

AIR POLLUTION HEALTH EFFECTS
OF ELECTRIC POWER GENERATION

A LITERATURE SURVEY

NORWEGIAN INSTITUTE FOR AIR RESEARCH
P.O. BOX 130, N-2001 LILLESTRØM

INSTITUTT FOR ATOMENERGI
P.O. BOX 40, N-2007 KJELLER

CONTENTS

	Page
PREFACE	11
SUMMARY, CONCLUSIONS AND RECOMMENDATIONS	13
SUMMARY OF FINDINGS BY OTHER INVESTIGATORS	13
<u>Pollutants Emitted</u>	13
<u>Health Effects of Air Pollutants</u>	14
<u>Estimating Health Effects</u>	16
<u>Comparing Health Effects of Electric Power Generation Methods</u>	17
NILU/IFA CONCLUSIONS FROM THIS WORK	18
RECOMMENDATIONS FOR FUTURE WORK IN NORWAY	20
RECOMMENDED TASKS FOR PHASE II OF THE PROJECT	21
CHAPTER 1: HEALTH EFFECTS OF ELECTRIC POWER GENERATION - AN INTRODUCTION	23
1.1 SCOPE	23
1.2 HEALTH EFFECTS FROM GENERATION OF ELECTRICITY	25
1.3 THIS PROJECT	26
REFERENCES	27
CHAPTER 2: LITERATURE SURVEY: HEALTH EFFECTS OF RADIATION	29
2.1 POWER PLANT EMISSIONS	29
2.1.1 <u>BWR, Maximum Release to Atmosphere</u>	33
2.1.2 <u>PWR, Maximum Release to Atmosphere</u>	34
2.1.3 <u>Actual Releases from BWR and PWR</u>	35
2.2 HEALTH EFFECTS OF RADIATION	39
2.2.1 <u>Acute Effects</u>	39
2.2.2 <u>Delayed Health Effects</u>	41
2.2.2.1 <u>Cancer Induced by high-level Radiation</u> ..	42
2.2.2.1.1 <u>Cancer Mortality. Hiroshima and Nagasaki</u>	43
2.2.2.1.2 <u>Mortality, Ankylosing Spondylitis</u> <u>Patients</u>	44
2.2.2.1.3 <u>Thyroid Cancer</u>	45

CONTENTS (cont.)

	Page
2.2.2.2 <u>Genetic Effects Induced by high-level Radiation</u>	47
2.2.2.2.1 <u>Genetic Effects. Hiroshima and Nagasaki</u> ..	48
2.2.2.3 <u>Health Effects of low-level Radiation</u> ...	51
2.2.2.3.1 <u>Study of the Lifetime Health and Mortality Experience of Employees of AEC-Contractors</u>	53
a) <u>Longevity</u>	54
b) <u>Differential Causes of Death, Cancer Death</u>	60
2.2.2.3.2 <u>Health of Workers in the United Kingdom Atomic Energy Authority</u>	61
2.2.2.3.3 <u>Sternglass</u>	63
2.2.2.3.4 <u>Kerala, India</u>	66
2.2.2.3.5 <u>Guarapari, Brazil</u>	67
2.2.2.3.6 <u>Argonne Radiological Impact Program</u>	68
2.3 HEALTH EFFECTS ESTIMATES	72
2.3.1 <u>ICRP, UNSCEAR and BEIR</u>	72
2.3.2 <u>The Linear Dose-Effect Relationship Hypotesis</u>	74
2.3.3 <u>Cancer</u>	77
2.3.3.1 <u>Risk Estimates</u>	77
2.3.3.2 <u>Gofman and Tamplin</u>	79
2.3.3.3 <u>Study on the Effect of Ionizing Radiation on the Fetus</u>	81
2.3.4 <u>Genetic Effects</u>	83
2.3.4.1 <u>Risk Estimates</u>	83
2.4 AN EXAMPLE OF RADIATION RISK ESTIMATION PROCEDURE..	84
2.4.1 <u>Radiation Doses</u>	84
2.4.2 <u>Acute Effects</u>	85
2.4.3 <u>Cancer</u>	85
2.4.4 <u>Genetic Effects</u>	85
2.4.5 <u>Calculated Radiation Risk</u>	86
REFERENCES	88

CONTENTS (cont.)

	Page
CHAPTER 3: LITERATURE SURVEY: HEALTH EFFECTS OF CHEMICAL AIR POLLUTANTS	95
3.1 INTRODUCTION	95
3.2 AIR POLLUTION FROM FOSSIL FUEL COMBUSTION	96
3.2.1 <u>Particulate Matter</u>	96
3.2.2 <u>Oxides of Sulfur</u>	96
3.2.3 <u>Oxides of Nitrogen</u>	96
3.2.4 <u>Carbon Monoxide</u>	97
3.2.5 <u>Hydrocarbons</u>	97
3.2.6 <u>Heavy Metals</u>	97
3.2.7 <u>Ozone</u>	97
3.2.8 <u>Radioactivity</u>	98
3.2.9 <u>Carbon Dioxide</u>	98
3.3 POLLUTANT EMISSION RATES	98
3.4 HEALTH EFFECTS STUDIES	103
3.4.1 <u>Types of Effect Studies</u>	104
3.4.2 <u>Epidemiologic Studies</u>	105
3.4.3 <u>Acute Effects</u>	112
3.4.3.1 <u>Mortality</u>	112
3.4.3.2 <u>Morbidity</u>	114
3.4.4 <u>Chronic Effects</u>	117
3.4.4.1 <u>Geographic Variations in Mortality</u>	117
3.4.4.2 <u>Geographic Variations in Morbidity</u>	118
3.4.5 <u>Studies on Children</u>	120
3.4.6 <u>Studies of Pulmonary Function</u>	122
3.4.7 <u>Studies on Special Panels</u>	123
3.5 HEALTH EFFECTS ESTIMATES	124
3.5.1 <u>Mortality Estimates</u>	124
3.5.2 <u>Morbidity Estimates</u>	129
REFERENCES	135

CONTENTS (cont.)

	Page
CHAPTER 4: LITERATURE SURVEY: METHODS OF COMPARING AIR POLLUTION HEALTH EFFECTS CAUSED BY FOSSIL FUEL AND NUCLEAR POWER PLANTS ...	149
4.1 INTRODUCTION	149
4.2 POLLUTANT EMISSION RATES	151
4.3 SPECIFIC HEALTH EFFECTS	152
4.4 OVERALL RISKS	153
4.5 AIR QUALITY STANDARDS	155
4.6 DISCUSSION	158
4.6.1 <u>Pollutant Emission Rates</u>	158
4.6.2 <u>Specific Health Effects</u>	159
4.6.3 <u>Overall Risks</u>	160
4.6.4 <u>Air Quality Standards</u>	161
4.7 CONCLUSIONS	163
REFERENCES	165
 APPENDIX: AN EVALUATION OF THE STUDY "HEALTH EFFECTS OF ELECTRICITY GENERATION FROM COAL, OIL AND NUCLEAR FUEL" BY LAVE AND FREEBURG	 167
A.1 BACKGROUND	167
A.2 STATISTICAL APPROACH	169
A.3 PARAMETERS USED	170
A.3.1 <u>Health Effects</u>	171
A.3.2 <u>Socioeconomic</u>	172
A.3.3 <u>Air Pollution</u>	172
A.3.4 <u>Other Variables</u>	173

CONTENTS (cont.)

	Page
A.4 DATA USED	173
A.4.1 <u>Health Data</u>	173
A.4.2 <u>Socioeconomic Data</u>	174
A.4.3 <u>Air Pollution Data</u>	174
A.5 RESULTS	176
A.6 APPLICATIONS	177
REFERENCES	179

Blank



PREFACE

Institutt for Atomenergi (IFA) and Norwegian Institute for Air Research (NILU) have undertaken a joint project with the ultimate purpose of comparing the relative air pollution health effects of gas-fired, oil-fired, and uranium-fueled electric power generating plants.

This report covers Phase I of the project, and includes a literature review on pollutant emissions and their related health effects. It also presents a review of the methods which have been used previously to compare the relative health effects. In this respect, one particular piece of work by L.B. Lave and L.C. Freeburg has been extensively evaluated.

David L. Brenchley has been the project leader at NILU, and is responsible for Chapters 1, 3, 4 and the Appendix in this report. (Dr. Brenchley is now at Battelle Memorial Institute, Pacific Northwest Laboratory, Richland, Washington.) Ulf Tveten and Karen Garder at IFA are responsible for Chapter 2.

-12-

Blank

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

The following is a summary of the results reported by various investigators in Chapters 2, 3, 4 and the Appendix. In addition conclusions from Phase I of this project are given, together with a list of recommendations for the tasks to be performed in Phase II. In all cases these comments must be considered in view of the scope and limitations of this project which are described in Chapter 1.

SUMMARY OF FINDINGS BY OTHER INVESTIGATORS

Pollutants Emitted

- Fossil-fueled power plants emit mainly particulate matter and oxides of carbon, nitrogen and sulfur. In addition, various hydrocarbon species and many different types of trace metals can also be emitted: benzo(a)pyrene, nickel, cadmium, cobalt, chromium, mercury, iron, zinc, copper, and others. The type and amount of pollutants emitted depend upon fuel properties, plant operation conditions, and the pollution control equipment in use. (Chapter 3, references 1, 2, 3 and others.)

- Chemical species emitted by fossil-fueled power plants undergo atmospheric chemical and/or photochemical reactions and form other types of pollutants. (Chapter 3, references 7 and 27 - 31.)
- Nuclear power plants emit radioactive noble gases and iodine to the atmosphere. The exact composition and amount emitted depends upon the type of plant and the type of off-gas equipment utilized. (Chapter 2, references 8, 10, 11 and 12.)
- Noble gases do not undergo chemical reactions and the exposure pathway is only by external radiation. The iodine exposure may be the result of direct inhalation or by the food chain pathway, e.g. air-grass-cow-milk-man. (Chapter 2, references 18 and 20.)

Health Effects of Air Pollutants

- The biological mechanisms whereby chemical air pollutants and ionizing radiation impair human health are not well-known. (Chapter 3, references 37 - 46 and Chapter 2, references 18 and 20.)
- Statistical relationships have been found which strongly correlate total mortality and morbidity with chemical air pollution variables. (Chapter 3, references 36, 38, 90 and others.)

- Investigators have had limited success in finding statistical correlations between chemical air pollution and disease-specific mortality. (Chapter 3, references 36, 90, 91, 92 and 100.)
- The health effects caused by ionizing radiation have been studied since early in this century and the knowledge compiled is extensive. (Chapter 2, references 18, 20 and others.)
- The health effects of air pollution exposure may be latent, i.e., they may not become evident until years after the period of exposure. However, for some types of pollutants, human health will improve quickly if the exposure is reduced or eliminated completely. (Chapter 2, references 18 and 20 and Chapter 3, references 38 and others.)
- The genetic effects of chemical air pollutants have not been systematically studied in human populations of any appreciable scale. (Chapter 3, references 41, 90 and others.)
- Statistical correlation analyses have revealed no connection between total mortality or malignant mortality and increased radiation level, except for very high radiation doses and dose rates (atomic bomb survivors and medical patients). (Chapter 2, references 18, 20, 27, 28, 29, 38 and others.)

- Statistical correlation analyses in human populations have revealed no connection between genetic damage and increased radiation level, even among atomic bomb survivors. These experiences are, however, not inconsistent with the theory of radiation induced genetic effects, since the expected genetic effects extrapolated from animal data would be low compared to the load of natural mutations. (Chapter 2, references 24, 36 and 37.)

Estimating Health Effects

- Investigators have developed statistical predictor equations which relate air pollution variables to total mortality. They have been less successful in relating air pollution variables to specific mortality causes. (Chapter 3, references 90, 97 and others.)
- By international agreement, linear prediction models for estimating the health response of low-level doses of radiation have been established. This method was developed for worker radiation protection purposes and it uses health effects data obtained after exposures to high doses and high dose rates. (Chapter 2, references 18, 20, 43 and others.)

- The likelihood of finding a statistical correlation between chemical air pollution and human health effects is quite dependent upon the statistical method used and the way the data is stratified. (Chapter 3, reference 93.)

- The statistical correlations which have related air pollution to adverse health effects do not necessarily prove that these air pollutants are "true causal parameters". (Appendix, references 1 and 5.)

- Data from recent epidemiologic studies have been used to estimate the extent of morbidity caused by sulfur dioxide and particulate sulfates. (Chapter 3, references 38 and 99.)

Comparing Health Effects of Electric Power Generation Methods

- Investigators have attempted to compare the relative health impacts of power plants by four methods:
 1. Comparing pollutant mass emission rates.
 2. Comparing specific health effects.
 3. Comparing air quality standards.
 4. Comparing overall risks. (See Chapter 4.)

NILU/IFA CONCLUSIONS FROM THIS WORK

- Air quality standards should not be used to compare the relative impact of various types of power plants. (See Chapter 4.)
- Any comparison method which considers only total mortality does not consider morbidity effects and possible genetic effects due to chemical air pollutants. (See Chapter 4.)
- Regression analyses, such as those used by Lave and Freeburg, may be used to quantify air pollution health effects. However, the ultimate usefulness of such a method is limited by the nature and quality of the data available. (See Appendix.)
- Any further statistical correlation work should use a greater variety of specific air pollution variables than those used by Lave and Freeburg. (See Appendix.)
- The specific results of the work by Lave and Freeburg probably could not be applied to Norway. However, if similar work were performed for Norway it is likely that relevant chemical air pollution variables would be found to be significantly related to adverse health effects. (See Appendix and Chapter 3, reference 98.)

