RM-76-23

EVALUATION OF HEALTH EFFECTS FROM SULFUR DIOXIDE EMMISSIONS FOR A REFERENCE COAL-FIRED POWER PLANT

W.A. Buehring

R.L. Dennis

A. Hölzl

September 1976

Preface

This report is one of a series describing a multi-disciplinary multinational IIASA research study on Management of Energy/Environment Systems. The primary objective of the research is the development of quantitative tools for energy and environment policy design and analysis--or, in a broader sense, the development of a coherent, realisitc approach to energy/environment management. Particular attention is being devoted to the design and use of these tools at the regional level. The outputs of this research program include concepts, applied methodologies, and case studies. During 1975, case studies were emphasized; they focused on three greatly differing regions, namely, the German Democratic Republic, the Rhone-Alpes region in southern France, and the state of Wisconsin in the USA. The IIASA research was conducted within a network of collaborating institutions composed of the Institut fuer Energetik, Leipzig; the Institut Economique et Juridique de l'Energie, Grenoble; and the University of Wisconsin, Madison.

This memorandum describes a model that quantifies certain health effects associated with emissions from coal-fired power plants.

Publications on the management of energy/environment systems are listed in Appendix C at the end of this report.

Wesley K. Foell

Evaluation of Health Effects from Sulfur Dioxide Emissions for a Reference Coal-Fired Power Plant

Abstract

Health effects from coal-fired power plants are causing growing concern. Interest is stimulated by delays in the use of nuclear power and the possibility of greater use of coal. A model to evaluate health effects has been developed, based on the concept of a reference 1,000 MW plant. This model has particular relevance to studies of alternative futures and analysis for long range planning. The model consists of two parts, dealing with health impact and dispersion to dosage. The health submodel is based on a study by Finklea et al. at the USEPA, and the dispersion submodel is based on results of detailed dispersion modelling and monitoring for a typical power plant. The human health impacts are parameterized in terms of SO_2 emissions, population and site characteristics, and background SO_2 concentration. Although these quantified impacts are only a partial indicator of the total air pollution impact, the results show that these impacts may be significant and that they should be considered in the analysis of power plant impacts.

Acknowledgement

The authors are indebted to several individuals for their aid in carrying out this research. We would like to especially thank Jim Bigelow (IIASA Biomedical Project) for providing valuable assistance in the mathematical development of the daily concentration distribution and Jackie Buehring for writing the program to evaluate premature mortality. We would also like to thank M. Benarie of IRCHA in France for his helpful comments. A special note of gratitude goes to J.F. Finklea for allowing us to quote the dose-response function he and his colleagures developed.

Table of Contents

List of Tables

Table

List of Figures

Figure

I. Introduction

Public health effects from coal-fired power plants are a primary concern as alternatives for future electrical generation are examined. Interest has been stimulated by delays in the use of nuclear power and the possibility of greater use of coal. The need for quantitative information on health effects on air pollution is evident as decisions that will affect air quality for some time to come are regularly being made.

The model presented in this paper has been developed to estimate certain human health effects that can be associated with the emission of sulfur dioxide from a coal- or oil-fired power plant that may consist of several units at a single site. The objective of this model's development is to provide a flexible tool for long-range environmental policy design and evaluation and for analysis of alternative futures; it was not designed to answer questions of a site specific nature. The model consists of two parts: one concerning the calculation of health impact and the other concerning the calculation of the ground-level concentration due to a given level of emissions.

Estimates of human health impact due to air pollution have been difficult to obtain and contain a great deal of uncertainty. Nevertheless, a recent study of health effects by Finklea, et al. at the USEPA [1] has provided quantitative relationships (dose-response functions) between increased levels of acid sulfate exposure and five categories of health effects. The Finklea study has served as the foundation for the health submodel, described in Section II. It must be emphasized that dose-response relationship are not certain and, furthermore, that the pollutant or pollutants responsible for the health effects are also uncertain.

The methodology and model that relates power plant emissions to ground level concentrations is based on previous studies with a Gaussian plume model and is presented in Section III. The

quantified health impacts as a function of SO_2 emission, background concentration, and population exposed are given in Section IV. In the final section the conclusions of the study are outlined with suggestions for further research.

II. Health Submodel

A difficulty in quantifying the health effects of air pollution is identification of the causal agent or agents. Statistical increases in mortality and morbidity have been shown to correspond to increases in indices of air pollution [2,3,4]. Although such studies indicate only an association between the index of pollution and health effects, they are convincing evidence that air pollution affects human health. Typically, studies have focused on the impact of short-term exposure to high levels of air pollution because these are the easiest effects to extract from the epidemiological data. Although one would like to know the acute as well as the chronic effects caused by both long- and short-term exposure to specific air pollutants, or combinations of air pollutants, in this report the basic epidemiological data are derived from previous studies and, hence, most of the quantified health effects are associated with short-term exposure to a single pollutant.

The dose-response relationships used in our model are based entirely on the Finklea study [1]. These relationships make it possible to estimate some of the health effects that correspond with various levels of sulfur dioxide concentration and associated exposures to acid-sulfate aerosols. Three categories of health effects are related to short-term (daily) pollutant exposure, and two others are related to long-term (several years) exposures to relatively high levels of pollution. The Finklea relationships are based on two primary assumptions,

- acid-sulfate aerosols, not SO_2, are the cause of the health effects, and
- the important averaging time for short-term exposures is one day.

The dose-response relationships remain highly uncertain, but evidence has been compiled that indicates increased emission of SO_2 can lead to

- premature mortality from acute exposure,
- aggravation of heart and lung disease in people over age 65,
- aggravation of asthma,
- excess acute lower respiratory disease in children aged 0 to 13, and
- excess risk of chronic respiratory disease symptoms in smoking and non-smoking adults.

Here SO_2 is considered to be an indicator of the impact rather than the causal agent, which is believed to be acid-sulfate aerosols. The relationship between SO_2 and sulfates is discussed in the next section, and this is followed by the dose-response functions used in our model.

II. 1 Relationships Between SO_2 and Sulfates

The desirable form of the dose-response relationship for power plant modelling is a function that relates excess adverse health effects to particular ground-level SO_2 concentrations. This is for several reasons: emissions of SO_2 from power plants and dispersion to ground-level concentrations can be well characterized; the transformation of SO_2 to acid-sulfate aerosol can be quite sensitive to the region being modelled due to catalytic effects of other aerosols on the transformation process and due to the intrusion of sulfates from other areas; and there are still uncertainties in the exact SO_2-sulfate conversion rate constants. Finklea has used suspended sulfates as a proxy for acid-sulfate aerosols and, based on studies of U.S. cities, has listed two possible conversions between 24-hour levels of suspended sulfates and SO_2 concentration. The equations are

$$\text{Suspended Sulfates } (\mu g/m^3) = 9 + 0.03 \cdot SO_2 (\mu g/m^3) \quad , \qquad (1)$$

$$\text{Suspended Sulfates } (\mu g/m^3) = 9 + 0.05 \cdot SO_2 (\mu g/m^3) \quad . \qquad (2)$$

The first equation is based on a study of Nashville, Tennessee, and is thought to be applicable when intruding sulfates from other regions are not a problem. The second equation is based on National Air Sampling Network data for several inland cities. The Nashville study is more representative for Wisconsin [5], which was the region used for the initial application of our model. Therefore, Equation 1 is used to relate both the annual and the 24-hour average SO_2 concentrations to the corresponding sulfate levels throughout the remainder of this report [6]. This equation corresponds to a conversion rate of SO_2 to acid sulfates of 0.13% per hour over a diurnal period. This is midway in the accepted range of 0.02 to 1.2% per hour for the rate of conversion [7].

The uncertainty in the relationship between SO_2 and sulfates directly affects the quantified health effects presented in later sections. If Equation 2 were used instead, the estimated health effects would be increased significantly.

II.2 Dose-Response Functions

Five dose-response functions linking acid-sulfate aerosol exposures to selected adverse health effects are given by Finklea and are reproduced here in Table 1. The main features are that below a threshold concentration, no health impacts are predicted (a point that has been debated [2], but the evidence presented by Finklea supports this conclusion), and that above the threshold the response increases linearly with increasing concentration. It should be noted that for all cities studied, particulate matter (P.M) was also present, so these relationships include synergistic interactions between P.M. and acid-sulfates. The reference power plant to which the dose-response functions are being applied emits both SO_2 and P.M., so a relationship that includes SO_2-P.M. interactions is more desirable than one which is for sulfates in the absence of P.M. The functions in Table 1 are not applicable in regions with large emissions of catalytically active metals or in regions with photochemical smog. In both cases, catalytically active metals (e.g. iron oxide) and smog, the atmospheric sulfate formation would be greatly enhanced [8]. In fact the system

of hydrocarbon/NO_x/SO_2 has been shown to be highly synergistic. Thus Equations 1 and 2 would greatly underestimate the suspended sulfate formation in such regions.

For use in our model the dose-response functions in Table 1 have been rewritten in terms of SO_2 concentration by applying Equation 1. In addition the population at risk has been specified for each adverse health effect in Table 1. The individual relationships are listed in the following subsections.

Table 1
Best Judgment Dose-Response Functions
(Source: Finklea [1])

Adverse Health Effect*	Threshold Concentration of Suspended Sulfates & Exposure Duration	Slope	Intercept
Increased daily mortality (based on 4 studies) (acute episodes)	25 $\mu g/m^3$ for 24 hours or longer	0.00252	-0.0631
Aggravation of Heart and Lung Disease in Elderly Patients (2 studies)	9$\mu g/m^3$ for 24 hours or longer	0.0141	-0.127
Aggravation of Asthma (4 studies)	6-10 $\mu g/m^3$ for 24 hours or longer	0.0335	-0.201
Excess Acute Lower Respiratory Disease in Children (4 studies)	13 $\mu g/m^3$ for several years	0.0769	-1.000
Excess Risk for Chronic Bronchitis (6 studies)			
Non-Smokers	10 $\mu g/m^3$ for up to 10 yr.	0.1340	-1.42
Cigarette Smokers	15 $\mu g/m^3$ for up to 10 yr.	0.0738	-1.14

*The excess adverse effects are given in terms of the fractional increase over the normal rate. Thus, a 100 $\mu g/m^3$ sulfate concentration for one day is expected to increase mortality on that day by 18.9 percent.

1. Premature Mortality

Fatalities from exposure to high levels of SO_2 are associated with 24-hour SO_2 (sulfate) concentrations. It is important to recognize that this relationship does not include fatalities from long-term low-level exposure. The fraction of total expected fatalities on day i that are premature is given by

$$F_1(i) = -0.0404 + 0.000076\, S_{24}(i) \qquad (3)$$

where

$F_1(i)$ = fraction of total expected deaths on day i that are premature, and

$S_{24}(i)$ = 24-hour SO_2 concentration in $\mu g/m^3$ on day i .

