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Non-Radiological Impacts of Transporting Radioactive Material

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NON-RADIOLOGICAL IMPACTS OF TRANSPORTING RADIOACTIVE MATERIAL*

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ABSTRACT

Estimates of health effects that result from exposure to air pollutants generated during normal (accident-free) transport of radioactive materials and from accidents are provided for use in preparation of environmental impact statements. The results are presented for truck and rail modes and uncertainties associated with these results are discussed. Since these health effects have no relation to the radioactive material being hauled, their measure is applicable to shipments of all similar weight loads. The pollutant health effects are calculated for and applicable to urban areas only while the accident health effects are averages over all population zones in the U.S.

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PREFACE

In preparing this report, we realize the uncertainties that exist in the analysis as well as the conservatism (upper limits) that the health-effects estimates reflect. The results are based on state-of-the-art information and are our view of the best estimates of health effects currently available. We present this analysis for review and for judicious use by others and hope that by publishing these results we will stimulate additional activity in this important area of predicting health effects from air pollution.

ACKNOWLEDGEMENTS

The authors wish to express their appreciation to R. L. Schlegel and B. W. Bartram of the NUS Corporation for their interest in this work. The authors also wish to express their appreciation to Dr. A. Roffman of Energy Impact Associates for discussions on road fugitive dust measurements.

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INTRODUCTION

In our technological society, judgments about the acceptability of risks are made continuously based on the best data and evidence available at the time the judgments must be made. For transportation of nuclear materials, judgments about the acceptability of risks should be made considering all aspects of transportation and not just the radioactive character of the load.

One hazard of transporting nuclear material (or any material) arises from the generation of non-radiological pollutants during travel. Unfortunately, this hazard, in terms of resultant health effects, has not been quantified for mobile sources but only for stationary sources, such as coal-fired power plants. While sufficient data are unavailable to allow estimates to be made with great confidence, the impacts from these pollutants may be important relative to the radiological hazard which has been evaluated in other reports. This report, recognizing the real and potential uncertainties of the analysis, relates the health effects of non-radiological pollutants generated during the transportation of radioactive materials to a distance traveled in an urban area.

A second hazard arises from accidents that occur during transport even if no radioactive material is dispersed. The deaths and serious injuries associated with the physical trauma of any serious vehicular accident are also quantified.

By analyzing both these hazards, decisions (particularly those based on environmental impact statements) can be made from a more complete basis of information.

NON-RADIOLOGICAL IMPACTS DURING NORMAL OPERATIONS

The magnitude of non-radiological impacts from pollutants is a function of which ones are produced, their dispersion, and the physiological response of persons exposed to them.

Pollutants

The principal pollutants that are the focus of this analysis are: sulfur oxides (SO_x), particulates, nitrogen oxides (NO_x), carbon monoxide (CO), hydrocarbons (HC) and photochemical oxidants (O_x). Oxidants, though not emitted directly, are included for completeness since they are secondary pollutants generated by interactions among the other pollutants. As would be suspected, they coexist in the atmosphere and interact with themselves and components of the atmosphere.

Historically, SO_x and particulates have been the most carefully studied pollutants because they were thought to cause the majority of the health effects. Thus, much is known about these pollutants. They are most often associated with emissions from stationary sources because, as Table 1 shows, the majority of SO_x and particulates in the ambient air are produced by stationary sources. Nevertheless, mobile sources do increase the SO_2 and particulate levels incrementally, and it is the incremental contribution of the mobile sources that is evaluated in this paper.

The other pollutants (NO_x, CO, HC, and O_x) are commonly associated with mobile sources. This latter group of pollutants is the subject of current study, but data describing their effects have not been amassed as they have been for SO_x and particulates. In addition, all are now "criteria pollutants", which are regulated by the EPA and for which primary standards have been developed and data are compiled. At this time, the contribution of NO_x, CO, HC, and O_x to health effects appears to be much less than for SO_x and particulates.

An appendix was prepared to acquaint the reader unfamiliar with basic pollutant chemistry and descriptions. In addition, another appendix serves as background for discussion of health effects and emission rates of these pollutants.

TABLE 1

Estimated Emissions of Air Pollutants
(Million Metric Tons Per Year)
(U.S., 1977)*

	SO_x	Particulates	CO	NO_x	Hydrocarbons
Highway Vehicles	0.4	0.8	77.2	6.7	9.9
Total (All Sources)**	27.4	12.4	102.7	23.1	29.3

*From Ref. 1

**Includes fuel combustion in stationary sources, industrial processes, solid waste disposal, and miscellaneous including transportation.

Pollutant Concentrations

The increase in pollutant concentration caused by the travel of a diesel truck or a locomotive must be calculated. The concentration is a function of two primary parameters: atmospheric dispersion and emission rates (source terms).