- Air pollution health effects can be estimated by regression analyses even though the "true causal factors" for adverse health are not positively known. This remains valid as long as the variables used remain related to the true causal factors. (See Appendix.)

- No adequate procedure has been developed for comparing the air pollution health impacts of various types of power plants for a particular location. Although a calculation method has been developed and agreed upon by international organizations for use on nuclear power plants, this has not been done for fossil fuel power plants. (See Chapter 2 and 3.)

- If air pollutants emitted from power plants undergo transformations in the atmosphere and produce additional pollutants, then these resulting secondary pollutants must also be included in any health effects evaluation. For example, particulate sulfates formed from the oxidation of sulfur dioxide and ozone produced by photochemical reactions. (See Chapter 3.)

- The results from many air pollution health effects studies are of questionable usefulness because the investigators did not account for variations in relevant parameters, e.g., smoking habits and occupation. (See Chapter 3.)

- The present measurement technology enables pollution emissions from nuclear power plants to be monitored with greater accuracy than those from fossil-fueled power plants. (See references on pollutant emissions in Chapters 2 and 3.)

RECOMMENDATIONS FOR FUTURE WORK IN NORWAY

- Regression analysis methods should be used to develop health effects predictor equations where adequate data are available.
- A procedure should be developed for comparing the relative air pollution health impacts of various types of power plants. The procedure should account for control, dispersion, and population exposure for various pollutants. Special attention should be given to identifying the various probable exposure pathways.
- Calculations should be made to estimate air pollution emissions and health effects from the types of nuclear and fossil fuel power plants which may be constructed in Norway. These calculations should consider the fuel properties and include estimates of emissions of reactive and polycyclic hydrocarbons and heavy metals, in addition to oxides of carbon, nitrogen and sulfur.
- Progress in further phases of the project requires participation by professionals from medical and other health related sciences.

RECOMMENDED TASKS FOR PHASE II OF THE PROJECT

- The Norwegian Health Directorate should be informed about the work in Phase I, and asked to contribute to further work either directly or through establishing contacts with interested medical professionals who can contribute to the project.

- Examine and evaluate present methods for calculating pollutant dose and health effects.
This work follows from the material presented in Chapter 2, 3 and 4.

- Ascertain if adequate data exists for using regression techniques to develop health effects predictor equations.

- Investigate the possibility of using health effects data from Denmark and Sweden in conjunction with any development of any health effects predictor equations. The following individuals have been involved in epidemiological studies by the World Health Organization and have already been contacted:

Dr. B. Holma
Institute of Hygiene
Faculty of Medicine
Copenhagen, Denmark

Dr. P.O. Petersson
Department of Pediatrics
Chief Medical Office
University Hospital
S-750 14 Uppsala
Sweden

- The U. S. Environmental Protection Agency is presently conducting epidemiological health effects studies. (CHESS Project.) A follow up of this work should be encouraged as it may provide some of the data on health effects needed for Phase II of this project.

- NILU and IFA should maintain close contacts and exchange information and ideas with individuals and organizations in other countries engaged in ongoing work on the evaluation of health effects from air pollution. For example, Dr. L. B. Lave, and other investigators (as reported in Chapter 4).

CHAPTER 1 HEALTH EFFECTS OF ELECTRIC POWER GENERATION - AN INTRODUCTION

1.1 SCOPE

An overall societal cost-risk-benefit analysis is desirable prior to planning and applying large scale technology, specifically large plants for energy production. In this analysis the following should be considered:

- Will application of the proposed technological activity lead to a net positive value for society (compared to no activity)?
- Does the proposed technological activity offer more advantages and fewer harmful side effects than alternative ways (alternative technologies) of achieving the same end result?

As for most other technologies, sufficient knowledge for such a full and quantitative analysis is lacking at this time, particularly in regard to the environmental and societal impacts that large scale electrical energy production might bring. This, however, should not deter us from attempting an evaluation of these aspects, to the extent that present knowledge permits, of those technologies which can be realistically expected to dominate added electrical generation capacity in Norway through the remainder of this century. The currently feasible technologically and economically alternatives are considered to be fossil fueled and nuclear power plants. The purpose of the work described in this report is to provide a partial background for an evaluation of the relative merits

and overall impact of fossil fueled power plants and uranium fueled power plants. This requires considering the economic, technical, political and social aspects.

The procedures for quantitative economic and technical comparisons are complex, but nevertheless defined. The construction and operating costs can be estimated and comparisons made. A number of engineering aspects can also be directly calculated and compared. In contrast, the societal implications of both technologies are not well known. The fossil fuel technology is familiar to us and we have accepted it on that basis. Yet, we actually know very little about its societal impact. It is a technology that was developed and used a long time before there was any interest and capability to study its negative effects. Specifically the study of human health effects from air pollutants emitted by fossil fueled power plants is in its infancy. On the other hand, nuclear fission entered history with a rather frightening use as a weapon. This precedent has caused the societal effects of nuclear technology to be questioned and closely scrutinized. Such wisdom is essential; we should be sure to take the same precautions with any energy technology.

Thus in this work we shall attempt to apply the same critical approach in the evaluation of health effects to the general public from normal operation of both nuclear and fossil fueled power plants. In executing this work, we have purposely coupled the somewhat different approaches of the nuclear energy experts and the air pollution experts, with the expectation that the specialists would benefit from working together towards a unified approach of estimating the adverse health effects of these two rather dissimilar power production forms.

1.2 HEALTH EFFECTS FROM GENERATION OF ELECTRICITY

The production of electricity by nuclear or fossil fuel power plants requires a number of important operations:

- exploration for fuel
- procuring the fuel
- preparation
- transportation
- use
- disposal or re-use

There are some detrimental aspects for each of these operations, but of course they vary depending upon the type of fuel and the way in which the operations are carried out. An over-all comparison of electricity generation methods should then consider all of these basic operations. In some specific application, however, there may be only one or two operations which are of greater importance.

In some cases it is possible to compare the relative effects of two operations without knowing the effect itself. For example the mass emission rates of sulfur dioxide from coal, oil and gas-fired power stations can be compared. One fuel may be selected on this basis without really having a complete understanding of the effects of sulfur dioxide and its reaction products upon health and welfare. Of course nuclear plants which emit the same type of radioactive species might be compared in a similar manner. However, it becomes quite complex to compare, for example, the air pollution aspects of Pressurized Water Reactor (PWR) with an oil-fired power plant. They emit different types of air pollutants. Hence if such comparisons are to be made, some "common denominator" must be found. Without such a common denominator only qualitative assertions, which are subject to bias, can be made.

1.3 THIS PROJECT

The purpose of this project is to identify how the relative health effects of nuclear, gas-fired and oil-fired electric power plants can be determined. If such methods can be found then they may be used as one input in the decision-making process to meet energy needs in Norway.

Phase I of this project consists of two main parts. The first part (Chapters 2, 3 and 4) involves a literature survey of health effects of pollutants from thermal electric power generation. The second part (Appendix) provides an evaluation of the work of Lave and Freeburg (1) in comparing the relative health effects of nuclear and fossil fuel electric power generation methods. This particular work was chosen because it was thought to be useful for the later phases of the project. In both of these parts the following constraints were used:

- consider only health effects due to direct exposure to air pollution
- consider public health effects only for the population in the region near the power plants
- consider only the normal operation of the power plants
- do not consider risks due to accidents

Thus, this project has a limited scope. There are many important aspects which have not been considered: nuclear fuel processing, mining and preparation of fuels, disposal of wastes, and others. Some of these should be included later. Specific recommendations for Phase II of this project are included in the summary section of this report.

REFERENCES

- (1) Lave, L.B., Freeburg, L.C. Health Effects of Electricity Generation from Coal, Oil and Nuclear Fuel. *Nuclear Safety*, 14, 409-428 (1973).

28

Blank!

CHAPTER 2
LITERATURE SURVEY:
HEALTH EFFECTS OF RADIATION

2.1 POWER PLANT EMISSIONS

During operation of a nuclear power plant radioactive materials are generated. Most of the generated radioactivity will stay inside the reactor fuel, but some will leak to the primary cooling water. Most of this will be trapped in the various waste treatment systems in the plant, but a small fraction will be released, under controlled conditions, to the atmosphere and the water recipient. The amount released and the composition will depend upon the degree of leakage from the reactor fuel and upon the waste treatment.

Only boiling water reactor (BWR) and pressurized water reactor (PWR) will be considered in this chapter, since these are the types that have been under primary consideration in Norway. In relation to the release of radioactive materials to the atmosphere during normal operations there is one important difference in the construction of the two types. In the PWR the water that passes through the reactor itself is enclosed in a primary circuit. The heat is transferred in a heat exchanger to water in a secondary circuit, and the steam produced goes to the turbine. In a BWR the steam that passes through the turbine comes directly from the reactor.

Due to this difference in construction it is natural to expect a larger release from a BWR than from a PWR, and the numbers reported in Table 2-3 show that this is indeed the case for most of the reactors referred to in that table. The release may, however, be reduced by delaying and filtering the radioactive gases, and BWRs constructed today have releases that are roughly of the same magnitude as releases from PWRs.

The routine releases to the atmosphere from nuclear power plants consist mainly of noble gases and smaller amounts of iodine. The releases are continuously monitored, and the amounts released are reported to the authorities. Radioactivity is easily measured and analyzed.

The limits to power plant emissions are in principle based upon recommendations from the International Commission on Radiological Protection (ICRP) (1). According to the recommendations the whole body dose to individuals in a population group should not exceed 500 mrem per year. In addition it is stated that the genetic dose to a population group should not exceed 5 rem per individual over one generation (30 years). This corresponds to an average of 170 mrem per year. These values are chosen in order to limit the risk of delayed effects (cancer and genetic damage). The values refer to the sum of "artificial" radiation exposure of all kinds, medical exposure excluded, and not only to exposure connected to nuclear energy. The fraction allotted to nuclear energy and to specific types of exposure connected to nuclear energy, is determined by the government of each country. That is why the actual limits concerning e.g. routine release from a nuclear power plant differ from one country to another.

In the following are summarized the limits used in some countries, regarding routine release to the atmosphere from nuclear power plants. Notice that the limits are not set upon the emission as such, but upon the resulting doses in the environment.

USA: The proposed guide lines as published in ref. (2) limits the radiation dose to members of the critical population group to

Whole body dose, atmospheric release	5 mrem/year
Thyroid dose, atmospheric release	15 mrem/year

These limits refer to individuals living in the immediate neighbourhood of the power plant. Using these limits and corresponding limits on emission with the cooling water, the United States Atomic Energy Commission has in a study of the future potential radiological implications (3) estimated that the average radiation exposure to the US population will be lower than 1 mrem/year, which is lower than 1% of the natural background radiation.

Western Germany: The German guide lines refer to the exposure from each separate power plant, and limits the exposure to individuals outside the power plant to

Whole body, atmospheric release	30 mrem/year
Thyroid dose, atmospheric release	90 mrem/year

According to the German authorities these limits together with corresponding limits for emissions with the cooling water, will assure that the average exposure to members of the public will not exceed 1 mrem/year (4).

The Nordic countries: The Radiation Protection Authorities in Denmark, Finland, Iceland, Norway and Sweden have established common principles for limitation of release of radioactive materials to the atmosphere. The actual choice of limiting values will, however, be done in each country. The recommended values for Sweden were formulated January 1975 by the Swedish Radiation Protection Institute as follows:

The whole body dose to an individual in the critical group near the power plant is limited to 10 mrem/year. Furthermore there is a limitation on the total population dose. The total dose commitment due to emissions from a nuclear power plant, summed over all individuals in Sweden, as well as in other countries, should not exceed 0.5 manrem/year per MW electrical (5).

In relation to these limits a clarification of the concept "dose commitment" is needed. The first time the concept was referred to by an international body was in 1962 by UNSCEAR (6). It was introduced in connection with the radiation dose increment resulting from the nuclear weapons tests. The yearly doses, that were measured and calculated, did not according to UNSCEAR give the full picture. Though longlived radioactive materials might account for only a fraction of the yearly doses, they might continue giving doses for a long time after the shortlived radioactive materials had disappeared. The total radiation dose over all coming years caused by a test series was defined as the dose commitment of this test series. The same reasoning is applied to other sources of radiation, like releases from nuclear power plants.

It is generally accepted that the only environmental effects of a routine release of radioactivity that need to be taken into consideration, are connected to exposure of man. This is expressed by EPA as follows (7): "Standards developed on this basis¹⁾ are believed to also protect the overall ecosystem, since there is no evidence that there is any biological species sensitive enough to warrant a greater level of protection than that adequate for man".

¹⁾ The basis referred to is the linear dose-effect relationship and estimates of the dose-effect relationship based upon observations at high doses and dose rates, similar to what is described in other sections of this chapter.

The US Atomic Energy Commission has published guides to limiting conditions for radioactive materials in effluents (8). Based upon these guides the gaseous release of radioactivity has been calculated in connection with an investigation performed by Institutt for Atomenergi, Norwegian Institute for Air Research, and Scandpower A/S for the Norwegian Water Resources and Electricity Board (9). The calculations were performed for a 3000 MWth power plant, and the results are presented in the following. It is important to keep in mind that these are limiting releases. Actual releases during normal operation of a power plant will be considerably lower, as shown by experience.