The minimum daily concentration to produce any effect is about 530 $\mu g/m^3$, which is a very high concentration. Therefore, on most days no mortality from SO_2 is predicted. The premature fatalities from SO_2 that occur each year can be determined by calculating the $F_1(i)$ for each value of SO_2 concentration above the threshold and accumulating as shown in Equation 4.

$$E_1 = (P)(R)(1/365) \sum_{i=1}^{365} F_1(i), \quad S_{24}(i) > 530\ \mu g/m^3 \qquad (4)$$

where

E_1 = premature fatalities per year from SO_2 exposure,

P = population exposed, and

R = death rate in deaths per person per year (about 9.3/1000 per year for Wisconsin in 1972 [9]).

The method used to determine the daily SO_2 concentration distribution, which is required to make use of Equation 4, is given in section III.2.

2. Aggravation of Heart and Lung Disease

The number of excess days of aggravation in people over 65 with pre-existing heart or lung disorders is assumed to be directly proportional to SO_2 concentration, with no SO_2 threshold, i.e.

$$F_2(i) = 0.000423\, S_{24}(i) \tag{5}$$

where

$F_2(i)$ = fractional excess days of aggravation on day i,* and

$S_{24}(i)$ = 24-hour SO_2 concentration in $\mu g/m^3$ on day i.

There is no threshold, so any additional SO_2 exposure will cause some additional effects. These elderly people typically suffer one day of aggravation out of every five days without any SO_2 exposure**. The population at risk is only about 2.7 percent of the total U.S. population. Therefore, the excess days of aggravation per year in the exposed population can be determined by calculating $F_2(i)$ over the 365 day distribution and accumulating,

$$E_2 = (0.2)(0.027)(P) \sum_{i=1}^{365} F_2(i) \quad , \tag{6}$$

or in terms of the 24-hour SO_2 concentration,

$$E_2 = (2.28 \times 10^{-6})(P) \sum_{i=1}^{365} S_{24}(i) \quad , \tag{7}$$

*F_2 is the number of days of aggravation associated with the SO_2 exposure divided by the number of days of aggravation that occur without SO_2 exposure. "Fractional excess" is used throughout this report as the ratio of the effects associated with SO_2 to the normal incidence rate without SO_2 exposure.

**The populations at risk and normal rates of incidence for all four categories of nonfatal health effects are based on average U.S. data supplied in Reference 1.

where

E_2 = excess days of aggravation from SO_2 exposure per year, and

$(0.027)(P)$ = total population over 65 with heart and lung disease.

Since there is no threshold, excess days of aggravation from SO_2 can be determined directly from the annual arithmentic mean, i.e.

$$E_2 = (8.34 \times 10^{-4})\ (P)(S_{365}) \qquad (8)$$

where

S_{365} = annual arithmentic mean SO_2 concentration in $\mu g/m^3$.

Equations 6 and 8 indicate that it takes an average exposure during the year (annual average exposure) of 32 $\mu g/m^3$ to cause one additional day of aggravation per year for a person over 65 with chronic heart or lung disease.

3. Aggravation of Asthma

The excess asthma attacks from SO_2 can also be related to 24-hour SO_2 concentrations,

$$F_3(i) = 0.00101\ S_{24}(i) \qquad (9)$$

where

$F_3(i)$ = fractional excess asthma attacks on day i, and

$S_{24}(i)$ = 24-hour SO_2 concentration in $\mu g/m^3$ on day i.

Since there is no threshold, any additional SO_2 exposure will increase the number of expected asthma attacks. The average number of attacks per year for an asthmatic without any SO_2 exposure is about seven, and about three percent of the total U.S. population are asthmatics. Therefore, the excess asthma attacks per year are given by

$$E_3 = (7/365)(0.03)(P) \sum_{i=1}^{365} F_3(i) \quad , \qquad (10)$$

or in terms of 24-hour SO_2 concentration,

$$E_3 = (5.81 \times 10^{-7})(P) \sum_{i=1}^{365} S_{24}(i) \quad , \qquad (11)$$

where

E_3 = excess asthma attacks per year from SO_2 exposure, and

$(0.03)(P)$ = total number of people with asthma in population P.

Since there is no threshold for effects, the excess asthma attacks can also be computed directly from the annual average concentration, i.e.

$$E_3 = (2.12 \times 10^{-4})(P)\ S_{365} \qquad (12)$$

where

S_{365} = annual arithmetic mean SO_2 concentration in $\mu g/m^3$.

These equations indicte that an annual average SO_2 concentration of about 140 $\mu g/m^3$ would be expected to cause one additional attack per asthmatic per year.

4. Excess Acute Lower Respiratory Disease in Children

The correlation for excess acute lower respiratory disease in children is given in terms of annual arithmetic mean SO_2 concentration,

$$F_4 = -0.308 + 0.00231\ S_{365} \qquad (13)$$

where

F_4 = fractional excess acute lower respiratory disease in children, and

S_{365} = annual arithmetic mean SO_2 concentration in $\mu g/m^3$.

Since the indicated threshold is 133 $\mu g/m^3$ for the annual average SO_2 concentration, this health impact will rarely occur in connection with power generation. The population at risk is all

children aged 0 to 13, or about 26 percent of the total U.S. population. The normal incidence rate is about six cases per 100 children per year. The total excess respiratory disease in children from SO_2 is therefore

$$E_4 = (3.60 \times 10^{-5})(P)(S_{365} - 133), \quad S_{365} \geq 133 \ \mu g/m^3 \qquad (14)$$

where

E_4 = excess acute lower respiratory disease in children per year from SO_2 exposure, and

$(0.26)(P)$ = total number of children aged 0 to 13 in population P.

5. Excess Risk for Chronic Respiratory Disease

The risk of chronic respiratory disease in adults aged 21 and over is also related to annual arithmetic mean SO_2 concentration. Separate relationships are given for non-smoking adults and for smoking adults,

$$F_5 = -0.214 + 0.00402\ S_{365}\ , \text{ and} \qquad (15)$$

$$F_6 = -0.476 + 0.00221\ S_{365}\ . \qquad (16)$$

where

F_5 = excess risk of chronic respiratory disease symptoms among non-smoking adults,

F_6 = excess risk of chronic respiratory disease among smoking adults, and

S_{365} = annual arithmetic mean for SO_2 concentration in $\mu g/m^3$.

The threshold for effects on non-smokers is about 53 $\mu g/m^3$ and for smokers is about 215 $\mu g/m^3$. The populations at risk in the U.S. are about 38 percent of the total for non-smoking adults and 23 percent of the total for smoking adults*. Without exposure

*Non-smokers are 62 percent of the U.S. population aged 21 and over [1], and 61 percent of the total U.S. population were 21 or older in 1970 [9].

to SO_2 about two percent of the non-smoking adults suffer from chronic respiratory disease symptoms; the corresponding figure for smoking adults is ten percent. The extra non-smokers and smokers exhibiting these symptoms because of SO_2 exposure above the respective thresholds are therefore

$$E_5 = (3.06 \times 10^{-5})(P)(S_{365} - 53.2), \quad S_{365} > 53.2 \ \mu g/m^3, \text{ and} \quad (17)$$

$$E_6 = (5.08 \times 10^{-5})(P)(S_{365} - 215), \quad S_{365} > 215 \ \mu g/m^3 , \quad (18)$$

where

E_5 = extra non-smokers showing chronic respiratory disease symptoms, and

E_6 = extra smokers showing chronic respiratory disease symptoms.

II.3 Limitations of the Health Submodel

The health effects parameterized in the previous section have been associated with daily average SO_2 concentration (mortality) or annual average SO_2 concentration (nonfatal health effects). The resulting calculated health impacts should be considered as only a partial indicator of the total health impact. More research is needed particularly on the effects of long-term exposures to low levels of pollutants. The quantified relationships themselves are not certain, and several important limitations [1] need to be considered:

- the chemical behavior over time of sulfur oxides and sulfates, mixed with other pollutants, is not well understood,
- measurements of suspended sulfates have been used as representative for acid-sulfate aerosols,
- historical data on emissions and air quality are limited,
- epidemiological studies, which are difficult to perform, are in their first stages and still have many uncertainties in their results,
- the total impact of air pollution includes not only the unquantified human health effects but also the effects on vegetation, animals, materials, climate, visibility, property values, etc.

The relationships used in our submodel are current best judgments. Finklea has also listed some alternative dose-response functions, based on least squares fits of the data, for the same health impacts (Appendix B). Analyses such as the Finklea study, crude as they may be at this time, are needed to provide better understanding of the implications for important decisions and to stimulate further research in related areas.

III. Emission and Dosage

The previous section provides certain quantified relationships between SO_2 exposure and health effects. It remains in this section to show how exposure has been related to emissions from a coal-fired power plant.

III.1 Annual Average Concentration

The annual average SO_2 concentrations within 80 kilometers of a power plant have been calculated using a Gaussian plume model [10]. The results depend strongly on whether the plant is in a rural or urban setting because the air flow over a city is typically more turbulent, thereby changing the dispersion coefficients. Therefore, we have treated urban plants separately from rural plants.

The reference plant calculations were based on the Moses and Carson plume rise formula [11] using Madison, Wisconsin, meteorological data for seasonal frequency of occurrence for wind speed, wind direction, stability category, and mixing height. The arithmetic average of the calculated seasonal concentration provides the annual average concentration of pollutants for the power plant. The particular characteristics for the reference plant were

- 1,000 MWe unit operating at 70 percent annual capacity factor, with equal quantities of generation in each of the four seasons,
- unit heat rate of 9,500 BTU per kWh (net efficiency of about 36 percent),
- urban or rural location (two separate calculations),
- emissions of SO_2 at a rate of 1.2 pounds per million BTU heat input (USEPA emission standard for coal power plants),

- stack height of 152 meters,
- stack diameter of 5 meters,
- stack gas exit temperature of 148.7° C,
- a volume flow rate of 334 m^3 per second.

These assumptions lead to a total SO_2 emission of approximately 35,000 short tons per year for the reference 1,000 MWe unit. It should be noted that the reference plant is not a typical coal-fired plant because most plants do not meet the USEPA emission standard. The average sulfur content of coal used by U.S. electrical utilities in 1970 was 2.5 percent [12]. If this average coal is assumed to have a heating value of 12,000 BTU per pound and is used in a power plant with characteristics similar to the reference plant, the resulting SO_2 emissions are over three times greater than those of the reference plant.

