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UCRL-51930
Vol. 2

**ENVIRONMENTAL EFFECTS OF ENERGY PRODUCTION
AND UTILIZATION IN THE U.S. VOLUME 2:
PUBLIC HEALTH EFFECTS**

Compiled and edited by Herbert W. Newkirk

June 1, 1976

Prepared for U.S. Energy Research & Development
Administration under contract No. W-7405-Eng-48

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Printed in the United States of America
Available from

National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road
Springfield, VA 22161

Price: Printed Copy \$; Microfiche \$2.25

Page Range	Domestic Price	Page Range	Domestic Price
001-025	\$ 3.30	326-350	10.00
026-050	4.00	351-375	10.50
051-075	4.50	376-400	10.75
076-100	5.00	401-425	11.00
101-125	5.25	426-450	11.75
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LAWRENCE LIVERMORE LABORATORY
University of California, Livermore, California 94550

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Foreword

Historically, concern in the United States over resources has focused on whether or not there would be enough food, energy, and materials to meet current and future needs. Today, there is also concern about the ability of our land, air, and water to absorb all the effluents that energy production and utilization generates. The first annual Environmental Quality report accurately epitomizes this fact: "The conflicts between the consumption patterns that we all want, and the environmental ill effects that we all wish to avoid, is sharper for energy than perhaps any other aspect of natural resource use."

The issues being debated are complex, and while the written pieces on the subject number in the thousands, to our knowledge there is not yet available a summary of the facts related to the environmental effects of energy production and utilization. We have attempted to compile this relevant information and publish it in three volumes. It is anticipated that these handbooks will provide a data bank of environmental information that will be helpful in making the many critical cost/benefit choices that energy production and utilization demands.

Volume I deals with sources (what the emissions are and where they come from), trends (quantities of emissions and their dispersion with time), and costs of control (what it takes in time, energy, and money to meet minimum standards). Volume II concerns itself with the public health effects of energy production and utilization. Volume III summarizes the various techniques for controlling emissions, technological as well as economic, social, and political.

Each volume is divided into sections dealing with the atmosphere, water, land, and social activities - each division indicating a particular sphere of man's environment affected by energy production and use. The sources of information that were used in this study included textbooks, journal articles, technical reports, memoranda, letters, and personal communications. These are cited in the text at the end of each subsection and on the applicable tables and figures.

Good writing always benefits from both criticisms and suggestions. Comments on this edition are solicited to guide the compiler in subsequent versions of these fact files.

Finally, in a project of this magnitude many people contribute both their time and talent and deserve special recognition. To our typists, Joan Rien, Betty Irving, Virg Jaramillo, and Donna Romero, who typed the rough drafts; to Virginia Caswell of LLL's Technical Information Division who performed a superb job of literature searching in this field; to Kent Cummings for his skill in editing the volumes; and to the LLL staff members who reviewed portions of the rough drafts, Stuart Winter (Energy Planning and Analysis Group), John Tewes (K Division), Hugh Ellsaesser (C Division), David W. Dorn and Carroll Maninger (Technical Application Group) we extend our thanks.

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Glossary

Coh	- Coefficient of haze
DL _{CO}	- Carbon monoxide diffusing capacity
FEV _{1.0}	- Forced expiratory volume (100%)
FEV _{0.25}	- Forced expiratory volume (75%)
FRC	- Functional residual capacity (Fixed)
GSD	- Genetically significant dose
LC ₁₀₀	- Lethal concentration (100%)
LC ₅₀	- Lethal concentration (50%)
MCP	- Maximum concentration permissible
MPL	- Maximum permissible limit
PTS	- Permanent threshold shift
TLV	- Threshold limit value
USW	- Ultra-shortwave therapy
VC	- Vital capacity

Section I

Summary of Public Health Effects

Although the acute effects of emissions on health are easily identified, it continues to be difficult to establish the presence or absence of harmful effects from permanent exposure to low dosage levels. It is likely that present localized levels of emissions in cities aggravate certain ailments and possibly facilitate their development in susceptible individuals or groups.

We gain the impression from reading the extensive documentation in this field that there is a tendency to exaggerate the associations between emission levels and health effects - making the emissions seem more hazardous than data supports.

The scientific arguments marshalled by opposing sides in debate continue to be so perplexing that there is a tendency to overlook a principal objection, simply, that it appears unpleasant. Nevertheless quantitative analysis of the effects of emissions on public health is still scarce, and the work being done on these topics is timely. Estimated health effects in 1973 associated with electric power production are given in Table 1.

Statistical correlation, the urban factor, and the classical air emission episodes of the Meuse Valley in 1930, Donora in 1948, and London in 1952 continue to remain the principal evidence defining health effects of air emissions.

The crucial question that still remains unanswered is whether or not these effects are noise in the data or are caused by misidentification of causal agents or variations in the susceptibility of individuals. The structure of our present control program continues to rest on the presumption that the latter premise is correct and on the philosophy that susceptible individuals must be protected.

The hypothesis is still not proven that some atmospheric emissions are mutagenic and increase the death rate from some chronic diseases. However, there is consistent statistical evidence from 38 metropolitan areas of the U.S. that increasing concentrations of air emissions and, in particular, of nitrogen dioxide and sulfur dioxide, are associated with increasing death rates from chronic diseases associated with senescence, such as various forms of cancer, total cancer, and diseases of the heart.

Because the Federal Clean Air Act of 1970 acknowledges that the statistical fluctuations of emissions and meteorological conditions can lead to severe episodes of concentrated air emissions with consequent acute illness and mortality, it requires each state to provide an *Episode Control Plan* for regions subject to significant discharge, to prevent ambient concentrations from reaching levels causing significant harm to health of persons. These levels are given in Table 2.

Table 1. Estimated health effects in 1973 associated with production of electric power.*

Fuel	1973 kWh(e) $\times 10^9$	Equivalent No. 1000 MW(e) plants	Estimated deaths	Estimated disabilities	Unknowns
Coal	846.0	128.2	2,000-16,000	26,000-39,000	Increased cancer, other chronic disease, mutations
Oil	310.7	47.1	100-5,000	4,000-9,000	Increased cancer, other chronic disease, mutations
Gas	336.0	50.9	7	700	Increased cancer, other chronic disease, mutations
Nuclear	83.3	12.6	9-20	60-300	Increased risk of catastrophic accident
Hydro	271.1	41.1	-	-	Increased risk from dam failure accident
Wood, waste, geothermal	2.3	0.3	-	-	-
<u>Totals</u>	1849.4	280.2	2,000-21,000	31,000-49,000	

Approximate annual total deaths in U.S. = 2,000,000

Percent associated with electricity production = 0.1 - 1%

Approximate number of deaths in U.S. ages 1-74 = 1,100,000

Percent associated with electricity production = 0.2 - 1.9%

*[Hamilton, L. D., and S. C. Morris, Brookhaven National Laboratory, Upton, NY, Rept. BNL-19265 (1974).]

Table 2. Significant levels for episode control.*

	Significant harm level	Alert level	Warning level	Emergency level
SO ₂ : 24-h average				
μg/m ³	2620	800	1600	2100
ppm	1.0	0.3	0.6	0.8
Particulate: 24-h average				
μg/m ³	1000	375	625	875
COH	8.0	3.0	5.0	7.0
SO ₂ particulate (product)				
μg/m ³ (× 10 ³)	490	65	261	393
ppm × COH	1.5	0.2	0.8	1.2
CO: 8-h average				
mg/m ³	57.5	17	34	46
ppm	50	15	30	40
4-h average				
mg/m ³	87.3			
ppm	75			
1-h average				
mg/m ³	144			
ppm	125			
Photochemical oxidants (O ₃) ^a				
4-h average				
μg/m ³	800			
ppm	0.4			
2-h average				
μg/m ³	1200			
ppm	0.6			
1-h average				
μg/m ³	1400	200	800	1200
ppm	0.7	0.1	0.4	0.6
Nitrogen dioxide				
1-h average				
μg/m ³	3750	1130	2260	3000
ppm	2.0	0.6	1.2	1.6
24-h average				
μg/m ³	953	282	565	750
ppm	0.5	0.15	0.3	0.4
Meteorological condition	Stagnation for 12 h			

^aProposed change to single significant harm level of 1200 μg/m³ (0.60 ppm), 1-h average, and emergency level of 1000 μg/m³ (0.50 ppm).

*[Federal Register 39, March 13, 1974, pp. 9672-9675.]

Section II

Air Emissions

CHEMICAL FACTORS

Carbon Monoxide

Carbon monoxide combines preferentially with red blood cells to form carboxy hemoglobin. This compound cannot carry oxygen, so the body suffocates. (Fig. 1).

Carbon monoxide is capable of reacting with other hemoproteins and may interfere with certain enzyme systems. Performance decrements and adverse cardiovascular effects become evident when there is more than 5% carboxy hemoglobin in the blood. The relationship between carbon monoxide exposure from smoking and carbon monoxide exposure from the ambient air, and the relationship between carbon monoxide exposure and cardiovascular disease, are not clear.

It is accepted by all workers that 20% carboxyhemoglobin (equivalent to saturation at about 120 ppm carbon monoxide) can cause symptoms and impair performance but healthy subjects rarely complain of any symptoms with saturations less than 10% (~60 ppm CO).

There is little convincing evidence that concentrations of gas giving less than about 8% saturation (~48 ppm CO) has any effect on perception or "intellectual" performance, and there is evidence that electroencephelogram activity remains unaffected until saturations approach 33% (~200 ppm CO).

The results of sound work on normal subjects have shown that saturations of about 5% (~30 ppm CO) have measurable effects on cardiac functions. For comparison, the present carbon monoxide standards, designed to prevent carboxyhemoglobin saturations exceeding 2%, are 9 ppm for 8 h and 35 ppm for 1 h.

References - Carbon Monoxide

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Lawther, P. J., in Proceedings of the Conference on Fuel and the Environment, November 26-29, 1973 (Institute of Fuel, London, 1973), Vol. 2, p. 123.

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Theodore, J., R. D. O'Donnell, and K. C. Back, "Toxicological Evaluation of Carbon Monoxide in Humans and Other Mammalian Species," J. Occup. Med. 13, 242 (1971).

Zimmerman, P. W., W. Crocker, and A. E. Hitchcock, "The Effect of Carbon Monoxide on Plants," Contrib. Boyce Thompson Inst. 5, 195 (April-June 1933).

Fig. 1. Carbon monoxide combines preferentially with red blood cells to form carboxy hemoglobin, which cannot carry oxygen. [Myers, P. S., "Nation's Cost/Benefit Ratio Weighs Heavily on Auto Emissions," SAE Journal (March 1970).]

Nitrogen Oxides

The significance to human health of currently encountered atmospheric levels of nitrogen dioxide is inadequately known. Very few epidemiological studies have been performed, and these have yielded conflicting results. In most instances, the studies in California have failed to correlate exposure to oxides of nitrogen with ventilatory dysfunction, respiratory symptoms, chronic lung disease, or mortality.

Although epidemiological data do not offer a definitive answer to the question of whether or not high ambient levels of nitrogen dioxide cause illness, the Chattanooga study, which bears most directly on this point, suggests that in certain populations exposure to high ambient concentrations (0.08 ppm) of nitrogen dioxide is associated with an increased risk of respiratory infection.

However, since more recent data indicate that (1) other pollutants, such as nitrates and sulfates, may have acted in concert with nitrogen dioxide at Chattanooga; (2) the nitrogen dioxide measurements were in themselves somewhat inaccurate; (3) follow-up studies of the affected population did not disclose sequelae; and (4) recent epidemiologic studies have not demonstrated illness due to oxidant exposure of similar degree, an increase in the standard to 0.08 ppm on an annual basis would be consistent with this new information.

Definitive evidence demonstrating that exposure to nitrogen dioxide can be deleterious to respiratory functions comes from two sources: (1) acute exposure of humans to high levels of nitrogen dioxide invariably results in respiratory disease and (2) experimental animals exposed to elevated levels of atmospheric nitrogen dioxide develop pathologic changes resembling emphysema and an enhanced susceptibility to bacterial infection.

A range of adverse effects has been described which correlate with the degree of exposure. Eye and nasal irritation occurs after exposure to 15 ppm of nitrogen dioxide. Pulmonary discomfort is noted at levels of 25 ppm, and bronchiolitis with focal pneumonitis occurs after exposures of 25 to 75 ppm of nitrogen dioxide. The duration of these exposures was put at less than 1 h. Comparable short periods of exposure to 150 to 200 ppm causes fatal pulmonary fibrosis. Higher exposures are associated with acute pulmonary edema, and sometimes death.

Assessments of the environmental hazard of nitrogen dioxide can be confined to the pulmonary effects of the emissions, because in humans extra-pulmonary abnormalities of biological significance have not been recorded, and in experimental animals the lung is the target organ for nitrogen dioxide induced injury.

The NO_x effect on the earth's ozone shield is based on highly complex, chemical models which at this time remain inadequately tested. Admittedly, several processes known to be significant are ignored; consequently, current results differ in several important respects from available observational data. (See the sections on photochemical oxidants and electromagnetic emissions.)

References - Nitrogen Oxides

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Grobecker, A. J., S. C. Coroniti, and R. H. Cannon, Jr., Report of Findings; The Effects of Stratospheric Pollution by Aircraft (Department of Transportation Climatic Impact Assessment Program, Washington, DC, 1974).

Photochemical Oxidants

Numerous epidemiological studies have been conducted on the effects of photochemical oxidants. The only convincing relationships (Table 3) have been those for eye irritation, at times accompanied by nasal irritation (when oxidant concentrations exceeded 0.10 ppm); for aggravation of symptoms among asthma patients (when oxidant concentrations exceeded 0.13 ppm); and for impairment of athletic performance (for hourly oxidant concentrations in the range 0.03 to 0.30 ppm).

No convincing relationships were observed between short-term variations in in oxidant levels and daily mortality or hospital admissions. Most of the reported studies do suggest, however, that photochemical oxidants are potentially hazardous environmental contaminants.

Recent work demonstrates the synergistic effect of ozone and sulfur dioxide enforces the concept that emissions cannot be considered separately when standards are promulgated and that complex interrelationships between them must be considered. It is also important to know whether phenomena well demonstrated in animals, such as tolerance or cross protection whereby exposure to ozone provides some protection to other irritant oxidants, occur in human beings.

Photochemical oxidants tend to exert their toxic effect on the body through inhalation. Alterations in the pulmonary function of the lungs are among the effect. Other effects include pathological, chemical, and biological changes in the lungs and other organs.

Occupational exposure to ozone has been documented by several studies; however, many of the studies are complicated by the presence of emissions other than ozone. Most of the studies were on subjects exposed to ozone concentrations on the order of 0.3 ppm and produced no convincing evidence that functional impairment was due to ozone; further studies should be conducted.

Laboratory experiments have shown more definitive results. The threshold level at which nasal and throat irritation will result in humans under prolonged exposure to ozone appears to be about $590 \mu\text{g}/\text{m}^3$ (0.3 ppm). For short-term exposures there is no firm information concerning exposure under $980 \mu\text{g}/\text{m}^3$ (0.5 ppm). Exposure to concentrations of 980 to $1960 \mu\text{g}/\text{m}^3$ (0.5 to 1.0 ppm) for periods of 1 to 2 h produces changes in the pulmonary function: increased airway resistance, decreased vital capacity, decreased

carbon monoxide diffusing capacity, and decreased forced expiratory volume (Fig. 2). Concentrations in the range of 1960 to 5900 $\mu\text{g}/\text{m}^3$ (1.0 to 3.0 ppm) produce extreme fatigue and lack of coordination in some people.

References - Photochemical Oxidants

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Goldsmith, J. R., and J. A. Nadel, J. Air Poll. Cont. Assoc. 19, 329 (1969).

McCarroll, J., Calif. Air Environment 4 (Winter 1974).

Zelac, R. E., et al., Environ. Sci. 4, 262 (1971); 4, 325 (1971).

Table 3. Summary of selected health effects on exposure to photochemical oxidant.*

Extent and duration of exposure	Observed effects
A. Demonstrated adverse effects	
0.1 ppm and above	Eye irritation in man
0.2 ppm for 3 h	Decreased visual activity for dark adaptation and middle vision ranges in man
0.25 ppm daily maximum hourly exposure	Increased frequency of asthmatic attacks in man
0.25-0.30 ppm daily maximum hourly exposure	"Threshold" for increased coughing and chest discomfort in man
0.3 to 0.8 ppm average for 8-h workday	Complaints of chest discomfort and throat irritation by workers
0.37 ppm for 2 h	Decreased pulmonary function after moderate exercise in man
B. No demonstrated adverse effects	
0.13 ppm daily maximum hourly exposure	No significant increase in asthmatic attacks in man
0.05-0.28 ppm daily average exposure	No effect on ventilatory performance of school children
0.2-0.25 ppm average for 8-h workday	No complaints of chest discomfort and throat irritation of workers
Ambient concentrations in Los Angeles	No significant effects on mortality due to occurrence of smog-alert days in man

*[Downing, P. B., Environ. Affairs 4, 738 (Fall 1975), reprinted by permission.]

Fig. 2. Air-quality criteria for photochemical oxidants. Conversion of effects data to criteria. [Lawrence Livermore Laboratory, staff compilation.]