2.1.1 BWR, Maximum Release to Atmosphere

The main sources of radioactivity release to the atmosphere for a BWR are the condenser air ejector, the gland seal system, and reactor and turbine building ventilation. It is assumed that the release from the condenser air ejector is delayed in coal filters, giving a delay time of 35 hours for krypton and 40 days for xenon. No delay is assumed for the ventilation air, and 2 minutes delay for the release from the gland seal system. All is eventually released from stack.

The resulting releases from stack are shown in Table 2.1.

Table 2.1. Calculated radioactivity release to the atmosphere, 3000 MWth BWR.

	Ci/y
Krypton- 83 m	8,980 x 10
- 85 m	6,735 x 10 ²
- 85	6,368 x 10 ²
- 87	4,409 x 10 ²
- 88	5,435 x 10 ²
- 89	1,355 x 10 ³
Xenon - 131 m	6,540
- 133 m	9,490
- 133	1,913 x 10 ³
- 135 m	7,460 x 10 ²
- 135	7,410 x 10 ²
- 137	2,401 x 10 ³
- 138	2,296 x 10 ³
Iodine - 131	5,150 x 10 ⁻¹
- 133	2,597
Sum	≈ 12 000 Ci/y

2.1.2 PWR, Maximum Release to Atmosphere

The main sources of radioactivity release to the atmosphere for a PWR are off-gases from the primary system water purification system, condenser air ejector, steam generator blow-down vent, the gland seal system, and ventilation air from the containment, turbine building and auxiliary building. It is assumed that the release from the purification system is delayed in coal filters, giving a delay time of 35 hours for krypton and 40 days for xenon. No delay is assumed for the other releases, and all is eventually released from stack.

The resulting releases from stack are shown in Table 2.2.

Table 2.2. Calculated radioactivity release to the atmosphere, 3000 MWth PWR.

	Ci/y
Krypton- 83 m	3,44
- 85 m	1,840 x 10
- 85	8,182 x 10 ²
- 87	1,024 x 10
- 88	3,225 x 10
- 89	7,70 x 10 ⁻¹
Xenon - 131 m	1,090 x 10 ²
- 133 m	3,562 x 10
- 133	4,028 x 10 ³
- 135 m	2,30
- 135	5,553 x 10
- 137	1,61
- 138	7,58
Iodine - 131	9,540 x 10 ⁻¹
- 133	3,280 x 10 ⁻¹
Sum	≈ 5 000 Ci/y

2.1.3 Actual Releases from BWR and PWR

The releases specified in the above, are based upon the guides given in ref. (8). It is assumed that the reactor is operating under limiting conditions, that is with the maximum number of leaky fuel rods. The actual radioactivity releases during normal operation are lower. In Table 2.3 is shown measured releases at a number of nuclear power plants in Europe and the United States. It is found by examining this table that the releases from most of the PWR plants are at a fraction of the maximum releases specified in Table 2.2. The releases from the BWR plants are, however, mostly higher than the releases specified in Table 2.1.

The reason is that these plants were built before the guides in ref. (8) were published, and a prolonged delay of radioactive gases before release was not required. A typical delay time for the gases in an older type BWR off-gas system is of the order of half an hour; in the type of off-gas system that is most frequently in use now, it is about forty days.

Table 2.4 gives the annual release of iodine 131 to the atmosphere, measured at a number of European nuclear power plants. These are also, with some exceptions, at a fraction of the maximum release specified in Table 2.1 and 2.2.

For some of the plants in Table 2.3 and in Table 2.4 are in addition shown the discharge limits set by the authorities.

Table 2.3. Measured annual release of noble gases to the atmosphere (curies) ref. (10), (11) and (12).

Facility	1970	1971	1972	Discharge Limit (Ci/year)
<u>Boiling Water Reactors</u>	(x1000)	(x1000)	(x1000)	
Oyster Creek	110	516	866	
Nine Mile Point	9.5	253	517	
Millstone 1	-	276	726	
Dresden 1	900	753	877	
Dresden 2, 3	-	580	429	
LaCrosse	0.95	0.53	30.6	
Monticello	-	75.8	751	
Big Rock Point	280	284	258	
Humboldt Bay	540	514	430	
Pilgrim	-	-	18.1	
Quad Cities 1, 2	-	-	132	
Vermont Yankee	-	-	55.2	
Garigliano	275	640	290	$6.3 \cdot 10^5$
Gundremmingen	7.35	6.78	11.105	$1.9 \cdot 10^6$
Lingen	114	9	5.3	$3.1 \cdot 10^6$
Würgassen	-	-	0.594	$3.2 \cdot 10^4$
Kahl	3.34	2.455	-	$8.8 \cdot 10^4$
Dodewaard	≈ 3	≈ 3	8.4	$3 \cdot 10^5$
<u>Pressurized Water Reactors</u>				
Maine Yankee	-	-	0.002	
Palisades	-	-	0.505	
Yankee	0.017	0.0128	0.0183	
Indian Point 1	1.7	0.36	0.543	
R.E. Ginna	10	31.8	11.8	
Connecticut Yankee	0.7	3.25	0.645	
H.B. Robinson	-	0.018	0.257	
San Onofre	0.42	7.67	19.1	
Point Beach 1, 2	-	0.838	2.81	
Surry 1	-	-	(0.0126) ¹⁾	
Yankee Rowe	-	-	0.018	
Haddam Neck	-	-	0.6	
Sena Chooz	0.003	4.5	31.342	$2.5 \cdot 10^6$
Trino Vercellese	0.019	585	1.031	$5 \cdot 10^5$
Obrigheim	7.7	1.456	3.202	$8 \cdot 10^4$
Stade	-	3	2.445	$6.1 \cdot 10^4$
BR 3	26.68	-	0.252	

1) Actual value, x1000 does not apply.

Table 2.4. Measured annual release of iodine 131 to the atmosphere ref. (12).

Facility	1970	1971	1972	Discharge Limit (Ci/year)
<u>Boiling Water Reactors</u>				
Kahl	0.6	0.0029	-	0.61
Gundremmingen	0.2	0.35	0.19	22
Lingen	0.26	0.38	0.15	16
Würgassen	-	-	-	-
Garigliano	0.06	0.13	0.06	1
Dodewaard	0.0063	0.0063	0.006	-
<u>Pressurized Water Reactors</u>				
Obrigheim	0.044	0.015	0.0062	15
Stade	-	-	0.04	0.21
Sena Chooz	-	-	0.023	1.5
BR 3	0.063	<0.00005	<0.001	-
Trino Vercellese	<0.00059	0.001	0.000001	0.5

2.2 HEALTH EFFECTS OF RADIATION

It has been known since early in this century that ionizing radiation may be hazardous to human health, and research in this field has been extensive. The development of research on radiation health effects and efforts to coordinate this research has been summarized in ref. (13), from which is quoted (translated from the original Swedish into English): "One knows much about radiation and radiation protection. Radiation from X-ray apparatus and radioactive materials has been known for close to 80 years. Organized cooperation regarding radiation protection questions has taken place within the International Commission on Radiological Protection for almost 50 years. In Sweden work was initiated in 1924 at the Laboratory that was later to become the government-directed Institute of Radiation Protection¹⁾. Radiation risks in connection with fission have been studied for more than 30 years. Since the beginning of the 1950ies extensive research has been and is taking place on how various radioactive materials are dispersed in nature and taken up by man: iodine, cesium, strontium and many others. The first large compilation - of thousands of research reports - about these problems was done in 1958 by the United Nations' Scientific Committee on the Effects of Atomic Radiation. This committee has been active for 20 years. Many research problems remain, but we have a rather good over-all picture of the radiation risks."

2.2.1 Acute Effects

Exposure to gamma radiation from radioactive materials in the passing air will give a radiation dose to the whole body (whole body dose). Radioactive materials may also be accumulated in organs and give specific organ doses. The only radioactive material of importance in this connection,

¹⁾ The Norwegian legislation pertaining to radiation protection goes back to 1938, and the State Institute for Radiation Hygiene was founded in 1939.

that is present in the routine gaseous release from a nuclear power plant, is iodine, which may give a radiation dose to the thyroid (thyroid dose). The acute effects that may be suffered are radiation syndrome, related to the whole body dose, and disturbance of the thyroid function, related to the thyroid dose.

Both these acute effects and the radiation levels at which they occur are well known. The radiation syndrome is known from the atomic bomb explosions over Hiroshima and Nagasaki, as well as a few cases from nuclear test facilities and medical treatment with radiation. Disturbance of the thyroid function has been observed in a number of cases where patients have received treatment with radiation in connection with thyroid as well as heart ailments. Furthermore there are numerous data available from animal experiments.

Both of these acute radiation health effects have dose levels below which the effects do not occur. The value of the "threshold dose" varies from one individual to another (14).

The value at which there is fifty percent mortality due to radiation syndrome is at around 450 rem. Below a value of about 200 rem there will be no deaths due to radiation syndrome, though some symptoms of syndrome (nausea, diarrhea) may occur. At doses below 100 rem only minor symptoms may occur (14).

After exposure to doses of above 100 000 rem the thyroid gland will entirely cease to function. This is not lethal. If proper treatment is applied the patient may lead a normal life. For lower doses the functioning of thyroid will be disturbed in larger or smaller degree. Below 1000 rem the functioning of the gland will not be noticeably affected (15).

All the above values apply only if the dose is received during one short time period. If the dose is received over an extended time period, the eventual acute radiation health effect will be less severe or absent. In connection with radiation therapy this relation has been expressed as follows (16)

$$NSD = D \cdot F^{-0.24} \cdot T^{-0.11}$$

where NSD is the Nominal Single Dose; the single dose that the fractionated dose is equivalent to, measured by the radiation effect in tissue. D is the dose. F is the number of fractions, and T is the number of days these fractions are extended over. Using this relationship we find that a dose given in seven fractions over four days is equivalent to half of what it would be if given in one exposure. Given in forty fractions over two months it is equivalent to quarter the dose.

The radiation doses due to routine release will be far below the levels at which there might be acute health effects. The highest whole body dose received by an individual outside the plant will typically be at least a factor hundred thousand below the lower threshold for radiation sickness. And the highest thyroid dose from radioiodine will be more than a factor one million below the lower threshold for thyroid function disturbance. The routine release doses referred to here are doses integrated over one year (9).

2.2.2 Delayed Health Effects

Exposure to radiation may give long-term health effects as well as the short-term effects described in the previous section. In ref. (13) this is expressed as follows (translated from the original Swedish into English): "... In the long term there is additionally, for many harmful chemical compounds as well as for radiation, the risk that a small number of the

persons exposed will get cancer. In some cases there may also be a possibility that the hereditary characteristics in the reproductive cells may be damaged. Such injury may be transferred to coming generations.

At low levels there are no longer any short-term effects. There is no direct illness or fatigue. But there may still be long-term effects from radiation as well as from dangerous chemical compounds. Cancer and genetic changes may occur. These effects may be very difficult to discover among all other cases of cancer and genetic changes in the population."

2.2.2.1 Cancer Induced by high-level Radiation

Radiation may induce cancer in humans and animals. The most important types of cancer in humans caused by radiation are cancer of the skin, blood-forming organs (leukemia), bone, lungs and thyroid. It may seem like a paradox, at first thought, that radiation is used in treatment of cancer, when it is in itself carcinogenic. But the fact is that cancer-induction after exposure to radiation is uncommon, as shown by data in the following sections.

Cancer after irradiation takes long to develop. For man the latency period of leukemia is about seven years, for other types of cancer longer, often several decades (14).

The most important data on the relationship between dose and cancer induction have been obtained from studies of atomic bomb survivors in Hiroshima and Nagasaki, studies of patients treated with radiation for ankylosing spondylitis in England, and some studies on thyroid cancer, cancer of the mammary glands, and studies of cancer in children after exposure to radiation.

2.2.2.1.1 Cancer Mortality. Hiroshima and Nagasaki

In Table 2.5 are given the results of studies among 108 767 atomic bomb survivors from Hiroshima and Nagasaki. The numbers in the table are actual deaths due to leukemia and other types of cancer. The numbers in parenthesis give the ratio between mortality rate due to leukemia and other types of cancer in this population group and in the rest of Japan (17).

Table 2.5. Mortality, leukemia and other types of cancer, Hiroshima and Nagasaki, 1950 - 1970.

Illness	Total	NIC ¹⁾	Dose (rad)					
			0-9	10-49	50-99	100-199	200	Unknown
Leukemia	147 (2.67)	13 (0.98)	45 (1.61)	19 (2.60)	7 (3.51)	13 (8.65)	42 (29.41)	8 (5.08)
Other cancers	3900 (1.09)	839 (1.02)	1990 (1.08)	561 (1.11)	166 (1.20)	106 (1.13)	127 (1.60)	111 (1.33)

¹⁾ NIC means not in city; persons who were not in the city at the time of the bombing.

These numbers are also reported in ref. (18) and are added up (Hiroshima and Nagasaki) in the way presented here in ref. (14), chapter 17.

It must be added that excess mortality from leukemia and from other types of cancer for radiation doses below 100 rem were found only in Hiroshima, where the neutron doses were significant.

.2.2.1.2 Mortality, Ankylosing Spondylitis Patients

In Table 2.6 are given deaths among 14 554 ankylosing spondylitis patients who have received radiation treatment. The table, as presented here is found in ref. (18), but the original reference is ref. (19).