Atmospheric dispersion. How the pollutants emitted during vehicle travel are dispersed through the atmosphere is determined using a line-source dispersion model as described in Reference 2. When the wind is blowing perpendicularly to the roadway, the concentration at any downwind distance, x, divided by the pollutant source strength is given by:

$$\frac{X_1(x)}{Q_1} = \left(\frac{2}{\pi}\right)^{\frac{1}{2}} \frac{K}{u\sigma_z} \qquad (1)$$

$X_1(x)$ = pollutant "1" concentration at a downwind distance, x $\mu g/m^3$

Q_1 = g/km-hr

$K = 0.28 \left(\frac{km}{m}\right)\left(\frac{\mu g}{g}\right)\left(\frac{hr}{sec}\right)$

$\bar{u}$ = mean wind speed $\frac{m}{sec}$

σ_z = vertical dispersion coefficient (m)

This equation is valid for a ground-level release and for a continuously emitting infinite line source. The physical representation of this calculation is shown in Figure 1.

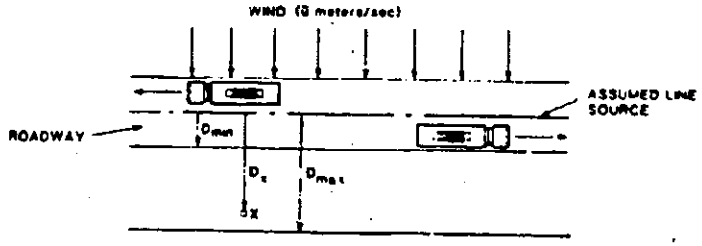

Figure 1. Physical Representation of Line-Source Calculation

The equation above is for a point at a distance, x, from the line source. In this analysis, the affected area over which the pollutants spread is determined by the area whose width is the distance between D_{min} and D_{max}. The value chosen for D_{min} is 30 meters and for D_{max} is 805 meters. These values were chosen to allow direct comparison to calculations in Reference 3. (The effect of using other values is examined in Appendix C.) To obtain the average concentration over this area, Equation (1) is evaluated:

$$\frac{\bar{X}_1}{Q_1} = \frac{\int_{D_{min}}^{D_{max}} \left(\frac{2}{\pi}\right)^{1/2} \frac{K dx}{(0.14)\bar{u}x(1 + 0.0003x)^{-1/2}}}{\int_{D_{min}}^{D_{max}} dx} \qquad (2)$$

where $\sigma_z = 0.14x(1 + 0.0003x)^{-1/2}$ for neutral atmospheric stability conditions in an urban area (Ref. 4) and x is the distance from the line source.

Performing the integration, the equation becomes:

$$\frac{\bar{X}_1}{Q_1} = \frac{K_1 ! \left(\frac{2}{\pi}\right)^{1/2}}{(D_{max} - D_{min})\bar{u}} \qquad (3)$$

$$\text{where } I = \int_{30}^{805} \frac{dx}{x(1 + 0.0003x)^{-1/2}}$$

$$\text{and } K_1 = \frac{K}{0.14} .$$

By integrating to obtain the value of I and by assuming a 3 m/sec wind speed (Ref. 5) and values for D_{max} and D_{min} that are given in Reference 3 for urban areas, the equation is reduced to:

$$\frac{\bar{X}_1}{Q_1} = 2.35 \times 10^{-3} \left(\frac{\mu g}{m^3}\right) \left(\frac{km - hr}{g}\right) \qquad (4)$$

where Q_1 has units of $\frac{g}{km - hr}$ and $\bar{X}_1$ has units of $\mu g/m^3$.

Source term. The source term (Q_1) of Equation (4) is described in terms of emission rates expressed on the basis of grams per kilometer of travel. The source term has three distinct components, as depicted in Figure 2: pollutants from the combustion of diesel fuel, particulates from tire wear caused by tires being abraded on a paved surface, and fugitive dust, generated in the wake of the vehicle, which includes resuspended particulates from combustion, brake bands/pads, tire wear, silt from wind-blown soil and other unspecified sources. Source terms are given in Table 2 for both truck and rail modes of transport.

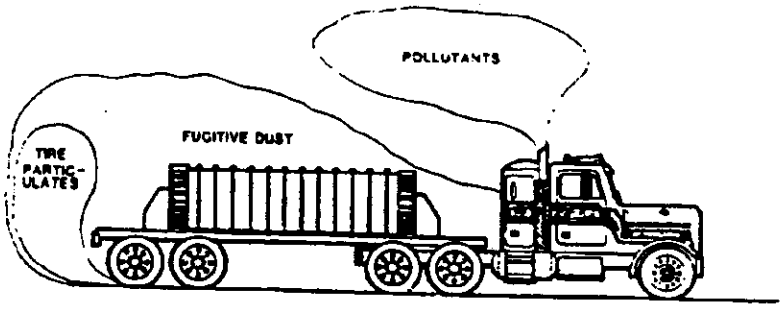

Figure 2. Components of Source Term

V_f is used to adjust emission rates to represent the assumed 24 kph (15 mph) speed. This velocity is assumed in order to be consistent with values in Reference 3. Where necessary, fuel efficiency was estimated to be

$22.3 \frac{\text{tonne-km}}{\ell} \left(58 \frac{\text{ton-mi}}{\text{gal}}\right)$ and $76.8 \frac{\text{tonne-km}}{\ell} \left(200 \frac{\text{ton-mi}}{\text{gal}}\right)$ for truck and rail modes, respectively (Ref. 8).