Sulfur Oxides

While energy needs have increased and clean sources of energy are limited, any reduction in standards for SO_2 and particulates that permits increased community emissions and concentrations will certainly result in a significant increase in the number of people who may become ill and die (Figs. 3, 4 and 5). If standards are not met, the excess number of asthma attacks have been estimated to be 6 million to 10 million each year, and could total over 50 million during the years 1975 through 1980.

The levels of SO_2 which are present in large urban areas appear to expose significant numbers of people to health risk during conditions of temperature inversion and air stagnation. Nasal mucus flow rate, airway resistance, and subjective response were studied in 15 young men during 6-h exposures to 1, 5, and 25 ppm sulfur dioxide. A significant decrease in nasal mucus flow rate during the 5- and 25-ppm exposures was observed. This decrease was greatest in the anterior nose and in subjects with an initially slow mucus flow rate. Pharyngeal air samples yielded less than 1% of the SO_2 inhaled, even after a 6-h exposure to 25 ppm SO_2 . An increased nasal airflow resistance and a fall in forced expiratory volume in 1 s and forced expiratory flow during the middle half of expired volume was found at all exposure levels, but there was no change in "closing volume." Discomfort was proportional to SO_2 concentration, but never excessive. Subjects with initially slow nasal mucus flow rates experienced the greatest discomfort.

Evidence has now begun to appear that significant drops in emission levels in recent years have not been accompanied by reductions in respiratory death rates. A recent study of SO_2 in New York City is informative. It found that during the period 1963 to 1972 the average level of SO_2 declined 60% (i.e., 2-1/2-fold). Over the same period there was no decline in death rates in any of the diseases usually attributed to SO_2 . But even more surprising, it found that the fraction of excess deaths "explained" by regression on SO_2 concentrations doubled. This seems to imply that SO_2 at the more recent levels is 5 times as lethal, yet is still causing the same death rate as when the mean level was 2-1/2 times higher.

In the Harlem area of New York City, no relationship between daily visits to emergency rooms and daily levels of either sulfate or sulfur dioxide have been observed, whereas in the Bedford-Stuyvesant area of Brooklyn, a relatively strong correlation between daily visits for asthma and daily levels of sulfur dioxide but not with sulfate have been observed over and above the

effect of temperature. In both areas there was a strong relationship between daily visits for asthma and the first cold spells of the Fall season. The average male/female ratio for asthma visits to hospital emergency rooms of both areas in the age group under 13 years of age was 1.7 and for over 13 years of age group was 0.6.

It appears from the data that Brooklyn presents a different picture for the environmental influence on asthma attacks. Even though it has been shown by previous investigators that asthmatics are sensitive to sulfur dioxide, we feel that in our case sulfur dioxide is not necessarily the causative agent, but might rather implicate some other confounding variable (most likely an environmental agent) that is disseminated in the general Bedford-Stuyvesant area in a day-to-day pattern similar to sulfur dioxide. We infer this from the fact that levels of sulfur dioxide were somewhat higher on the average in the Harlem area than they were in the Brooklyn area.

New York City hospitals reported a rise in respiratory infections and cardiac diseases during a 10-day period with daily 0.07 to 0.86 ppm SO_2 ; the rise in hospital admissions was evident before 0.25 ppm was reached. A similar occurrence was noted in Rotterdam during the few days with 0.11 to 0.19 ppm SO_2 .

As for long-term studies, one in Genoa, Italy, demonstrated a higher incidence of respiratory disease in an area with an annual mean of 0.092 ppm SO_2 vs areas with annual means of 0.037 ppm and 0.028 ppm, respectively. Another study, done in areas near a common smelter, demonstrated again the correlation between SO_2 concentration and respiratory symptoms; after the smelter installed a higher stack for better dispersion, the respiratory symptoms disappeared.

Sulfur dioxide, particulates, and, even more, their products - sulfuric acid and acid sulfates - seriously affect the health of significant numbers of humans, even at the levels currently considered to be safe. The major target organs are the heart and lungs; therefore, large numbers of individuals in the population are in a high-risk category regardless of the source of stress. These emissions may act over long periods of time to produce respiratory tract changes in normal individuals. Their major acute or short-term action appears to be as disease and death accelerators, significantly shortening the lives of those who have poor adaptive capacities.

Studies of episodes suggest that a rise in the daily death rate occurred when the concentrations of sulfur dioxide rose abruptly to levels at or about $715 \mu\text{g}/\text{m}^3$. Daily concentrations of sulfur dioxide in excess of $1500 \mu\text{g}/\text{m}^3$ for 1 day in conjunction with levels of suspended particles exceeding $2000 \mu\text{g}/\text{m}^3$ appear to have been associated with an increase in the death rate of 20% or more over baseline levels. This same effect has been observed at lower sulfur dioxide levels when the maximum emission levels lasted for longer and shorter periods.

At concentrations ranging from 300 to $500 \mu\text{g}/\text{m}^3$ of sulfur dioxide (24-h mean), with low particulate levels, increased hospital admissions of older persons for respiratory disease have been noted; absenteeism from work, particularly with older persons, also has been noted. At lower levels, i.e., at concentrations as low as 105 to $120 \mu\text{g}/\text{m}^3$, there have been observations of increased frequency and severity of respiratory symptoms and lung disease.

Studies carried out in four distinct geographic areas of the U.S. involving 20,472 children and adults clearly linked excessive, acute respiratory disease with communities heavily concentrated with sulfur dioxide and suspended sulfates (Table 4). Exposure of 3 years or longer was an important component in increased risk for acute respiratory disease. Laryngotracheobronchitis was the major condition productive of excess, acute, lower-respiratory disease in children in heavily concentrated communities in the Salt Lake Basin and Rocky Mountain communities. The effect of emissions on excess, acute respiratory disease was found to be independent of socioeconomic status and cigarette smoking.

The levels of air emissions for sulfur compounds and particulates decreased somewhat between two sampling periods 5 to 6 years apart in Berlin, New Hampshire. Population surveys showed that there was a lower prevalence of respiratory symptoms in 1967 than in 1961. These differences persisted after age standardization and when comparisons were made within a smoking category. Changes in inhaling habits or the use of filter-tip cigarettes did not seem to be likely explanations for this decrease in prevalence. Tests of pulmonary function were less clear cut but did indicate that a slight improvement in pulmonary function had possibly occurred. It is believed that these changes may be related to the decrease in the levels of air emissions.

At sulfuric acid concentrations of $1100 \mu\text{g}/\text{m}^3$, workers and exposed volunteers immediately experienced irritation of the throat and nose. At 2400- to $6000 \mu\text{g}/\text{m}^3$ sulfuric acid concentrations, all subjects had severe

throat irritation and cough. Sulfur dioxide, on the other hand, will not cause such symptoms in most humans even at concentrations of $13,400 \mu\text{g}/\text{m}^3$ (5 ppm). Sulfuric acid levels of $500 \mu\text{g}/\text{m}^3$ provoked rapid increases in the respiratory rate, and the effect continued for several minutes after exposure.

A study of asthmatics carried out in the Metropolitan New York area showed that when minimum temperatures were 30 to 50°F , dose-related increments in asthma attack rates were associated with increments in total suspended particulates and suspended sulfates but not sulfur dioxide. The threshold level for morbidity excess occurred at daily sulfate levels of $7.3 \mu\text{g}/\text{m}^3$ when minimum temperature exceeded 50°F , while the excess morbidity threshold increased to $12 \mu\text{g}/\text{m}^3$ sulfate when minimum temperatures were -2 to 10°C (30 to 50°F). No firm evidence could be found to associate elevations in sulfur dioxide (100 to $180 \mu\text{g}/\text{m}^3$) with excessive asthma attack rates on either cold or warm days. A linear regression of asthma attack rate on temperature showed that temperature alone had little effect on asthma attack rates.

Adverse health effects are associated principally (80%) with the particulate measure (smoke shade); only a small fraction (20%) are associated with the SO_2 measure (Table 5).

If we regress mortality on temperature and SO_2 alone, the effects attributable to the SO_2 measure are increased approximately threefold. This shows that SO_2 serves as an index of particulate concentration and, presumably, of volatile emissions also. Moreover, it shows that other mortality and morbidity studies which use SO_2 as a principal measure of air emissions are undoubtedly measuring the effects of particulates, or even of other volatile components, rather than those of SO_2 .

Within the limits of the accuracy of the data, despite a 70% reduction in SO_2 levels in the latter period (1970-1972) as compared to the earlier years, there has been no reduction in the adverse health effects associated with SO_2 measure. This tends to confirm that SO_2 is serving as a day-to-day indicator of air quality rather than functioning as a harmful emission.

Daily emission levels reflect the joint effects of the volume of emissions which are emitted into the atmosphere, and meteorological conditions.

Whether fuels contain 0.3% sulfur or 3% sulfur, the daily SO₂ level is responsive to these joint effects. Of course, its absolute level will be much lower if low sulfur fuel is used; however, this would not necessarily result in a reduction of adverse health effects unless SO₂ was itself a harmful emission.

Statistical studies suggest that SO₂ is not only serving as an indicator of air quality but also is not injurious, at least at the ambient levels encountered in New York City in the 1960's - namely, average levels of 0.2 ppm and peak levels of 0.6 ppm.

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Table 4. Summary of findings in CHES pulmonary function studies.*

Location	Time	Age group tested	Findings
Cincinnati	1967-1968	Second grade	<p>White children exposed to average suspended sulfate levels of 9.5 $\mu\text{g}/\text{m}^3$ had lower $\text{FEV}_{0.75}$ than white children exposed to average suspended sulfate levels of 8.3 $\mu\text{g}/\text{m}^3$</p> <p>The $\text{FEV}_{0.75}$ of black and white children was lowest in winter</p> <p>The $\text{FEV}_{0.75}$ of black children did not vary with air emission exposure</p> <p>The $\text{FEV}_{0.75}$ of black children was consistently lower than that of whites</p>
New York	1970-1971	All elementary grades	<p>The $\text{FEV}_{0.75}$ of white children aged 9 through 13 years, who had been exposed to high levels of sulfur oxides and particulates during the first decade of life, was lower than that of children who had not been so exposed.</p> <p>This finding was statistically significant in males, but not in females</p> <p>The $\text{FEV}_{0.75}$ of children aged 5 through 8 years did not vary consistently with emission exposure</p> <p>The $\text{FEV}_{0.75}$ of children in all grades was lowest in winter</p>

* [Health Consequence of Sulfur Oxide: A Report from CHES 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Table 5. Estimates of "% premature deaths" associated with air emission, New York City, 1963-1972.^a
 "% premature deaths" is a sensitive indicator of adverse health effects. Adverse health effects are associated principally (80%) with the particulate measure (smoke shade) and only a small fraction (20%) with SO₂.^{*}

Period (years)	Season (months)	J	Regression On					Regression ^b coefficient on temp × 100 ²
			TP ₁	TP ₂	T, P ₁ and P ₂			
					P ₁ share	P ₂ share	Total	
1963-1972	1-12	0	1.35(.43)	2.68(.66)	0.58(.51)	2.20(.79)	2.78(.67)	54.2(7.6)
1963-1972	1-12	5	1.39(1.17)	3.22(1.71)	0.43(1.33)	2.89(1.96)	3.31(1.76)	-6.1(17.0)
1963-1966	1-12	0	1.72(.68)	2.22(.96)	1.22(.82)	1.26(1.16)	2.48(.98)	65.1(11.3)
1963-1966	1-12	5	0.66(1.86)	1.47(2.50)	0.21(2.15)	1.16(2.88)	1.37(2.60)	-7.1(25.2)
1967-1969	1-12	0	1.16(1.00)	3.30(1.34)	-0.62(1.28)	3.82(1.72)	3.20(1.35)	39.7(16.2)
1967-1969	1-12	5	2.67(2.78)	6.29(3.43)	-0.19(3.33)	6.58(4.12)	6.39(3.61)	-54.5(37.9)
1970-1972	1-12	0	1.47(.76)	2.64(1.19)	0.81(.89)	1.97(1.40)	2.78(1.20)	52.4(12.7)
1970-1972	1-12	5	2.29(2.02)	2.86(3.18)	1.58(2.27)	2.01(3.59)	3.59(3.27)	35.2(28.3)
1963-1972	1-2	0	0.39(1.03)	2.27(1.69)	-0.43(1.20)	2.64(1.98)	2.21(1.70)	26.7(14.7)
1963-1972	1-2	5	1.81(2.93)	-0.31(4.32)	3.12(3.33)	-3.29(4.93)	-0.18(4.48)	-47.6(31.3)
1963-1972	3-4	0	2.24(.85)	3.62(1.21)	1.18(1.03)	2.67(1.47)	3.85(1.23)	35.9(12.7)
1963-1972	3-4	5	1.05(2.42)	6.48(3.35)	-2.00(2.82)	7.88(3.93)	5.88(3.47)	9.8(28.5)
1963-1972	5-6	0	2.48(.85)	3.14(1.04)	1.58(.99)	2.15(1.21)	3.74(1.11)	103.2(15.0)
1963-1972	5-6	5	3.68(1.95)	2.21(2.60)	3.69(2.19)	0.05(2.91)	3.74(2.74)	101.0(32.8)
1963-1972	7-8	0	0.21(1.00)	1.70(1.07)	-0.82(1.16)	2.16(1.25)	1.34(1.19)	228.2(19.9)
1963-1972	7-8	5	-2.11(2.31)	1.47(2.33)	-4.29(2.82)	4.04(2.85)	-0.26(2.62)	317.7(49.2)
1963-1972	9-10	0	1.40(.70)	2.65(1.14)	0.75(.83)	1.98(1.36)	2.74(1.15)	57.5(13.8)
1963-1972	9-10	5	1.83(1.84)	7.35(3.05)	-0.12(2.04)	7.40(3.40)	7.28(3.12)	-4.3(33.4)
1963-1972	11-12	0	1.56(1.01)	2.97(1.70)	0.77(1.27)	2.19(2.13)	2.96(1.70)	30.0(17.5)
1963-1972	11-12	5	1.48(3.04)	6.98(4.92)	-0.67(3.45)	7.54(5.61)	6.87(5.02)	-71.0(42.2)

^aObtained by regressing total daily mortality on seasonally corrected mean daily temperature (T), SO₂ (P₁), and smoke shade (P₂). Figure in parentheses is the standard error of the estimate. Two estimates are given, one for same day regressions for which J = 0 and a second estimate for which J = 5, and daily mortality is regressed on same day and five previous days of T, P₁, and P₂. See original study for discussion of methods and qualifications [Schimmel, H., and L. Greenburg, "A Study of the Relation of Pollution to Mortality, New York City, 1963-1968," *J. Air Poll. Cont. Assoc.* 22: 607 (1972)].

^bThis information has been included to show the complexity of mortality, temperature, and emission relationships. In a previous study when lagged temperatures were not included the "percent premature death" estimate with lags was approximately double that of the same day estimate (same reference as above).

^{*}[Schimmel, H., and T. J. Murawski, *J. Air Poll. Cont. Assoc.* 25, 739 (1975).]

Fig. 3. Mean residual mortality by SO₂ classification, New Jersey Metropolitan Area, 1962-1966. High SO₂ days differ from low SO₂ days in many ways. Which of these differences is responsible for the correlation with mortality remains to be determined. [Buechley et. al., Arch. Environ. Health 27, 137 (1973), copyright 1973, American Medical Association.]

Fig. 4. Effects of sulfur oxides on health. The numbers refer to specific incidents, for example, the London, England, smog of 1952 is represented by point 31 (152 deaths). (o) morbidity in man; (•) mortality in man; (Δ) morbidity in animals; (▲) mortality in animals. The hatched area indicates the range of concentrations and exposure times in which deaths have been reported in excess of normal expectation. The crosshatched area indicates the range of concentrations and exposure times in which significant health effects have been reported. The stippled area indicates the ranges of concentrations and exposure time in which health effects are suspected. This is the upper bound of the error envelope. [From U.S. Department of Health, Education, and Welfare, Publ. No. 1619, USGPO, Washington, DC.]

Fig. 5. Deaths due to cardiovascular disease and levels of SO₂ (averaged over 212 mi²) in males 55 and over, Chicago, January 1966. The SO₂ averages shown are derived from the 24-h SO₂ readings (ppm), as monitored by the city. The significant increases in cardiac disease and the absence of any increase in other disease categories further support the validity of these studies, since those suffering from cardiac difficulties would be expected to be the most affected during high air pollution episodes. [Carnow, B. W., Calif. Air Environment 4, 7 (1974).]

Nonradioactive Particulates

Modeling of Deposition in the Respiratory Tract

Quantitative modeling studies have been made of the fraction of particles deposited in the various regions of the respiratory tract. These regions include the alveolar region (for both nose and mouth breathing); the tracheo-bronchial tree (for both nose and mouth breathing); the oral cavity, pharynx, and larynx (for mouth breathing); the nasopharynx, pharynx, and larynx (for nose breathing); the ciliated nasal passages (for nose breathing); and the anterior unciliated nares (for nose breathing). The various models differ somewhat in their quantitative predictions, depending on the assumptions made, but qualitatively their results are much the same.

Three conclusions can be drawn about deposition in the alveolar region:

- There is a maximum efficiency of deposition at a particle size between 1 and 2 μm .
- There is a minimum efficiency at about 0.5 μm .
- The percentage of particle deposition for sizes less than 0.1 μm is just as great as for sizes more than 1 μm .