The Gaussian plume model results have been simplified for further use in this model by dividing the area around the plant into quadrants, with a single quadrant representing the area that has the highest annual concentration and the other three quadrants representing the remaining area that has lower annual average concentrations. This approximation is not necessary but reduces data handling and other calculations that depend on the annual average concentrations. These simplified results for the reference plant emissions are given in Table 2 for the urban and rural sites. The increased turbulence associated with the urban site results in higher ground-level concentration near the plant and lower concentration at large distances. The annual average SO_2 concentration at any point around a new plant with different emissions is assumed to be proportional to the concentration for the appropriate reference plant, i.e.

$$S_{365}(\vec{r},E,u) = \frac{E}{E_o} S_{365}(\vec{r},E_o,u) \qquad (19)$$

where

$S_{365}(\vec{r},E,u)$ = annual average SO_2 concentration ($\mu g/m^3$) at point $\vec{r}$ that results from power plant emission, E, at site type u,

Table 2

Annual Arithmetic Average SO_2 Concentrations
At Ground Level for a 1000 MWe Reference Coal Plant
($\mu g/m^3$)

Distance From Plant (km)	Urban Site		Rural Site	
	High Quadrant	Other Three Quadrants	High Quadrant	Other Three Quadrants
5	8.5	3.8	1.4	0.77
10	4.6	2.3	2.4	1.1
15	3.0	1.6	2.0	1.0
20	2.2	1.2	1.8	0.82
25	1.8	1.0	1.6	0.70
30	1.5	0.9	1.4	0.60
35	1.2	0.8	1.2	0.55
40	1.1	0.7	1.1	0.51
45	1.0	0.6	1.0	0.48
50	0.9	0.5	0.95	0.45
55	0.8	0.4	0.91	0.42
60	0.7	0.4	0.90	0.39
65	0.6	0.4	0.88	0.36
70	0.6	0.3	0.86	0.35
75	0.5	0.3	0.85	0.33
80	0.5	0.3	0.85	0.32

The reference plant meets USEPA emission standards and operates at 70% capacity factor. Total SO_2 emission is 35,000 tons per year.

$\vec{r}$ = particular ground-level point,

E = annual SO_2 emission (tons/year),

E_o = annual SC_2 emission for reference plant (35,000 tons/year),

u = site type (urban or rural), and

$S_{365}(\vec{r},E_o,u)$ = annual average SO_2 concentration ($\mu g/m^3$) at point $\vec{r}$ that results from reference plant emission, E_o, at site type u (Table 2).

Thus, a plant emitting twice as much SO_2 would be expected to cause ground-level concentrations two times larger than those in Table 2.

The results in Table 2 can be coupled with an assumed population distribution and the relationships of the health submodel for nonfatal effects to yield the quantified nonfatal health impacts for the reference plant. The dose-response function for premature fatalities requires a 24-hour concentration distribution; the methodology for determining doses that can be used in the mortality dose-response function is outlined in the next section.

III.2 Daily Concentration Distribution

Since the mortality dose-response function (Equation 4) requires 24-hour average concentrations, it was necessary to employ a method for linking the annual average concentrations with a distribution of daily average concentrations. Pollutant concentrations that result from power plant emissions are assumed to be log-normally distributed for a daily averaging time [13,1]. The standard geometric deviation (S), arithmetic mean (A), and geometric mean (M) for a log-normal distribution are therefore related according to the equation

$$M = A \exp[-\tfrac{1}{2}(\ell n S)^2] \quad . \tag{20}$$

The annual geometric mean can be determined for each corresponding combination of A and S. The annual geometric mean is a key parameter in the relationship used to obtain daily concentrations.

The annual arithmetic mean is known from the previous calculations (Table 2); the standard geometric deviation for a daily

averaging time is not known. However, we have developed an empirical relationship for S, the standard geometric deviation, as a function of distance from the plant and angle around the plant, based on actual Wisconsin data [14,15].

In the region near the point source where there are high ground-level peaks in the annual average concentrations, S is relatively large -- approximately five for SO_2 concentration based on a 24-hour averaging time*. At comparatively large distances from the plant, e.g. 50 to 80 kilometers, the plume is no longer distinguishable above the background, and S is approximately 1.75 for SO_2. For the intermediate and lower level peaks in the ground-level concentration S has an intermediate value of approximately three. Beyond the ground-level peaks around the plant the concentration decreases approximately as an exponential, leading one to expect that S will also decrease nearly as an exponential to the value 1.75. We have assumed the standard geometric deviation varies as a function of distance from the power plant as shown in Figure 1. However, the actual location and extent of the regions of high concentration gradients depends on the meteorology and the surface roughness (whether the plant is in a rural or urban setting).

The daily distribution for average concentrations can now be determined. The derivation of the relationship is given in Appendix A. Only the results are outlined here.

The probability (normalized frequency) of occurrence can be plotted against concentration in a way such that the concentration that is exceeded with probability p can be read directly off the chart. The probability scale can be divided into 365 equal parts such that the concentration that corresponds with p of 1/365 is the concentration that is exceeded only one day per year, the concentration that corresponds with p of 2/365 is exceeded two days per year, etc. For the calculation of health impact we need to associate a concentration with each day, rather than the concentration exceeded on that day. Therefore we have defined the

---

*A similar observation of this behavior of the standard geometric deviation for the air pollution concentration frequency distribution near a point source with a tall stack is also reported in Knox and Lange [16].

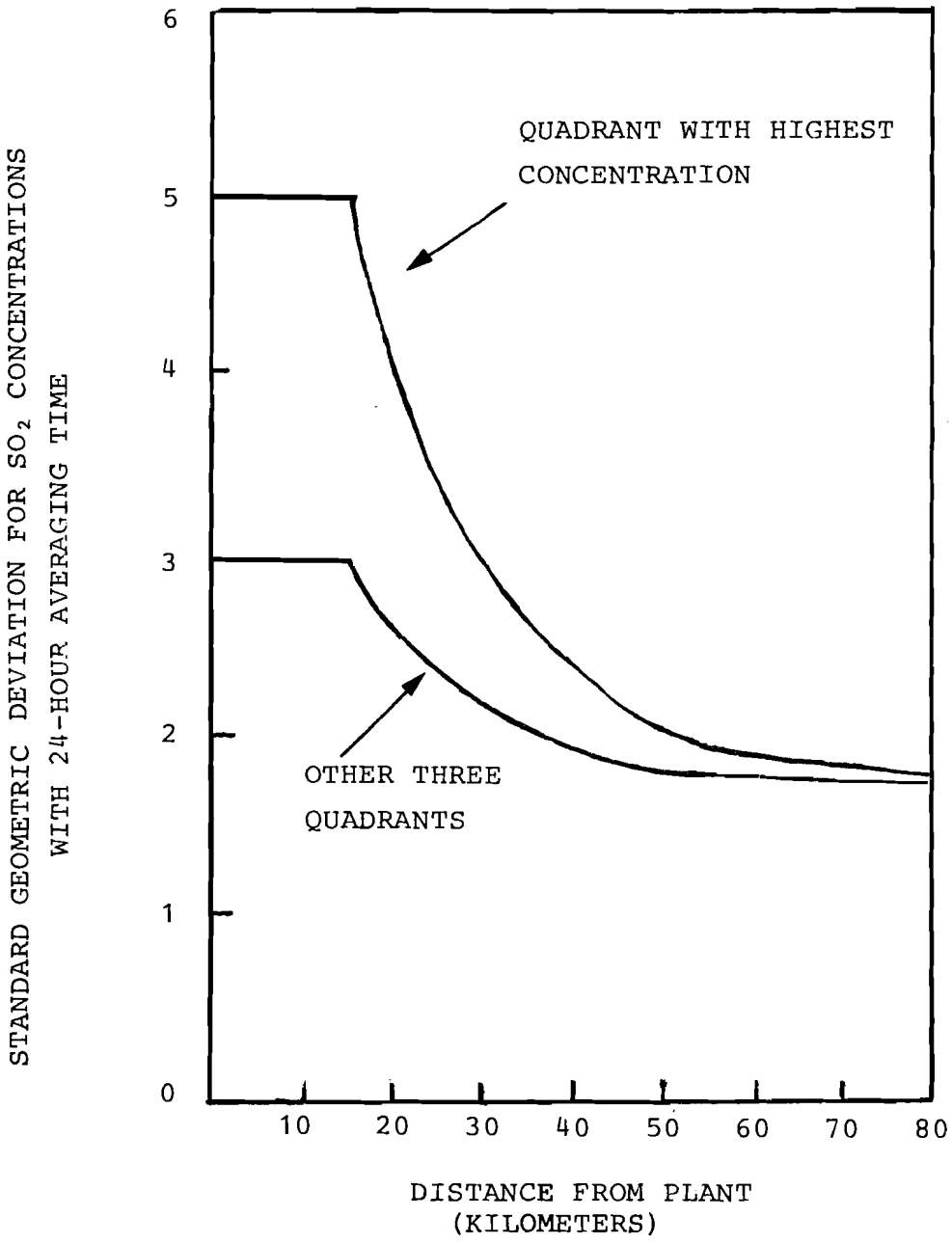

Figure 1: Assumed SO_2 Standard Geometric Deviation as a Function of Distance from the Reference Plant.

probability that gives a more representative concentration for the day with the highest concentration as 0.5/365*. Thus the cumulative probability distribution is given by

$$p_i = \frac{i - \frac{1}{2}}{365} \qquad i = 1,2,\ldots, 365 \quad . \tag{21}$$

The corresponding daily concentrations are given by

$$C_i = MS^{\Phi^{-1}(1-p_i)} \qquad i = 1,2,\ldots, 365 \tag{22}$$

where

C_i = average SO_2 concentration for day i,

M = annual geometric mean SO_2 concentration,

S = standard geometric deviation for a daily averaging time, and

Φ^{-1} = inverse of the normal cumulative function ($\Phi^{-1}(X)$ is the argument A that gives $\Phi(A) = X$).

Thus, C_1 is the highest daily average concentration, C_{183} is the geometric mean concentration ($p_{183} = 0.5$), and C_{365} is the lowest daily average concentration.

In this manner the daily distribution can be determined whenever the annual arithmetic average and standard geometric deviation are known. Since the standard deviation is raised to a power that becomes as large as 3.0 for the day with highest concentration, the functional relationship between standard geometric deviation and distance from the plant (Figure 1) has a significant effect on the estimated fatalities.

---

*This concentration is expected to be exceeded one day every two years, while the concentration corresponding to a probability of 1/365 is expected to be exceeded one day every year. Use of 1/365 would be taking the lower bound for the concentration, and hence the lower bound for premature mortality, on the day with highest annual average concentration.