The tire-particulate source term is derived from Table 0.4-15 of Reference 7. The truck-trailer is assumed to have 18 wheels. Obviously, no tire wear is included for the rail mode.

The third component of the source term is fugitive dust. The quantity of fugitive dust entrained by a truck is given by the following equation (Ref. 9):

$$EF = 0.026\ (I)\left(\frac{4}{n}\right)\left(\frac{S}{10}\right)\left(\frac{L}{280}\right)\left(\frac{W}{2.7}\right)^{0.7} \qquad (6)$$

EF = fugitive dust emissions, kg/km

I = industrial road augmentation factor (1)

n = number of traffic lanes (4)

S = silt content on highway (10%)

L = surface dust loading on traveled portion of road (42 kg/km)

W = weight of truck-trailer (34 tonnes)

Because no empirical relation exists, the fugitive dust entrained by a passing railcar cannot be calculated. For this report, it is assumed that the quantity entrained is 10% of that entrained behind a truck (based on work that is presented in Reference 10). In reference 10, it was shown that the tire contact on a road surface caused much more dust to be entrained than just the air turbulence created by a passing truck. Specifically, a truck passing next to a dusty surface produced 10% of the entrained dust that a truck passing directly over the surface did. The wheels of a railcar touch only a narrow strip of rail and do not make contact with the dusty roadbed. For the above reasons, a fugitive dust source term was assumed for a railcar that is 10% of the value for truck.

From this point, the particulates from fugitive dust, tire wear, and combustion will be combined into one particulate source term. Implicit in so doing is the assumption that the health effect of the particulates from each component will be the same. This is not necessarily the case but can be justified because air sampling measurements do not currently distinguish among particulate sources or compositions. Only 50 weight percent of the fugitive dust particulates obtained from the above equation will be used to compute health effects because this is the fraction that lies in the less than 5 micron size range (References 9 and 11).

Concentration of pollutants. Combining the dispersion model results and source terms provides an average concentration of pollutants that results from the passage of truck and railcar shipments.

As an example, using a speed of 24 kph for an urban area (Ref. 3), the source term required by the dispersion equation will be:

$$Q_i = \frac{\left[\frac{\text{DIST TRAVELED, URBAN}}{\text{HR}}\right]\ [\text{EMISSION RATE, URBAN}]}{[\text{LENGTH OF SOURCE}]}$$

$$= \frac{\left(\frac{24\ \text{km}}{\text{hr}}\right)\left(q_i\ \frac{g}{\text{km}}\right)}{[24\ \text{km}]} = q_i\left(\frac{g}{\text{km-hr}}\right) \qquad (7)$$

where Q_i = source term rate $\frac{g}{\text{km-hr}}$ and

q_i = source term in $\frac{g}{\text{km}}$ for ith pollutant.

This can be interpreted to mean that vehicles pass by a point once an hour and that in an hour each vehicle travels 24 km (15 miles). The values for Q_i in the dispersion equation are as indicated in Table 3. These source terms can be input to the dispersion equation (4):

$$X_i = 2.35 \times 10^{-3}\ Q_i,$$

to obtain an average concentration in the urban area of interest.

The resultant concentrations are presented in Table 4. As can be seen in the table, the calculated mean concentrations for all pollutants are much lower than the primary National Ambient Air Quality Standards that have been established by the EPA.

Table 3. Values for $Q_i\left(\frac{g}{\text{hr - km}}\right)$

POLLUTANT	TRUCK	RAILCAR
Particulates	13	5
SO_2	5.1	10
NO_x	13	65
HC	3.3	19
CO	22	24

Table 4. Calculated Mean Pollutant Concentrations

	CONCENTRATION $\frac{\mu g}{m^3}$		
POLLUTANT	TRUCK	RAILCAR	PRIMARY STANDARD
Particulates	3.1×10^{-2}	1.4×10^{-2}	75 (annual geometric mean)
SO_2	1.2×10^{-2}	2.4×10^{-2}	80 (annual arithmetic mean)
NO_x	3.1×10^{-2}	1.5×10^{-1}	100 (annual arithmetic mean)
HC	7.8×10^{-3}	4.5×10^{-2}	160 (3-hour maximum)
CO	5.2×10^{-2}	5.6×10^{-2}	1×10^{4} (8-hour maximum)

These concentrations are really the incremental change in mean concentration caused by the continuous passage of one vehicle per hour.