These conclusions must be weighed by the particle size distribution for any particular case of interest.

Figures 6 and 7 illustrate the clearing mechanisms and particle termination points in the human body. Figure 8 represents the calculated efficiencies of deposition of particles of various sizes, in the tracheobronchial and alveolar regions of the respiratory system, and shows the size for minimum efficiency. A minimum in the removal efficiency with respect to particle size arises in the transition range between removal by Brownian motion and by impaction. Figure 9 shows the deposition efficiencies.

Epidemiological Effects

At concentrations of 750 $\mu\text{g}/\text{m}^3$ and higher for particulates on a 24-h average, accompanied by sulfur dioxide concentrations of 715 $\mu\text{g}/\text{m}^3$ and higher, excess deaths and a considerable increase in illness may occur.

A decrease from 140 to 60 $\mu\text{g}/\text{m}^3$ (annual mean) in particulate concentrations may be accompanied by a decrease in mean sputum volume in industrial workers.

If concentrations above 300 $\mu\text{g}/\text{m}^3$ for particulates persist on a 24-h average and are accompanied by sulfur dioxide concentrations exceeding 630 $\mu\text{g}/\text{m}^3$ over the same period, chronic bronchitis patients will likely suffer acute worsening of symptoms.

At concentrations over 200 $\mu\text{g}/\text{m}^3$ for particulates on a 24-h average, accompanied by concentrations of sulfur dioxide exceeding 250 $\mu\text{g}/\text{m}^3$ over the same average period, increased absence of industrial workers due to illness may occur.

Where concentrations range from 100 to 130 $\mu\text{g}/\text{m}^3$ and above for particulates (annual mean) with sulfur dioxide concentrations (annual mean) greater than 120 $\mu\text{g}/\text{m}^3$, children residing in such areas are likely to experience increased incidence of certain respiratory diseases.

At concentrations above 100 $\mu\text{g}/\text{m}^3$ for particulates (annual geometric mean) with sulfation levels above 30 $\text{mg}/\text{cm}^2\cdot\text{mo}$, increased death rates for persons over 50 years of age are likely. These sulfation levels correspond to SO_2 concentrations of 120 $\mu\text{g}/\text{m}^3$, or 0.04 ppm.

The EPA cites sulfates as being the sulfur oxide most suspected of causing adverse health effects. However, since the assumption that the health effects of sulfur-bearing air emissions are largely due to the formation of sulfates is theoretical, further confirmation is needed and no air quality standard for sulfates has yet been established.

Table 6 relates infant and total mortality rates for 114 Standard Metropolitan Statistical Areas in the U.S. to air emissions and other factors. As the biweekly minimum level of suspended particulates increases, the death rate rises significantly. Moreover, the death rate increases with (1) the population density of the area, (2) the proportion of nonwhites, (3) the proportion of people over age 65, and (4) the proportion of poor families.

Toxicological and Physiological Effects

Particulate matter may exert a toxic effect through one or more of three mechanisms:

- The particle may be intrinsically toxic due to its inherent chemical or physical properties.
- The particle may interfere with one or more of the clearance mechanisms in the respiratory tract.
- The particle may act as the carrier of an adsorbed toxic substance.

Results of a study of asthmatics in the Salt Lake Basin showed the highest morbidity rates were associated with the highest suspended sulfate levels. However, the most consistently linked emission was total suspended particulates and the least impressive effect could be attributed to sulfur dioxide exposure (Fig. 10). The threshold for aggravation of asthma by suspended sulfates was $1.4 \mu\text{g}/\text{m}^3$ on cooler days (30 to 50°F minimum temperature).

In a study of cardiopulmonary patients in the New York metropolitan area, the strongest and most consistent emission effects were found for suspended sulfates which were linked to a worsening of symptoms, particularly shortness of breath, cough, and increased production of phlegm. The worsening of symptoms which was attributed to suspended sulfates always persisted after the correction for the effects attributable to temperature and other emissions.

A study of school children in Cincinnati found that the ventilatory function of children residing in an industrial valley of Cincinnati was significantly lower than the performance of children living in a clean area of the metropolitan region. Differences in suspended sulfate levels were found to be most closely associated with differences in ventilatory performance.

Two significant toxicological findings have resulted from the few studies of sulfuric acid and sulfate particulates. First, sulfuric acid and sulfate particulates are much more potent irritants than SO_2 gas alone. Secondly, the mass weight of sulfates is an insufficient basis on which to predict irritant potency; particle size and chemical composition determine the toxic potential of sulfates (Tables 7-10).

A highly variable dose-response relationship exists between SO_2 and particulate levels and health impact on different populations (Fig. 11 and Table 11). In summary, there is no threshold for health effects. The outcome - health or disease, living or dying - depends upon the number of environmental stressors and the intensity of each, matched against the resistance of the host and the host's adaptive ability.

The effects of other emissions, particularly cigarette smoking and heavy occupational exposure to irritant gases, act synergistically with SO_2 and particulates. The very large population already at risk because of such other factors increases the need for controlled levels of SO_2 and particulates necessary to protect the health of these people.

Of particular interest for coal-fired power plants, a synergistic potentiating effect has been observed between some particulates and sulfur dioxide. However, not all particulates potentiate the response to sulfur dioxide, nor does not particulate potentiate the response of all irritant gases. Both the solubility of sulfur dioxide and its catalytic oxidation to sulfuric acid play a major role in its potentiation by certain particulates. The presence of carbon or soot as a common particulate is noteworthy, inasmuch as carbon is an efficient adsorber of a wide range of organic and inorganic compounds.

Few particulates commonly found in the atmosphere appear to be intrinsically toxic; from the point of view of coal-fired power plants, the most important toxic aerosol is sulfur trioxide, either as the free oxide or hydrated as sulfuric acid. Recently, there has been increased concern about some less common toxic particulates, including lead, beryllium, and asbestos.

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Table 6. Regressions relating infant and total mortality rates for 114 Standard Metropolitan Statistical Areas in the U.S. to air emissions and other factors. Values in parentheses are the t statistic. For means and standard deviations (S.D.) of the variables, see Footnote b.*

Category	K ^c	Air emission (minimum) concentrations	Socioeconomic			
			P/m ^{2d}	Non- white (%)	Over 65 (%)	Poor (%)
Total death rate						
1.Particulates	.804	0.102 (2.83)	0.001 (2.58)	0.032 (3.41)	0.682 (18.37)	0.013 (0.93)
2.Sulfates	.813	0.085 (3.73)	0.001 (1.86)	0.033 (3.56)	0.652 (17.60)	0.006 (0.49)
Death rate for infants of less than 1 year						
3.Particulates	.545	0.393 (3.07)		0.190 (6.63)		0.150 (3.28)
4.Sulfates	.522	0.150 (1.91)		0.200 (6.83)		0.123 (2.70)
Death rate for infants less than 28 days old						
5.Particulates	.260	0.273 (2.48)		0.089 (3.61)		0.063 (1.60)
6.Sulfates	.263	0.170 (2.57)		0.097 (3.96)		0.047 (1.23)
Fetal death rate						
7.Particulates	.434	0.274 (2.02)	0.004 (2.01)	0.171 (5.70)		0.106 (2.11)
8.Sulfates	.434	0.171 (1.95)	0.004 (1.82)	0.181 (5.87)		0.085 (1.71)

^aThe t statistic: for a one-tailed t-test, a value of 1.65 indicates significance at the .05 level.

^bTotal death rate per 10,000: mean, 91.5; S.D., 15.2. Infant death rate (age, <1 year) per 10,000 live births: mean, 255.1; S.D., 36.1. Infant death rate (age, <28 days) per 10,000 live births: mean, 188.0; S.D., 24.4. Fetal death rate per 10,000 live births: mean, 153.9; S.D., 34.4. Suspended particulates ($\mu\text{g}/\text{m}^3$), minimum reading for a biweekly period: mean, 45.2; S.D., 18.7. Total sulfates ($\mu\text{g}/\text{m}^3$) ($\times 10$), minimum reading for a biweekly period: mean, 46.9; S.D., 30.6. Persons per square mile: mean, 763.4; S.D., 1387.9. Percentage of nonwhites in population ($\times 10$): mean, 125.2; S.D., 102.8. Percentage of population over 65 ($\times 10$): mean, 84.2; S.D., 21.2. Percentage of families with incomes under \$3000 ($\times 10$): mean, 181.6; S.D., 65.7.

^cThe coefficient of determination: a value of .804 indicates a multiple correlation coefficient of .90, and indicates that 80 percent of the variation in the death rate is "explained" by the regression.

^dPersons per square mile.

*[Lave, L. B., and E. P. Seskin, *Science* 169, 725 (1970).]

Table 7. Best judgment estimates of emission thresholds for adverse effects of short-term exposures.*

Effect	Threshold ($\mu\text{g}/\text{m}^3$)		
	Sulfur dioxide (365) ^a	Total suspended particulate (260) ^a	Suspended sulfates (no standard) ^a
Aggravation of cardiopulmonary symptoms in elderly	>365	80-100	8-10
Aggravation of asthma	180-250	70	8-10

^aNational Primary Air Quality Standard.

* [Health Consequences of Sulfur Oxides: A Report from CHES, 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Table 8. Best judgment estimates of emission thresholds for adverse effects of long-term exposures.*

Effect	Threshold, annual average ($\mu\text{g}/\text{m}^3$)		
	Sulfur dioxide (80) ^a	Total suspended particulates (75) ^a	Suspended sulfates (no standard) ^a
Increased prevalence of chronic bronchitis in adults	95	100	15
Increased acute lower respiratory disease in children	95	102	15
Increased frequency of acute respiratory disease in families	106	151	15
Decreased lung function of children	200	100	13

^aNational Primary Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about $85 \mu\text{g}/\text{m}^3$.

*[Health Consequences of Sulfur Oxides: A Report from CHESS, 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Table 9. Summary of CHES studies relating long-term emission exposures to adverse effects on human health.*

Adverse effect	Type of estimate	Duration of exposure (y)	Annual average levels linked to adverse health effects ($\mu\text{g}/\text{m}^3$)		
			Sulfur dioxide (80) ^a	Total suspended particulates (75) ^a	Suspended sulfates (no standard) ^a
Increase in prevalence of chronic bronchitis in adults	Worst case	3	62	65	12
	Least case	10	374	179	20
	Best judgment	6	95	100	15
Increases in acute lower respiratory tract infections in children	Worst case	3	92	65	7.2
	Least case	3	177	102	15
	Best judgment	3	95	102	15
Increase in frequency or severity of acute respiratory illness in families	Worst case	1	50	104	14
	Least case	3	210	159	16
	Best judgment	3	106	151	15
Subtle decreases in childhood ventilatory function	Worst case	1	57	96	9
	Least case	9	435	200	28
	Best judgment	8-9	200	100	13

^aNational Primary Ambient Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about $85 \mu\text{g}/\text{m}^3$.

* [Health Consequences of Sulfur Oxides: A Report from CHES, 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Table 10. Summary of threshold estimates for adverse effects of short-term exposure.*

Adverse effect	Type of estimate	Minimum temperature (°F)	Daily average levels linked to adverse health effects ^a (µg/m ³)		
			Sulfur dioxide (365) ^b	Total suspended particulate (260) ^b	Suspended sulfur (no standard) ^b
Aggravation of cardiopulmonary symptoms in elderly					
"Well" panel	Worst case	20-40	81-365	NPE	<1
	Least case		NE	NE	NE
	Best judgment		NPE	NPE	8-10
	Worst case	>40	81-365	68	2
	Least case		NE	NE	10
	Best judgment		NPE	80-100	8-10
"Heart" panel	Worst case	>40	NPE	76-260	10
	Least case		NE	NE	10-20
	Best judgment		NPE	NPE	10
"Lung" panel	Worst case	20-40	NPE	76-260	6
	Least case		NE	NE	NE
	Best judgment		NPE	NPE	10
	Worst case	>40	NPE	76-260	11
	Least case		NE	NE	12
	Best judgment		NPE	NPE	12
"Heart and lung" panel	Worst case	20-40	181	47	9
	Least case		NE	NE	10
	Best judgment		NPE	80-100	10
	Worst case	>40	NPE	76	6
	Least case		NE	NE	17
	Best judgment		NPE	NPE	10
Aggravation of asthma	Worst case	30-50	NPE	61-75	8
	Least case		NE	NE	NE
	Best judgment		NPE	105	9-10
	Worst case	>50	23	61-75	<1
	Least case		NE	NE	10
	Best judgment		180-250 ^c	70	8

^aNE: no effect below primary standard, or simply no effect for suspended sulfates, for which no primary standard has been established. NPE: no proven effect below primary standard, or simply no proven effect for suspended sulfates.

^bNational Primary Air Quality Standard.

^cThis judgment is based on presently summarized studies and on a previously reported CHESS study of asthma in New Cumberland, West Virginia.

*[Health Consequences of Sulfur Oxides: A Report from CHESS, 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Table 11. Summary of dose/response relationships for effects of particles and SO₂ and health (old results, leading to original standards).*

Averaging time for measurements	Place	<u>Approximate concentration levels</u>		Effect
		Particles ($\mu\text{g}/\text{m}^3$)	SO ₂ ($\mu\text{g}/\text{m}^3$)	
24 h	London	2000	1144	Mortality
24 h	London	750	700	Mortality
24 h	London	300	600	Deterioration of patients
Weekly mean	London	200	400	Prevalence or incidence of respiratory illnesses
24 h	New York	6 ^a	1500	Mortality
Winter mean	Britain	100-200	100-200	Incapacity for work from bronchitis
Annual	Britain	70	90	Lower respiratory infec- tions in children
	Britain	100	100	Upper and lower respira- tory infections in children
	Britain	100	100	Bronchitis prevalence
	Britain	100	100	Prevalence of symptoms
	Buffalo	100	0.30 ^b	Respiratory mortality
	Berlin, NH	180	0.73 ^b	Increased respiratory symptoms Decreased pulmonary function

^aCOHS.

^b $\text{mg}/\text{cm}^2/30$ days.

* [Health Consequences of Sulfur Oxides: A Report from CHESS, 1970-1971, Environmental Protection Agency, Research Triangle Park, NC, Publ. EPA-650/1-74-004 (1974).]

Fig. 6. Particle clearing mechanisms in the human body. The body's defenses against invasion by foreign particles are considerable, but can be overwhelmed by excessive amounts of particulate emissions, by breathing particles in combination with gases, or by particles that are infectious, irritating, or toxic. [Brodine, V., Air Pollution (Harcourt, Brace, and Jovanovich, New York, 1973), p. 122.]

1. Lung tissue. If soluble, it may be dissolved in lung fluid and chemically bound with tissue; if insoluble, it may penetrate tissue and remain there.

3. Blood stream. Particle reaches blood via nasal clearing; or by passing into pulmonary capillaries with inhaled oxygen; or after penetrating lung tissue, passes into lymph and from lymph to blood; or is taken up by phagocyte in alveolus and absorbed into blood; or is swallowed with mucus, passes through digestive system, is absorbed into blood, and may reach other parts of the body in the blood flow.

2. Lymph nodes. Particle passes from lung tissue to lymph, is stored in lymph nodes.

4. The gastrointestinal system. After reaching the system, the particle remains in one of the organs of the system instead of passing into the bloodstream.

Fig. 7. Termination points of particle not cleared by one of the particle clearing mechanisms in the human body. [Brodine, V., Air Pollution (Harcourt, Brace, and Jovanovich, New York, 1973), p. 123.]

Fig. 8. Calculated fraction of particles deposited in the respiratory tract as a function of particle radius. [From Air Quality Criteria for Particulate Matter, Department of Health, Education, and Welfare, National Air Pollution Control Administration, Washington, DC, Publ. AP-49 (1969), p. 114, and Findeisen, W., Arch. Ges. Physiol. 236, 367 (1935).]

Fig. 9. Fraction of particles deposited in the three respiratory tract compartments as a function of particle diameter. [Air Quality Criteria for Particulate Matter, Department of Health, Education, and Welfare, National Air Pollution Control Administration, Washington, DC, Publ. AP-49 (1969). p.115.]

Fig. 10. Air quality criteria for particulates. $26 \mu\text{g}/\text{m}^3 \text{SO}_2 = 0.01 \text{ ppm (vol)}$.
[Lawrence Livermore Laboratory, staff compilation.]

Fig. 11. Dose response curve for sulfur dioxide and total suspended particulates. [Ferris, B., personal communication (September 1973).]

Organic Gases

The principal problems are not created by the hydrocarbon directly, but by compounds derived from the atmospheric reactions of hydrocarbons and their derivatives with other substances (Tables 12-15).

Reactive hydrocarbons become involved in photochemical air emissions not because of their exposure to sunlight, but because of their reactions with atomic oxygen, electronically excited O_2 , O_3 , and free radicals generated by the action of sunlight on other components in the atmosphere, most particularly NO_2 .

The photochemical reactions have the net effect of producing photochemical oxidants (sometimes reported as "ozone"). These are the compounds that in association with NO_2 and particulate matter produce the recognizable smog-type air pollution that reduces visibility, colors the atmosphere, irritates the eyes and respiratory system, and is toxic to flora and fauna.

Certain hydrocarbons - in particular, aromatics with three to six benzene rings - constitute the most important group of known or suspected carcinogens, and potentially harmful effects may result from repeating exposure. The more reactive ingredients, such as ethylene, disappear quite rapidly (within 24 h or less), while the less reactive components may have lifetimes measured in weeks or months.