III.3 Population Distribution

In order to minimize additional complexities, only three population alternatives have been examined. The assumed population within 80 kilometers (50 miles) for the three siting alternatives are

	Population within 80 km	Population Density (people/km^2)
Urban	6,300,000	313
Average	2,250,000	112
Rural	300,000	15

In all cases population density is assumed to be independent of distance or direction from the plant. In addition the characteristics of the population are assumed to be independent of location for a given siting alternative, i.e. the age distribution is not a function of distance or direction from the plant. The urban population has been associated with the urban reference plant concentration in Table 2, and the rural site corresponds to the rural reference plant in Table 2. The health effects for the "average" site have been determined by averaging the per capita health effects for the urban and rural sites and multiplying by the population shown above. Since all calculations are on a per capita basis, the estimated health effects for a particular site can be modified for different populations within 80 kilometers simply by multiplying by the population ratio. If a population distribution with density or population characteristics, such as age, varying with distance or direction is desired, additional calculations are necessary.

III.4 Background Exposures

Since several of the dose-response functions have thresholds, the background, defined here as the SO_2 concentrations from all other sources, has an effect on the predicted health impacts. Therefore, the results presented later in this report are given

for different annual average background concentrations. In the case of premature fatalities, a daily background distribution was calculated from the annual average background concentration by assuming a log-normal distribution with a standard geometric deviation of 1.75, a typical value in rural areas away from large point sources. The day with the highest expected background was assumed to occur the same day as the highest daily average concentration from the power plant. This will not be strictly true at every point because weather conditions do not affect the daily background concentration distribution in exactly the same way as the power plant concentration distribution. However, it was felt that this was a reasonable approximation of the average effect within an 80 kilometer radius of the power plant.

The threshold phenomena raises the issue of whether background concentrations or concentrations associated with power plant emissions are responsible for the health effects. The share of responsibility for the health effects depends on which contributor uses the concentration below the threshold. The approach used in this report is to list the total quantified health impact corresponding to the indicated combination of background and power plant concentrations. For those health impacts that have no SO_2 threshold, namely asthma and aggravation of heart and lung disease, the effects of background and the power plant are separable, and only the power plant impacts have been determined. Only when annual average SO_2 background concentrations become greater than 50 $\mu g/m^3$ is the background by itself sufficient to produce any of the health effects with SO_2 thresholds, as determined from the best judgment dose-response functions.

III.5 Summary of Key Assumptions for the Dosage Methodology

The dose estimates for the reference plants have relied on particular data for Wisconsin but are thought to be reasonably typical of other regions. The most important assumptions employed in these calculations are

- Gaussian plume model for calculating annual average concentrations,

- log-normal distribution for daily concentration due to power plant emissions,
- standard geometric deviation as a function of distance from the plant as shown in Figure 1,
- homogeneous population per unit area (age, asthmatics, etc.), and
- log-normal distribution for daily background concentrations.

IV. Quantified Impacts for Coal-Fired Power Plants

Quantified human health impacts that are associated with SO_2 emissions from a coal-fired power plant have been parameterized as a function of SO_2 emissions, background concentrations, and type of site (urban, rural, or average). Results are presented for each of the five health impacts in the following subsections.

Premature Fatalities

The premature fatalities were calculated for a particular site, population, quantity of SO_2 release, and background concentration with the aid of a computer program. After calculating many such individual cases, the results were plotted as shown in Figures 2,3, and 4. The premature fatalities are plotted versus the ratio of SO_2 emitted to the emission for the reference plant. If 350,000 tons of SO_2 were emitted, the ratio would be 10. If two 1,000 MWe plants, at the same site with the same efficiency and capacity factor as the reference plant, used coal with 11,000 BTU per pound and five percent sulfur with no SO_2 controls, the SO_2 emission would be about 15 times greater than the reference plant. The results shown in Figures 2, 3, and 4 indicate the number of premature fatalities per year associated with SO_2 emission is relatively small for low emissions and low background concentrations of 0 to 20 $\mu g/m^3$, which are typical for most areas in Wisconsin.

Tables 3 and 4 show the results of the calculations over a wider range of backgrounds and emissions. The higher backgrounds are more typical of the heavily industrialized areas in the U.S. The expected premature fatalities for the urban site are given in Table 3 and for the rural site in Table 4. The fatalities for