Miscellaneous Inputs

In order to complete the health effects calculations, two additional inputs not previously discussed are needed: urban population densities and average pollution levels for each of the pollutants in the urban environment.

Population density. The assumed population density is consistent with Reference 3 by using a value of 3861 persons/km^2 (10,000 persons/mi^2). Appendix J of Reference 3 indicates that this value is representative of urban centers in the United States. The reason for selecting only urban areas is discussed in Appendix B.

The total population living in an urban roadside zone (area: $(D_{max} - D_{min})$ x length of line source) would be:

$$\frac{3861 \text{ people}}{\text{km}^2} \; (.775 \text{ km}) \; (24 \text{ km}) \approx 72{,}000.$$

Mean pollutant levels. Since health effects are calculated using elasticities (see Appendix B) that are valid near the mean pollutant levels for which they were calculated, the mean pollutant levels from References 12 and 13 are used for particulate and SO_2 levels. The values are shown in Table 5.

At this point, the effects of CO, NO_x, and HC will no longer be considered because elasticities have only been calculated for SO_2 and particulates (refer to Appendix B). Mean values for CO and NO_2 are shown in Table 5 for information only. There is no substantial evidence which imputes chronic health effects (refer to Appendix B) to these omitted pollutants and, in some cases, their effect is likely to be overshadowed by the contribution of the SO_2 and particulates (Ref. 14).

Table 5. Monitored Mean Pollutant Levels in Urban Environments

Pollutant	Concentration($\mu g/m^3$)
Particulates	103[a]
SO_2	33[a]
NO_2	49.6[b]
CO	2.6[b]

[a]From Ref. 12 and 13, approximate midpoint of pollutant concentration range.

[b]From Ref. 15.

Health Effects

The inputs necessary to calculate the health effects have been obtained as discussed in Appendix B. Important assumptions had to be made, three of which are taken from the appendix and emphasized here because they form the basis for the health effects calculations:

1) The elasticities calculated for the pollutant dose-response behavior are assumed to be valid only over a small increment from the mean concentrations used to calculate them.

2) The elasticities are assumed to be valid for both particulates and SO_2.

3) The health effects of CO, NO_x, and HC are not considered in the health effects calculations because relationships between exposure to them and health effects have not been established. They are believed not to be significant contributors to health effects.

With these assumptions in mind, the health effects can be calculated according to the following relationship:

$$\frac{\text{change in mortalities}}{\text{mortalities from all causes}} = \sum_{\text{pollutants}} (\text{ELASTICITY}) \quad \frac{\text{change in concentration}}{\text{mean concentration}} \qquad (8)$$

The changes in concentrations for both the particulates and SO_2 are found in Table 4, and their mean concentrations are found in Table 5. From Reference 16, the annual mortality rate in the U.S. for all causes (denominator on right side of equation (8)) is 8.8 deaths per 1000 people. Thus, for an urban area consisting of 72,000 persons, about 635 would be expected to die each year from all causes. Since the elasticity value used (described in Appendix B) is 0.05, the additional number of deaths caused by the passage of one additional truck per hour continuously for a year would be 0.02 (numerator of right side of equation (8)) and the number caused by the passage of one additional loaded railcar would be 0.028. In other words, the passage of about 50 trucks per hour continuously for 1 year would result in one additional mortality in the population considered here.

These results can be translated into a more useful format: deaths per kilometer of travel in an urban area. Since the average velocity of travel is 24 kph

(15 mph), the truck or railcar would travel 210,000 kilometers running continuously for one year. Therefore, the unit-consequence factors (deaths per kilometer travel in an urban area) are 1.0×10^{-7} for truck and 1.3×10^{-7} for a railcar. The deaths referred to here do not occur immediately, but rather, may occur after some latency period of several years. It is not possible, using available statistical methods, to detect on a specific individual basis the life shortening that may result from an increase in pollution levels, and the deaths cannot be attributed to the passage of a specific shipment. However, each shipment contributes to the likelihood of a fatality at some time in the future by adding its increment of pollution.

NON-RADIOLOGICAL IMPACTS FROM ACCIDENTS

Traumatic injury and death that are in no way related to the radioactive properties of the cargo can occur from vehicle accidents. As a result, accident statistics can be used that reflect the statistics for general commerce. Truck and rail accident statistics are compiled regularly by the Department of Transportation; Reference 17, which analyzes data for 1976 and 1977, was used to produce fatality unit-risk factors shown in Table 6. The injuries per kilometer of travel are calculated by multiplying the fatality factors by ratios of injuries to deaths obtained from Reference 18. From the table, it can be seen that 5.1×10^{-7} injuries and 3.0×10^{-8} deaths are expected to occur per kilometer of truck travel and 4.6×10^{-7} injuries and 3.4×10^{-8} deaths per kilometer of railcar travel. These values are averaged over all population zones and are not restricted in application to urban areas as the factors for pollutants are. The deaths and injuries predicted here occur immediately - either at the accident scene or during initial hospital treatment - and, unlike the deaths from pollutants, can be attributed to a specific shipment.

