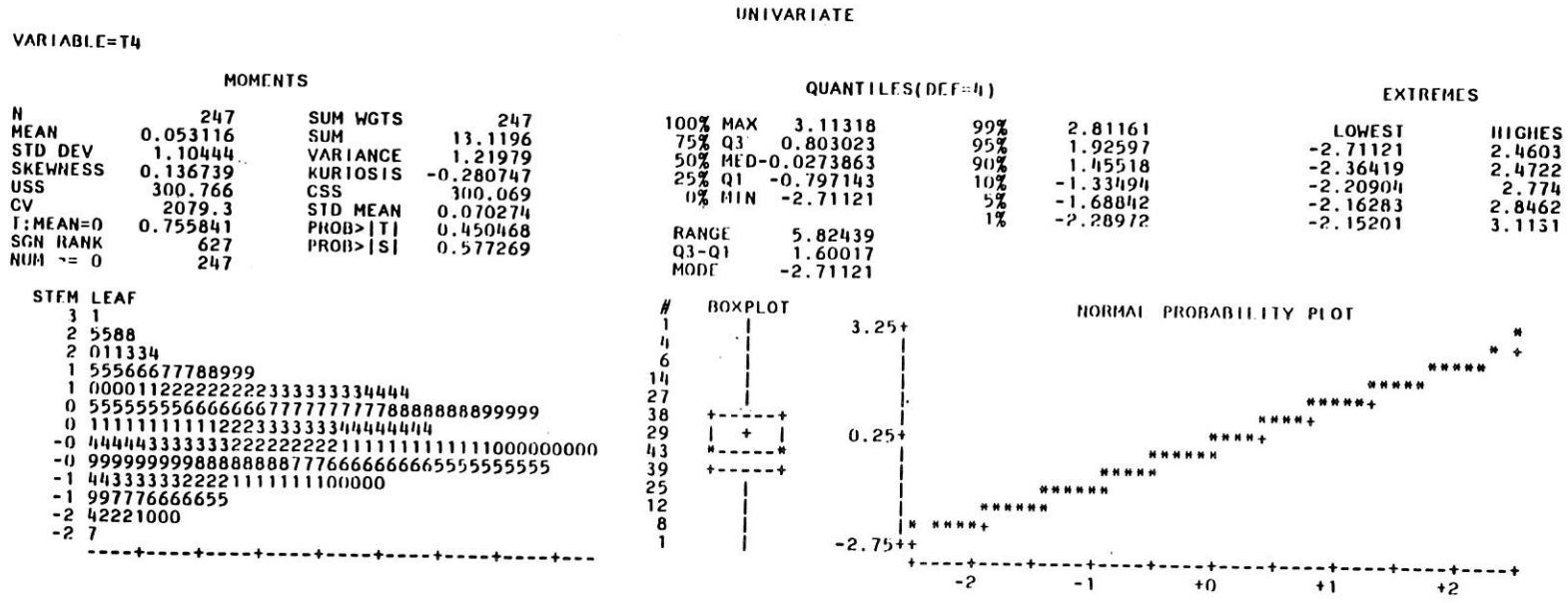


Fig. B.18—Further summaries of individual z statistics for NO_2

UNIVARIATE

VARIABLE=T5

MOMENTS

N	247	SUM WGTS	247
MEAN	-0.160018	SUM	-39.5245
STD DEV	1.0748	VARIANCE	1.15521
SKEWNESS	0.0326311	KURTOSIS	-0.877207
USS	290.505	CSS	284.181
CV	-671.676	STD MEAN	0.0683882
T:MEAN=0	-2.33985	PROB>|T|	0.0200918
SGN RANK	-2464	PROB>|S|	0.0284011
NUM ^= 0	247		

QUANTILES(DEF=4)