Table 2.6. Observed and expected number of deaths by cause* among ankylosing spondylitis patients.

Cause of death	Deaths				Excess mortality per million per year
	Observed	Expected	Excess	Obs./Exp.	
Leukemia	52	5.48	46.52	9.5	328.1
Cancer of heavily irradiated sites**	200	127.27	72.73	1.6	512.9
Cancer of lightly irradiated sites***	60	52.42	7.58	1.1	53.5
Causes with no obvious relation to ankylosing spondylitis****	752	555.41	196.59	1.4	1 386.4

* Followed to 1st January 1960.

** Cancer of pharynx, esophagus, stomach, pancreas, larynx, bronchi, ovaries, skin, bones. Hodgkin's disease, and cancer of other lymphatic and hemopoietic tissues except leukemia.

*** Cancer of brain and central nervous system, mouth, liver and gall bladder, rectum, breast, uterus, prostate, testes, kidneys, and urinary bladder.

**** Such as peptic ulcer, cerebro-vascular disease, bronchitis, violence, etc.

"Expected deaths" as expressed in the table, refer to the number of deaths to be expected in a similar, non-irradiated control population.

2.2.2.1.3 Thyroid Cancer

Risk estimates of thyroid cancer are based upon two types of data material; persons treated with X-rays in infancy for thymic enlargement, and A-bomb survivors from Hiroshima and Nagasaki. The observations of the Marshallese irradiated as children primarily with iodine from an H-bomb explosion in 1954 is not used, since the number of cases of thyroid cancer found was only one. None of the reported cases of thyroid cancer in these studies were fatal.

In Table 2.7 is given basis for risk estimates for thyroid cancer as summarized by BEIR (20). The data shown are taken from ref. (21) and (22), and refer to childhood exposure. The number of observed cases of thyroid cancer are found in column 16 of the table, where the two numbers given in each "box" are the number of cases observed in the exposed population and the number of cases in an unexposed population, based upon data from the control group specified in column 15 of the table.

Table 2.7. Basis of Risk Estimates for Thyroid Cancer, Childhood Exposure.

Study population	Reference	Type of radiation	Duration of radiation exposure	Duration of follow up (years)		Period after irradiation, on which risk estimates are based (years)	Number of subjects	Number of person-years	Dose (rads)		Age at irradiation (years)		Sex	Nature of control	Relative risk (O/E)	RBE	Percent increase in relative risk per rem	Absolute risk, deaths or cases/10 ⁶ /year/rem		Footnotes or other comments	
				Range	Mean				Range, External	Mean, to tissue	Range	Mean						Best	Limits Lower 90% Higher 90%		
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)	
Thymus x-ray 1926-57	21	X	min. to weeks	6-37	16	6-37	2,878	33,060	5-1,200	229	0-1.5	0	M & F	Upper & N.Y. State Incidence	$\frac{19}{0.14} = 136$	1	59	2.5	1.8	3.4	Morbidity; see footnote (b)
Thymus (high-risk) 1931-46	21	X	min. to weeks	17-32	24	6-32	233	4,577	80-1,200	329	0-1	0	M & F	Upper & N.Y. State Incidence	$\frac{14}{0.60}$	1	71	9.3	6.2	13.3	Morbidity; see footnote (b)
A-bomb H + N 1945	22	Y + n	<10"	24	24	10th to 24th	811	12,069	10-600	143	0-9	5	M & F	0-9 rads	$\frac{6}{1.60} = 3.75$	5	1.2	1.6	.14	4.1	Morbidity RBE for n taken as 5, gamma as 1

a This table does not provide a complete compendium of all studies.

b The incidence figures (obtained by mail survey) refer to surgically excised thyroid cancers, the histological diagnosis of which has been verified and in most cases reviewed by the authors (20). An attempt was made to estimate thyroid doses, but estimates were undoubtedly in error in many cases.

Although the relative risk at high dose levels is definitely increased for persons under 20 years at the time of exposure among the Japanese A-bomb survivors, the risk is not clearly increased for persons 20 years of age or older at exposure (22). Another study of persons treated with X-radiation as adults (23) showed no cases of thyroid cancer, although if the best estimates from Table 2.7, column 19 were to apply, one would have expected a minimum of 16 and a maximum of 93 cases in the group studied. These studies confirm the lesser susceptibility of the adult thyroid to radiation-induced carcinogenesis, as compared with the thyroid of the infant or child.

2.2.2.2 Genetic Effects Induced by high-level Radiation

Radiation may cause two different types of genetic effects: mutation of genes and chromosomes. Both of these types of damage do occur naturally, and the cases induced by radiation do not differ from the natural ones.

Mutation of genes implies a change in the genetic information, and will cause a change, which may be significant or insignificant, in the hereditary characteristics. Spontaneous, natural mutations occur all the time, and they are transferred to coming generations. On the other hand harmful mutations are eliminated gradually, if they give characteristics to the individuals that will impede their ability to survive. A population will contain a certain amount of hereditary ailments and diseases, caused by earlier gene mutations, and it has been expressed that 5 to 10% of the population suffer such ailments or diseases. It appears that in the case of radiation-induced gene mutations, the number of mutations are proportional to the dose, and there is no lower threshold. The gene mutation risk has frequently been expressed by the

doubling dose; that is the dose that will cause a doubling compared to the natural mutation rate, and which may in the long run lead to a doubling of the number of cases of hereditary diseases.

While gene mutations means that a change has occurred in the information contained in the gene, chromosome mutations imply loss, duplication or shuffling of genes. Like gene mutations, chromosome mutations occur naturally. Mongolism is caused by a naturally occurring chromosome mutation. Many cells with chromosome mutations will be eliminated. If such a cell takes part in a conception, the fertilized egg may die at an early stage, or it may lead to an early abortion. Unlike gene mutation, cases of chromosome mutations will mainly appear only in the first generation. The number of radiation-induced chromosome mutations is not directly proportional to the dose. Low doses give relatively fewer mutations (14).

All estimates of the dose-effect relationship of radiation-induced genetic effects are based upon experiments with lower organisms, up to and including mice. Radiation-induced genetic changes in man have not been observed with certainty.

2.2.2.2.1 Genetic Effects. Hiroshima and Nagasaki

In 1947 the Atomic Bomb Casualty Commission initiated a programme to investigate genetic effects among the atomic bomb survivors. The results of this programme has recently been published by the Commission. The data material in this programme was 70 000 births. The statistical analysis was performed on various indicators of genetic damage. These were the following:

- Deformities,
- Still births,
- Infant mortality,
- Body weight at birth,
- Growth rate,
- Child mortality,
- Ratio between boys and girls.

The investigations of the last indicator, the ratio between the sexes, was carried out over a larger material, including 140 000 births. The results of the investigations have been published by the Commission in a number of reports, and these were compiled in a report published by the American Academy of Sciences in 1956. The investigations of sex ratio were published in American Journal of Human Genetics in 1966, and the investigations on child mortality were published in Genetics in 1974. All of these reports are presented in a condensed version in ref. (24).

Only one of the indicators gives some results that might be a statistically significant indication of genetic damage, namely ratio between the number of boys and the number of girls born. This was investigated during the time period 1948 to 1955. The results were of such a nature that the investigation was extended in order to give a better indication, and the ratio was followed up in a second time period extending from 1956 to 1962. The effect is explained in the references, and is connected to the fact that girls have two X-chromosomes, while boys have one X- and one Y-chromosome. The observable result of irradiation would be that there would be more boys born in marriages where the father has been irradiated, while there would be more girls born in marriages where the mother has been irradiated, compared to a non-irradiated control group. The value of the sex ratio as an indicator of genetic damage is, however, increasingly disputed (25) and (20).

In the first time period it was found that there were 522 boys born per 1000 births in the control group. In cases where the mother was moderately irradiated the number is 518, while for highly irradiated mothers the number is 514. This is just the effect one would expect from the genetic theory. In cases where the father has been moderately irradiated the number has dropped, however, to 516, which is contrary to theory. But for highly irradiated fathers it is 532, again as would be expected from the theory. Altogether these results seem to give an indication of a genetic effect. In the second time period, in which one should expect the same effects, they are absent. In fact the numbers (though the differences are smaller) show a clear tendency opposite to what would be expected from theory. The birth ratio in the control groups of the two time periods also differ. In the second time period only 514 boys were born per 1000 births. The group which differs most from the control group (births in the first time period with highly irradiated fathers) was also the smallest group (2062 births).

It should be mentioned that a definite increase of chromosome mutations in somatic cells was observed among the survivors. This does not, however, affect the individual, and it is not transferred to coming generations. That may happen only if mutations have occurred in the reproductive cells. Such chromosome mutations were not observed.

The Atomic Bomb Casualty Commission has concluded from the investigations that one could find no indication of genetic damage in the exposed population. If all observed effects are added, genetic damage is indicated as being slightly more frequent in the unexposed group than in the exposed group, but the difference is not statistically significant.

2.2.2.3 Health Effects of low-level Radiation

From the data material presented in the preceding sections of this report it may be concluded that high-level radiation may cause cancer in man. The same conclusion cannot be drawn regarding genetic damages to man, but it has been shown that such damage may be induced in lower animals.

It is frequently assumed that cancer and genetic damage are also caused by low-level radiation. A number of studies have been and are being conducted in order to establish such a relationship, and those that are generally regarded as the most important of these studies are treated in the sub-sections of this section. One study (Sternglass, sub-section 2.2.2.3.3 is generally considered of dubious quality, but is included because of the wide publicity it has achieved. One study by Stewart (on the effects of ionizing radiation on the fetus), should be considered together with the studies in this section, but has for editorial reasons been placed in section 2.3.3.3) of this chapter.

In the first two sub-sections are treated two studies among occupationally exposed personell in the United States and the United Kingdom.

In the last three sub-sections are treated studies of health effects caused by variation in the natural background radiation level. In this way there is a possibility of examining the influence of increased radiation exposure on large population groups. The geographic variations in the natural background radiation, as illustrated in Figure 2.1 (26), are much higher than the increase that may be caused by a nuclear power plant.

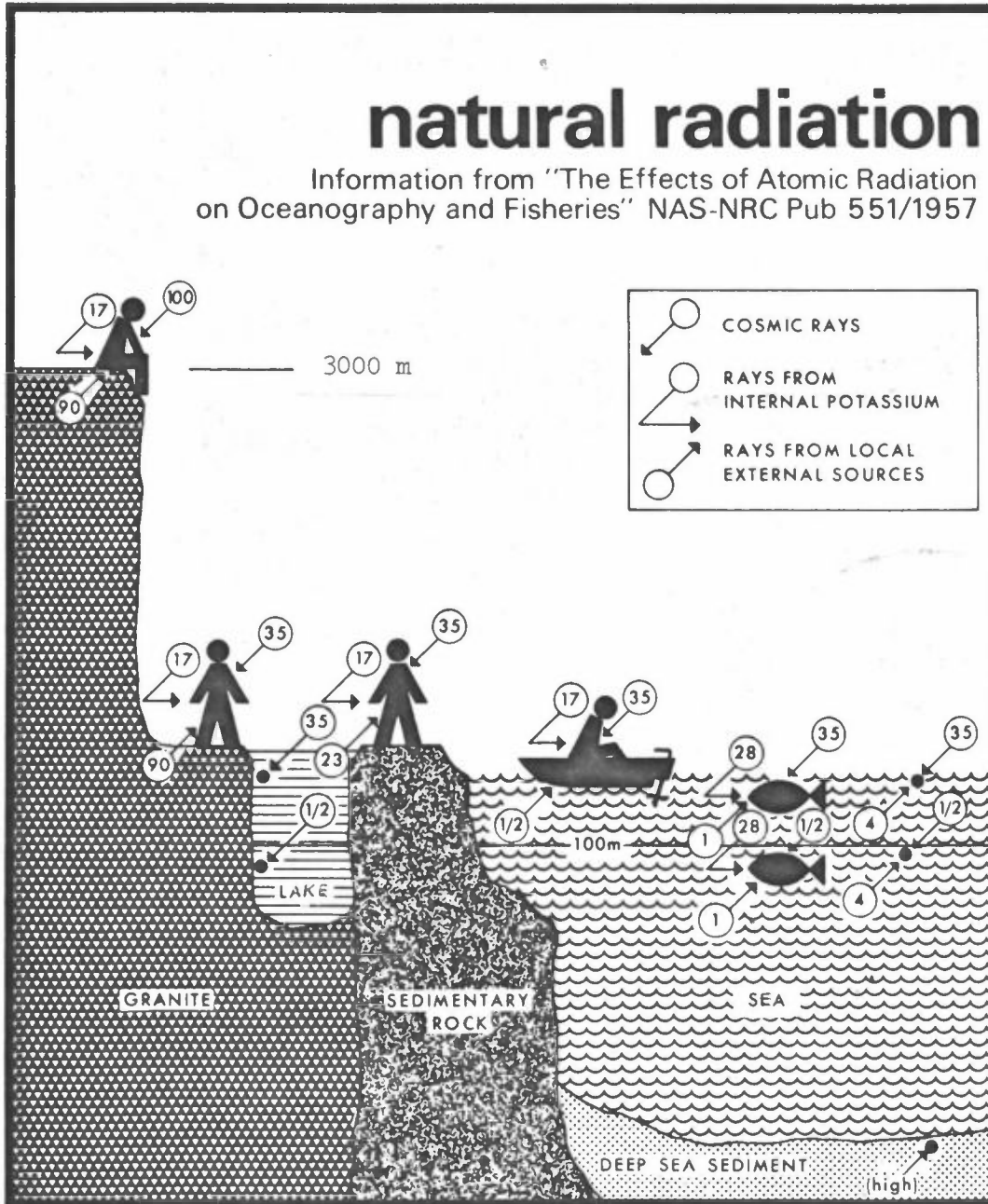


Figure 2.1. Natural Background Radiation (mrad/year).