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Table 12. Toxicity of saturated aliphatic hydrocarbons (through octane).*

Hydrocarbon	Concentration (ppm)										
	100,000	50,000	40,000	30,000	20,000	15,000	10,000	5,000	2,000	1,000	500 0
Methane CH_4	No effect										
Ethane C_2H_6		No effect									
Propane C_3H_8	No irritation noticed Dizziness in a few min				Odor not detected		No symptoms after brief exposure				
Butane C_4H_{10}							Drowsiness in 10 min	Odor not detectable			
Pentane C_5H_{12}	Narcosis in 5-60 min							Odor readily detectable; No irritation or symptoms in 10 min		TLV	
Hexane C_6H_{14}			Convulsions and death	Narcosis				Dizziness, giddiness, in 10 min	No symptoms in 10 min		TLV
Heptane C_7H_{16}					Convulsions, death in 30-60 min	Narcosis in 30-60 min		In 4 min: marked vertigo, hilarity, incoordination In 15 min: uncontrolled hilarity or stupor	Slight vertigo in 4 min		TLV
Octane C_8H_{18}							Narcosis in 30-90 min				TLV

* [Kamens, R. M., and A. C. Stern, J. Air Poll. Cont. Assoc. 23, 592 (1973).]

Table 13. Comparative effects of acute and chronic exposure to aromatic hydrocarbon vapors in air.*

Compound	Subject	Concentration		Daily exposure (h)	Exposure (days)	Effect
		ppm	mg/m ³			
Benzene	Man	25	80	Acute	--	Threshold limit value (TLV)
	Man	100	319	Acute	--	Mucous membrane irritation
	Mice	370	1,180	Acute	--	Threshold for affecting the central nervous system
	Man	3,000	9,570	Acute	--	Endurable for 1/2-1 h
	Mice	4,700	14,993	Acute	--	Prostration
	Mice	7,400	23,606	Acute	--	LC ₅₀
	Man	7,500	23,925	Acute	--	Dangerous after 1/2-1 h
	Mice	14,100	44,979	Acute	--	LC ₁₀₀
	Rats	17,800	56,782	Acute	--	LC ₁₀₀
	Man	20,000	63,800	Acute	--	Fatal after 5-10 min
	Man	50-100	188-377	Acute	--	No effect
Toluene	Man	200	753	8	1	Mild fatigue, weakness, confusion, skin paresthesias
	Man	300	1,130	8	1	Symptoms more pronounced
	Man	400	1,506	8	1	Also: mental confusion
	Man	600	2,259	3	1	Also: nausea, headache, dizziness
	Man	600	2,259	8	1	Also: loss of coordination, staggering gait, pupils dilated
	Mice	2,700	10,166	Acute	--	Prostration
	Mice	6,700	25,226	Acute	--	LC ₅₀
	Mice	9,500	35,768	Acute	--	LC ₁₀₀
	Rat	13,500	50,828	Acute	--	LC ₁₀₀
Styrene	Man	100	418	Acute	--	Threshold limit value (TLV)
	Guinea pig	650	2,714	8	180	No effect
	Rat	1,300	5,428	8	180	Eye & nasal irritation only
	Rabbit	1,300	5,428	8	180	Eye & nasal irritation only
	Guinea pig	1,300	5,428	8	180	10% deaths
	Guinea pig & rat	2,500	10,438	8	1	Some fatalities; varying degree of weakness, stupor, incoordination, tremor, unconsciousness (in 10 h)
	Guinea pig & rat	5,000	20,875	1	1	Unconsciousness in 1 h
	Guinea pig & rat	10,000	41,750	1/2-1	1	Unconsciousness in 10 min; deaths in 30-60 min
Xylene	Mice	174	755	Acute	--	Threshold for affecting central nervous system
	Man	200	868	Acute	--	Threshold limit value (TLV)
	Rat & rabbit	690	2,995	8	150	No hematological effects

*[Air Quality Criteria for Hydrocarbons, Department of Health, Education, and Welfare, National Air Pollution Control Association, Washington, DC, Publ. AP-64 (1970).]

Table 14. Toxicity of unsaturated aliphatic hydrocarbons.*

Hydrocarbon	Concentration (ppm)											
	350,000	30,000	200,000	100,000	50,000	40,000	20,000	8,000	5,500	5,000	4,000	1,000
Ethylene									MPL			
Propylene											MPL	TLV
1-butene											MPL	
1,3-butadiene											MPL	
2-methyl-1,3-butadiene								Irritation of eye and upper respiratory tract in man				
Acetylene	Unconsciousness in 5 min	Inordinat	Marked intoxication	Slight intoxicating effect on man						MPL		

*[Air Quality Criteria for Hydrocarbons, Department of Health, Education, and Welfare, National Air Pollution Control Association, Washington, DC, Publ. AP-64 (1970).]

Table 15. Toxicological comparison of methanol and gasoline.*

	Methanol	Gasoline
Chemical nature	CH ₃ OH	Mixture of C ₄ - C ₁₂ aliphatics plus aromatics
Threshold limit value (TLV) and maximum allowable concentration (MAC)	200 ppm	500 ppm 25 ppm benzene
Acute inhalation	Slight hazard	Slight hazard
Chronic inhalation	Moderate hazard	Slight hazard
Aspiration into lungs	Slight hazard	Very toxic
Ingestion	Moderate hazard	Slight hazard
Skin contact	Both defatting and irritating Absorbed through skin	Little absorbed
Warning odor	Inadequate	Adequate

* [Berger, B. J., Environmental Aspects of Methanol as Vehicular Fuel: Health and Environmental Effects, Lawrence Livermore Laboratory, Rept. UCRL-76076 (1974).]

Trace Metals

Table 16 lists the major energy sources of trace metals in the air and their effects on human health.

Beryllium

Beryllium produces a variety of effects depending upon the dose and the duration of exposure. A prominent lesion caused by beryllium is a granulomatous nodule found in the skin, subcutaneous tissues, and lungs. The most extensive changes are usually found in the lungs.

At present there is no evidence that community or industrial exposure to beryllium compounds is associated with an increase in the incidence of carcinoma in humans. The pathogenesis of health effects produced by beryllium is not clear.

Cadmium

Cadmium is toxic to man and to other living things in virtually all of its chemical forms. Although we do not know the threshold values at which chronic effects begin to be noticeable, there is little indication that cadmium at any level serves any useful function in living organisms. Cadmium accumulates in the body, especially in the kidney and the liver, over many years.

Cadmium does not degrade in the environment, but accumulates in soils and sediments where it can enter various food chains. Such accumulations resulting from mining operations caused itai-itai disease in Japan through the contamination of food and water.

Cadmium in the air is absorbed from the lungs and deposited in the kidney, liver, and arteries. It can produce destructive kidney changes, and liver damage has been caused by chronic exposure to large amounts. Rats exposed for a lifetime to small doses of cadmium in drinking water exhibited arterial hypertension and the pathological counterpart of the human disease.

Cadmium is not important as a constituent of coal, but it reacts with sulfhydryl enzymes such as the metals mercury and lead that are present in coal. It is important to consider that the metals in coal have the same mode of action as other metals that enter the atmosphere from other sources,

and these combinations of metals from different sources have the potential for causing additive toxicity.

We do not know the extent to which present environmental levels of cadmium in the U. S. are a health hazard, although some studies have implicated cadmium in connection with hypertension and increased mortality. We do know that some 4% of surface waters sampled by the USGS in 1970 exceeded the Public Health Service drinking water standard of 0.01 ppm. Furthermore, the Bureau of Mines projects a two- to threefold increased demand for cadmium by the year 2000.

Mercury

The symptoms of chronic mercury poisoning may appear after a few weeks of exposure or they may be delayed much longer. The characteristic serious effects are psychic and emotional disturbances, loss of memory, tremors, and renal disease.

In considering the hazards of mercury, it is important to recognize that its principal biochemical action is combined with sulfhydryl groups of important enzymes. In this respect, mercury resembles arsenic, lead, and cadmium.

Known as a poison to industrial workers since ancient times, primarily as the metal or in simple inorganic form, it has only recently been recognized as a lethal material in the environment in the form of salts of the alkyl compounds. These alkyl salts may be formed from inorganic mercurials directly by chemical conversion or by the action of certain microorganisms living in soils or sediments under fresh-water bodies.

The widespread occurrence of methylmercury, the most toxic form of mercury in man's food chain, catalyzes a continuing need to understand its effects. One of the major problems is the definition of "safe" levels. Many of the answers needed to define "safe" levels will come from the study of humans exposed to inordinately high levels.

Nickel

Nickel as nickel carbonyl causes cancer in animals and humans. Cancer of the respiratory tract has been induced in rats by exposing them to nickel carbonyl. Nickel refinery workers are prone to develop lung cancer when exposed to nickel dust and fumes or nickel carbonyl for many years. Death

rates from these causes are 2 to 25 times the rates expected in the general population. Nickel carbonyl is readily picked up from the lung by the blood although some nickel combines with lung proteins.

It is not known whether airborne nickel contains the carbonyl form, but the conditions are right for forming it in some power-plant combustion equipment. It is formed when hot carbon monoxide passes over nickel.

Because of the carcinogenicity of nickel in appropriate doses, it represents a potential hazard to urban dwellers, and the sources of atmospheric nickel should be decreased as fast as possible.

Lead

Lead is an element with no known beneficial function in human metabolism. It has been known for 2000 years that it is toxic; but in spite of its recognition as a poison it is widely used in modern technology.

Acute toxicity results from excessive exposure, primarily from industrial hygiene problems among occupationally exposed adults and from the pediatric lead poisoning associated largely with lead-based paints.

To date, no cases of acute plumbism have been reported in adults with blood-lead below 80 $\mu\text{g}/100\text{ cc}$. Some authorities believe that biological changes are exhibited at all lower exposures, that any change is undesirable, and that no threshold exists below which no damage results.

Lead is excreted from the body at nearly the same rate as introduced, the balance accumulating slowly in the body with age, principally in the bones, aorta, liver, kidney, pancreas, trachea, larynx, lungs, and skin. The bones on the average contain 91% of the total lead in the body. As lead impairs the production of hemoglobin, it also causes oxygen starvation and anemia.

Lead has been observed to cause deleterious effects in the liver, kidney, brain, and nervous system. These effects are associated with blood lead levels in excess of 100 $\mu\text{g}/100\text{ ml}$ of blood in acute encephalopathy and 80 $\mu\text{g}/100\text{ ml}$ of blood in chronic encephalopathy.

Persons most prone to lead poisoning are workers occupationally exposed and children. Occupations include industrial lead-processing operations and employment near high traffic densities. Children exhibiting symptoms of lead toxicity have been observed eating chips of old paint and putty.

Other factors such as urban atmospheric concentrations, ingestion of street dirt, ethnic diet, and ethnic and cultural factors have not been adequately evaluated. It is a curious fact that about 10 times as many cases of pediatric lead poisoning occur in midsummer, August, as occur in the period from January through March.

Conclusive evidence is lacking that lead is emitted in auto exhaust in concentrations threatening human health, or that it has built up in the environment to such concentrations. Further, until very recently, no comprehensive systems approach to the distribution and environmental effects of lead had been made, particularly at the ecosystem level.

Other Metals

Numerous other metals are present in coal, and they are emitted into the atmosphere at various concentrations. Most of them will interfere with physiological processes at appropriate concentrations. Thus, bismuth, antimony, tin, aluminum, barium, strontium, zirconium, niobium, and vanadium are all present in coal and are released into the atmosphere by the burning of coal.

A number of metals that are essential for man are also found in the air, but the intake of these metals is usually below 1% of the amounts in food and water.

The available information indicates that most of the metals present in the atmosphere do not represent a hazard to human health, but a few are present at levels that are near those known to produce adverse effects.

The estimation of the safety of various levels of metals in the air usually has to be based on experimental work done to establish safe levels for industrial exposure. The permissible levels are based on daily 8-h exposures rather than to the 24-h exposures that the general population receives from air emissions. Furthermore, the permissible industrial levels usually do not consider possible additive or synergistic effects from combinations of metals which are always present in the atmosphere.

It is clear from the information available at the present time, even though it is incomplete, that reasonable efforts should be made to reduce the emission of metals and other toxicants from coal and other sources.

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Table 16. Major energy sources of trace metals in the air and their effects on human health.*

Element	Threshold limits (mg/m ³)	Sources	Effects on human health
Be	0.002 as Be	Metallurgical industry, rocket fuels, and coal	Chemical pneumonitis, berylliosis, chemical ulcer, and carcinogenesis
Cd	0.2 as Cd and Cd in its salts; 0.1 as CdO fume	Electroplating, alloys, solders, and coal	Pulmonary emphysema, hypertension, kidney damage, and carcinogenesis
Hg	100 µg/m ³	Coal, geothermal sources	Hypertension, psychic and emotional disturbances
Ni	1.0 as Ni and Ni in its salts; 0.007 as nickel carbonyl	Steel and nickel-alloy industry, nickel plating, asbestos, coal, fuel oil, and incineration	Dermatitis, respiratory disorder, and carcinogenesis
Pb	0.2 as Pb	Automobile exhaust, coal, incineration, and pigments	Lead poisoning

*[Adapted from Hwang, J. Y., Anal. Chem. 44, 21A (1972).]

Hydrogen Sulfide

Hydrogen sulfide gas is very toxic to humans and at concentrations over 1,000,000 $\mu\text{g}/\text{m}^3$ quickly causes death by paralysis of the respiratory tract (Table 17). At lower concentrations it causes conjunctivitis with reddening and lachrymal secretion, respiratory tract irritation, pulmonary edema, damage to heart muscle, psychic changes, disturbed equilibrium, nerve paralysis, spasms, unconsciousness, and circulatory collapse.

One outstanding episode in Mexico involving accidental release of hydrogen sulfide killed 22 people and 50% of the exposed animals, and hospitalized 320 persons.

The gas has a very obnoxious odor at low concentrations (1 to 45 $\mu\text{g}/\text{m}^3$).

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Table 17. Effects of hydrogen sulfide on humans.*

Concentration ($\mu\text{g}/\text{m}^3$)	Effects
1-45	Odor threshold; no reported injury to health
10	Threshold of reflex effect on eye sensitivity to light
150	Smell slightly perceptible
500	Smell definitely perceptible
15,000	Minimum concentration causing eye irritation
30,000	Maximum allowable occupational exposure for 8 h (ACGIH Tolerance Limit)
30,000-60,000	Strongly perceptible but not intolerable smell; minimum concentration causing lung irritation
150,000	Olfactory fatigue in 2-15 min; irritation of eyes and respiratory tract after 1 h; death in 8 to 48 h
270,000-480,000	No serious damage for 1 h but intense local irritation; eye irritation in 6 to 8 min
640,000-1,120,000	Dangerous concentration after 30 min or less
900,000	Fatal in 30 min
1,160,000-1,370,000	Rapid unconsciousness, respiration arrest, and death, possibly without odor sensation
1,500,000+	Immediate unconsciousness and rapid death

*[Miner, S., Air Pollution Survey of H_2S , Department of Health, Education, and Welfare, National Air Pollution Control Association, Raleigh, NC, Publ. APTD 69-37 (1969).]

Water Vapor

Plumes and fogs from towers and ponds become an environmental hazard if they restrict visibility on streets, highways or railroads, or produce icing (when the temperature is below freezing) on streets, sidewalks, or power lines. Vision-obstructing fogs may extend downwind 1500 ft or more from wet cooling towers.

Problems of fog and icing have usually been associated with mechanical-draft towers which release their effluent plumes relatively close to the ground (50 to 75 ft). The natural-draft, hyperbolic towers release their plumes so high, usually above 350 ft that they seldom if ever sink to ground level.

The combination of vapor from cooling towers and stack gases from fossil-fueled plants can cause special problems involving the generation of acid droplets downwind from the plant.

Continual release of water vapor into the stratosphere might increase the prevalence of cirrus clouds, which could alter the earth's heat balance, however, the long-term climatic consequences of increases in the earth's high cloud cover are still unknown.

Stratospheric water vapor will increase, on a global average, by 0.2 ppm by mass (from 3.0 to 3.2 ppm). Since there will be more water vapor added to the north temperate latitudes, parts of this region may perhaps have a contribution to standing concentration as much as tenfold higher than the increase of the global average (that is, grow from 3 to 5 ppm water vapor).

Water vapor and molecular hydrogen released between 88 km and orbit insertion at 442 km by Skylab's rocket excavated a 1000-km hole in the ionosphere on May 14, 1973.

Water vapor in the stratosphere does not absorb much solar radiation. But it absorbs and emits strongly in the ir region of the spectrum and, by the "greenhouse effect," warms the lower atmosphere (Fig. 12). The present estimate of this warming effect is characterized by about the same uncertainty as the estimate of the cooling effect due to SO_2 . At this time, the water vapor effect is estimated to be about one-half the cooling effect due to the SO_2 emitted using present-day fuels. Note that while the SO_2 effect can be reduced directly by using fuel with a smaller sulfur content, there is no way to reduce water vapor emissions from an operating aircraft.