Table 6. Unit-Risk Factors for Vehicle Accidents

Mode	Injuries/km	Deaths/km
Truck	5.1×10^{-7}	3.0×10^{-8}
Rail	4.6×10^{-7}	3.4×10^{-8}

SUMMARY

In making decisions about projects involving the risk of transporting radioactive material, the non-radiological health effects have not been considered. To obtain a more complete picture of the risks involved, the non-radiological health effects from pollutants were estimated using what is believed to be the best available information, and the injuries and deaths from the trauma caused during accidents were calculated using general commerce transportation statistics.

Of all the pollutants considered, including particulates, SO_x, NO_x, CO, and hydrocarbons, only particulates and SO_2 could be used to calculate a total number of health effects. Their impact was assumed to represent the impact from all air pollutants generated by the vehicles.

The pollutant dispersal away from the route of travel was calculated using a line-source Gaussian dispersion model. Average meteorological data were input to the model. Data used to describe physical travel parameters were obtained from Reference 3. Other data and assumptions needed to complete the correlation from pollutant source terms to health effects were obtained from referenced public documents.

The effects models and the atmospheric models were combined with other appropriate data and assumptions to produce unit-consequence factors, for both truck and rail travel as summarized in Table 7. The values are 1.0×10^{-7} mortalities per kilometer in an urban area for truck and 1.3×10^{-7} mortalities per kilometer in an urban area for a railcar. Much uncertainty surrounds these values and their use requires proper caution and awareness of the assumptions that were made in calculating them. In fact, the assumptions and models used for calculating the health effects are such that the results must be considered as upper limits to the non-radiological impacts of pollutants emitted during transportation.

The unit-risk factors for accidents are also shown in Table 7. These non-radiological factors are calculated using average values for urban and rural population zones throughout the United States and are, therefore, not restricted just to urban population zones.

Table 7. Unit-Consequence and Unit-Risk Factors

Source	Factor	Health Effects Per Kilometer Truck	Rail
Pollutants	Unit Consequence[a,b]	1.0×10^{-7} (urban travel only)	1.3×10^{-7} (urban travel only)
Trauma from Accidents	Unit Risk - Injuries	5.1×10^{-7}	4.6×10^{-7}
	Unit Risk - Fatalities[c]	3.0×10^{-8}	3.4×10^{-8}

[a]Fatalities expected after some latency period, e.g., cancer death.

[b]If urban travel distance is assumed to be 5% of the total travel distance (90% rural and 5% suburban) as discussed in Reference 3, the unit-consequence factors due to pollutants for truck and rail shipments are 0.5×10^{-8} and 0.65×10^{-8}, respectively. These factors are less than one-fifth of the fatality risk factors resulting from accidents.

[c]Deaths at the scene of an accident or during initial hospital treatment.

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APPENDIX A

DESCRIPTION OF POLLUTANTS

PARTICULATES

The category of pollutants called particulates can include a vast array of particles with differing size, composition, and origin. The size of a particulate is very important in determining its dispersibility and potential health effects. Very large particles (> 100 μm diameter) will settle within a few meters of their source Smaller particles, in the range of 30 to 100 μm diameter, will settle more slowly and deposit within a few hundred meters, but particles in this size range and greater are not as hazardous as smaller particles because, if inhaled, they would be deposited upon entry to the respiratory tract in the nose or throat. These particles are coughed up or passed through the digestive system. Particles that are smaller than 30 μm will remain suspended for long periods of time and will be transported much greater distances. They are of concern in this report because they can penetrate deeper into the respiratory tract and have a longer residence time in the body. A particle 30 μm in diameter is also about the largest particle captured by typical particulate samplers generally used (Ref. A-1) by regulatory agencies, as well as utilities, for monitoring airborne particulates. Particles less than about 15 μm are considered respirable (Ref. A-1). That is, they can penetrate to and can be deposited in the lung. Particles less than 2 to 4 μm typically result from combustion and contain the majority of the sulfate salts. Larger particles, between 4 and 15 μm, are usually generated by abrasive and grinding actions (e.g. brake applications, tire wear, and roadway use) and not by combustion (Ref. A-2).

The chemical composition of the particulate pollutants is as varied as their size since they act as scavengers of other air pollutants. Indeed, particulates act as carriers of trace elements and hydrocarbons and may contain known carcinogens such as benzo(a)pyrene and its relatives (Ref. A-3). Sulfates may appear in the atmosphere as particulates. Small particles often contain adsorbed sulfates, trace heavy metals and organics, while larger particulates often contain silicates and aluminosilicates.