At ocean level and on sedimentary rock the background level is at 75 mrem/year. At ocean level and on granite it is 142 mrem/year. At elevation 3000 meters and on granite it is 207 mrem/year. Some places on earth the background level may be up to twenty times what is considered the "normal" background level. The studies in sections 2.2.2.3.4 and 2.2.2.3.5 were conducted in such places of exceptionally high background level.

2.2.2.3.1 Study of the Lifetime Health and Mortality Experience of Employees of AEC-Contractors

A very comprehensive study of the lifetime health and mortality experience of employees of AEC-contractors is being carried out by Thomas F. Mancuso and Barkev S. Sanders at the University of Pittsburg, Department of Occupational Health.

This study, although not complete yet, is one of the most extensive studies up till now concerning low level chronic radiation effects on human populations.

As stated in the project's progress report no. 10 (27) and in previous reports the rationale for this project was based on the need to document whether there were any detectable adverse health effects resulting from occupational exposure levels prevailing in the various AEC facilities, at the average exposure levels that were well below 5-15 rem per year limits, or in special exposure groups.

It is explained in previous project reports (28) and illustrated by numerical calculations, that this project was not based on any à priori expectation of finding statistically significant increases in any of the causes of mortality known to be associated with radiation exposure at higher dose levels. The project is based on the need to ascertain the presence or absence of measureable effects to human population from chronic exposure at low dose rates.

The study is at present confined to Hanford and Oak Ridge contractor operations and some feed mill facilities, and it is planned to be extended to other contractor facilities as rapidly as resources permit.

The study of Oak Ridge employees comprise a total of about 105 000 employees, however, the results were not completed in time to be included in the progress report no. 10. The report provides findings on Hanford employees, and as shown in Table 2.8, the total study population at Hanford was 55 348. The analyses up to now include some 32 000 employees, about 18 000 identified siblings (brothers and sisters) of these employees, nearly 2 500 Hanford job applicants who were examined and offered a job, but who declined the offer, and almost 1 400 identified siblings of these applicants.

The 16 689 exposed employees had an aggregate recorded dose of 54 840 054 mrem, and a mean annual exposure per exposed person per year of 440 mrem. For 3 444 female employees the corresponding figures were 2 755 200 mrem as the total and a mean of 200 mrem per exposed person per year. The radiation effects considered in this report are restricted to external penetrating whole body ionizing radiation.

a) Longevity

The study started with a comparative analysis of longevity for Hanford employees and their siblings, on the assumption that if exposure to radiation, to which many Hanford employees have been exposed, had serious life-shortening effect, this should become apparent in comparing the longevity of siblings and employees.

These comparisons showed no marked differences in the longevity of the siblings and that of Hanford employees. Small excesses in longevity that were found in favor of the

siblings were not considered to be of importance since the proportion of identified siblings was much smaller for the earliest cohorts.

Comparative longevity of nonstarts (those who applied for jobs at Hanford, but did not accept the offer when it was made to them) showed, for males, a significantly shorter longevity when compared with Hanford employees in five years age groups. For females, the nonstarts had a slight advantage which was not statistically significant.

When Hanford employees were divided into two groups, those with and those without a record of exposure to external whole body radiation at Hanford and/or any other installation included in the present study, the exposed consistently showed a statistically significant excess in longevity, both for males and females. This disadvantage in longevity of the non-exposed was strongly reinforced when their longevity was compared with that of their siblings. This comparison showed statistically significant advantage in favor of the siblings. There was no such advantage in favor of the siblings when the longevity of radiation-exposed employees was compared with that of their siblings.

The study reported on in ref. (27) is concentrated on the comparative dose level of exposed workers with comparable exposure opportunities up to a specified time, and the mean dose levels of those from this population who die in an ensuing year, with the mean dose of those who survive such a year. The logic of this being, that if dose levels of exposure had an adverse effect on longevity, we should find, on the average, higher dose levels among those who die, in comparison with the mean dose levels of those who survive a specific year. The reader must keep in mind that the mean dose for those who die and those who survive is the cumulative dose acquired by each over the same time interval preceding the year of death for the deceased.

Table 2.8. The White¹ Study Population of Hanford Utilized to Date (4/74) for Various Comparative Analyses to Determine Whether Exposure to Ionizing Radiation² Among Hanford Employees Incident to their Employment in Atomic Plants³ has been Injurious to their Health⁴ - Production⁵ Employees Hired from 1943 through 1971.

Characterization of the Population	Number	Reported Deaths ⁶	Death Certificates	
			Number	%
Male Hanford Employees (white)	23 425	3 418	3 342	97.78
Those with recorded exposure to radiation ⁷	16 976	2 089	2 056	98.42
Those without recorded exposure to radiation	6 449	1 329	1 286	96.76
Identified siblings of employees ⁸	14 174	1 618	1 453	89.80
Nonstarts ⁹	1 901	307	297	96.74
Identified siblings of nonstarts ⁸	1 447	189	167	88.36
Total Males	40 947	5 532	5 259	95.07
Female Hanford Employees (white)	8 731	365	332	90.96
Those with recorded exposure to radiation ⁷	3 485	109	102	93.58
Those without recorded exposure to radiation	5 246	256	230	89.84
Identified siblings of employees ⁸	4 954	130	107	82.31
Nonstarts ⁹	434	15	13	86.67
Identified siblings of nonstarts ⁸	282	8	6	75.00
Total Females	14 401	518	458	88.42
Total Study Population - Male and Female ¹⁰	55 348 ¹¹	6 050 ¹¹	5 717	94.50

¹ Hanford has had a small nonwhite employee population (1 311) which is included in the present study. This nonwhite group was included in some of the earliest gross comparative analyses of relative longevity of employees with identified siblings, and with the nonstarts and their siblings. These analyses are found in the Annual Report to AEC, 1970-71. They represent less than 3 percent of the Hanford study population, and almost nil in the earliest cohorts. For economy, most detailed analyses have been confined to whites only.

- 2 To date, the only radiation exposure data that have come to us from Hanford, or any other installation included in our study, have been restricted to external whole body penetrating radiation - almost all of it from badges.
- 3 External whole body penetrating radiation data for Hanford employees utilize all such information with respect to those employees from Oak Ridge and other installations included in our study.
- 4 To date, the only health criterion that we have used has been comparative longevity as such or its complement, post death years. Differential causes of death have also been used.
- 5 The study population of Hanford has been restricted, to date, to plant production employees. It fails to include six to seven thousand employees of contract construction who are exposed to ionizing radiation in the course of their employment at Hanford. These employees, whose radiation records at Hanford are kept on the same basis and by the same staff that keeps radiation records for the production workers, must be differentiated from construction workers who put up the original plant, who were of no interest to the study since they would have no exposure to radiation.
- 6 The deaths used in this study are restricted to deaths identified for us by the Social Security Administration. This way, any deficiency in identifying deaths would be uniform for employees and their controls within random variations, thus avoiding possible bias. We estimate Social Security records possibly miss at least 3 percent of the male deaths and 9 percent of the female deaths. The true error may turn out to be somewhat larger because of erroneous identification of deaths.
- 7 An employee (of Hanford) is considered exposed to radiation if there is any record at Hanford, or any other installation included in our study, where such employee may have worked between 1943 and 1971 inclusive.
- 8 Siblings are those of the same sex as Hanford employees, or the nonstarts, whom the Social Security Administration was able to identify for us on the basis of the information that we could supply.
- 9 Nonstarts are individuals who applied for employment at Hanford, passed the pre-employment examination, but declined the job offer when it became available.

¹⁰ Excludes a small number of nonwhites - see footnote ¹, and it excludes construction contract workers - see footnote ⁵.

¹¹ The tabulations which were run for these analyses were based on data available in Pittsburgh as of September 1973 and now differ from the latest known figures at Oak Ridge, where an up-date is in progress, by 1 662 total whites with 59 reported deaths, 53 male and 6 female.

This analysis comparing the mean cumulative external radiation of employees dying in a specified year following their year of hire with the mean exposure of employees surviving such a year of death is the initial attempt to explore any possible correlation between dose levels of exposure to external penetrating whole body radiation and longevity.

In addition to the overall comparison, the exposed employees were subdivided on the basis of their cumulative exposure up to the year or a year before the year in which the death occurred and a comparison made between the mean exposure of the deceased and the survivors within each of these subgroups.

The dose levels in the 10 subgroups ranged from below 500 mrem to 15 000 mrem and over in the highest exposure group.

The results are presented in 62 tables, comparing the dose levels for deceased and the survivors year by year and for the different subgroups. The analysis also includes various tests for statistical significance.

The most striking results of the study is that the comparison of the mean doses for the deceased and the survivors when resulting in statistically significant differences, more often indicates higher dose level for the survivors vis-à-vis the dose level of the deceased.

As stated in ref. (27):

"This is totally incompatible with any hypothesis that exposure to external whole body penetrating radiation at the levels experienced by Hanford employees could have reduced their longevity. In fact, on the basis of our findings for male employees for certain selected large cohorts, we find evidence of a strong negative association between radiation dose levels and probability of death. Even when we eliminate the size of cohorts, we find, for most years and most cohorts, the probability of death lowest among employees with highest dose, vis-à-vis those with lower doses."

The authors conclude that the relationships appear to remain consistent - the "exposed" have significantly greater longevity. It is also stated that "the findings for the male employees at Hanford are totally incompatible with any notion of a linear dose effect of radiation without any threshold" (27).

However, the authors are very careful not to make any premature generalization (27):

"In closing, we would like to emphasize again that we are not prepared, on the basis of this evidence, to conclude that no employee health at Hanford was adversely affected on account of his exposure to radiation, but merely that there is no indication that employees in general suffered adverse health effects because of their exposure to external whole body penetrating radiation - which is still consistent with our previous conclusion:" ... if there are any harmful effects from employment at Hanford, they could not be gross in character, nor could they affect large numbers of workers. However, it would be premature with the information we have now and findings surfaced so far, to conclude that radiation,

within permissible limits, has had no adverse effect at all. In closing, we wish to emphasize again that even if our study of Hanford were to prove conclusively no adverse effects, it would be unwarranted with presently available insights to generalize such findings to apply to all forms of ionizing radiation in all other AEC plants."

The tabulations at Oak Ridge which were not completed in time to be included in ref. (27), but the authors assert that the data consistently support the findings of earlier reports which showed a statistically significant higher longevity for radiation exposed employees as compared with employees with no known record of exposure to whole body external penetrating radiation.

b) Differential Causes of Death, Cancer Death

The first preliminary analysis (27) with respect to differential causes of death between Hanford employees and their identified siblings is completed. The initial gross examination of the data was in terms of proportionate representation of the causes of death for employees and their siblings. These causes showed close similarities. The detailed analysis for the underlying cause of death, equating for the age of employees and the siblings at the time of hire and the interval of years of observation in which the deaths occurred, confirmed this general observation: no statistically significant occurrence of death among Hanford employees vis-à-vis that of their siblings that could in any way be attributed to the radiation exposure of such employees.

The preliminary analysis shows that there is no indication whatsoever from these comparison that there are certain categories of diseases such as neoplasms, that are found with a significantly higher frequency among employees as compared to the siblings.

Preliminary tabulations (27) also indicates similar findings when dose levels for those who died from cancer are compared with the dose levels of survivors. Dose levels of those who died from cancer do not differ significantly from the dose level of those dying from causes other than cancer.

2.2.2.3.2 Health of Workers in the United Kingdom
Atomic Energy Authority

Detailed statistics of the morbidity and mortality experience of workers employed by the United Kingdom Atomic Energy Authority have been maintained for some years. A summary of the data for the years 1962-68 have been presented by K.P. Duncan and R.W. Howell (29). In this study the death rates from different causes for male employees at U.K.A.E.A. are compared with "expected" death rates. The expected death rates are those for the general population of males and are corrected for age distribution.

The results are presented in Table 2.9, and as can be seen from the table the death rates from "All malignant neoplasms" are considerably lower for the U.K.A.E.A. employees compared with the "expected".

The authors are making following comments:

"The figures for all malignant neoplasms provide no evidence of any adverse occupational effect. While it is difficult to attach much statistical weight to this observation, it is, as far as it goes, confirmatory evidence that present levels of radiation exposure are not causing significant variation in the mortality pattern."

In this study, however, no comparison is made between those workers with radiation exposure and those without exposure, or between different dose levels. The radiation exposure of U.K.A.E.A. employees is given in Table 2.10.

Table 2.9. Health of Workers in the United Kingdom Atomic Energy Authority. Actual and expected deaths - All males.