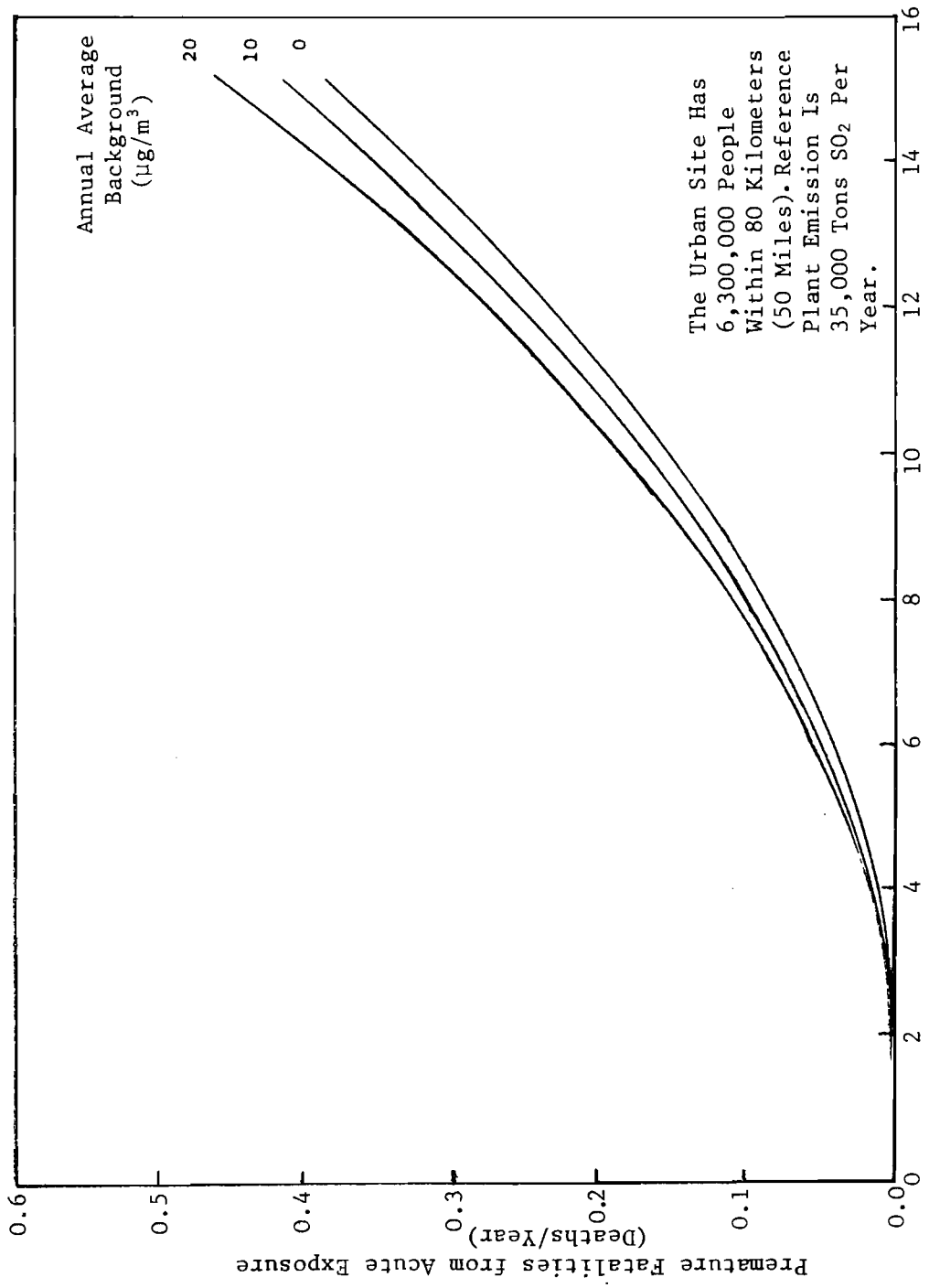

Figure 2: Premature Fatalities From Acute SO_2 Exposure for the Urban Site

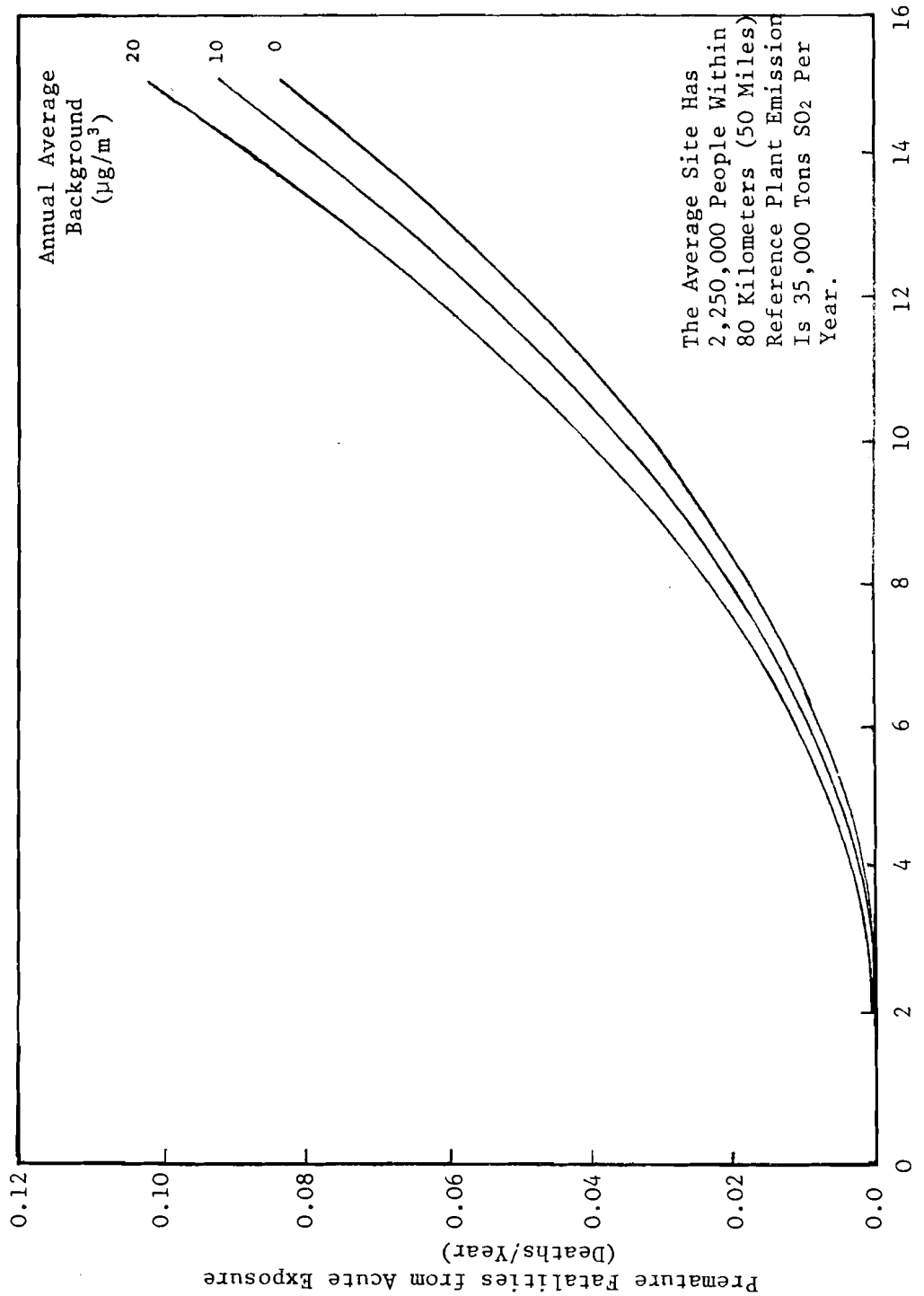

Figure 3: Premature Fatalities From Acute SO_2 Exposure for the Average Site

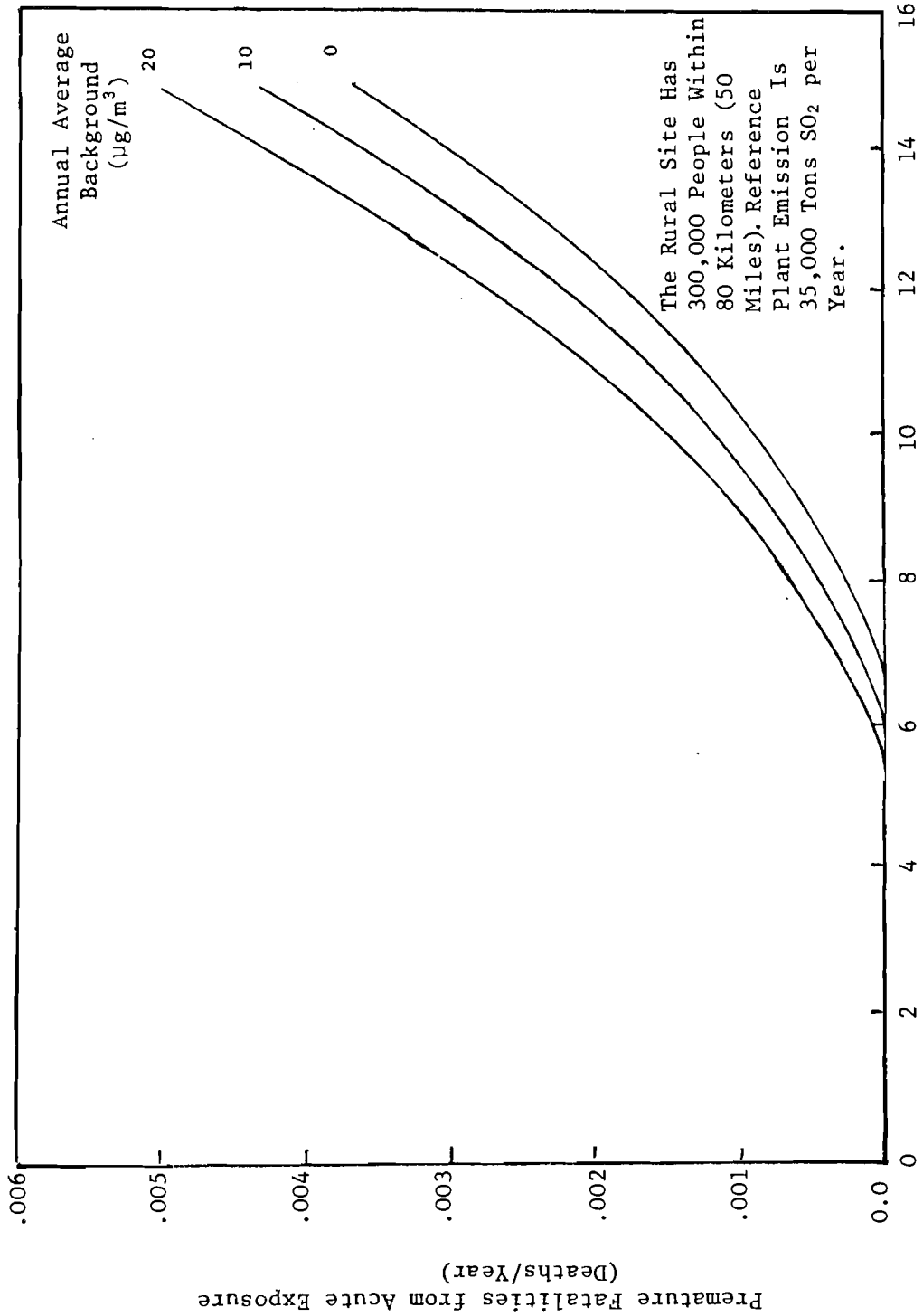

Figure 4: Premature Fatalities from Acute SO_2 Exposure for the Rural Site

Table 3

Annual Premature Fatalities at the Urban Site
As a Function of Background Concentrations and
Power Plant Emissions

Ratio of SO_2 Emission To Reference Plant Emissions	Annual Average Background SO_2 Concentration ($\mu g/m^3$)					
	0	20	40	60	80	100
1	0.0	0.0	0.0	0.0004	0.0015	0.0072
2	0.0006	0.0016	0.0027	0.0061	0.014	0.030
3	0.0040	0.0065	0.012	0.020	0.034	0.067
5	0.024	0.035	0.050	0.068	0.10	0.18
10	0.15	0.19	0.24	0.31	0.42	0.60
15	0.39	0.47	0.57	0.71	0.90	1.2
20	0.74	0.86	1.0	1.2	1.5	2.0
25	1.2	1.4	1.6	1.9	2.3	3.0

N.B. The urban site has a homogeneous population density of 313 people/km^2.
The reference plant emission is 35,000 short tons of SO_2 per year.

Table 4

Annual Premature Fatalities at the Rural Site
As a Function of Background Concentrations and
Power Plant Emissions

Ratio of SO_2 Emission To Reference Plant Emissions	Annual Average Background SO_2 Concentration ($\mu g/m^3$)					
	0	20	40	60	80	100
1	0.0	0.0	0.0	0.0	0.0	0.00001
2	0.0	0.0	0.0	0.0	0.0	0.0004
3	0.0	0.0	0.0	0.0	0.0002	0.0010
5	0.0	0.0	0.0001	0.0005	0.0012	0.0028
10	0.0009	0.0014	0.0024	0.0038	0.0060	0.011
15	0.0037	0.0050	0.0070	0.0097	0.015	0.025
20	0.0082	0.011	0.014	0.019	0.027	0.043
25	0.014	0.018	0.023	0.031	0.042	0.066

N.B. The rural site has a homogeneous population density of 15 people/km^2.
The reference plant emission is 35,000 short tons of SO_2 per year.

the average site can be computed from these tables by averaging the per capita effects for the urban and rural sites and then multiplying by the assumed average site population of 2,250,000. Table 4 indicates that even in the cases with the highest background there was no direct contribution by the background itself to the premature fatalities, i.e. the worst day for an arithmetic mean of 100 $\mu g/m^3$ and a standard geometric deviation of 1.75 does not exceed the threshold value for premature fatalities. Therefore, the premature fatalities are entirely attributable to the power plant if the power plant concentrations are assumed to be an addition to the background.

It is evident in Tables 3 and 4 that increasing background with fixed power plant emissions has a nonlinear effect on the number of expected fatalities. For example, if background is increased from 0 to 100 $\mu g/m^3$ for an emissions ratio of 2 at the urban site (Table 3), the expected fatalities increase by about 0.03, while the same change in background at an emissions ratio of 25 results in an increase in fatalities of 1.8. This is a result of the threshold form of the dose-response function.

Excess Heart and Lung Disease Aggravation in the Ederly

Since the dose-response functions for heart and lung disease aggravation and asthma attacks are linear with no threshold, the effects associated with the power plant emissions are not affected by the background SO_2 concentration. The health effects attributable to the background are an addition to those calculated for the power plant.

The calculations for heart and lung disease aggravation in the elderly are shown in Figure 5. Urban siting can result in about 51,000 excess days of aggravation per year for a site with 15 times as much SO_2 emitted as the 1,000 MWe reference plant. This compares with a normal incidence without SO_2 exposure of about 12 million days of aggravation per year at the urban site. The number of days of aggravation for an emission ratio of 15 is about a 0.4 percent increase in the normal rate at all three sites.