References - Water Vapor

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Fig. 12. Surface air temperature change due to increased stratospheric water vapor, with average cloudiness and corrective-radiative equilibrium. [Manabe, S., and R. T. Wetherald, "Thermal Equilibrium of the Atmosphere with a Given Distribution of Relative Humidity." J. Atm. Sci. 24, 241 (1967).]

RADIOLOGICAL FACTORS

Gaseous Radioactivity

The hazard of tritium is not from penetrating radiation, but from tritium behaving chemically as hydrogen. There are isotope effects on chemical kinetics and equilibria, but as far as we know, these are of minor importance in its radiotoxicity. Tritium readily converts to tritiated water, HTO. In this chemical form it can quickly pass through the lungs and skin and become uniformly dispersed throughout the body fluids. For this reason, tritium that is chemically bound in water is several hundred times more hazardous than tritium as an elemental diatomic gas. In addition, it can become incorporated into the cells of plants, both by isotopic exchange and photosynthesis. The plants are eaten and tritium is incorporated into the molecular structures of our bodies.

Radioactive decay of tritium within the body has two damage mechanisms. One is the usual chemical changes induced by ionizing radiation. The 5.6-keV tritium beta particle can produce about 160 ionizations in tissue. In this respect, the hazard associated with tritium is substantially the same as other kinds of radiation, such as gamma rays when compared on a rad-(100 erg/g tissue)-for-rad basis. The other mechanism results from a tritium atom in a molecule (DNA, for example) which suddenly converts to a helium atom. The molecule becomes a free radical and can react chemically in an abnormal biological fashion. Rats that have been given doses of tritiated thymidine, a precursor of the DNA molecule, show a very high incidence of various cancers. In the studies done to date, however, this second mode of damage appears to be heavily overshadowed by the more conventional damage mechanisms, and the effectiveness factor for tritium is only about 1.7.

Since the noble gases are chemically inactive they remain in the atmosphere in elemental form until they decay and are not concentrated or retained anywhere in the body. The health effects would be those due to gamma radiation.

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Radioactive Particulates

The primary environmental effects arise when radioactive nuclear aerosols, resulting from atmosphere nuclear explosives, are precipitated or rained out on the earth's surface. Human exposure to fallout is from three sources: ingestion, inhalation, and whole-body external exposure. Of the nuclides ingested, the main contributors to dose are ^{90}Sr and ^{89}Sr (to whole-body and to bone); ^{131}I (to thyroid); ^{137}Cs (to whole-body); and ^{14}C (to whole-body and to bone). Because strontium is deposited permanently in bone, the ^{90}Sr doses have been estimated based upon a 50-year (or lifetime) delivery of dose. The whole-body dose from inhalation is quite small; the 50-year respiratory lymph-node dose from plutonium is large indeed.

From a biological standpoint, the most important long-term fallout constituents are ^{90}Sr and ^{137}Cs , because of their long half-lives (28 and 30 years, respectively) and relatively high uptake and damage in biosystems. Strontium is chemically similar to calcium, which is used for bone formation, while cesium simulates potassium, crucial for the function of cells.

Estimates of ^{90}Sr intake via the total diet in New York City and San Francisco have been made since 1960 from quarterly food samplings and average consumption statistics. The dietary intakes of ^{90}Sr have decreased from the maximum levels attained during 1963 and 1964, but the declines have become more gradual in recent years due to the continuing small amounts of ^{90}Sr deposition and the little-changing cumulative deposit of ^{90}Sr in soil. The annual intake in 1973 averaged 9.7 pCi/day in New York and 3.2 pCi/day in San Francisco, slight decreases from the previous year. Further gradual reductions in ^{90}Sr intake are anticipated during 1974.

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PHYSICAL FACTORS

Thermal Inputs

The weather pattern of the classical air emissions episode is a prolonged period of near constant conditions: little or no wind or turbulence, temperature reduced both in mean level and diurnal range, high humidity accompanied by a several-hundred-foot layer of water-drop fog or low-level unbroken stratus clouds.

Practically all of the vital endocrinological and other physiological functions of the body are affected by sudden changes in environmental temperature modulated by humidity, wind or turbulence, and lighting. Some adjustments are rapid and accommodate the diurnal cycle, others are of longer term and may be measurable over periods of weeks.

Four aspects of the thermal regulating system are important to air emission episode problems:

- Morbidity Limit. The specific physiological condition of a healthy person which leads to disease if this particular condition is surpassed. This limit is temperature and humidity dependent, is different each day for different people, and differs in the same person from one day to the next.
- Age Dependence. The thermal regulatory mechanism is poor in both the very young and the aged. A correlation between heat waves, low temperatures, and mortality of those under 1 and over 44 has already been noted. Alcoholism, arteriosclerosis, and metabolic diseases are cited as usual concomitant disturbances in the older heat-wave deaths.
- Psychosomatic Forces. The unconscious and conscious psychic life modulates the thermal regulating functions of the rhinencephalic hypothalamus-pituitary system.
- Prior Conditioning. The control and operational functions of the thermal regulating systems are highly dependent on seasonalization, thermal tempering, and previous physiological stimulus (Wilder's Law).

Through the interaction of the above factors something comparable to Wilder's Law of the initial value applies to populations as well as to an individual nervous system. Succeeding temperature extremes in the same season are less likely to cause mortality because of a longer period of seasonalization and through both thermal tempering and removal of susceptible individuals by the preceding periods of stress. On the other hand, the lethality of a particular cold wave (heat wave) can be greatly enhanced by the survival, i.e., accumulation in the population, of susceptible individuals through one or more preceding mild winters (summers).

Statistics show that mortality spikes appear in association with early or preseasonal heat and cold waves even though the ambient temperature reached during such periods may not be extreme. The fall and early winter rise in cardiovascular and respiratory mortality is, in general, steeper than the spring and early summer decline. These phenomena are particularly noticeable when the preceding contrasting season is extreme or prolonged and/or when the preceding like season is mild. Similarly, periods of extreme temperature occurring late in season or preceded by significant periods of similar temperature anomaly generally show only normal or even subnormal seasonal mortality. (Figures 13-15 and Table 18.)

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Table 18. Heat wave mortality.*

Year	Hot spell	Average daily deaths	No. days × daily excess	Excess deaths
1949	20-27 Jun	234	8 × 17	136
	19-22 Jul	223	4 × 20	80
	27-30 Jul	241	4 × 38	152
	7-12 Aug	236	6 × 45	270
1952	15-18 Jun	229	4 × 12	48
	23-28 Jun	320	6 × 103	618
	13-24 Jul	239	12 × 36	432
1953	4-8 Jun	251	5 × 34	170
	19-23 Jun	251	5 × 34	170
	16-19 Jul	233	4 × 30	120
	24-31 Aug	275	8 × 84	1002
	1-5 Sept	269	5 × 66	
1955	1-9 Jul	266	9 × 63	567
	16-25 Jul	235	10 × 32	320
1956	12-16 Jun	288	5 × 71	355
1957	12-19 Jun	276	8 × 59	472
	20-24 Jul	262	5 × 59	245

*[Kutschenseuter, P. H. Ph.D. thesis (Rutgers, New Brunswick, NJ, 1960).]

Fig. 13. Response of daily mortality to temperature during heat waves that exceed minimum conditions. The conditions defining a heat wave which resulted in increased mortality (with a 1-day phase lag) were found to be: (a) three or more consecutive days with temperatures consistently above the seasonal normal and with 3-day mean departures from normal $\geq 5^{\circ}\text{F}$ and (b) maximum temperature on at least one day greater than 90°F and the departure from normal on at least one day $\geq 10^{\circ}\text{F}$. [Kutschenreuter, P. H., Ph.D. thesis, Rutgers University, New Brunswick, NJ (1960).]

Date	T_x	T_n	T	ΔNT	Wind
15	93	68	81	+10	SW 7.8
16	85	69	77	+7	SE 6.0
17	95	70	83	+11	SW 8.4
18	88	70	79	+7	W 10.2
19	87	66	77	+5	SW 10.7
20	78	59	60	-3	NW 8.8
21	64	59	62	-11	NE 8.0
22	62	58	60	-13	NE 9.4
23	64	58	61	-12	E 7.7
24	85	62	74	+1	SW 7.0
25	99	75	87	+14	S 8.6
26	100	81	91	+17	W 7.7
27	90	72	81	+7	N 7.9

T_x Maximum temperature

T_n Minimum temperature

T Mean temperature

ΔNT Departure from normal temperature

Wind Prevailing direction and average hourly velocity

Temperature, °F

- Weekly means of daily maximum temperatures in 1934 period.
- - - Normal weekly means of daily maximum temperature based on corresponding weeks of 40 to 60 years.
- Actual weekly maximum temperature in 1936 period.

Death rate from all causes per 1000 population (annual basis)

- Weekly rate in 1934 period.
- - - Average weekly rate for corresponding weeks of 1930-1933.

Fig. 14. Weekly death rates from all causes and weekly temperature records in the summer of 1934 for nine cities in the drought area and three cities not affected by the heat wave. Simple averaging of the weekly death rates in the eight cities with definite mortality peaks (Chicago, Cincinnati, Columbus, Des Moines, Indianapolis, Kansas City, MO, Omaha, and St. Louis) gives a mean death rate of 25.5 per 1000 population (annual basis) as compared with an expected normal rate for this season of the year of 12.4. This represents a weekly mortality excess of 106% due to hot weather for the combined populations of these eight cities. [Collins, S. D., Public Health Reports 49, 1015 (1934).]

Fig. 15. Nursing home deaths, maximum daily temperatures, and smog alert days, Los Angeles County, California, July 1955 through December 1955. Unlike London's pollution, the worst Los Angeles smog attacks occur during hot weather. Some of the attacks occurred, for example, just prior to and following a heat wave in late August and early September in 1955. About 1000 excess deaths occurred during that period, but it is generally agreed that it was the heat (a week with maximal temperatures over 38°C) that was the major contributor. † indicates days of official alerts (ozone at 0.50 ppm or higher). [State of California, Department of Public Health.]

Electromagnetic Emissions

Radio- and Low-Frequency Radiation

An area of investigation which is related to and often spoken of in the same context as electromagnetic waves and electromagnetic fields, is high-frequency electric currents. Their ability to heat tissue simulates the effect of electromagnetic waves. Consequently, high-frequency alternating currents have their medical application in diathermy, and have been erroneously given the name of "ultra-shortwave" (USW) therapy.

There is sufficient evidence in the literature indicating that electromagnetic radiation in the radiofrequency and very-low-frequency range is capable of producing thermal effects which are, therefore, absorbed-dosage dependent as well as nonthermal effects which are field dependent and not necessarily related to heating. These effects are primarily concerned with central nervous system and peripheral nervous system.

A long-term study was made of these effects with systematic medical examinations of about 250 men working at 500- and 750-kV substations. These results were compared with medical examinations of men working at lower voltage substations. The studies showed that long-time work at 500-kV substations without protective measures resulted in changes of the dynamic state of the central nervous system, heart, and blood-vessel system, and in changing blood structure. The severity of these effects depended directly on the length of stay in the field.

Another study showed that some types of cardiac pacemakers are susceptible to disruption even from very small electromagnetic fields. These results have been confirmed by other experiments. Currents as small as 50 μ A disrupt the operation of demand-type pacemakers, and currents of this size are running continuously in the bodies of people standing on the ground under the 765-kV lines.

An experiment on nerve and brain tissue demonstrated that electric fields over a certain critical intensity can affect the function of these very delicate tissues. Brain organelles were found to be especially sensitive to electrical effects. More research is needed to define the exact level at which these disruptive effects begin to occur.

In another experiment, monkeys were exposed to electric fields in air of power-line frequency and there was a significant alteration of the interresponse time of the exposed animals. This change in time perception is believed to be a very sensitive measure of conditions which, if long-term exposure occurred, would result in important behavioral and neurological changes.

Ultrasonic Waves

It is suspected that the biological effects of ultrasonic waves are related to the local generation of heat. Whether any cellular destruction is associated with cavity formation in tissues is not known.

Hearing loss and effects on the heart and blood vessels have been observed, such as vasoconstriction, that may affect the adrenal cortical function. Observations of the mother and fetus in the course of several thousand obstetric procedures did not indicate any hazard.

However, no long-term follow-up studies have been done on those receiving ultrasound therapy in medicine. Experimental biological studies at reasonably low exposure intensities (as opposed to clinical studies) suggest strongly that undetected biological damage is associated with diagnostic exposure.

Where ultrasound passes into the air, as in certain industrial applications, the probability of tissue damage is lower than in the case of direct contact, since the coupling obtained between ultrasonic transducers and air is very poor.

The dangers of airborne hearing damage, systemic effects, and psychological effects necessitate caution even in the case of airborne ultrasound. Such airborne ultrasound is sometimes emitted by certain machines, so that surveillance is necessary based on appropriate measurements.

Microwave Radiation

The biological effects generated by exposure to microwave radiation (Fig. 16) are usually designated as thermal or nonthermal in nature. Thermal effects are those interactions which are caused by the heating of the biological specimen and can be duplicated using conventional heating techniques.

Nonthermal effects are due to the direct interaction of the electromagnetic field of the incoming microwave radiation and the biological specimen and are more difficult to detect than the thermal effects.

The two organs of the body which are particularly sensitive to elevation in temperature are the testicles and the eyes (Table 19). Research on dogs, rabbits, and rats has shown that at 10 mW/cm^2 (power density in milliwatts per square centimeter), pathological damage to the testes include a degeneration of the epithelium lining of the seminiferous tubules, and a sharp reduction in the number of maturing spermatocytes. The reduction in testicular function due to the heating effect at 10 mW/cm^2 appears to be temporary and reversible.

Cataracts have been produced in the eyes of experimental animals. Several investigators have used the eyes of rabbits to establish threshold for cataract formation. For cw radiation, the threshold in rabbits for long-term exposure was measured to be approximately 100 mW/cm^2 . Several cases of eye cataracts in man due to microwave exposure have been reported at power densities of the same order of magnitude.

Modeling studies indicate that the human vestibular apparatus (the inner ear) may be responsive to low-level microwave radiation via indirect, caloric stimulation.

The most often reported effects of low-level microwave radiation are neurological in nature (Table 20). Effects on animals include changes in EEG patterns, changes in the conditioned reflexes, alterations in sensitivity to light, sound, and olfactory stimuli, alterations in the biocurrents of the cerebral cortex, and changes in behavior. Many subjective symptoms in humans working around microwave equipment have been reported by USSR and Eastern European researchers.

Genetic effects have been reported by some investigators. Exposure of chick embryos to microwave radiation induced abnormal development, while conventional heating to the same temperature did not cause abnormalities. In general, the abnormalities appeared to be caused by the inhibition of growth and cell differentiation. Abnormal development of the "mealworm" beetle pupae has also been detected when exposed to microwave radiation. This study also indicates that the abnormalities were not due to thermal effects but due to the inhibition of cell differentiation caused by a direct interaction with the electromagnetic field.

Theoretical calculations demonstrate that radiation levels from powerful VHF broadcasting stations could be high enough to cause concern over pacemaker interference and electroexplosive devices under certain circumstances. Epigastric distress and/or nausea may occasionally occur at as low as 5 to 10 mW/cm² and are most commonly associated within the frequency range from 8×10^3 to 12×10^3 MHz.

As an added factor, the radiation may be up to 10,000 times higher than the levels which Soviet Bloc researchers believe to have direct effects on the central nervous system and other biological functions.

Laser Radiation

The main biological effects of laser radiation are due to heating of the tissues and are essentially local. There may also be nonthermal effects, such as crythema of the skin due to photochemical reactions. Nonthermal effects occur mainly with high-power-density pulsed lasers. No genetic effects or late effects such as skin cancer, have been reported.

The primary hazard from laser radiation is exposure of the eye. Eye damage can range from mild retinal burns, with little or no loss of visual acuity, to severe lesions with loss of central vision, and ultimately to gross overexposure with total loss of the eye.

The other organ of concern, besides the eye, is the skin. It is not as sensitive as the eye and, if damaged, is usually more easily repaired. However, it may be also subject to severe damage from laser impact when energy densities approach several joules per square centimeter (J/cm²). Laser damage to the skin may range from a mild erythema to a surface charring, and ultimately to a deep hole literally burned and blown into the skin.

Under certain circumstances, the organ of concern may also be the blood vessels or the underlying organs. Blood vessels may be easily occluded or cauterized by a laser hit.

Ultraviolet Radiation

The biological effects of uv radiation depend on the absorbed dose and on the wavelength and intensity of the radiation. They result mainly from

the absorption of energy by the proteins and nucleic acids of the exposed organism. Skin erythema and burn is produced by uv radiant energies with wavelengths from about 100 to 300 nm.

The extent of penetration by uv radiation is usually slight, except for such tissues as the lens and humors of the eye, so that the critical organs are usually the skin and eyes. Exposure of the surface layer of the skin to such radiation may result in erythema, skin cancer, rapid skin aging, and photosensitization.

In the case of the eye, the radiation is absorbed primarily in the cornea and may produce lesions that could lead to blindness. Some portions of the uv spectrum are mutagenic and may be lethal to cells:

- Keratitis in the eye (inflammation of the cornea) occurs predominantly at wavelengths below 330 nm.
- Conjunctivitis in the eye. The action spectrum for this effect appears to be similar to that for skin erythema.
- Lenticular fluorescence occurs in the eye at wavelengths around 360 nm (black-light region). This is a fluorescence of the lens which occurs only during exposure and produces no adverse effects at exposure levels which are common today.
- Skin carcinogenesis can occur at extremely high exposure levels. Statistically, it is found most often in people exposed to the sun for large fractions of their lives.