Causes of deaths	International standard classification Nos. (7th revision)	1968 only								1962-1968	
		Manual staff		Non-manual staff		Pensioners		All male staff		All male staff	
		Actual	Expected†	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected
All malignant neoplasms	140-205	28	33	11	22	16	17	55	72	331	438
Ca. lung bronchus*, trachea, etc.	162-3	10	15	5	10	4	7	19	32	141	190
Leukemia and* aleukemia	204	1	3/4	0	1/2	0	1/4	1	1 1/2	6	11 1/4
Lymphosarcoma reticulo-* sarcoma, Hodgkin's disease, other lymphoma and multiple myeloma	200-203	4	1	0	1	0	1/4	4	2 1/4	14	14 1/2
Arteriosclerotic and degenerative heart disease (including coronary artery disease)	420-422	43	39	19	26	28	22	90	87	522	502
Road traffic accidents	E810-825	3	3	1	3	0	1/2	4	6 1/2	41	43 1/2
Suicide and self-inflicted injury	E970-979	0	2	1	2	3	1/2	4	4 1/2	26	33 1/2
Other causes		16	47	8	31	28	30	52	108	315	676
All deaths, all causes		90	124	40	84	75	70	205	278	1235	1693

* These conditions are also included in the first line of the table.

† The "expected" figures are quoted as single numbers instead of as a range to simplify the table.

Table 2.10. U.K.A.E.A. whole body gamma doses.

	Year		
	1965	1966	1967
Number of people wearing films	19003	17949	16863
Number exceeding annual dose of 5 rads	64	111	74
Percentage exceeding annual dose of 5 rads	0.34	0.62	0.44
Total annual man-rads	9620	10020	10270

2.2.2.3.3 Sternglass

In well over thirty technical papers, spanning nearly ten years, Sternglass has presented results of his investigations, which he takes as indication that very low level radiation is responsible for "excess" infant and adult mortality as well as a number of diseases. His probably most well-known paper was first published in reference (30).

The concept of "excess" mortality requires a short explanation. Sternglass shows among other figures a semilog graph of 0-1 year infant death rate in the United States for the years 1935 - 1968, included in this report as Figure 2.2 (31). Up till about 1948 the infant death rate has been steadily decreasing, and in the semilog scale, it follows roughly a straight line. After 1948 the infant mortality continues to drop, but not fast enough to follow an extrapolation of this straight line. The difference between the actual infant mortality and the extrapolated line is called "excess" infant mortality by Sternglass, and is claimed to be caused by exposure to low level radiation from the atomic bomb tests. Why the infant mortality should follow a straight line on semilog paper, is, however, not explained.

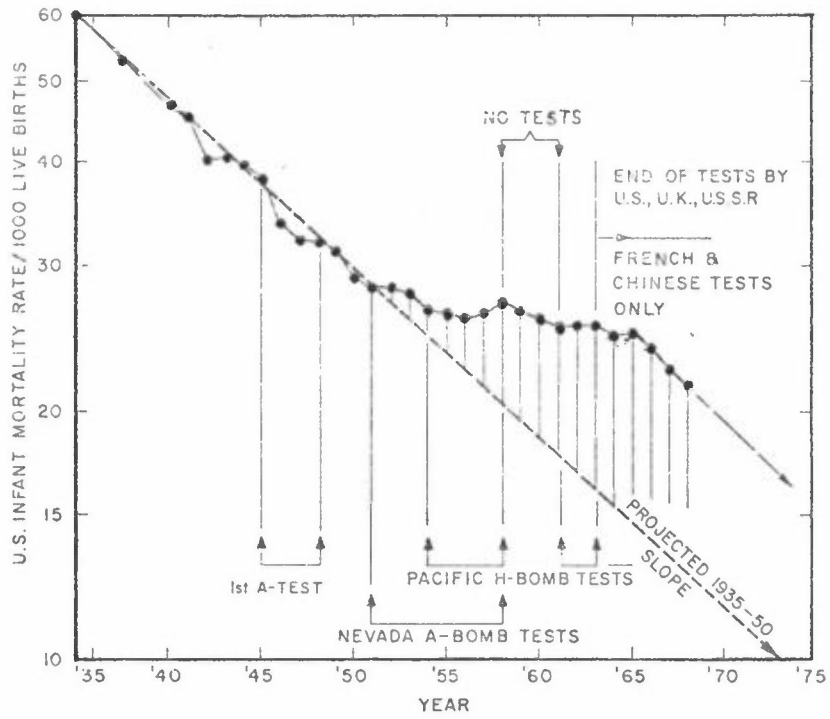


Figure 2.2. Mortality rate in the U.S. for infants 0 - 1 year.

Sternglass has also published several papers about the claimed adverse health effects of low-level radiation from nuclear reactors.

Sternglass' treatment of the statistical material has been criticized and his results are claimed to be unfounded e.g. in references (32, 33, 34, 35). The following statement was read by Health Physics Society President D. W. Moeller to the Society's Annual Meeting in New York on July 14th 1971: "On the third such occasion since 1968, Dr. Ernest J. Sternglass, at an annual meeting of the Health Physics Society, presented a paper in which he associates an increase in infant mortality with low levels of radiation exposure. The material contained in Dr. Sternglass' paper has also been presented publicly at other occasions in various parts of the country. His allegations, made in several forms, have in each instance been analyzed by scientists, physicians, and biostatisticians in the federal government, in individual states that have been involved in his reports, and by qualified scientists in other countries.

Without exception, these agencies and scientists have concluded that Dr. Sternglass' arguments are not substantiated by the data he presents. The U.S. Public Health Service, the Environmental Protection Agency, the States of New York, Pennsylvania, Michigan and Illinois have issued formal reports in rebuttal of Dr. Sternglass' arguments. We, the President and Past Presidents of the Health Physics Society, do not agree with the claim of Dr. Sternglass that he has shown that radiation exposure from nuclear power operations has resultated in an increase in infant mortality."

2.2.2.3.4 Kerala, India

Along the South-West coast of India there exist some monazite-bearing high-radiation areas. The area that has been studied is about 55 km long, and is densely populated. About 70 000 persons live in this region, and it was found that about 20 000 of these were likely to receive radiation doses between five to ten times normal background.

Data were collected on the following effects: fertility index, sex-ratio, abortions and still-births, congenital abnormalities, infant mortality, multiple births. Results of these studies were reported in ref. (36). Studies of chromosomal patterns are being carried out, but results have not yet been published.

The population studied was divided in four radiation exposure groups that were intercompared, as well as compared to other rural areas in India. Only a fraction of the population in the area was actually included in the survey. 2420 couples with 13 720 pregnancies were analysed.

None of the effects studied showed any statistically significant variation between the different exposure groups. The results show, however, that the actual numbers for infant mortality and still-births are higher in the group that has received the highest exposure (more than twenty times normal background), and the fertility index is lower in this same group. But it must be kept in mind that the group consists of only nine couples, and the difference might easily be caused by random variations, or by individual health defects that are not related to radiation.

2.2.2.3.5 Guarapari, Brazil

Along the Atlantic coast states of Espirito Santo and Rio de Janeiro and in the inland states of Minas Gerais, all in Brazil, there are areas of high background radiation. Results of an investigation carried out in the coastal town of Guarapari, population 12 000, are reported in ref. (37). This town is situated on, and the buildings contain, monazite sand, which contains as much as 6% thorium and 0.3% uranium.

It is stated that this population group is not expected to exhibit epidemiologic manifestations; but it was also evident, before the investigations were initiated, that the necessary health data were not available. Guarapari was a rather primitive society, with so poor hygienic conditions, that out of four infants born, one died in the first year and another before the fifth year. This of course was so in numerous similar communities in Brazil at that time, and is not related to the high background. Instead it will tend to cover any possible radiation effects on the population, as only the healthiest individuals will survive. Of the death certificates for Guarapari at that time, some 70% were registered as "cause of death unknown", simply meaning that no medical doctor was in attendance.

Instead of direct epidemiological investigations, it was decided to perform a cytogenic survey, in which the chromosomes of the radiosensitive peripheral blood lymphocytes were microscopically analyzed for aberration rates.

Increased somatic chromosome aberration (mutation) rates were observed, but they appeared to be higher than what could be explained by external whole body dose only. Comparisons with a similar survey among workers in a monazite sand processing mill and a lamp factory (where ThO_2 is used), indicate that the aberrations are mainly caused by inhaled alpha-emitting radioactive materials of short half-life.

Chromosome mutations have no genetic effect, unless they appear in the reproductive cells. This is one of the reasons why it is difficult to relate observed chromosome mutation rates to eventual genetic damage. Such a relationship must be established before the observations may yield any dose effect relationship for genetic damage.

2.2.2.3.6 Argonne Radiological Impact Program

In late 1971 the Environmental Statement Project was formed at Argonne National Laboratory to aid the U.S. Atomic Energy Commission in the preparation of environmental statements for the nuclear facilities being licensed by the Commission. Development of the necessary methods and programs formed the Argonne Radiological Impact Program.

Ref. (38), published as part of the program, concerns itself with evaluation of the carcinogenic hazard that might be associated with the radiation and radioactivity from nuclear facilities.

In recent years, the hypothesis has been advanced that a significant fraction of human cancer mortality may be due to the natural radiation background. (Ref. (39), (40) and others.) Ref. (38) is devoted to "the examination of the degree to which these hypotheses could be justified from current vital statistics and from the known variations in the radiation background."

The investigation is one of the largest epidemiological studies of the health effects of radiation that has been performed. It includes the entire US population in the years between 1950 and 1967.

The results of the investigation are summarized in Table 2.11. The population has been divided into groups. Group A includes the seven US states with natural background radiation higher than 165 mrem/year. Group B includes the fourteen states with background higher than 140 mrem/year, and group C includes the fourteen states with the lowest background. The group designated U.S. contains the population of the 50 US states.

In Figure 2.3 are shown malignant mortality rates as a function of natural background radiation. The indicated malignancy rates in the table and in the figure are per 100 000 persons. The data base of the figure is very large. Each point in the figure represents an average of 100 000 deaths, and each point represents one state. No error bars are shown, since the standard errors are less than the size of the points.

The investigations showed that a higher radiation background is linked with a lower malignancy rate. It is hard to accept that radiation is actually healthy, and one tried to find correlation between malignancy rates and various socio-economic factors like pollution, income, availability of physicians and hospital beds etc. A number of factors that were investigated are included in Table 2.11, but none of these furnish an explanation.

Urbanization has been associated with increasing malignant mortality. A separate comparison of 16 metropolitan areas in groups A, B and C gave the same results as those of the groups themselves, i.e. urban areas in A and B showed much lower malignancy rates than those in C, even for areas of common socio-economic and ethnic factors.

It should also be mentioned that a similar and even more dramatic effect was found in the non-white population; namely that there is a negative correlation between malignancy rate and background radiation.

The conclusion of the study was (quoted from the abstract of ref. (38)): "Various models and predictions of carcinogenic hazard are examined and compared with actual experience in U.S. and foreign populations. All of the models predict a significant increment in malignant mortality with increasing background. Observation of the actual populations at risk shows not only no increment, but an actual decrement, so that these predictions are left quite without observational support. It is concluded that extrapolation of high-rate and usually high dose-level studies to low rates and low levels is probably invalid, and that radiation at such levels and rates does not constitute an environmental carcinogen of significance."

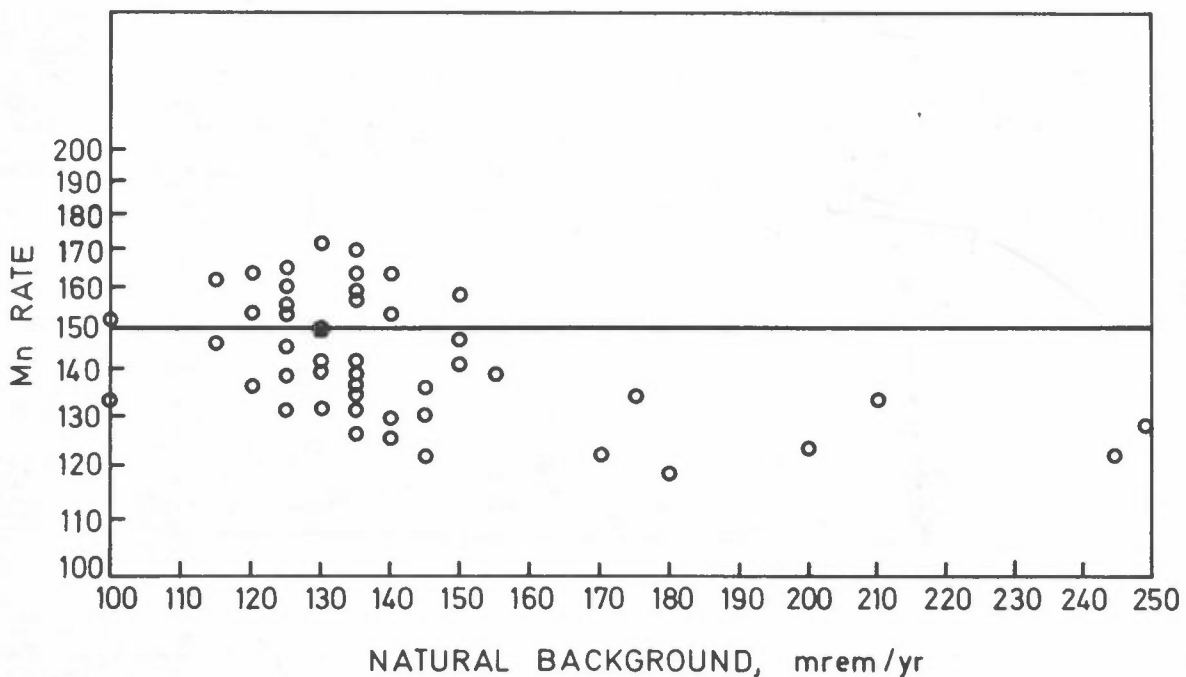


Figure 2.3. Malignant Mortality Rates for the U.S. White Population, 1950-1967, by State and Natural Background. The horizontal line and filled circle indicate the rate and background for the U.S. as a whole.