Because their compositions are so varied, it would be suspected that the sources of the particulates would also be varied. For example, particulates in rural areas will differ in composition from particulates in urban areas where they may come chiefly from industrial sources. In general, the sources of particulates may be industrial, agricultural, or transportation related. The mix of these sources, in turn, determines the composition of the particles in the atmosphere; particles will not have the same chemical composition in each community and, therefore, may not have the same health effect.

SULFUR POLLUTANTS

In order To describe sulfur pollutants, a number of sulfur compounds must be discussed. Aside from natural sulfur emissions, such as H_2S, the majority of anthropogenic sulfur is emitted during combustion as gaseous SO_2, which is a colorless, nonflammable, and nonexplosive gas. The gas may be detected by taste at concentrations of about 1 ppm and has an acrid odor at greater levels (> 3 ppm). Some SO_3 may be emitted directly, but the relative emission of SO_2 to SO_3 that results from combustion of fossil fuels is 40-80 to 1 (Ref. A-4). The SO_2 is converted at a rapid rate (1-5% per hour in daylight and less than 1% at night, Ref. A-5), to an intermediate product such as SO_3 and then to a sulfate or sulfuric acid. SO_2 is very hygroscopic so that, if humidity levels are high, SO_2 can be very rapidly converted to sulfuric acid aerosols.

CARBON MONOXIDE

Carbon monoxide is the only pollutant that has a quantifiable biologic measure of exposure or dose. The carbon monoxide molecule, which is similar in structure to the oxygen molecule, displaces oxygen from the hemoglobin in the blood and thereby lowers the oxygen-carrying capacity of the blood; the physiological effect is similar to that of anemia. Small amounts of CO in the blood are normal, but as exposure to heightened levels in a polluted atmosphere increases, the level in the blood increases (and symptoms increase with increased exposure) since the uptake is determined by the difference in partial pressures of the gas in the blood and in the alveolar air.

Sources of CO can be quite varied since CO results from the incomplete combustion of organic substances, but vehicular emissions are, by far, the major contributor to CO pollution.

HYDROCARBONS

The hydrocarbons referred to are those non-methane gases which interact with ozone and oxides of nitrogen to form photochemical air pollution. Generally, little is known about the health effects of these gases, which result from combustion of organic substances, except that the majority of them are irritants to the eyes and lungs (Ref. A-7). These pollutants are also strongly associated with vehicular sources.

PHOTOCHEMICAL OXIDANTS

Oxidants can be considered secondary products of fossil fuel combustion, especially the combustion associated with transportation. They result from the interaction of effluent hydrocarbons, oxides of nitrogen, and ultraviolet light. Ozone and PAN are examples. Oxidants irritate the eyes, nose, and throat.

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APPENDIX B

HEALTH EFFECTS

This appendix describes the potential health effects that may result from increased concentrations of the pollutants in Appendix A. Unfortunately, this prediction is not an easy or simple task because the needed data relating mortality or morbidity to pollutant exposure are difficult and expensive to obtain. Morbidities are not considered because fewer studies have been completed for morbidity than for mortality and the uncertainties are greater accordingly.

In principle, data can be gathered in three ways: quantitative laboratory studies on man and animals, clinical studies, and epidemiological studies. The first two are scientifically acceptable but are not generally effective because they require extrapolation from very controlled environments to the environments that people actually live in. These techniques isolate important parameters but then require interpretation to relate the results from ideality to reality. Epidemiological studies deal with the complex environment people live in, but they are fraught with problems because the results reflect a complex exposure environment that must be simplified so that an analysis can even be attempted. In short, epidemiological studies consider the actual environment where all influences and synergisms are considered and results from them are not readily interpreted.

The basis for the non-radiological impacts estimated in this report is the substantial body of literature on laboratory and epidemiological studies that has established a relationship between air pollution and ill health and increases in the mortality rate. This report makes no attempt to describe this assortment of literature because such descriptions are presently available in many references (Ref. B-1,2,3,4, 5,6,7) and relies on suggestions from Reference B-7 as to which quantitative relationships should be used to estimate health effects. The fact that uncertainty exists regarding dose-response relationships for these pollutants is emphasized.

Determining the health effects of atmospheric pollutants is a complex and confounding problem as manifest in epidemiological studies that have been performed to determine what the effects of some pollutants are. The strongest evidence from the studies indicates that air pollution does have an effect on mortality rates (Ref. B-3,7,8), but that the reliability of the analyses is insufficient to state selectively that the effects are a result of particular pollutants.

The majority of data exists for sulfur compounds and particulates. Historically, they have been monitored because they were present in high concentrations during acute air pollution episodes where premature mortality was observed. SO_x and particulates were also measured because they were easy to measure and because simple measurement techniques were available. It has become more apparent that they were not the only pollutants that were present during these episodes.