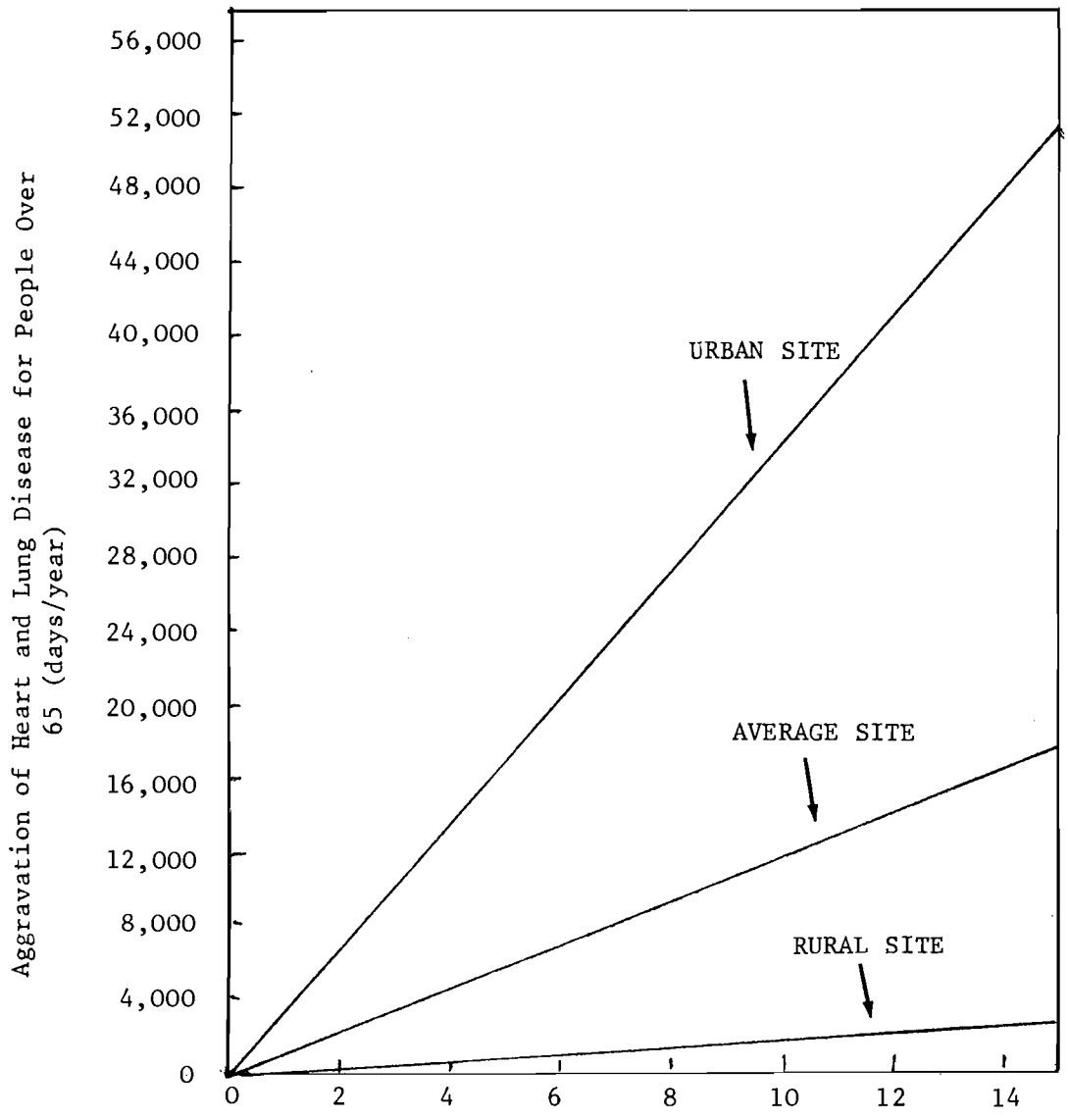

Figure 5: Heart and Lung Disease Aggravation from Sulfur Dioxide Emissions at Coal-Fired Power Plants

Excess Asthma Attacks

The excess asthma attacks are plotted in Figure 6 as a function of SO_2 release for the three sites. The excess asthma attacks per year for a plant with 15 times as much SO_2 release as the reference plant represents about a one percent increase in the expected number of asthma attacks in that population.

Excess Acute Respiratory Disease in Children

According to the dose-response function given by Equation 14, no excess acute lower respiratory disease in children will occur for annual average SO_2 concentration less than 133 $\mu g/m^3$. Therefore, these extra health effects are relatively rare except when background and emissions are very high, as shown in Table 5.

Excess Chronic Respiratory Disease Symptoms in Adults

The minimum annual average SO_2 concentration that results in extra chronic respiratory disease symptoms for non-smoking adults is 53 $\mu g/m^3$ and for smoking adults is 215 $\mu g/m^3$. The threshold for non-smokers is low enough that many extra cases occur when background or emissions are high, as shown in Table 6. However, the smokers threshold is so high that only very few cases result from the combinations of the greatest emissions and backgrounds, as shown in Table 7. For the rural site the threshold for smokers is not exceeded for any combination of background and emissions shown in Table 7.

Finklea's alternative dose-response functions, based on least squares fits, have also been used to compute expected health impacts. In Appendix B some results are compared to the results obtained with the best judgment dose-response functions. In general the best judgment functions result in lower levels of predicted health effects. The comparison demonstrates the considerable uncertainty that is associated with the dose-response functions.

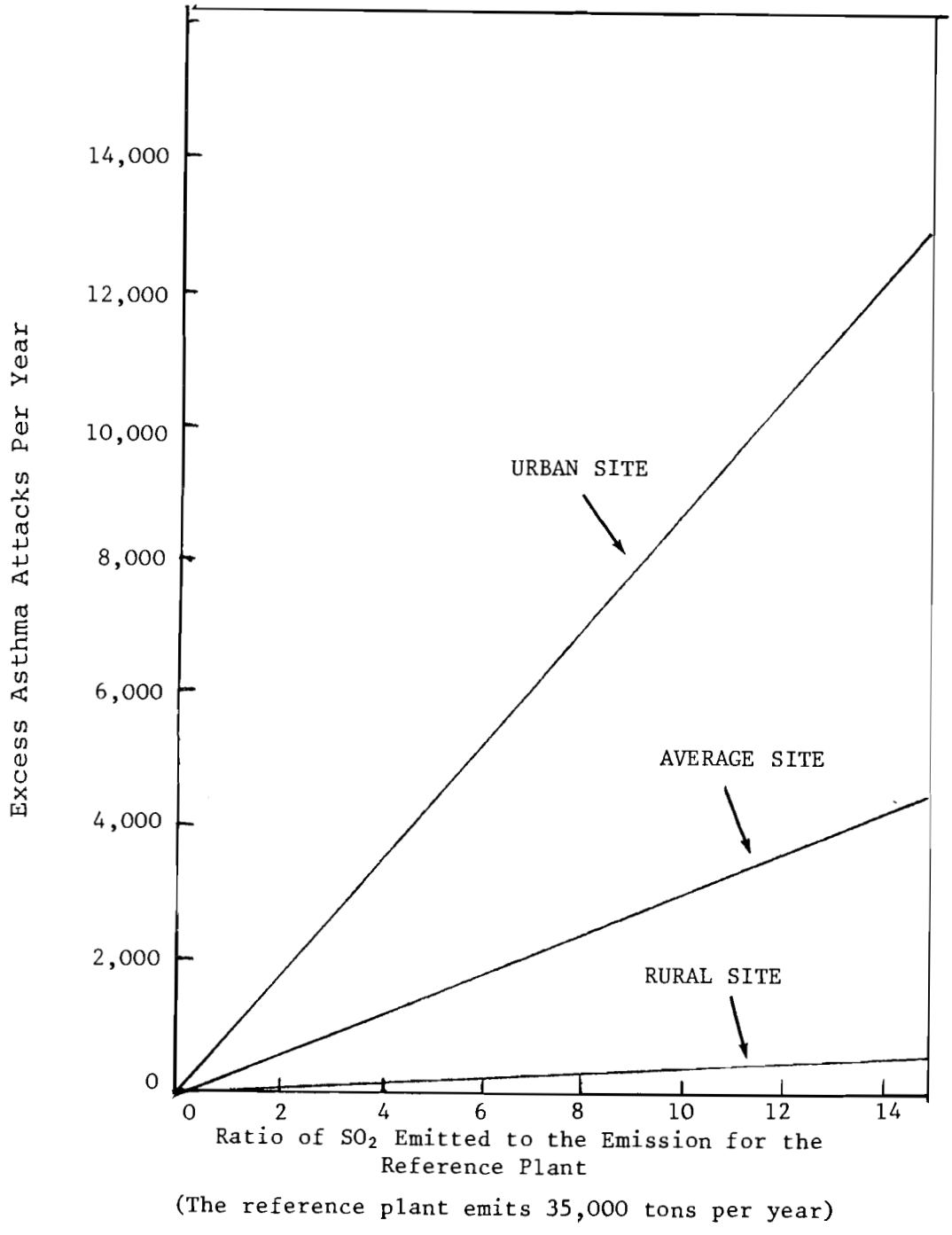

Figure 6: Excess Asthma Attacks from Sulfur Dioxide Emissions At Coal-Fired Power Plants

Table 5

Excess Acute Lower Respiratory Disease in Children as a Function of Background Concentration and Power Plant Emission (cases per year)

Ratio of SO_2 Emission To Reference Plant Emission / Urban Site[1]	Annual Average Background SO_2 Concentration ($\mu g/m^3$) 0	20	40	60	80	100
1 (35,000 tons/yr)	0.0	0.0	0.0	0.0	0.0	0.0
5	0.0	0.0	0.0	0.0	0.0	2.1
10	0.0	0.0	0.0	2.7	7.1	23.
15	0.0	3.2	7.6	12.	30.	77.
20	8.2	13.	17.	36.	75.	180.
25	18.	23.	42.	76.	140.	320.
Rural Site[1]						
1 (35,000 tons/yr)	0.0	0.0	0.0	0.0	0.0	0.0
5	0.0	0.0	0.0	0.0	0.0	0.0
10	0.0	0.0	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0	0.0	0.09
20	0.0	0.0	0.0	0.0	0.0	1.1
25	0.0	0.0	0.0	0.0	0.22	3.6

[1]The assumed population densities were 313 people/km^2 for the urban site and 15 people/km^2 for the rural site.

Table 6

Extra Non-Smokers Showing Chronic Respiratory Disease as a Function of Background Concentration and Power Plant Emission.

Ratio of SO_2 Emissions to Reference Plant Emission	Annual Average Background SO_2 Concentration[2] ($\mu g/m^3$)					
Urban Site[1]	0	20	40	60	80	100
1 (35,000 tons/yr)	0.0	0.0	0.0	1,400.	5,300.	9,000.
5	0.0	1.8	16.	1,900.	5,800.	9,700.
10	6.0	20.	110.	2,600.	6,400.	10,300.
15	25.	65.	300.	3,200.	7,000.	10,900.
20	63.	150.	620.	3,800.	7,700.	11,500
25	120.	270.	1,000.	4,400.	8,300.	12,200.
Rural Site[1]						
1 (35,000 tons/yr)	0.0	0.0	0.0	68.	250.	440.
5	0.0	0.0	0.0	90.	270.	460.
10	0.0	0.0	1.2	120.	300.	480.
15	0.0	0.075	6.1	140.	330.	510.
20	0.0	0.88	20.	170.	360.	540.
25	0.18	3.0	35.	200.	380.	570.

[1]The assumed population destinies were 313 people/km^2 for the urban site and 15 people/km^2 at the rural site.

[2]Since the threshold for health effects is 53$\mu g/m^3$, the background concentration, without any contribution from the power plant, causes some health effects for background concentrations above this level.