Table 2.11. U.S. Low and High Background White Populations, 1950-1967.

No.	Characteristics	A	B	U.S.	C
1	Natural background, mrem/year	210	170	130	118
2	White population, thousands	5735	16 897	158 051	59 683
3	r, Mn 140-159*	42.9	45.6	52.4	50.3
4	r, Mn 160-164	15.8	16.9	22.3	23.4
5	r, Mn 170-181	36.8	38.2	41.5	40.1
6	r, Mn 190-205	30.8	31.5	33.3	33.0
7	r, All malignancies	126.3	132.2	149.5	146.8
8	Residence altitude, ft	4510	2650	900	730
9	Urbanisation, %	63	57	69	74
10	Per capita personal income, \$	2021	1922	2215	2255
11	Median family income, \$	5600	5400	5660	5650
12	Physicians/1000 population	1.27	1.25	1.49	1.49
13	Hospital beds/1000 population	8.24	8.82	9.49	8.76
14	Median years of school completed	11.8	11.7	10.9	10.8
15	Poor diet household, %	16.5	21.2	19.1	19.1
16	Population on Federal Food Assist, %	2.6	3.2	3.2	3.3
17	Unemployment, %	4.3	3.9	3.9	2.5
18	Accepted, Military Selective Service	65	63	56	53
19	Life expectancy, male	67.7	67.7	67.6	67.5
20	Life expectancy, female	74.5	74.7	74.2	74.3
21	Urban air, particulates, $\mu\text{gm}/\text{m}^3$	129	119	115	116
22	Urban air, benzene soluble, $\mu\text{gm}/\text{m}^3$	10.1	9.3	9.5	9.6
23	Urban air, radioactivity, pCi/m^3	8.5	7.7	6.8	6.3
24	Urban air, beta, pCi/m^3	5.5	5.2	4.4	4.2
25	r, Mn 140-205, age 0-9	8.11	8.31	8.54	8.31
26	r, Mn 140-205, age 10-19	6.80	6.61	6.82	6.72
27	r, Mn 140-205, age 20-29	10.46	10.73	11.09	11.19
28	r, Mn 140-205, age 30-39	27.61	28.39	31.45	32.27
29	Mortality rate, all causes	892.0	893.2	928.5	903.9
30	U.S.-group, all causes	36.5	35.2	-	24.6
31	U.S.-group, malignancy	23.2	17.3	-	2.7
32	r, Stomach, 151	11.7	11.6	11.8	11.0
33	r, All G.I., 150-159	40.7	43.0	49.0	46.7
34	r, Lung, 163-164	14.5	15.5	20.4	21.5
35	r, Breast, female, 170	21.5	22.6	25.3	24.4
36	r, Thyroid, 194	0.055	0.054	0.057	0.054
37	r, Bone, 196	0.92	1.03	1.12	1.07
38	r, Leukemia, 204	7.03	7.23	7.13	6.91

* r is the observed rate of age-adjusted mortality Mn 140-159 etc. refers to the international classification of malignant diseases.

2.3 HEALTH EFFECTS ESTIMATES

In this section are collected attempts to quantify the relationship between health and radiation at the low levels relevant to exposure due to normal operation of nuclear power plants. There is serious lack of adequate data in this dose region. Use of available data for high exposure levels has, however, together with other available information, made it possible to formulate an estimation procedure, that is judged to give upper limits for the health effects (confer references in the following sections).

These procedures were formulated and agreed upon by ICRP and other international bodies for use for radiation protection purposes. They have also frequently been used for making risk estimates, but were not originally meant to be used for this purpose.

2.3.1 ICRP, UNSCEAR and BEIR

Extensive reviews on the health effects of radiation, prepared by international committees have frequently been published. The assembled knowledge about these effects is presented in these reviews, and it has therefore not been necessary in the present report to perform such an extensive literature survey in relation to these health effects, as has been required in connection with pollutants from oil- and gas-fired power plants.

The most recent reviews were published by UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (18), and by the National Academy of Sciences and the National Research Council, USA. The latter is usually referred to as the BEIR report (Biological Effects of Ionizing Radiation (20)).

Because of the central position of these reports, as well as of the reports from the ICRP (International Commission on Radiological Protection), a short presentation of the background is pertinent.

ICRP (International Commission on Radiological Protection) was founded in 1928 in Stockholm. The commission has prepared a number of publications giving recommendations on dose levels or levels of content of radioactive materials, based upon the accumulated world-wide research in the field. The most recent ICRP publication relevant to this chapter is ref. (41).

The existence of UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) is a direct result of the atomic bomb tests conducted after the Second World War. Fear of the health effects of radioactive fall-out from the tests led to the establishment of the committee in 1955 by the United Nations. UNSCEAR has been active since 1956, and has among many other tasks reviewed the current knowledge of delayed radiation health effects, the latest of these reports being ref. (18).

The BEIR report is the most recent in a series of reports published by the Committee on the Biological Effects of Ionizing Radiations of the National Academy of Sciences and the National Research Council. The previous reports were published from 1956 to 1961. The present report was prepared in response to a request to the National Academy of Sciences from the Federal Radiation Council (whose activities have since been transferred to the Radiation Office of the EPA) for information relevant to an evaluation of present radiation protection guides. The request was made in the summer of 1970. Publication of the report was made jointly by the Department of Health, Education, and Welfare and the Environmental Protection Agency (EPA) (20).

2.3.2 The Linear Dose-Effect Relationship Hypotesis

Common for delayed health effects due to radiation is that all information on the dose-effect relationship relate to doses and dose rates that are very much higher than the ones pertinent in connection with routine releases from nuclear power plants. It is not known whether there are dose levels connected with these effects, under which the effect will not occur. Almost universally the conservative assumption has been adopted for radiation protection purposes that there is proportionality between dose and effect, regardless of the size of the dose or the dose rate.

In the UNSCEAR report published in 1964 (42) it is stated that extrapolation to low doses on the basis of the linear (proportional) non-threshold hypotesis could be considered to give only an upper limit of the risk at low radiation levels. The Committee concluded that at low radiation levels the lower limit of risk could approach a value of zero.

The National Council on Radiation Protection and Measurements in the United States has quite recently published a review (43) in which the linear relationship is referred to as follows: (The actual wording is taken from a News Release published by the Board of Directors of the Council in connection with the publication of the review.)

" ... There is also increasing evidence that with X- and gamma rays, at least, the frequency with which cases are produced is determined to some extent by the rate at which the dose is delivered, i.e., the effects are usually very much less when a given dose is protracted over the lifetime of the animal as compared with the same dose delivered within a few minutes or hours.

The human data on which the risk of cancer production by X- and gamma rays is based were derived largely from studies at high doses and high dose rates.

... Some fifteen years ago, because of the absence of such information (data for low doses and dose rates), organizations concerned with radiation protection, including the NCRP, began to employ the cautious assumption that there is a proportional relation between dose and effect and that the effect is independent of dose rate in the lower ranges of exposure.

... It should be emphasized that the cautious approach, whereby it is postulated that the risk from radiation is proportional to dose, does not imply acceptance of this hypothesis as either proven or highly probable. Numerical estimates of risk based on this conservative assumption could prove eventually to be very unrealistic.

With respect to induction of cancer, there is increasing evidence that as a given dose of X-rays or gamma rays is delivered over a longer period of time, not only does the frequency of cancer induction diminish, but the latent period, i.e., the length of time it takes the cancer to develop, also increases. For low doses and dose rates, it is possible that the latent period may be so long that the manifestation of the effect will not occur within the lifetime of the individual exposed. We cannot speak unequivocally about these matters, but the Council has reviewed the emerging data and concludes that the value of permissible doses adopted by the NCRP in 1971, have, in fact, been overly conservative, though prudence dictates that these values not be completely discarded as yet.

The linear hypothesis, by its very nature, makes it possible to calculate risk coefficients, i.e., the number of cancers or other effects that would be expected to occur in an exposed population, of a given size, per unit of radiation dose. It

must be emphasized, as noted earlier, that risk coefficients derived from the linear hypothesis are based on data obtained at high doses and high dose rates. The National Academy of Sciences (BEIR Committee), in its 1972 report, cautioned against the use of risk coefficients at doses and dose rates orders of magnitude lower than those at which observations were made. The United Nations Committee, in its 1972 report, expressed a strong opinion that the uncertainties in the linear hypothesis are such as to make it inadvisable to use risk coefficients except in regions where data exist. The evidence for both dose rate effect and departure from linearity are such that the NCRP believes that the concern expressed by each of the committees is warranted.

... Most of the public's concern with radiation hazard in recent years has been associated with exposure from the nuclear power industry. In this regard, NCRP notes reports from the Environmental Protection Agency and the Atomic Energy Commission which indicate that the exposure to residents living near such facilities has, up to now, been a small fraction of what the NCRP denominated permissible in its 1971 report. Further, the Environmental Protection Agency estimates that, by the end of the century, the per capita dose for this type of radiation in the United States will be but a very small fraction of the radiation we already receive from cosmic rays and radioactive materials in the earth's crust."

The views of the U.S. Environmental Protection Agency regarding this estimation procedure is stated as follows (7):

"For the purpose of setting radiation protection standards the most prudent basis for relating radiation dose to its possible impact on public health continues to be to assume that a potential for health effects due to ionizing radiation exists at all levels of exposure and that at the low levels

of exposure characteristic of environmental levels of radiation the number of these effects will be directly proportional to the dose of radiation received (a linear non-threshold dose-effect relationship). Even under these assumptions, the range of estimates of the health risks associated with a given level of exposure derived from existing scientific data is broad. It is recognized that sufficient data are not now available to either prove or disprove these assumptions, nor is there any reasonable prospect of demonstrating their validity at the low levels of expected exposure with any high degree of certainty. However, the Agency believes that acceptance of the above prudent assumptions, even with the existence of large uncertainties, provides a sound basis for developing environmental radiation standards which provides reasonable protection of the public health and do so in a manner most meaningful for public understanding of the potential impact of the nuclear power industry ...".

2.3.3 Cancer

2.3.3.1 Risk Estimates

Estimates of the dose - cancer relationship are presented in ref. (18) and ref. (20), the UNSCEAR and the BEIR reports. These two estimates are presented here, since they are undoubtedly the present estimates that are based upon the most complete compilation of data. The data material is presented in section 2.2.2.1.

In UNSCEAR (18) excess mortality from radiation induced cancer is estimated to be 32 - 100 cases per million man-rem. (One million man-rem may be one million persons who each have received a dose of one rem, or ten million persons who each have received a dose of one tenth of a rem etc.)

In BEIR (20) excess mortality from radiation induced cancer is estimated to be 50 - 165 cases per million man-rem.

The risk of thyroid cancer is estimated to be 1.6 til 9.3 cases per million children exposed per rem (20). The risk to persons exposed as adults is considerably less. The data material is presented in section 2.2.2.1.3.

All these numbers are based upon data obtained at high doses and high dose levels. Coupled with the linear dose-effect relationship hypotesis described in section 2.3.2., they are frequently used to obtain estimates of cancer risk, for radiation protection purposes. These estimates will be upper limits to the actual cancer risk, when doses and dose rates are low.

Concerning the reliability of these numbers it is stated in the conclusion of the UNSCEAR report: " ... The Committee wishes to re-emphasize that all the estimates apply to short-term exposures at high dose rates and ... are likely to be over-estimates of the risks per unit dose that may result from protracted irradiation at low dose rates of low-LET radiation. The estimates given ... are all subject to revision, both because the total risk of any malignancy can only be assessed by observing a cohort of irradiated people until extinction, and in no case has there been an opportunity for such prolonged observation yet, but also because of the basic uncertainties of the data. ... These reflect a still inadequate knowledge of the tissue doses received by all groups of irradiated people, but even more our ignorance of the RBE values that must be applied in obtaining risk estimates from these groups that were exposed to mixed neutron and gamma radiation and that have so far provided the largest amount of information on the induction of malignancies in man."

And in the summary of the BEIR report chapter on somatic effects it is likewise stated: "Cancer induction is considered to be the only source of somatic risk that needs to be taken into account in setting radiation protection standards for the general population. Despite many uncertainties, an approximate estimate of overall cancer mortality can be made on the basis of follow-up studies on Japanese atomic bomb survivors and patients treated with radiation for diseases other than cancer."

Other view points on the risk estimates, that are discussed in the BEIR-report (20), also comprise suggestions for alternatives to the linear hypothesis. BEIR also mention the risk estimates by Dolphin and Marley (44), who specifically recommended that the risk coefficient should not be applied to dose levels below 10 rads.