Human sensitivity to sunlight is controlled by heredity. This is exemplified by genetic deficiencies in melanin formation and thus the absence of tanning (as in Irish, Scottish, and Welsh peoples); deficiencies in cellular capacity to repair solar radiation damage, as in the inherited disorder xeroderma pigmentosum; metabolic over-production of porphyrin (a natural photosensitizer); and altered tryptophan metabolism.

The wavelengths of sunlight below 320 nm, those modulated by the presence of ozone in the stratosphere, are the most detrimental to biological systems (Fig. 17). Yet it is this same wavelength region of light that produces the essential vitamin in the skin of man - vitamin D. Thus, the situation is one of balance; sunlight is necessary for life, yet in excess it is harmful.

Currently three major enzymatic pathways are known for the repair of uv-induced damage to DNA:

- The damaged part of the deoxyribonucleic acid molecule is restored to its functional state in place.
- The damaged sections of a DNA molecule are cut out and replaced with undamaged nucleotides to restore the normal function of the DNA.
- The damaged section of DNA is not directly repaired but is bypassed during replication.

While uv radiation produces very few breaks directly in DNA molecules, in the process of repairing uv-radiation-induced damage to DNA, enzyme-induced breaks are produced in the DNA. These breaks are then subsequently repaired.

The therapeutic value of uv radiation has been recognized for a long time. "Rest and sunshine" has been prescribed for ills since prehistory. Classic examples of the medical use of uv radiation are the treatment of tuberculosis and rickets.

Ultraviolet light's germicidal properties are employed in hospitals and operating rooms and in restrooms. Ultraviolet radiation can kill or inactivate viral strains which are responsible for influenza and other upper respiratory infections in humans.

The assessment of the changes caused by impact of increased solar uv on humans is complicated by the nature of the organisms, the water body under study, and the quality of water which is considered tolerable.

There can be no reasonable doubt, on the basis of animal experiments and human epidemiologic data, that frequent and prolonged exposure to sunlight is the major cause of basal-cell and squamous-cell carcinoma in man, and that the shortest uv wavelengths penetrating the atmosphere are mainly responsible.

The most conservative estimates suggest that a 5% decrease in ozone would result in an eventual increase of from 30,600 to 44,000 additional cases of skin cancer per year (approximately 10 to 15% of the present incidence rate).

Under these field conditions more subtle yet important physiological processes were altered. Mutation rates of stamen hairs on a special test line of Tradescantia were significantly enhanced by the higher-uv-radiation regime. Also, ultrastructural modification of chloroplast membranes (as shown by electron microscopy) was also apparent in soybean leaves exposed to the enhanced uv radiation in the field.

A 400-W high-intensity mercury vapor discharge lamp, whose outer uv absorbing envelope is broken, emits sufficient radiation to cause severe photokeratitis and facial erythema in humans at distances of more than 20 ft.

Ultraviolet radiation from the sun is usually of no importance, since the natural ozone layer in the stratosphere absorbs the most damaging wavelengths. However, it has been estimated that if the ozone layer were depleted 0.5% by energy-related chemical emissions, skin cancer occurrence among whites would increase 1%.

Visible Light

Because of its narrow depth of penetration, visible light in general does not manifest itself as a potential hazard. However, there are situations in which it can become hazardous. Probably the one of greatest danger is that of high-intensity light which may cause (1) transient loss of visual function (flash blindness) or (2) irreversible thermal injury of the retina.

Flash blindness is a relatively new problem to man occurring only since man has developed sources of light brighter than the sun. It results from the bleaching of the visual pigments. Flash blinding can be prevented with proper filtering of light when the occurrence is expected. The second of these effects on the eye, irreversible thermal injury, has been known for years. For example, lookouts for airplanes have on occasion suffered retinal lesions which appear to be caused by heat generated at the sun's image on the retina.

With regard to the eye there appear to be three predominant factors controlling the potential of the hazard: (1) intensity, (2) pupil dilation (i.e., area of exposure), and (3) length of exposure. If these factors can be controlled to keep the absorbed energy below the threshold of thermal burning, reported to be between 40 and 50 cal/cm²·min⁻¹, no eye injury would be expected.

Directly beneficial effects of light produced by artificial sources include the photorepair of uv damage, treatment of jaundice of the newborn infant with blue light, destruction of the virus of herpes simplex when stained by certain photosensitizing dyes, diagnosis of some hereditary diseases in utero by activation of fluorescent dyes in fetal cells, and, recently, the destruction of certain cancer cells by visible light irradiation after their incorporation of photosensitizing agents.

The psychological effects of light, particularly of colored light, are well known but not well understood. These effects may bear a causal relationship to purely biological processes in the brain induced by light, which in turn will affect psychic behavior. Light intensity as well as wavelength specificity may alter productivity and mood. In the infant, sensory overload by prolonged exposure to highly intense illumination may produce undesirable effects on development. Indeed, the manipulation of the lighting environment of adults as well as of infants can have consequences of which we may be quite unaware.

The penetration of visible light in tissues deeper than the skin has not been adequately measured. The penetration of uv radiation has been measured with some success, so that we do know much about the photochemical actions within these superficial cells, but the photochemical action of visible light upon deep tissues is an area of study that needs much further work.

Effects of Near Ultraviolet Light (320 to 400 nm)

The wavelengths of sunlight which are modulated by the ozone in the stratosphere, i.e., 280 to 320 nm, are the wavelengths of light that produce sunburn and skin cancer. Wavelengths longer than 320 nm are very ineffective in this regard. However, if both types of light are used together they produce a greatly enhanced sunburn reaction in human skin.

Electromagnetic Field Radiation

In one study, 10 linemen who did repair work on 345-kV lines were checked throughout a 9-year period with 7 medical examinations. At the end of that time 3 of the 10 linemen had a reduced sperm count; however, it would be difficult to draw any conclusion on this effect from such a small sample.

In 1962, after the first 500-kV lines had been operating in the Soviet Union for several months, men working at the substations began to complain of headaches and a general feeling of malaise. Abnormal fatigue and sleepiness mentioned by a majority of the workers were associated with exposure to the electric fields.

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Table 19. Thermal-biological effects of microwaves.*

Frequency (MHz)	Wavelength (cm)	Site of major tissue effects	Major biological effects
>10,000	<3	Skin	Skin surface acts as reflector or absorber with heating effects
10,000	3	Skin	Skin heating with sensation of warmth
10,000 to 3,300	3 to 10	Top layers of skin, lens of eye	Lens of eye and testicles particularly susceptible
10,000 to 1,000	3 to 30	Lens of eye	Critical wavelength band for eye cataracts and testicular damage
1,200 to 150	25 to 200	Internal body organs	Damage to internal organs from overheating
<150	Above 200		Body is transparent to waves above 200 cm

* [McRee, D. I., J. Air Poll. Cont. Assoc. 24, 122 (1974).]

Table 20. Clinical manifestations of chronic occupational exposure of humans to microwave radiation.*

Neurological effects

1. Bradycardia
2. Disruption of the endocrine-humoral process
3. Hypotension
4. Intensification of the activity of thyroid gland
5. Exhausting influences on the central nervous system
6. Decrease in sensitivity to smell
7. Increase in histamine content of the blood

Subjective complaints

1. Increased fatigability
2. Periodic or constant headaches
3. Extreme irritability
4. Sleepiness during work

Central nervous system of animals

1. Changes in the conditioned reflexes
 2. Alterations in sensitivity to light, sound, and olfactory stimuli
 3. Changes in structure of skin receptors of the digestive and blood carrying system
 4. Alteration in the biocurrents of the cerebral cortex
 5. Reversible, structural changes in the cerebral cortex and diencephalon
 6. Appearance of various vegetative reactions
-

*[McRee, D. I., J. Air Poll. Cont. Assoc. 24, 122 (1974).]

Fig. 16. Parts of the body affected by microwave radiation. (a) Radiation at frequencies of 150-1000 MHz is absorbed by internal body organs shown. Approximately 40% of the energy is absorbed by the deep organs; the rest is either reflected or passes harmlessly through the body. Overheating and damage to organs with few blood vessels, such as the gall bladder, urinary bladder, and portions of the gastrointestinal tract, can result from microwave exposure in this range. (b) Radiation at frequencies of 1000-3000 MHz is absorbed by both the skin and deep organs. The lens of the eye is particularly sensitive to 3000-MHz energy. From 20-100% of the microwave energy absorbed, depending on the fat and water content of the tissues hit by radiation. (Fat is a poor absorber of microwave radiation.) The remaining radiation is either reflected or transmitted. (c) Radiation at frequencies of 3,000-10,000 MHz is absorbed only by the top layers of skin and the lens of the eye. Approximately 40% of the microwave energy is absorbed; the rest is reflected. [Aaransen, T., Environment 12, 2 (1970).]

Fig. 17. Comparison of atmospheric ozone content with incidence of skin cancer and melanoma. Skin cancer and melanoma among whites decreases in the higher latitudes as the atmosphere ozone content increases. (a) Variation of ozone with latitude at height of 11.4 mi. (b) Variation of melanoma with latitude. (c) Variation of skin cancer (apart from melanoma) with latitude. The role of atmospheric ozone in protecting susceptible whites from uv radiation and skin cancer is believed illustrated by the combination of a decrease in such cancer at higher latitudes and a rise in ozone quantity. The ozone measurements were made last year by U-2 plane. Cancer incidence is for 1970 in the U.S. and is age adjusted. [The New York Times, February 7, 1975, p. 8. Copyright 1975 by the New York Times Company. Reprinted by permission.]

Noise

General Health/Social Impact

Noise-induced hearing loss is the most significant physical health problem from exposure to excessive noise. It is estimated that in 1975 7 million industrial workers in the U.S. were exposed to occupational noise levels that could damage their hearing. Lower levels of noise encountered in the everyday environment of the 130 million residents of metropolitan areas may also cause some degree of hearing loss (Fig. 18).

Noise produces physical and mental stress, at least in some people; more than half the people responding to surveys in major U.S. metropolitan areas admit to being annoyed by the levels of noise to which they are exposed. The complexity of the problem of annoyance indicates that progress will continue to be slow and that no easy or immediate solution is probable.

Pathological Effects of Noise

Although there is no doubt that noise causes annoyance and may adversely affect well-being, there is as yet no clear epidemiological proof that the various physiological and psychophysiological responses to noise will, in the long term, lead to pathological changes. Long-term comparative studies are needed in order to gain a fuller understanding of this problem. Deafness is, of course, the major hazard involved in exposure to noise that is too intense or too prolonged.

Deafness may occur suddenly due to acoustic trauma following exposure to very-high-intensity noise, but more usually appears gradually as a result of repeated exposure to noise. The most typical case is that of occupational deafness, in the early stages of which hearing impairment, as determined by audiometric methods, occurs in the 3000- to 6000-Hz octave bands. The individual concerned is in most cases unaware of this impairment. Damage then becomes more serious and its extent increases; it becomes particularly troublesome when the voice frequencies are affected. Impairment progresses in an irregular manner toward severe deafness, associated with very high levels of mean hearing loss. Although there are some similarities between effects, such as temporary threshold shift, auditory fatigue, and acoustic trauma,

no conclusive evidence exists of any definite relationship between them.

Although the immediate effects of noise exposure on some body functions, such as pupillary dilation, peripheral circulation, and respiratory changes, are reasonably well understood, the practical implications on the general health of the average individual have not been determined. Generally, humans adapt very well to stimuli such as noise, and most extra-auditory response changes measured appear to be transitory or short-term alterations.

Perhaps of greater importance is the question concerning possible adverse effects due to regular long-term exposure to noise over many years. With the exception of hearing, evidence is not sufficient to permit us to determine if long-term exposure on a regular basis is either harmless or potentially harmful to health. Consequently, it may be wise to proceed on the assumption that long-term exposure might only be harmful to health and well-being and to continue under this assumption until it can be proven otherwise.

Psychological and Physiological Effects

It has not been demonstrated that people are having their lives shortened by exposure to audible noise. Perhaps the stress of continued exposure to high levels of noise can produce disease or make us more susceptible to disease, but, overall, the evidence is not convincing.

The effects of noise on people have not been successfully measured in terms of excess deaths, shortened lifespan, or days of incapacitating illness. There are only hints that such effects might exist. Of course, there may be accidental deaths or injuries because warning signals were not heard or were misunderstood because of noise.

There is clear evidence that exposure to noise of sufficient intensity and duration can:

Permanently damage the inner ear with resulting permanent hearing losses that can range from slight impairment to nearly total deafness.

- Result in temporary hearing losses; repeated exposures to noise can result in chronic hearing losses.

It is also apparent that noise can (Figs. 19-22 and Table 21):

- Interfere with speech communication and the perception of other auditory signals.
- Disturb sleep.
- Be a source of annoyance.
- Interfere with the ability to perform complicated tasks and, of course, can especially disturb those tasks that demand speech communication or response to auditory signals.

These latter effects are difficult to quantify, since they affect the essential nature of human life - its quality. But alone they are sufficient to require more efforts toward controlling the problem.

References - Noise

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Table 21. Subjective responses to various airborne ultrasonic exposures generated by ultrasonic instrumentation.*

Ultrasonic Instrument type	Operating characteristic	Subjective response	Sound pressure levels (dB) at midpoint frequencies (third octave bands) (kHz)					
			10	12.5	16	20	25	31.5
Drill	Input freq. -23.1 -26.1 kHz 400 mA	Pain in ears after few minutes. Headache after 1 h. Nausea after 2 h.	91	85	90	81	107.5	84
Welder	Input freq. -15,750 Hz, intermittent exposure, on 1.5 s, off 5 s 1 transducer	Malaise, backache, individual wore earplugs and symptoms disappeared, operator had a cold when symptoms were experienced.	76	95	115	93	73	69
Cleaner	Input freq. -20 kHz	Operator didn't use the machine, the machine noise was so intense.	125	123.5	119.5	113	133.5	110
Cleaner	Input freq. -40 kHz	Headache, malaise, fatigue, when necessary to use, the operator leaves the room.	77	78	79	96	77	77
Cleaner	Input freq. -20 kHz 1 transducer, water circulating at 20°C	Fatigue and malaise. Shrill sound of 94 dB at 4 kHz midpoint frequency.	81	78	84	93	76	76

*[Johnson, Lavern, "Auditory Response Thresholds and Habituation During Sleep," paper presented at the 1971 Annual Meeting of the Committee on Hearing, Bioacoustics, and Biomechanics, Cocoa Beach, Florida, March 31, 1971.]

Highway vehicles (typical number of hours use per day on day of use)

Aircraft

Rail vehicles

Recreational vehicles (typical)

Probable region
of concern for
nonoccupational
exposures

Occupational
safety and
health act
criteria

50 60 70 80 90 100
Equivalent 8-h exposure level - dBA

Fig. 18. Potential hearing damage from transportation system components in terms of equivalent 8-h exposure levels, for passengers or operators. Numbers at left-hand side of bars are averages; numbers at right-hand side are maxima. [Report to the President and Congress on Noise, Document No. 92-63 (USGPO, Washington, DC, 1972).]

Fig. 19. Performance on a postural equilibrium task during whole-body (chamber) and auditory (earphone) exposures to broadband noise. Asymmetrical exposures used two hearing protectors (earplug and earmuff) at one ear to provide about 20 dB less noise and only one hearing protector (earplug) in the other ear. Symmetrical exposures employed only earplugs in both ears. [Nixon, C. W., C. S. Harris, and H. E. van Gierke, Rail Test to Evaluate Equilibrium in Low-Level Wide Band Noise, U.S. Air Force, Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, Ohio, Rept. No. AMRL-TR-66-85 (1966).]

Fig. 20. Representative low-frequency and infrasonic noise environs and human subjective responses to the exposures. [Mohr, G. C., J. N. Cole, E. Guild, and H. E. von Gierke, "Effects of Low Frequency and Infrasonic Noise on Man," Aerospace Medicine 36, 9 (1965).]

Tolerance data	
Exposure	Observed behavior
0-50 Hz Up to 145 dB	Chest wall vibration, gag sensations, respiratory rhythm changes, postexposure fatigue; voluntary tolerance not exceeded
50-100 Hz Up to 154 dB	Headache, choking, coughing, visual blurring, fatigue; voluntary tolerance limit reached
Discrete frequencies	Tolerance limit symptoms
100 Hz at 153 dB	Mild nausea, giddiness, subcostal discomfort, cutaneous flushing
60 Hz at 154 dB 73 Hz at 150 dB	Coughing, severe sub-sternal pressure, choking respiration, salivation, pain on swallowing, giddiness

2

Fig. 21. Variations in amplitude of the finger pulse and in pupil size of subjects exposed to white noise at several different sound-pressure levels. [Jansen, G., Noise as a Public Health Hazard, OSHA Publication No. 4 (USGPO, Washington, DC, 1969).]

Fig. 22. Amount of noise-induced permanent threshold shift (PTS) experienced in groups of men who worked from 4 to 33 years in noise environments. [Glorig, Aram, W. Dixon Ward, and James Nixon, "Damage Risk Criteria and Noise Induced Hearing Loss," Arch. Otolaryngology 74, 413 (1961).]