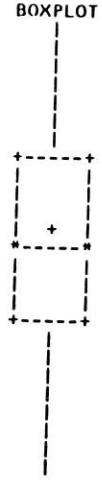
100% MAX	2.07517	99%	1.95882
75% Q3	0.687989	95%	1.5934
50% MED	-0.224522	90%	1.36819
25% Q1	-1.01386	10%	-1.52739
0% MIN	-2.6601	5%	-1.88183
		1%	-2.42912
RANGE	4.73528		
Q3-Q1	1.70185		
MODE	-2.6601		

EXTREMES

LOWEST	HIGHES
-2.6601	1.8083
-2.61631	1.8805
-2.22632	1.9372
-2.22472	1.9787
-2.15678	2.0751

```
STEM LEAF                            #
  20 8                               1
  18 1848                            4
  16 0015017                         7
  14 12348113358                    11
  12 0141269                         7
  10 22256712555699                 14
   8 11138013479                    11
   6 7899137779                     10
   4 03455677800234555566677        22
   2 03661255                        8
   0 22588012469                    11
  -0 753220988643322O               16
  -2 9975443398766221               16
  -4 997665540766644321             18
  -6 6522187330                     10
  -8 9887640099866430O              17
 -10 762105432221 10                14
 -12 9775322195422211O              17
 -14 5209887720                    10
 -16 865096331                      9
 -18 5431973                        7
 -20 664                            3
 -22 32                             2
 -24                                0
 -26 62                             2
     ----+----+----+----+--
     MULTIPLY STEM.LEAF BY 10**-01
```

BOXPLOT



NORMAL PROBABILITY PLOT

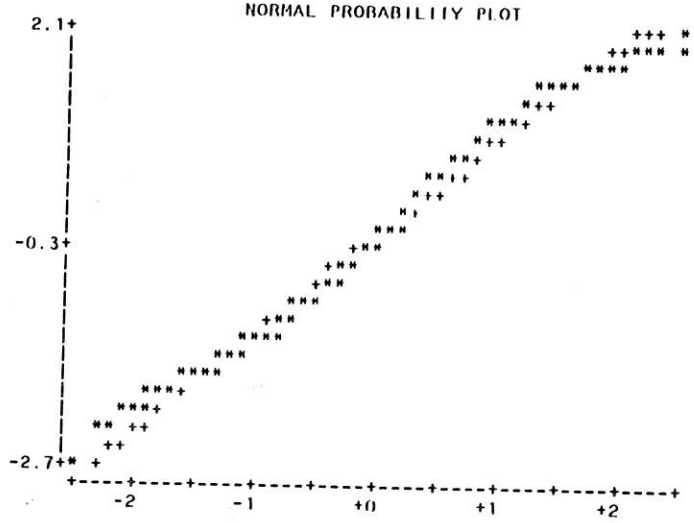


Fig. B.19—Further summaries of individual z statistics for CO

```
                                                            UNIVARIATE
VARIABLE=T6

                     MOMENTS                                              QUANTILES(DEF=4)                                      EXTREMES

N              247      SUM WGTS         247         100% MAX     2.8531      99%      2.20305          LOWEST      HIGHES
MEAN     -0.188916      SUM         -46.6622          75% Q3    0.552141      95%      1.74812         -2.7606      2.0776
STD DEV    1.08347      VARIANCE      1.1739          50% MED. -0.222738      90%      1.30632        -2.46919      2.1038
SKEWNESS  0.173106      KURTOSIS   -0.487398          25% Q1    -0.98112      10%     -1.64332        -2.28249      2.1736
USS        297.594      CSS          288.779           0% MIN    -2.7606       5%     -1.89795        -2.23894       2.230
CV        -573.518      STD MEAN   0.0689393                                 1%     -2.37957         -2.2216       2.853
T:MEAN=0  -2.74032      PROB>|T| 0.00658782      RANGE        5.6137
SGN RANK     -3141      PROB>|S| 0.00520593      Q3-Q1       1.53326
NUM ^= 0       247                                MODE        -2.7606

 STEM LEAF                                     #   BOXPLOT                     NORMAL PROBABILITY PLOT
    2 9                                        1      0         2.75+                                                     *
    2 001122                                   6      |             |                                                ***** *
    1 56677788889                             11      |             |                                            ****++
    1 0000111222223334444                     19      |             |                                        ****+
    0 55555555556666677777777777788889999     35   +-----+          |                                   ******
    0 11111111122222222233333333344444        32   |     |          |                               ****+
   -0 444444444333333222222211111000000000    36   *--+--*          |                           ****
   -0 9999888888888888877777777666665555555555555  43   +-----+     |                      ******
   -1 444444444433322222111111111100000       33      |             |                 ******
   -1 99888777777776666555                    20      |             |           *******
   -2 322110000                                9      |             |*  *****++
   -2 85                                       2      |       -2.75++ ++
      ----+----+----+----+----+----+----+---                        +----+----+----+----+----+----+----+----+----+
                                                                         -2        -1        +0        +1        +2
```