Air pollution control in the U.S. has focused on SO_2. Unfortunately, there is no statistical evidence that the SO_2 controls (for example, in New York City) have reduced mortality rates (Ref. B-6) even though many epidemiological studies impute SO_2 as having an impact on mortality rates. Without exception, conflicting results for each of the pollutants considered can be found. Currently, however, the pollutants that are supported by the majority of the literature (recently in Ref. B-9) as having a positive correlation between increased concentrations and increased mortality are particulates, including sulfates. The direct health effects of SO_2 and NO_x are weakly substantiated, and CO, hydrocarbons, and photochemicals (especially ozone) may not have a detectable effect at ambient concentrations.

SO_x and particulates together may have an impact on human health (mortality), but it is not possible, using present analytical techniques, to disaggregate their

effects. Indeed, because their effects may be synergistic, one might not want to separate them or may be in error by doing so. Despite this fact however, British scientists believe that their air quality control measures for particulates alone (they do not control sulfur dioxide as the U.S. does) have reduced particulate concentrations which in turn have reduced mortality rates (Ref. B-3). Earlier epidemiological studies have indicated strong correlation between SO_2 and mortality rates (Ref. B-10) but recent evidence suggests that this correlation is not so strong (Ref. B-6).

NO_x has been imputed to have an effect on mortality, but its effect may result more from its reaction products. Evidence relating photochemicals and health effects is inconclusive; ozone, however, has no apparent effect. That is not to say that their health effects may not be significant for sensitive portions of the population. It is interesting to note, that even though Los Angeles has a severe photochemical pollution problem, it cannot be demonstrated that residents of Los Angeles have a higher mortality rate because of it (Ref. B-1, B-3).

Despite all of the information above, an underlying concern of many scientists is that the air pollutants measured today may simply be a proxy for pollutants that are currently not measured, or for some other, as yet unrecognized, variable that may not be related to air pollution. Trace metals are now becoming suspect for their role in increasing health effects (Ref. B-2, 5, 12).

Why is there such uncertainty? The techniques used to analyze epidemiological data are not adequate to disaggregate the effects of individual pollutants and cannot prove causation. Almost all epidemiological studies have used multivariate linear regression analysis or some variation of it to analyze the epidemiological data. Problems with these techniques have been documented in numerous places many times before (Ref. B-7, 10, 11, 12, 13, 14).

Before discussing the problems, it is important to indicate that multivariate linear regression analysis is based on an equation that can typically be represented as:

$$\text{Health Effects} = Ax + By + Cz + Dw + Eu + \ldots \qquad (B\text{-}1)$$

This equation can contain as many parameters as required. In this example, x, y, z, w, and u may represent data such as pollution levels, population density, income, race, doctor availability, and smoking habits. The coefficients A, B, C, D, and E are weighting factors for the data that are obtained by a "best fit" of observed health effects to the presumed variables. The number of variables can be very large (a condition which leads to possible spurious relationships) or the number can be small (a condition which may result in biased estimation from a priori elimination). If few variables are specified, the unaccounted for variables are, in effect, presumed to be constant. Which variables to include is a major problem. The adjustment for factors such as weather, smoking, and population density has a large impact on results. Regression analysis "fishes for" a good fit and may or may not produce meaningful correlations.

The linear regression model assumes linearity, but dose-response relationships may be curvilinear. Thus, results from linear analysis should be applied over limited ranges of pollutant concentrations near the mean concentration.

Other problems are associated with epidemiological studies themselves: nonuniform response to pollutants throughout the population; adjustments for individual behaviors (including health related items as smoking, occupation, socioeconomic status, air pollution in the home); mobility of populations; lack of data regarding historical pollution levels (time-integrated levels); sampling techniques; and limited sampling

locations that do not provide representative air pollution levels.

Despite the problems with how dose-response relationships are generated and despite their shortcomings, the relationships are the best that are available and can be valuable provided that they are used with caution.

One additional aspect regarding dose-response relationships, the concept of a threshold, must be discussed. If a threshold exists, a concentration below the threshold level will produce no effect and one above would produce an effect. Generally, a threshold for the effects from air pollution levels is not believed to exist (Refs. B-4, 7, 10, 14), that is, there is no level at which mortalities will not occur. In this respect, air pollution control limits set by the Environmental Protection Agency are misleading because one might be lead to believe that no effects will occur below the control levels. In any event, implicit in linear regression analysis is that there is no threshold level.