Magnetic Field Emissions

The human community has been living with 60-Hz magnetic fields of moderate, but ever increasing, intensity for over 50 years without any demonstrated cases of injury, disability, or even noticeable discomfort. Therefore, it is very unlikely that weak magnetic fields are doing anything very injurious to us individually or as a population. Studies in Europe, Asia and Australia with 50-Hz magnetic fields and several other frequencies show a similar lack of noticeable effect.

However, human subjects can, under some conditions, sense the presence of a 60-Hz magnetic field. Researchers have reported a wide variety of such effects, ranging from interference with normal embryonic development of frogs, to a statistical relationship between psychiatric disturbances and natural magnetic field intensity. Major biological effects of a basic nature have been cited in the literature.

The production of magnetophosphores by the application of alternating fields is a well-accepted phenomenon. The major question, at this time, seems to be the mode of action responsible.

Medium-to-high-strength, nonuniform steady-state fields have well-documented effects upon rapidly growing tissues. Steady-state fields with strengths approximating the natural geomagnetic field are reported to produce alterations in the pattern of biological cycles.

There is general agreement in the literature that whole-body exposure to fields of a few thousand gauss should be avoided. This is primarily based on the evidence that genetic effects occur in insects and animals at such levels. However, there was no clear evidence on how serious such effects would be for humans.

References - Magnetic Field Emissions

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X Radiation

Ionizing Radiation

There are two kinds of biological effects of ionizing radiation:

- Genetic effects, which are transmitted to the offspring of the exposed individual by mutations in the genes. Most experts agree that any exposure to radiation, including natural sources, can cause some genetic alteration, although long-term genetic changes are very difficult to measure for small doses.
- Somatic effects, which impair the health or shorten the life of those exposed to radiation. There is considerable debate over whether exposure to very low radiation levels causes somatic damage such as cancer. Some scientists say that any dose of radiation is harmful. Others hold that low doses of radiation are not necessarily harmful below a certain threshold level. Unfortunately, the evidence is not clearcut in deciding between the two hypotheses. As a result, radiation exposure standards must be set as low as possible while balancing benefits (such as medical x rays) against the probability of harm from low doses.

Genetic Effects - Concern over radiation exposure arises from the fact that radiation is a mutagenic agent. Each new mutant added may result in harm to some descendant, and any additional number of defective individuals, of course, constitutes a greater burden to society. Some idea of the delicacy of the DNA molecule can be obtained by considering that there are only 14 mg of DNA in the whole world, which took hundreds of centuries to evolve, and would stretch from the earth to the moon.

Evidence from the experiments with mice suggest that the frequency of mutations increases linearly with dose, but is also a function of dose rate. In male mice at high dose rates (90 rad/min) the doubling dose - the amount of radiation which would double the incidence of mutations - is approximately 90 rem, while at lower measured dose rates (1 to 9 rad/min) the mutation frequency is one-third to one-quarter of this rate. Using these data, estimates have been made of the mutation frequencies and genetic deaths in man which may be contributed by natural background radiation, diagnostic

x rays, and other sources of radiation. It has been estimated, and not without contention, that the number of genetic deaths introduced into the U.S. population each year by the genetically significant population dose from diagnostic x rays - 5 arem/y in 1964 - could range from 1,100 to 44,000 deaths per year.

Somatic Effects - The neoplastic diseases, in particular leukemia and cancer, are the somatic health effects that are of most concern. Neoplasia may be a long-delayed consequence of a large acute exposure or may follow chronic irradiation. It is by no means an inevitable result of any form of human exposure. A single dose of 200 rads roughly triples one's chances of developing leukemia within a period of 10 years following irradiation. Low doses have been implicated in the case of childhood leukemia and other neoplasms resulting from diagnostic irradiation of the embryo during later stages of development. There is evidence that the leukemia risk of those children exposed prenatally to doses of not more than a few rads is at least 40% higher than the leukemia risk in children not so exposed.

When portions of the body are irradiated with higher doses than can be administered to the whole body without death, a variety of tumors may be produced, depending on the region being irradiated. Occupational exposure of the hands in earlier decades produced many instances of skin lesions, precancerous changes, and malignancies. Neoplasms of the skeleton and immediately surrounding tissues resulting from long-term retention of radium in radium dial painters have received particular attention. The incidence of lung cancer in uranium miners is another example of a malignant neoplastic disease produced by radiation.

All forms of cancer can be increased by ionizing radiation, and one way to describe the phenomenon is either in terms of the dose required to double the spontaneous incidence rate of each cancer or, alternatively, as the increase in incidence rate of such cancers per rad of exposure:

For adults

{ ~100 rads as the doubling dose.
~1% increase in incidence rate
per year per rad of exposure.

For youthful subjects
(<20 years of age)

{ Between 5 and 100 rads as the
doubling dose.
Between 1 and 20% increase in
incidence rate per year per
rad of exposure.

For infants in utero

{ ~6 rads as the doubling dose.
~17% increase in incidence rate per
year per rad of exposure.

Cataracts - Sufficient exposure (on the order of 200 rads from x or gamma rays) of the lens of the eye to ionizing radiation may cause cataracts. Radiation-induced cataracts differ from radiation-induced leukemias and other effects because they can be differentiated in many cases from the corresponding defect due to other causes. A long latent period (5 years or more) exists between the time of exposure and the onset of the development of cataracts; however, for very high doses the interval can be reduced to months.

Growth and Development - At exposure levels substantially higher than those contemplated for occupational exposure, serious effects on growth and development are demonstrable in animals and man. For example, Japanese children exposed to atomic bomb irradiation showed some retardation of both growth and maturation. While growth and development have no special significance for adults occupationally exposed to radiation, the situation is markedly different in the prenatal child. As many as 24 types of major abnormalities have been reported among children irradiated in utero at dose levels between 50 and 200 rads.

Life Shortening - Although a prominent feature of radiation effects is the ability of cells, tissue, and organs to recover, at least partially, from sublethal changes, some changes are induced at the cellular or higher level that may lead to late effects. Irreversible injury, which does not

produce a recognizable, specific disease, may be expected to decrease the vitality or reserve capacity of these cells and organs in which it occurs. Thus, following sufficient exposure to radiation, recognizable shortening of life span may be expected to occur either in association with increased or earlier incidence of specific disease or from more general causes, at least superficially similar to premature aging. No life shortening has been observed in humans, but it can be estimated from experimental effects in animals. The expected human life shortening from nuclear plant effluents at their current release levels is estimated to be about 24 seconds. It is important to compare this with the life-shortening effects of other agents (Tables 22 and 23 and Fig. 23).

Mutagenesis in its broadest sense - including somatic and germinal effects, point mutagenesis, chromosome breakage (clastogenesis), and carcinogenesis - is currently the dose-limiting biological criterion for setting radiation standards. This same dose-limiting genetic criterion will probably apply to many other energy effluents and to the spectrum of commonly used drugs, food additives, and the chemicals of industry and agriculture. Understanding such effects and developing tools for their assay present a critical challenge to a technological society and are exciting areas for biological research.

Our greatest capability for measuring genetic effects is in micro-organisms. There is a pressing need to relate such effects to higher organisms and to develop methods of sufficient sensitivity and power to measure genetic damage directly in man.

References - X Radiation

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Hickman, R. G., Tritium in Nuclear Fusion Power, Lawrence Livermore Laboratory, Rept. UCRL-75546 (1974).

Report from the National Research Councils Committee to Study the Long-Term Worldwide Effects of Multiple Nuclear Weapons Detonations, NAS-News Report (1975), p. 9.

Schurr, S. H., Energy Research Needs, Resources for the Future, Inc., Washington, DC, Publ. PB-207516 (October 1971).

Table 22. U.S. population exposure from different sources of radiation, 1970.*

Source	U.S. population at risk in 1970		Average per capita dose to population at risk in 1970 ^a	Average per capita dose to U.S. popu- lation in 1970 ^a	Total man-rem per year in 1970 (millions)	Total estimated man-rem per year in 2000 ^e (millions)
	Percent	Millions	(mrem)	(mrem)		
A. Natural						
1. Cosmic.....	100	205	45	45	9.2	14.4
2. Terrestrial:						
a. External.....	100	205	80	80	22.5	19.3
b. Internal.....	100	205	25	25	5.1	8.0
Subtotal (natural).....	100	205	130	130	26.6	41.7
B. Radioactive contamination in the environment^c						
1. Worldwide (allow except tritium (H)).....	100	205	4.0	4.0	0.82	1.4
2. Worldwide PM ^g	100	205	0.07	0.07	0.015	0.004
3. Operating nuclear facilities: ^c						
a. Reactors.....	23	47.6 ^h	0.0092	0.0021	0.00043	3.036
b. Fuel reprocessing.....	0.7	1.4 ^h	0.113	0.0008	0.00017	0.045
c. Worldwide RTR.....	100	205	0.0004	0.0004	0.00008	0.012
d. AEC facilities.....	0.6	1.0	1.5	0.01	0.0023	3.0018
Subtotal (nuclear facilities) ⁱ				0.01	0.0032	3.117
Subtotal (radioactive contamination).....				4.1	0.84	1.4
C. Man-made sources excluding "environmental" contamination						
1. Medical irradiation:						
a. Diagnostic x rays						
(1) Medical.....	31	61.3 ^h	230 ^h	10.7 ^h	14.7 ^h	45 ^h
(2) Dental.....	22	45.9 ^h		11	7.2	
b. Radiotherapy (external or sealed source) ^j	0.01	0.025 ^h		5.1 ^h	2.5	
c. Internally administered radioisotopes.....	0.7	1.5		1.1	3.2	
Subtotal (medical irradiation).....				110	14	47
2. Occupational irradiation.....	0.4	0.772	212	0.14	0.14	0.1
3. Consumer devices ^k	100	205	2.0	0.2	3.4	4
Subtotal (man-made sources) ^l				117	20	48

*Whole-body dose unless otherwise noted.

^bBased on an estimated U.S. population in 2000 equal to 321×10^6 .

^cDoes not include small radioactive releases from fossil-fuel power plants.

^dTritium is produced naturally by nuclear explosives, and in nuclear reactors. In 1970 the vast majority of the worldwide tritium was from fallout. By 2000 approximately one-half of the estimated worldwide tritium will be reactor produced.

^eWithin 50 miles of reactor site.

^fWithin 100 km of plant site.

^gPopulation at risk in 1965.

^hThe whole-body dose is taken as the gonad dose.

ⁱThe estimated 1970 mean genetically significant dose (GSD) to entire U.S. population is 61 mrem per capita in the U.S. In 2000 the projected GSD = 93 mrem per capita in the U.S.

^jAssumed to equal 1.5 times the projected GSD.

^kOnly GSD for treatment of malignant disease reported.

^l1966 data.

^mAverage dose to 93.5 million, the U.S. population under 30.

ⁿTelevision, watches, air transport, etc.

*[Based on data contained in a June 1971 draft, "Estimates of Ionizing Radiation Doses in the United States 1960-2000," Special Studies Group under the auspices of EPA.]

Table 23. Comparison of estimated deaths due to man-made radioactivity in nuclearly stimulated natural gas with similar estimates for other sources of radiation exposure of the public in the U.S.*

Sources of exposure	Estimated deaths per million individuals exposed ^a
Natural background radiation ^b	17
Radioactivity in natural gas ^c	
Natural (radon + daughters)	0.3
Man-made	0.03
Other man-made sources of radiation	
Medical diagnostic x rays	20
Fallout from nuclear weapons	0.4
Consumer devices	0.4
Industrial uses of radiation	<0.2
Power reactors	<0.2

^aObtained by summing estimated somatic and genetic effects; therefore, some of these estimated deaths will occur among the exposed individuals or the first generation of their offspring, but a large majority (over 80%) will occur in succeeding generations.

^b0.1 rem/y.

^cBased on projected radionuclide concentrations used in this study.

*[Rohewer, P. S., C. J. Barton, R. E. Moore, and S. V. Kaye, Third International Radiological Protection Association Meeting, Washington, DC (September 1973).]

Fig. 23. Observed and expected annual lung cancer mortality per 10,000 miners and 95% confidence limits in relation to exposure. [Federal Radiation Council, Washington, DC, Rept. FRC No. 8, Revised (1967).]

Section III Water Effluents

CHEMICAL FACTORS

Trace Metals

From the standpoint of environmental effects, toxic elements can be considered (1) noncritical, (2) toxic and relatively accessible, or (3) toxic, but very insoluble or very rare. Toxic elements move through the environment under natural conditions by way of the "geocycle" from which the elements become available to plants, animals, and humans. Our activities provide new sources of toxic elements that influence the geocycle and, consequently, the availability of these elements to biological processes (Fig. 24 and Table 24).

One of the factors determining toxicity concerns the chemical form to which the organism is exposed. Some inorganic compounds are more or less toxic, depending on whether they are in the trivalent or pentavalent state; metallic-organics vary considerably in their toxicity; and the form of an element present in high concentration in shrimp and other seafoods is relatively nontoxic. For example, toxicities vary with the various forms of mercury, depending on whether it is mercury metal (quicksilver), inorganic mercury, or an organic compound containing the metal. Each form of a metal presents its own unique spectrum of toxicity.

The metabolic activity of microorganisms plays a significant part in the mobility of toxic elements in the environment. Microorganisms are exceedingly versatile in the way they metabolize natural substances, and it can be said that if they do not degrade a particular compound, then it is unlikely that higher organisms will have the capacity to do so.

In the case of metals with no biological role, however, plants and animals have generally failed to develop a similar homeostatic defense. Consequently, even small amounts of such toxic metals as lead and mercury are absorbed and readily accumulated by the organism. These metals, therefore, tend to be cumulative poisons.

The tendency for accumulation is reflected in the long biological half-life for these metals in humans -- 1460 days for lead and 10 days for mercury. It should be recognized that the biological half-life has somewhat different meanings, depending on whether one is talking about the whole body, the liver, the bone compartment, or some other tissue. A long biological half-life can obviously be an important factor influencing the toxicity of a metal.

Lead

Food furnishes 50 to 95% of the daily dose of lead. An urban dweller may take up an average of 20 $\mu\text{g/day}$ from food, and 10 $\mu\text{g/day}$ from air inhaled in the city. The rural dweller receives the same dose from food, presumably, but inhales much less lead in the air so that his uptake from the atmosphere is only 0.5 μg . Water contributes only a fraction of 1 $\mu\text{g/day}$ in either case. Smoking 30 cigarettes per day may add another 10 μg of lead to the daily dose.

Plumbism, or lead poisoning, has been reported for adults with less than 80 $\mu\text{g}/100$ cc of blood, while children have been reported to have lead poisoning with blood levels as low as 50 $\mu\text{g}/100$ cc. The average U.S. citizen has about 25 $\mu\text{g}/100$ cc, with the range from 5 to 40 $\mu\text{g}/100$ cc for persons not occupationally exposed. The margin of safety appears to be low.

Children and workers who are occupationally exposed are most subject to acute lead poisoning. From 6 to 10% of the children in lower socioeconomic groups in Chicago have been found to have blood levels of 50 $\mu\text{g}/100$ cc or more. In downstate cities, the Illinois Department of Public Health recently found 20 to 30% of such children had 40 $\mu\text{g}/100$ cc blood lead, a value near the onset of overt symptoms of plumbism. Children less than 2 years of age seem to be most susceptible. There is some evidence that certain individuals are most susceptible to lead poisoning due to genetic factor.

Mercury

The toxicity of mercury is attributed to its high affinity for sulfur-containing compounds and its lesser affinity for organic ligands. Interference by mercury in the synthesis and function of enzymes and other proteins can result in a variety of adverse effects.

Alkyl mercury poisoning has somewhat different characteristics than other mercury poisoning. Symptoms may be dormant for weeks or months after acute exposure, and if brain damage occurs, effects may be irreversible. Inhalation of alkyl mercury can produce dryness and irritation in the nasopharynx and mouth.

Alkyl mercury poisonings have caused permanent neurological damage resulting in impaired vision and hearing, sensory loss in limbs, ataxia, and tremor. Neurological disorders such as mental retardation and convulsive cerebral palsy have occurred in infants whose mothers were exposed to methylmercury during pregnancy. Fetal nerve tissue may be especially sensitive to methylmercury.

Ingestion of 1.0 to 2.0 g mercuric chloride is frequently fatal to human beings. It produces corrosion of the intestinal tract, injury to the kidney, and ultimately death due to kidney failure. The kidney appears to be the concentrator organ for inorganic mercury.

Miscellaneous Metallic Elements

A peculiar complication of the metal effluent picture is that some of the metals considered to be dangerous are, in fact, needed in trace quantities by all plants and animals, including man. The metals vanadium, chromium, manganese, iron, cobalt, copper, zinc, selenium, molybdenum, and tin are usually incorporated into proteins and serve as catalysts which initiate or assist in many biological reactions. In addition to enzymes such as these, other biologically important molecules also contain essential metals. In animals, for example, iron is an integral part of the hemoglobin used for oxygen transport, while in plants the chlorophyll molecule contains magnesium. No fewer than 51 metals have been detected in the human body, and undoubtedly some of these additional metals will eventually be shown to be necessary for life.

Essential metals are, therefore, required in small quantities for the well being of living organisms. However, the same metals released to the environment in larger quantities through man's activities or from natural sources can create toxic conditions. Zinc and copper from mining operations, for example, can easily kill fish and other aquatic organisms at very low concentrations. For essential metals, toxic levels may be 40 to 200 times greater than that required for proper nutritional response.