Fig. B.20—Further summaries of individual z statistics for minimum temperature

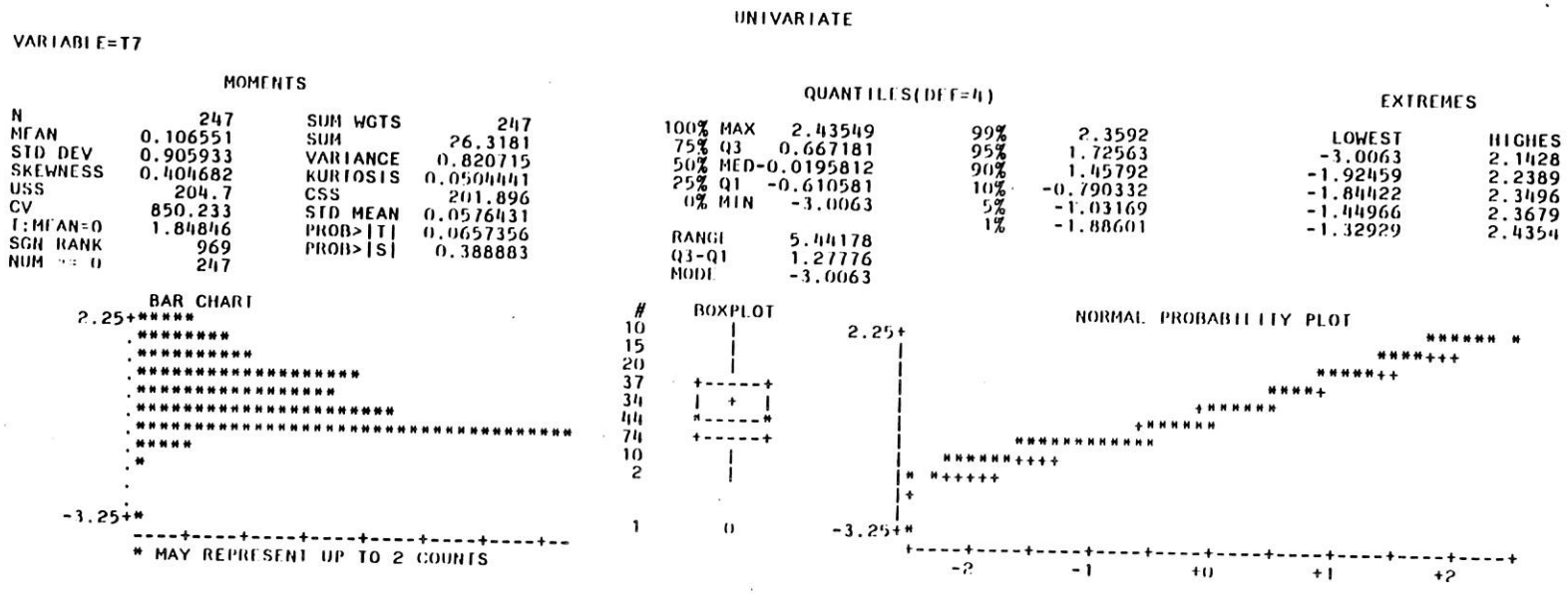


Fig. B.21—Further summaries of individual z statistics for precipitation

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