With the techniques and problems associated with the dose-response relation described, a quantitative estimate is given in the body of the report to relate the increase in average air pollution levels to increase in mortalities. This is done using a construct called an elasticity, which can be described by referring to Equation (B-1). After a regression analysis is completed, weighting factors (A, B, C, D, E, . . .) have been determined for a set of mean values of the data (x, y, z, u, w, ...). Using these weighting factors and mean values, the mortalities calculated from Equation (B-1) should correspond to the observed number of mortalities in a study area. An elasticity is used to describe quantitatively the the change in mortalities due to perturbations from mean values of data inputs. If one of the mean values is perturbed by some value while all others are held constant, a new value for health effects results. As an example, an elasticity for pollutants is described as the percentage change in mortalities that would be predicted should the pollutant concentration change by 1% from its mean value while all other data are held constant at their mean value. This relationship is represented by Equation (B-2). The elasticity value to be used in this report is 0.05, and the basis for this value is an interpretation of results from References B-10 and B-13 as described in Reference B-7. This value means that a 1% increase of pollution level from mean levels will result in a 0.05% increase in mortality rate.

$$\frac{\Delta \frac{\text{mortality}}{\text{mortality}}}{\Delta \frac{\text{pollutant concentration}}{\text{mean pollutant concentration}}} = \text{Elasticity} \qquad \text{(B-2)}$$

It will be applied to total pollutant levels (more exactly to the total for SO_2 and particulates only).

It is important to delineate the problems with applying an elasticity value to a generic population. By using a specific value, it is assumed that value applies to all of the relevant population. It should be noted here that only metropolitan area population subgroups are examined in References B-10 and B-13. The elasticity also assumes that all population units will experience the same reduction. In addition, use of one elasticity value assumes that the elasticity is constant for the pollutants considered and over a range of mean concentrations. However, the value is only relatively constant about a range near the mean pollutant levels for which it was calculated. As a result, mean pollutant levels will be used as reported in the references from which this elasticity value was generated.

In summary then, an elasticity value has been chosen that represents the results from current epidemiological studies. This value will be applied to the SO_2 and particulate incremental concentrations. It is assumed that air pollutants have an adverse affect on mortality rates but that disaggregated effects of individual pollutants cannot be determined. Elasticities will be applied to incremental increases to mean concentrations as determined in the studies which are the basis for the elasticity value.

The primary concern of transportation planners and health officials involves any health consequences to the people living along the transportation routes. Since pollutants from mobile vehicles are emitted near ground level, people in the immediate vicinity of the transportation routes will be exposed to greater concentrations and, as a consequence, may suffer most of the health effects. No threshold will be assumed for health effects, except that a distance chosen for the maximum distance from a highway may have the effect of imposing a threshold. The effect of varying the maximum distance on the number of health effects predicted is examined in Appendix C.

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APPENDIX C

EFFECT OF VARYING D_{max}

The ultimate values of the unit-consequence factors for the pollutants are dependent upon the value selected for D_{max} used in Equation (C-1), which can be applied to calculate pollutant concentrations.

$$\frac{\bar{X}_i}{Q_i} = \frac{K_1 \, I}{(D_{max} - D_{min})\,\bar{u}} \left(\frac{2}{\pi}\right)^{1/2} \qquad \text{(C-1)}$$

where

$$I = \int_{D_{min}}^{D_{max}} \frac{dx}{x(1 + 0.0003x)^{-1/2}}$$

By selecting a value for D_{max}, the user is really specifying a distance beyond which the effects of the pollutants are not considered. If the health effects associated with pollutants do not have a threshold of minimum concentration as discussed in Appendix B, then the resultant unit-consequence factors may underpredict the number of health effects. Nevertheless, certain practical factors must also be considered that necessitate selecting a finite value for D_{max}: Equation (C-1) (Gaussian dispersion model) is accurate over short distances from the source (not beyond 5,000 to 10,000 m); urban dispersion coefficients (σ_z) used in Equation (C-1) are also not valid beyond 10,000 m (Ref. C-1); urban area population densities begin to decrease at some finite distance; and meteoro logical and dispersion parameters such as $\bar{u}$ and σ_z (see main text, Equation 1, for description) in Equation (C-1) maybe more important in determining the ultimate concentrations.

Since many values for D_{max} are possible, Table C-1 is presented to allow the reader to select the unit-consequence factors that most closely apply to the practical problem with which he is dealing.

Table C-1. Unit-Consequence Factors for a Selection of Values for D_{max}

D_{max}(m)	Rail $\left(\frac{\text{deaths}}{\text{km}}\right)$	Truck $\left(\frac{\text{deaths}}{\text{km}}\right)$
805	1.3×10^{-7}	1.0×10^{-7}
1000	1.4×10^{-7}	1.1×10^{-7}
1500	1.5×10^{-7}	1.2×10^{-7}
2000	1.7×10^{-7}	1.3×10^{-7}
2500	1.8×10^{-7}	1.4×10^{-7}
3000	1.9×10^{-7}	1.5×10^{-7}
4000	2.0×10^{-7}	1.6×10^{-7}
5000	2.1×10^{-7}	1.7×10^{-7}
6000	2.3×10^{-7}	1.8×10^{-7}
7000	2.4×10^{-7}	1.8×10^{-7}
8000	2.4×10^{-7}	1.9×10^{-7}
9000	2.5×10^{-7}	2.0×10^{-7}
10,000	2.6×10^{-7}	2.0×10^{-7}

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