Thus, for each of the metallic elements in the environment, to which all living organisms experience a finite level of exposure, the critical question of toxicity cannot be answered with a "yes" or "no," but by how much. What level of exposure to a specific metal is consistent with safety? In order to determine the levels that may be tolerated in air, water and food, it is also necessary to know the level of unavoidable background exposure from all sources.

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Table 24. Classification of elements according to their toxicity. Elements omitted from this table should not be neglected in the environmental sense. For example, iodine and manganese are important elements, but they fit more than one category for the above classification.*

Noncritical			Very toxic and relatively accessible			Toxic but very insoluble or very rare	
Na	C	F ^a	Be	As	Au	Ti	Ga
K	P	Li	Co	Se	Hg	Hf	La ^b
Mg	Fe	Rb	Ni	Te	T	Zr	Os
Ca	S	Sr	Cu	Pd	Pb	W	Rh
H	Cl	Al	Nz	Ag	Sb	Nb	Ir
O	Br	Si	Sn	Cd	Bi	Ta	Ru
N				Pt		Re	Ba

^aSome may argue with this designation, but we do add fluoride to drinking water.

^bAll the lanthanides are very insoluble and some are very rare.

*[Wood, J. M., Science 183, 1049 (1974).]

Fig. 24. A comparison of the lethal dose of various metal ions according to their position in the periodic table and their charge. Dose was administered to mice intraperitoneally. [Pierre Bienvenue, M. M., Claude Nofre, and André Cier, "Toxicite Generale Comparee des Ions Metalliques. Relation avec la Classification Periodique," *Comp. Rend.* 256, 1043 (1963).]

RADIOLOGICAL FACTORS

Tritium

The effects of interest of tritium are solely those due to the impact of radioactivity on living things. Because of its relatively short biological half-time and its relatively low energy, tritium is one of the least hazardous radionuclides produced in nuclear reactors. At the projected levels of activity for the year 2000, worldwide population exposure rates from ingestion of water would be somewhat less than 0.1 mrem/yr (compared with about 125 mrem/yr from all natural sources).

References - Tritium

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Other Radioactivity

The health effects of radioactive materials ingested by humans depend in a complicated way on the half-life of the radionuclide, its mode of decay, the organ in which it is concentrated, the quantity present, the half-life (residence time) of the element in the body, and the age of the person, as well as other factors (Fig. 25).

Iodine and strontium isotopes, in particular, are concentrated by the body: infants and children are the critical sectors of the population. Iodine is taken up by the thyroid gland; strontium is incorporated in newly formed bone. Cesium, on the other hand, is distributed in the soft tissues.

Although we know that high concentrations of radioiodine can destroy the thyroid function and high concentrations of radiostrontium could produce bone cancer or changes in bone structure, no data are available as to the effects on man of extremely low concentrations such as would result from exposure of human populations to maximum permissible concentrations (MCPs) of ^{131}I , ^{90}Sr , or ^{137}Cs .

The main radiological impact of radiostrontium in man is due to its biochemical resemblance to calcium and its long half-life. When ingested, some of the strontium becomes fixed in the bones where it remains for a long time. Its subsequent decay produces radiation dose directly at the site (the 2.27-MeV highest-energy beta of the daughter ^{90}Y has a maximum range of only about 1.0 g/cm^2). Because of this, the recommended MCPs in air and water are quite small.

References - Other Radioactivity

Forbes, G. B., "Significant Isotopes in Foods," in Conference on the Pediatric significance of Peacetime Radioactive Fallout, supplement to Pediatrics 41 [1], Part II, 207 (January 1968).

Fig. 25. Pathways between radioactive materials released to ground and surface waters (including oceans) and man. [*Instrumentation for Environmental Monitoring, RAD-REA Nuclear Reactors*, Appendix C, p. 34, Lawrence Berkeley Laboratory, University of California, Berkeley, CA.]

PHYSICAL FACTORS

Thermal Inputs to Water

Field studies made to date have demonstrated that as water temperature rises it holds less dissolved oxygen. Also, as the water temperature rises aquatic organisms require more dissolved oxygen in order to maintain a normal existence, or to live at all.

The temperature requirements of a given species of fish vary throughout its life history. The requirements differ for spawning and the development of eggs and fry. The sensitivity or tolerance of aquatic organisms to temperature changes or levels also varies with age, size, and season.

Lethal high and low temperatures vary widely for different species. The difference in upper lethal levels for different species varies as much as 32°F, e.g., 41°F for the goldfish and 75°F for the pink salmon. Water temperatures do not have to reach lethal levels, however, to wipe out a species. Temperatures which favor competitors, predators, parasites, and diseases can destroy a species at levels far below those which are lethal. As water temperatures increase, bacterial action and the so-called natural purification process are speeded up. Then, oxygen depletion may occur in summer in certain areas where dissolved oxygen conditions are satisfactory in other seasons.

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Section IV

Land Use

CHEMICAL FACTORS

Acid Fallout

Effects may or may not include changes in the leaching rates of nutrients from plant foliage, changes in the leaching rates of soil nutrients, effects on predator/prey relationships and effects on the metabolism of organisms.

Increased input of acid to soil may lead to increased leaching of calcium and other nutrient elements. Acidity is also correlated with humus content. If causal, then the acid would help return minerals. Although such losses would be small for some ecosystems and unlikely to result in any significant short-term damage to arable land, they do represent other added stresses to the ecosystem. During the growing season in forested areas most of the acid rain impinges directly on the foliage where ion exchange occurs. Thus the rain that reaches the forest soil is far less acid than the incident precipitation.

Serious fish mortality, particularly early age classes in salmonoids, has been attributed directly to increased acidity from precipitation. Similar acidification of aquatic ecosystems must be occurring in other parts of the world, but it is unrecognized or is often confounded by other sources of pollution.

So long as there is sufficient lime, it is impossible to harm soil with excess SO_2 over and above crop needs. It merely causes conversion to calcium sulfate. Nor does any excess of sulfur cause an equivalent lime loss in the drainage. Most of the loss of lime from arable land is caused by solution of bicarbonate resulting from the high CO_2 concentration in the air in the soil. Sulfate, whether from SO_2 or from fertilizers, serves partly to displace bicarbonate. A sphagnum moss actually takes minerals out of rain, replacing them by hydrogen (acid) so that the runoff from such land is naturally acid. Lakes in such high-rainfall districts, such as North Wales and southwest Sweden, have been naturally acid for centuries.

The lakes in Sweden which have created the alarm are those in the high-rainfall area of the southwest, with very little buffer capacity, and consequently low productivity. There are perhaps 3000 of them, medium to small, among Sweden's 100,000 lakes. It has been known, and practiced, for almost 50 years that the lakes must be limed to give a reasonable yield of fish.

References - Acid Fallout

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Solid Effluent

Solid effluent dumped on the land is aesthetically objectionable.

Some components of spent shale effluent and sludge effluent from power plant scrubbers are significantly water soluble and carcinogenic, indicating the need to guard against uncontrolled leaching. Tests have indicated that water after intimate contact with spent shale will be highly alkaline and contain high concentrations of calcium, sodium, and potassium in the form of sulfates, whether or not mineral decomposition has occurred in the retorting process.

Additional salinity in the Colorado River system and at Hoover Dam would come from: leaching of spent shale both during waste pile buildup and after revegetation; reinjection of mine water and upward movement and surface discharge of saline waters; accidental release of low-quality mine water, including failure of evaporation ponds; and any return flows of saline water. Contributions of salts from these sources could increase the salinity at Hoover Dam to as much as 50% (1140 mg/liter).

Erosion of shale oil disposal piles (both during buildup and after revegetation) and of off-site construction areas such as utility corridors, transportation networks, and town site expansion will lead to increased sediment loads in local streams.

Up to 10 gallons of water will be produced from each ton of shale during the retorting process, and this water will have sizable quantities of polycyclic aromatic hydrocarbons, some of which will be carcinogenic. The water could be treated to remove the contaminants, but industry officials propose using the water to wet down and thus stabilize the shale refuse piles. This procedure, of course, would add to the carcinogenic material already in the piles.

The wastewater from the processed shale is different from additional industrial water which also will cause a waste disposal problem after being used in the overall mining, retorting, and disposal operation. For that operation, some three barrels of industrial water will be needed for every barrel of shale oil produced. To ranchers and farmers in the arid West, the water may be more precious than the oil.

How readily the native grasses and shrubs can grow on refuse piles is a question that will largely determine the environmental effects of solid waste. In the case of shale oil refuse dumps, many small plots show that at least three native grasses grow profusely, after watering heavily for the first year, using commercial fertilizer, and mulching. Two native shrubs have also grown, but so far (1975) none of the woody species used as browse by deer have achieved maturity.

References - Solid Effluent

Environment 16 (July/August 1974).

RADIOLOGICAL FACTORS

High-Level Storage

The principal concern with respect to high-level radiation effluent is accidental leakage into the biosphere. Modes by which this might occur include: (a) leakage from liquid-storage tanks; (b) rupture of solid-waste containers en route from processing plant to burial site; (c) migration of radioactive materials during storage. Such accidents could result in local or widespread contamination of populated areas depending on the circumstances and whether or not radioactive materials had entered the hydrosphere.

Atoms of medium atomic weight, formed by fission of uranium or plutonium, form one category of effluent. The isotopes ^{90}Sr , ^{137}Cs , and to a lesser extent ^{85}Kr , are the main elements; ^{93}Zr , ^{129}I , ^{135}CS , and others are much less important. In 700 years, less than one ten-millionth of these radioactive elements remains.

The other category of radioactive effluent consists of the actinides (the elements actinium, thorium, uranium, neptunium, plutonium, and so on), which are formed not by fission but by neutron absorption into the original uranium (or thorium) fuel. All are very toxic and most have long half-lives — for example, about 25,000 years for ^{239}Pu , the most abundant transuranium actinide, which is formed either in conventional light-water reactors or in proposed liquid-metal fast breeder reactors (Figs. 26 - 28).

The actinides cause effluent management difficulties at two distinct points in the nuclear fuel cycle; some are carried over with the fission products during nuclear fuel reprocessing, and some highly dilute plutonium wastes will appear from fuel manufacturing plants. Two of the most important things about this category of effluent are that (1) the offending actinides are relatively toxic and (2) although initially far less radioactive than the fission products, they become dominant at 500 years because of their much longer half-lives.

References - High-Level Storage

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Fig. 26. Toxicity of effluent from light-water reactors, for an equilibrium fuel cycle, with 99.5% removal of uranium and plutonium. Each metric ton of fuel is assumed to deliver a total thermal energy of 33,000 MW/day during its operating lifetime. The turn-up at 10⁶ years arises from growth of daughter products not present in the original material, which is not in decay equilibrium. [Kubo, A. S., and D. J. Rose, Science 182, 1205 (1973).]

Fig. 27. Risk of radiation-induced cancer death per μCi of Pu deposited in lung vs age at inhalation. The scale at the right is the lethal dose (i.e., dose/cancer induces) converted to mass of Pu and corrected for deposition efficiency of inhaled Pu (25%). The curves for bone and liver cancer have been corrected at young ages for reduced breathing rate in children. The curves without this correction are used for calculating effects of injection in the bloodstream and are shown by the dot-dash lines. [Fish, B. R., et al., ORNL-NSIC-74, p. 108.]

Fig. 28. Toxicity of effluent from light-water reactors, for different degrees of actinide extraction. [Kubo, A. S., and D. J. Rose, Science **182**, 1205 (1973).]

PHYSICAL FACTORS

Strip- and Surface-Mining Activities

Adverse

The ecology of streams in runoff areas is disturbed by acidic materials and silting, and the land-damaging and air emission effects can extend for miles beyond the immediate area of mining operations (Table 25). When area mining has been completed and not reclaimed, the land is left corrugated with a pattern of long parallel ridges and valleys caused by erosion and land slides.

In California, extracting 2 million tons of gravel along the Russian River has resulted in altering the channel of the river, caving in banks, washing away adjoining land, and muddying water downstream, harming both fishery and recreational resources. The Sonoma County Water Agency reports that gravel extraction also imperils the water supplies for 150,000 people in Sonoma and northern Marin counties.

Mine tailings and low-grade ore left untreated from an abandoned copper mine near Keswick in Shasta County, California, leached poisonous copper residues into the Sacramento River. After construction of Shasta Dam cut the water flow, the increased concentrations of copper became lethal to migrating salmon. As a result, the Federal government spent more than a million dollars trying to impede the flow of poisoned water, but the project has been only partially successful and additional water has had to be released from Shasta Dam to dilute the copper further.

Near the city of Sonora, in Northern California, milling wastes and tailings from a recently abandoned asbestos mine have produced dangerous dust particles. While the mine dumps are not near populated areas, the local official responsible for the enforcement of air emission standards has not ruled out potential health hazards to surrounding communities.

Beneficial

In the western U.S., some surface mines have exposed ground-water sources and made water available where none existed before. This water has proved invaluable to livestock and wildlife. At some surface-mining operations along mountainsides, the pits impound surface runoff from torrential rains, minimize the sediment load of streams draining the area, and effect considerable ground-water recharge as well.

In California, piles of dredge tailings are quite permeable. However, because of their irregular conformation, they undoubtedly inhibit surface runoff to a greater degree than the original slopes, thus making some contribution to flood control and ground water recharge.

In Alaska, dredge mining for gold has destroyed the permafrost and the resulting tailings and mined areas are considered premium property for residential and industrial development.

Surface mining has created many opportunities to develop recreational areas where none existed before. Water in the form of small ponds or lakes, and the spoil piles themselves, frequently provide a pleasant topographic change in areas of virtually flat land. Examples may be found in flat coastal areas in such states as Kansas, Illinois, Indiana, Ohio, and California.

References - Strip- and Surface-Mining Activities

Man's Impact on Environment, T. R. Detwyler, Ed. (McGraw-Hill Book Co. New York, 1971), p. 348.

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Table 25. The coal miner and his health - facts and figures (1971), Daphne A. Roe, M. D.*

Total number of living coal miners	250,000
Total number of pensioners	67,203
Total number of pensioners receiving income from social security viz: 51,330 on old age pension and 8,756 on disability pension	60,086
Total number of pensioners receiving Black Lung benefits	17,901
Total number of pensioners receiving workmen's compensation	3,243
Total number of pensioners receiving UMWA pensions	66,658
Total number of pensioners employed in nonmining jobs	4,714
Total number of miners in work force	182,797
Total number of working miners receiving Black Lung or total disability benefits	1,192
Total number of working miners receiving workmen's compensation for injuries sustained in mining accidents	2,197
Average monthly income from old age pension	\$127.42
Average monthly income from disability pension	162.97
Average monthly income from Black Lung pension	210.21
Average monthly income from WCB pension	151.10
Average monthly income from UMWA pension	150.00
<u>Annual estimates of health costs (based on January 1971 figures)</u>	
It has been estimated that disability pensions cannot be regarded as occupational health costs, because they are awarded for a variety of diseases which are not occupational.	
Occupational health costs include:	
Black Lung benefits for pensioners	\$45,155,630.52 p.a.
Black Lung benefits for miners	3,006,843.84 p.a.
WCB payments for pensioners	5,880,207.60 p.a.
WCB payments for miners	3,983,600.40 p.a.
Total	\$58,026,282.36 p.a.

*[Computed from Report of United Mine Workers of America Welfare and Retirement Fund, year ending June 30, 1971.]

Power-Plant Siting

A principal concern regarding the siting of power plants and transmission systems is the incompatibility of these systems with competitive land uses, particularly residential and recreational uses. The aesthetics of current designs leaves much to be desired, and residential communities fear for the effect on property values; the public in general and environmentalists in particular deplore the despoiling of wilderness and otherwise uncontaminated natural areas. Unfortunately, the most desirable regions for these alternative land uses, which tend to be the areas of interface between land and water, are also the most desirable for the siting of power plants because of the availability of cooling water.

A transmission system passing through wild and forested areas requires brush and tree clearing along the right-of-way in order to provide physical clearance for the transmission lines and access for inspection and maintenance activity. This clearing plus the use of herbicides for maintenance of the right-of-way are matters of considerable environmental concern.

Long-term safety of nuclear power plants is concerned with the permissible radiation levels in the vicinity of the power-plant sites and the disposal of the long-lived radioactive wastes resulting from the nuclear fuel reprocessing cycle. Short-term concerns pertain to the destructive release of radiation in reactor accidents or acts of God.

Doubts still exist that backup cooling systems and reactor containment designs are adequate to handle potential emergencies.

References - Power-Plant Siting

Environmental Effects of Producing Electric Power (Joint Committee on Atomic Energy, USGPO, Washington, DC, 1969).

Power-Plant Siting and Environmental Protection (House Committee on Interstate and Foreign Commerce, USGPO, Washington, DC, 1971).

Power Plant Siting, Its Impact on System Planning and the Environment (IEEE, New York, 1971).

Hydroelectric Dams

Construction of a large dam in southern India has raised the water table in the surrounding district making the soil more alkaline and thereby upsetting the trace element balance in the diet of local inhabitants. The tragic consequence is genu valgum, a crippling bone disease affecting young people.

Downstream from the Aswan Dam, water now flows year round contributing to increased snail population and schistosomiasis.

References - Hydroelectric Dams

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