Table 7

Extra Smokers Showing Chronic Respiratory Disease Symptoms As a Function of Background Concentration and Power Plant Emission

Ratio of SO_2 Emission to Reference Plant Emission	Annual Average Background SO_2 Concentrations ($\mu g/m^3$)					
Urban Site[1]	0	20	40	60	80	100
1 (35,000 tons/yr)	0.0	0.0	0.0	0.0	0.0	0.0
5	0.0	0.0	0.0	0.0	0.0	0.0
10	0.0	0.0	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0	0.0	3.9
20	0.0	0.0	0.0	4.7	11.	17.
25	0.0	5.5	12.	18.	24.	30.

[1]The threshold of $215 \mu g/m^3$ was not exceeded at the rural site for any combination of background up to $100 \mu g/m^3$ and a ratio of emissions up to 25.

V. Conclusions

Relationships have been developed for estimation of some quantified human health impacts of SO_2 emissions from coal-fired power plants. The five categories of health impacts are premature fatalities, aggravation of pre-existing heart and lung disease in the elderly, excess asthma attacks, excess acute lower respiratory disease in children, and excess risk of chronic respiratory disease symptoms in adults. The following critical assumptions, which are subject to considerable unresolved uncertainty, have been made:

- the dose-response functions providing expected health impacts that result from exposure to sulfates, shown in Table 1,
- the conversion between SO_2 and sulfate concentration given by equation 1,
- the empirical relationship between standard geometric deviation for 24-hour SO_2 concentration and distance from the power plant provided in Figure 1, and
- the log-normal distributions for both the daily background SO_2 concentrations and the SO_2 concentrations that result from power plant emissions.

The last two assumptions are used only for calculation of premature fatalities.

The health impacts are parameterized in terms of SO_2 emission, population and site characteristics, and background SO_2 concentration. This parameterization gives the health impact model its strength and flexibility for use in environmental policy analysis and long range planning.

The results have shown that excess days of aggravation of heart and lung disease in the elderly and excess asthma attacks are the quantified effects that occur most frequently as a result of SO_2 emission from coal-fired power plants; premature mortality, excess acute lower respiratory disease in children, and excess risk of chronic respiratory disease symptoms in adults occur less frequently. It should be noted that these quantified effects are either the result of short-term exposure or long-term exposure to relatively high levels of pollution. The impacts of long-term exposure to low levels of pollution have not yet been quantified. Therefore, the quantified impacts of air pollution included in this report are only a partial indicator of the total health impact and are not the total impact of air pollution.

This study has indicated that the quantified health impacts of SO_2 emissions from a single coal-fired power plant can amount to thousands of days of human illness, and some premature fatalities. Additionally, for the combination of circumstances that result in few quantified health effects, one cannot say these impacts are negligible without a review of alternatives and consideration of other impacts, unquantified effects, and conventional costs [17]. The quantified relationships provided in this report are a partial indicator of the total impact, and the results indicate they are a significant addition to power plant impact analysis.

REFERENCES

[1] Finklea, J.F., et al., "Health Effects of Increasing Sulfur Oxides Emissions," Draft Report, National Environmental Research Center, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina (1975).

[2] Lave, L.B., and E.P. Seskin, "Air Pollution and Human Health," Science, Vol. 169, No. 3947, 21 August 1970, pp. 723-733.

Lave, L.B., and L.C. Freeburg, "Health Effects of Electrical Generation from Coal, Oil, and Nuclear Fuel," Nuclear Safety, Vol. 14, No. 5, September-October 1973, pp. 409-428.

[3] Hickey, R.H., et al., "Ecological Statistical Studies Concerning Environmental Pollution and Chronic Disease," IEEE Trans. on Geoscience Electronics, Vol. GE-8, No. 4, October 1970, p. 186.

[4] Carnow, B.W., et al., "Health Effects of Fossil Fuel Combustion: A Quantitative Approach," selection from report Health Effects of Energy Systems: A Quantitative Assessment, submitted by American Public Health Association to Ford Foundation, March 1974.

[5] Finklea, J.F., Private communication.

[6] Finklea, J.F., Private communication. Reference 1 has different annual average and 24-hour average conversion rates. This was mainly the result of data scatter and insufficient data.

[7] R.S. Berry and P.A. Lehman, "Aerochemistry of Air Pollution," Adv. in Phys. Chem., 22, 47-84 (1971).

[8] P.F. Fennelly, "The Origin and Influence of Airborne Particulates", American Scientist, 64, 46-56 (January-February, 1976).

[9] U.S. Department of Commerce, Statistical Abstract of the United States, U.S. Government Printing Office, 1974.

[10] Dennis, R.L., Model Calculations for the Portage Power Plant Project, U. of Wisconsin, 1974 (unpublished).

Buehring, W.A., et al.,"Environmental Impact of Regional Energy Use: A Unified Systems Approach, "Energy Systems Planning, Forecasting and Pricing, Ed. by C.J. Cicchetti and W.K. Foell, Univ. of Wisconsin Press, 1975.

[11] H. Moses and M.R. Kraimer, "Plume Rise Determination--a New Technique Without Equations", Journal of the Air Pollution Control Association, 22 (8), 621-630 (1972).

[12] Evans, R.J., "Potential Solid Waste Generation and Disposal from Lime and Limestone Desulfurization Processes," Bureau of Mines Information Circular 8633, U.S. Dept. of Interior, 1974.

[13] Benarie, M., "Sur la Validite de la Distribution Logarithmico-Normale des Concentrations de Polluants", Proceedings of the Second International Clean Air Congress, Ed. by H.M. England and W.T. Beery, Academic Press, 1971.

Larsen, R.I., "United States Air Quality", Arch. of Envir. Health, 8, 325-333, 1964.

Larsen, R.I., "A Mathematical Model for Relating Air Quality Measurements to Air Quality Standards", U.S.E.P.A. Pub., WP-89, 1971.

[14] Ragland, K.W., and Miller, M.H., "Air Pollution Emissions Inventory in the Madison, Wisconsin Area," University of Wisconsin Institute for Environmental Studies Working Paper 5, 1972.

Ragland. K.W. and Kitson, K.L., "Air Pollution due to Point Sources in Madison, Wisconsin," University of Wisconsin Institute for Environmental Studies Working Paper 6, 1972, Revised by R.L. Dennis, 1974.

Ragland. K.W.., et al., "Boundry Layer Model for Transport of Urban Air Pollutants," Paper presented at the AIChE National Meeting, March 18, 1975.

[15] (Anonymous), "State of Wisconsin Department of Natural Resources 1973 Air Quality Data Report," 1974.

[16] Knox, J.B., and Rolf Lange, "Surface Air Pollutant Concentration Frequency Distributions: Implications for Urban Modeling," Journal of the Air Pollution Control Association, 24 (1) 48-53 (1974).

[17] Buehring, W.A. and W.K. Foell, "Environmental Impacts of Electrical Generation: A Systemwide Approach," IIASA RR-76-13, 1976.

APPENDIX A

Derivation of the Daily Concentration Distribution

If the dose-response function given by Equation 4 in Section II.2 is used to compute the number of premature fatalities, the daily distribution of SO_2 concentrations is needed. The purpose of this appendix is to outline the method used to determine that distribution.

The premature fatalities per day is calculated from the relationship

$$DPF = \mu \cdot \begin{cases} c - c_o & \text{if } c > c_o \\ o & \text{if } c \leq c_o \end{cases} \qquad (1)$$

or

$$DPF = \mu \cdot \max \{o, c - c_o\}$$

where

DPF = premature fatalities per day,
μ = dose-response coefficient,
c = daily average SO_2 concentration,
c_o = threshold SO_2 concentration below which no premature fatalities are expected, and
max{a,b} = the larger of the two quantities inside brackets.

The daily average SO_2 concentration, c, is assumed to have a log-normal distribution. If one denotes the probability density function with f(c), the expected premature fatalities per year (APF) can be expressed as

$$APF = 365 \cdot \mu \cdot \int_o^\infty \max\{o, c - c_o\} \cdot f(c)\, dc \quad , \qquad (2)$$

where the integral is the expected excess of daily average SO_2 concentrations over the threshold value c_o. One can divide the range of possible concentrations into intervals (a_{i-1}, a_i) such that the probability of finding $a_{i-1} \leq c < a_i$ is 1/365, with i = 1,2,...,365. Therefore, the expected number of days in a year with an average of SO_2 concentration in the interval

(a_{i-i}, a_i) is equal to 1. The "center", c_i of each of these intervals can be defined by

$$\text{pr}\{c < c_i\} = p_i = \frac{i-\frac{1}{2}}{365} \qquad (3)$$

$$i = 1,2,\ldots,365$$

where pr should read as "the probability that". This definition implies that the probability that $a_{i-1} \leq c \leq c_i$ is equal to the probability that $c_i \leq c \leq a_i$, i.e. the daily average concentration, c, is equally likely to fall above or below c_i within the interval (a_{i-1}, a_i). Thus c_i is really representative of the i^{th} highest range of concentrations.

The values c_i, i=1,...,365 can be interpreted in an intuitive way. The concentration on the day with the highest average concentration is associated with c_1, the concentration level which is expected only one day every two years,..., the concentration on the day with the mean value is associated with c_{183}, the concentration which is expected to be exceeded half of the time,..., and the concentration on the day with the lowest average concentration is associated with c_{365}, the concentration which is expected to be exceeded all but one day in two years. We have defined the probability for the day with the highest concentration as 0.5/365 rather than 1/365. This concentration is expected to be exceeded one day every two years rather than one day every year and is a more representative value for the day with the highest concentration. Use of 1/365 for the day with the highest concentration would simply set a lower bound on premature mortality.

The integral in Equation 2 can now be approximated by a sum, i.e.

$$\text{APF} = 365 \cdot \mu \cdot \sum_{i=1}^{365} \max\{o, c_i - c_o\} \cdot \frac{1}{365}$$

$$= \mu \sum_{i=1}^{365} \max\{o, c_i - c_o\} \quad . \qquad (4)$$

The relationship in Equation 4 indicates that the premature fatalities per year are simply the sum of the premature fatalities that occur each day. To evaluate the c_i with $c_i < c_o$, Equation 3 can be rewritten as

$$pr\{c \leq c_i\} = 1 - p_i$$

$$= \int_o^{c_i} f(c)dc \quad . \tag{5}$$

When c has a log-normal distribution, then ℓnc has a normal distribution that is completely specified by two parameters, the expected value (or mean), m, and the variance, v. Estimates for these parameters from n observations ℓnc_j, $j=1,\ldots,n$ are

$$\bar{m} = \frac{1}{n} \sum_{j=1}^{n} \ell nc = \ell n \left(\prod_{j=1}^{n} c_j \right)^{\frac{1}{n}} = \ell nM \tag{6}$$

$$\text{thus } M = \exp(\bar{m})$$

and

$$\bar{v} = \frac{\sum_{j=1}^{n} (\ell nc - \ell nM)^2}{(n-1)} = \ell n^2 S \tag{7}$$

$$\text{thus } S = \exp(\bar{v})^{0.5}$$

where M is the geometric mean and S is the standard geometric deviation of the n observations c_j, $j=1,\ldots,n$. Equation 7 shows that S tends toward 1.0 as the variation of the log of the daily concentrations around the geometric mean tends toward zero, i.e. $S \geq 1.0$ for all distributions.

As ℓnc is a strictly monotonic function of c, Equation 5 can be modified to give

$$pr\{c \leq c_i\} = pr\{\ell n\, c \leq \ell n\, c_i\} \quad . \tag{8}$$

The probability relationship is not affected by subtracting a constant, ℓn M, and dividing by a positive constant, ℓn S, i.e.

$$\text{pr}\{c \leq c_i\} = \text{pr}\left\{\frac{\ell n\ c - \ell n\ M}{\ell n\ S} \leq \frac{\ell n\ c_i - \ell n\ M}{\ell n\ S}\right\}$$

$$= 1 - p_i \qquad (9)$$

Let x be defined as $(\ell n\ c - \ell n\ M)/\ell n\ S$. Then x has a normal distribution with zero mean and unit variance. The cumulative function $\Phi(X)$, is given by the integral

$$\Phi(x) = \int_{-\infty}^{x} f(x')dx' \qquad (10)$$

where f(x) is the probability density function. As x approaches $+\infty$, the integral converges to 1.0. When $x_i = (\ell n\ c_i - \ell n M)/\ell n\ S$, the integral in Equation 10 is just $1 - p_i$. Therefore

$$\Phi\left(\frac{\ell n\ c_i - \ell n\ M}{\ell n\ S}\right) = 1 - p_i \quad . \qquad (11)$$

The daily average concentration can now be determined using Equation 11, giving

$$c_i = M \cdot S^{\Phi^{-1}(1-p_i)} \qquad i = 1,2,\ldots,365 \qquad (12)$$

where Φ^{-1} is the inverse function of the cumulative function for the normal distribution. Since M, S, and the arithmetic average concentration, A, are linked by the relationship given by Larsen [13],

$$M = A \cdot \exp(-\tfrac{1}{2}\ \ell n^2 S), \qquad (13)$$

Equations 12 and 13 can be used to solve for the daily average concentration distribution whenever two of M, S, and A are known.

For example, the calculations for three days of the year are indicated below for the particular case when the geometric mean is 1.37 $\mu g/m^3$ and the standard geometric deviation is 5.0*.

---

*The annual arithmetic average is 5.0 $\mu g/m^3$ for these values of M and S.

Day with highest concentration:

$$1 - p_1 = 1 - \frac{1 - \frac{1}{2}}{365} = 0.99863$$

$$\Phi^{-1}(0.99863) = 2.9956$$

$$c_1 = 1.37(5^{2.9956}) = 170 \mu g/m^3$$

Day with the mean concentration:

$$1 - p_{183} = 1 - \frac{183 - \frac{1}{2}}{365} = 0.5$$

$$0.5 = \Phi(0)$$

$$c_{183} = 1.37(5^0) = 1.37 \text{ µg per } m^3$$

Day with the lowest average concentration:

$$1 - p_{365} = 1 - \frac{365 - \frac{1}{2}}{365} = 0.00137$$

$$0.00137 = \Phi(-2.9956)$$

$$c_{365} = 1.37(5^{-2.9956}) = 0.011 \text{ µg per } m^3$$

APPENDIX B

Some Results For the Least Squares Model

The dose-response functions that were presented in Table 1 are the "best-judgment" functions as given by Finklea [1]. These relationships are highly uncertain and are based on a limited number of observations. A different set of dose-response functions for the same health impacts is also presented by Finklea. The second set of dose-response functions are based on a least squares fit. Finklea preferred the best judgment functions because the data points available for the assessment of a single adverse effect were not independent and because of differences among the original studies.

The SO_2 concentration thresholds, below which no excess health effects are expected, are compared in Table B-1 for the two sets of dose-response functions. The primary differences with the best judgment thresholds are premature mortality, acute lower respiratory disease in children, and chronic respiratory disease in non-smokers. In addition the slopes of the linear functions based on least squares are not identical with the best judgment slopes.

The dose-response functions using the least squares fit were applied to several combinations of SO_2 emissions and background. Results with the least squares functions are compared with the best judgment results for these particular cases in Table B-2. It should be noted that in some cases the background would produce impacts without any power plant emission. For example, the least squares function for premature mortality yields about six premature fatalities for a background of 60 $\mu g/m^3$ with no emission and nearly ten premature fatalities with a background of 60 and an emission ratio of 10. The lowest annual average SO_2 background concentration, with a standard geometric deviation of 1.75, that produces premature fatalities without power plant emissions is 116 $\mu g/m^3$ for the best judgment function and only 24.5 $\mu g/m^3$ for the least squares function. As shown in Table B-2, the least squares functions result in greater quantified helath impacts except for slight reductions in asthma attacks and aggravation of heart and lung disease in the elderly.

The annual premature fatalities at the urban site for several emission ratios and background levels are given in Table B-3. Comparison with Table 3 in Section IV indicates that the least squares function predicts significantly greater premature fatalities than the best judgment function. Also, with the lower threshold for the least squares function, it is also evident that an annual average background concentration of 60 or 100 $\mu g/m^3$ is responsible for a significant number of fatalities without any power plant emissions.

The primary conclusions of the comparison are that the least squares functions predict significantly greater premature fatalities, cases of acute lower respiratory disease in children, and chronic respiratory disease in non-smokers. In each case the primary reason for the difference is a lower threshold used in the least squares functions. Use of the best judgment functions, with the conversion of SO_2 to sulfates given by Equation 1 in Section II.1, generally results in fewer predicted health effects. The actual dose-response relationships remain uncertain.

Table B - 1

Comparison of Best Judgment and Least Squares

Dose-Response Functions

Adverse Health Effect	SO_2 Concentration Averaging Time	Inferred SO_2 Threshold ($\mu g/m^3$) Below Which No Excess Health Impacts are Expected		Slope, or Percent Excess Health Impacts Resulting From a 1 $\mu g/m^3$ Increase in SO_2 Concentrations above the Threshold*	
		Best Judgment	Least Square	Best Judgment	Least Squares
Increased daily mortality	Daily	530	112	.0076	.0096
Aggravation of heart and lung disease in elderly patients	Daily	0	0	.0423	.0393
Aggravation of asthma	Daily	0	0	.101	.0062
Excess acute lower respiratory disease in children	Annual	133	10	.231	.137
Excess risk for chronic bronchitis					
Non-smokers	Annual	53	20	.402	.444
smokers	Annual	215	222	.221	.236

* The percent (not fractional) excess is with respect to the normal rate for the health impact. The slope with respect to SO_2 depends strongly on the assumed relationship between sulfates and SO_2. Equation 1 in Section II.1 has been used here.

Table B - 2

Comparision of Selected Results for the Best Judgment and Least Squares Dose-Response Functions[1]

	Best Judgment	Least Squares	Best Judgment	Least Squares	Best Judgment	Least Squares
Ratio of SO_2 Emitted to Reference Plant Emissions	1	1	5	5	10	10
Annual Average Background SO_2 Concentration ($\mu g/m^3$)	0	0	20	20	60	60
Annual Health Impacts:						
Premature Fatalities	0.0	0.001	0.006	0.17	0.70	9.9*
Days of heart and lung disease in elderly patients[2]	1,200	1,100	5,800	5,400	12,000	11,000
Asthma attacks[2]	300	180	1,500	920	3,000	1,800
Cases of acute lower respiratory disease in children	0.0	0.0	0.0	630*	0.47	2,700*
Chronic respiratory disease in non-smokers	0.0	0.0	0.3	270*	900*	3,500*
Chronic respiratory disease in smokers	0.0	0.0	0.0	0.0	0.0	0.0

[1]The population within 80 kilometers corresponds to the average site (2,250,000).

[2]These two health impact categories have no threshold. Therefore, only the impacts associated with the power plant emission are tabulated. The impacts produced by the background are omitted.

*The background produces some health impact without any power plant emission.

Table B - 3

Annual Premature Fatalities at the Urban Site as a Function of Background Concentrations and Power Plant Emissions - Least Squares Model*

Ratio of SO_2 Emission to Reference Plant Emissions	Annual Average Background SO_2 Concentrations ($\mu g/m^3$)			
	0	20	60**	100**
1 (35,000 tons/yr)	0.0056	0.036	17.6	102
2	0.036	0.13	18.7	105
3	0.093	0.27	19.9	107
5	0.28	0.68	22.3	112
10	1.2	2.3	28.9	124
15	2.6	4.6	36.2	136
20	4.4	7.5	44.1	149
25	6.6	11.0	52.5	161

*Compare with best judgment results in Table 3, Section IV.

**The background is responsible for some premature fatalities without any power plant emissions.

APPENDIX C

PAPER IN THE IIASA PUBLICATION SERIES ON MANAGEMENT OF ENERGY/ENVIRONMENT SYSTEMS

Keeney, R.L. Energy Policy and Value Tradeoffs, RM-75-76, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1975.

Foell, W.K., Scenario Writing: One Component of a Systems Approach to Energy/Environment Management, RM-76-20, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Born, S., C. Cicchetti, R. Cudahy, J. Pappas, P. Hedrich, K. Lindner, D. Ufer, J.-M. Martin, D. Finon, Energy/Environment Models and their Relationships to Planning in Wisconsin, the German Democratic Republic, and Rhone-Alpes, RM-76-21, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Foell, W.K., The IIASA Research Program on Management of Regional Energy/Environment Systems, RM-76-40, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Buehring, W.A., W.K. Foell, Environmental Impact of Electrical Generation: A Systemwide Approach, RR-76-13, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Buehring, W.A., W.K. Foell, R.L. Keeney, Energy/Environment Management: Application of Decision Analysis, RR-76-14, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Stehfest, H., A Methodology for Regional Energy Supply Optimization, RM-76-57, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Buehring, W.A., R.L. Dennis, A. Hölzl, Evaluation of Health Effects from Sulfur Dioxide Emissions for a Reference Coal-Fired Power Plant, RM-76-23, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Forthcoming

Dennis, R.L., Regional Air Pollution Impact: A Dispersion Methodology Developed and Applied to Energy Systems, RM-76-22, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.

Buehring, J., WISSIM: An Interactive Computer Simulation Control Language, RM-76-24, International Institute for Applied Systems Analysis, Laxenburg, Austria, 1976.