2.3.3.2 Gofman and Tamplin

Different view points on risk estimates is discussed in the BEIR-report - among others also the reports by Gofman and Tamplin (39, 45). These authors have arrived at a considerably higher risk estimate than what is referred above. Estimates have been made that if the maximum "allowable" dose from all non-medical sources of 0.17 rem per year to the general population is reached, the additional cancer death rate in USA may be as high as approximately 104 000 per year or an increase of 34.3% over the current rate. (The comparable figure from the conclusion on the BEIR-report is 3 000 - 15 000 cancer deaths annually, depending on the assumption used in the calculations. The Committee considers the most likely estimates to be approximately 6 000 cancer deaths annually, an increase of about 2% in the spontaneous cancer death rate, which is an increase of about 0.3% in the over all death rate from all causes.)

The basis for the estimates of Gofman and Tamplin rest on three generalizations:

- 1) All forms of cancer, in all probability, can be increased by ionizing radiation, and the correct way to describe the phenomenon is either in terms of the dose required to double the spontaneous mortality rate for each cancer or, alternatively, of the increase in mortality rate of such cancer per rad of exposure.
- 2) All forms of cancer show closely similar doubling doses and closely similar percentage increase in cancer mortality rate per rad.
- 3) Youthful subjects require less radiation to increase the mortality rate by a specified fraction than to adults.

As basis for their estimates they have used the studies of A. Stewart (46) and of MacMahon (47). They have also used the data of Court Brown and Doll (19) on patients treated for ankylosing spondylitis, but since these patients did not receive whole body irradiation, the dose estimates for different organs become crucial. These dose estimates were evaluated by the BEIR Committee - and it was concluded that the doses must have been 5 - 10 times higher than the estimates used by Gofman and Tamplin, which again implies that the risk estimates for the age-group 21 - 30 are a factor of 5 - 10 too high.

Furthermore, the lifetime risk estimate calculated by Gofman and Tamplin are at least 10 times higher than that which can be derived from the ABBC studies (17), the most unequivocal data on the induction of neo-plasms in humans.

The BEIR Committee concluded that Gofman-Tamplin had over-estimated the relative risk of tumor induction following irradiation by factors of 4 - 10 (depending on age) and they had made the unreasonable assumption of a life-long plateau of additional cancer incidence following in utero irradiation.

2.3.3.3 Study on the Effect of Ionizing Radiation on the Fetus

Animal studies suggest that the fetus may be more sensitive to the effects of radiation and therefore might be expected to demonstrate effects at lower exposure levels. Evidence for such an increase sensitivity has been accumulated by Alice Stewart (46) since 1958 and by Brian MacMahon (47).

Both investigators studied children who had been exposed to diagnostic radiation while in utero, and both found that these children have a risk of cancer and leukemia 50 percent greater than children who were not so exposed.

The risk estimates given by A. Stewart is 300 - 800 cases of radiation induced cancer per million children exposed to 1 rad in utero (47). The highest value (800 per million and rad) is about 5 times higher than the risk estimates given for adults. A. Stewart's study also indicates a linear relationship between increased cancer frequency and dose for dose levels down to a few hundred mrad.

However, increased sensitivity of the fetus is not accepted by all investigators as conclusively proven. It has been suggested (48) that the data may be biased in that the children of pregnant women who find it necessary to undergo diagnostic X-ray examination may be at higher risk of leukemia and cancer for reasons other than the X-ray exposure itself. The estimate

given by Stewart is not borne out by the survey of survivors of *in utero* irradiation at Hiroshima and Nagasaki, which did not show increased cancer mortality to be expected on the basis of the estimate (49). The Commission reviewed the juvenile cancer experience of 1292 children exposed prenatally in 1945 to the atomic bombs in Hiroshima and Nagasaki. There was no significant excess of mortality from leukemia or other cancers.

The question of the higher sensitivity of the fetus to ionizing radiation has been carefully discussed both in the BEIR-report (20) and the UNSCEAR-report (18) and similar conclusions have been drawn:

BEIR-report page 166:

"The studies reported to date indicate that diagnostic exposures during fetal life are associated with an increase in cancer deaths under 10 years of age. Whether or not radiation is causally related to the increase in cancer is open to question, since neither laboratory research nor clinical observations as yet support the concept that very low doses of irradiation might increase the relative frequencies of all categories of childhood cancer by about 50%."

2.3.4 Genetic Effects

2.3.4.1 Risk Estimates

Estimates of the dose - genetic effects relationship are presented in the UNSCEAR report (18) and the BEIR report (20). These two estimates are presented here, since they are the present estimates that are based upon the most complete compilation of data. The data material is presented in section 2.2.2.2.

In the UNSCEAR report (18) total radiation-induced genetic effects are estimated to 300 cases per rad and per million zygotes (zygote is the union of an egg cell and a sperm cell). Of these 6 - 15 are expected to show up in the first generation. It is implied that each person involved has received one rad at the time of the zygote.

In the BEIR report (20) the estimates are given in a somewhat different form, but when they are transferred, the estimates are that there will be 30 - 750 cases of radiation-induced genetic effects per rad and per million zygotes. Of these 6 - 100 are expected to show up in the first generation.

The UNSCEAR report expresses the same estimate in another way, by stating that, assuming above values to be valid for man, the doubling dose would be about 100 rad. This corresponds to a 1% increase on mutation frequency per rad.

All these numbers are based upon data obtained at high doses and high dose levels. None are obtained from exposure of man, but are obtained from experiments with lower organisms. Coupled with the linear dose-effect relationship hypothesis described in section 2.3.2, they are frequently used to obtain estimates of genetic risk, for radiation purposes. These estimates will be upper limits to the actual genetic risk, when doses and dose rates are low.

2.4 AN EXAMPLE OF RADIATION RISK ESTIMATION PROCEDURE

This section is included to give an illustration of how the available information on radiation health effects is frequently used in order to give an upper limit estimate of the health effects of radiation.

The cited radiation doses apply to a 3000 MWth nuclear power plant in the Oslofjord area, and are published in ref. (9). They are calculated assuming maximum release. The radiation doses experienced during actual normal operation will be considerably lower, as reported in section 2.1.3.

The health effects are calculated based upon the estimates given in section 2.3.3.1 and 2.3.4.1, coupled with the linear dose-effect relationship hypothesis discussed in section 2.3.2.

2.4.1 Radiation Doses

In ref. (9) are calculated individual doses as well as total regional population doses due to maximum routine releases to the atmosphere of radioactive materials from a nuclear power plant. The releases are specified in section 2.1.1 and 2.1.2. The dose calculations are based upon meteorological and population data typical of the Oslofjord area.

Assuming release from a 100 meter stack, the highest individual doses of maximum release were found to be 1.5 mrem/year for a BWR and 0.02 mrem/year for a PWR. If these doses are compared to the numbers in Figure 2.1, it is observed that they are less than one percent of the dose from the natural background radiation in the Oslofjord area.

The population doses summed over the complete population of the region (about one million persons) were found to be 0.72 manrem/year for a BWR and 0.11 manrem/year for a PWR.

The doses are whole body doses.

2.4.2 Acute Effects

There are no acute effects due to routine releases to the atmosphere of radioactive materials from a nuclear power plant.

2.4.3 Cancer

In section 2.3.3.1 is indicated that the best available information on dose effect relationships point to a value of about 100 cases per million manrem for excess mortality due to radiation-induced cancer.

If this value is used, along with assumption of linearity of the dose effect relationship, the excess cancer mortality due to the population doses in section a) is 0.00007 deaths (BWR) or 0.00001 deaths (PWR), in a population of one million persons in the Oslofjord area.

2.4.4 Genetic Effects

In section 2.3.4.1 it is indicated that the best available information on dose effect relationships point to a value of about 300 cases of genetic effects per rad (one rad to each person involved) and million zygotes, summed over all coming generations. Roughly ten of these are expected to show up in the first generation.

If these values are used, along with assumption of linearity of the dose effect relationship, the genetic effects are determined as follows. It is assumed that each couple has an average of 2.5 children. Furthermore it is assumed that one generation is 30 years. It is also assumed that these 2.5 children are born at the same time, after the parents both have been exposed for 30 years. In the above population the dose received in the course of 30 year will, on the average, be 0.02 mrem for a BWR and 0.003 mrem for a PWR. The resulting genetic effects summed over all coming generations will be 0.008 cases (BWR) or 0.001 cases (PWR). Of these 0.0003 cases (BWR) or 0.00004 cases (PWR) are expected to show up in the first generation. These numbers result from exposure of a population of one million persons in the Oslofjord area for a time period of 30 years (one generation).

2.4.5 Calculated Radiation Risk

The radiation risks calculated in the preceding are presented in Table 2.12. It must be remembered that the numbers have been calculated assuming maximum release, and that the procedure used to calculate the health effects can be expected to give an upper limit only.

The naturally present risks of the same health effects in the population of the Oslofjord area, based upon health statistics, are also presented in the table.

Table 2.12. Upper limits to health effects. 3000 MW thermal nuclear power plant in the Oslofjord area.

	BWR	PWR
Radiation-induced cancer	0.00007 cases per million persons per year	0.00001 cases per million persons per year
Naturally occurring cancer	≈ 1000 cases per million persons per year	
Radiation-induced genetic effect	0.008 cases per million persons and generation exposed	0.001 cases per million persons and generation exposed
Naturally occurring genetic effect	≈ 40 000 cases among the total off-spring of one million persons	

REFERENCES

- (1) Recommendations of the International Commission on Radiological Protection, ICRP, Publication 9. Pergamon Press, Oxford, 1966.
- (2) *Federal Register*, May, 1975.
- (3) Potential radiological implications of nuclear facilities in the upper Mississippi River Basin in the year 2000. (The year 2000 study.) USAEC. WASH-1209, 1973.
- (4) Schwiback, J. Strahlenschutzrichtwerte für die Genehmigung der Ableitung radioaktiver Stoffe. *Atomwirtschaft - Technik* 17, 153-154, 196-198, 280-282, (1973).
- (5) *Reaktornr. 2*, AB Atomenergi, Studsvik, Sweden, 1975.
- (6) Report to the UN General Assembly, UNSCEAR, United Nations Scientific Committee on the Effects of Atomic Radiation, 1962.
- (7) Radiation protection for nuclear power operations, Proposed standards. Environmental Protection Agency. *Federal Register*, May 29, 1975.
- (8) Final environmental statement concerning proposed rule making action: Numerical guides for design objectives and limiting conditions for operation to meet the criterion "as low as practicable" for radioactive material in light-water-cooled nuclear power reactor effluents, USAEC, WASH-1258, 1973.

- (9) Killerud, K.,
Tveten, U. Atmosfærisk sprednings- og
stråledoseberegning for kjerne-
kraftverk. Scandpower Report,
Kjeller, 1974.
- (10) Radioactive effluents from
nuclear power stations in the
community, Commission of the
European Communities, Direc-
torate for Health Protection,
Doc. V/1973/74 e, 1974.
- (11) Report on releases of radio-
activity in effluents from
nuclear power plants for 1972,
USAEC, Directorate of Regula-
tory Operations, 1973.
- (12) Vinck, W. et al. Connection between siting,
normal operating practices,
structural lay-out and quality
assurance; the present situ-
ation and the future outlook
for nuclear power plants,
Commission of the European
Communities, IAEA - SM - 188/3,
Vienna, 1974.
- (13) Den här skriften handlar om
strålning och strålskydd.
Kungl. Vetenskapsakademien,
Stockholm, 1975.
- (14) Devik, F. Strålevirkinger.
In: *Strålefysikk, stråleterapi,
strålehygiene, strålebiologi.*
The State Institute for Radi-
ation Hygiene, Oslo, 1973,
Chapter 15.
- (15) Närförläggning av kärnkraftverk,
Statens offentliga utredningar
1974:56, Stockholm, 1974.
- (16) Ellis, F. Dose, time and fractionation:
A clinical hypothesis,
Clinical Radiology 20, 1-7, 1969.

- (17) Jablon, S.,
Kato, H. Mortality among A-bomb survivors, 1950-1970. ABCC-TR-10-71, Atomic Bomb Casualty Commission, Hiroshima, Atomic Bomb Casualty Commission, Nagasaki, 1971.
- (18) Ionizing radiation: Levels and Effects. UNSCEAR. A report of the United Nations Scientific Committee on the Effects of Atomic Radiation. New York, 1972.
- (19) Bourt Brown, W.M.,
Doll, R. Mortality from cancer and other causes after radiotherapy for ankylosing spondylitis, *Brit. Med. J.*, 1965, ii: 1327-1332.
- (20) The effects on populations of exposure to low levels of ionizing radiation, Report of the advisory committee on the Biological Effects of Ionizing Radiation (BEIR), National Academy of Sciences, National Research Council, Division of Medical Sciences, Washington, D.C. 1972.
- (21) Hempelmann, L.H. et al. Neoplasms in persons treated with X-rays in infancy for thymic enlargement, A report of the third follow-up survey, *J. Nat. Cancer Inst.* 38, 317-341 (1967).
- (22) Jablon, S. et al. Cancer in Japanese exposed as children to atomic bombs, *Lancet* 1971, I, 921-932.
- (23) DeLawter, D.S.,
Winship, T. Follow-up study of adults treated with roentgen rays for thyroid disease, *Cancer* 16, 1028-1031 (1963).

- (24) Frydenberg, O. Hiroshima og Nagasaki -
atombombernes følger,
Naturens Verden, København, 1975.
- (25) Private comm. F. Devik. State
Institute for Radiation Hygiene,
Oslo, 1975.
- (26) *IAEA Bulletin* 12, No. 5, 1970.
- (27) Mancuso, T.F.,
Sanders, B.S. Study of the Lifetime Health
and Mortality Experience of
Employees of AEC Contractors.
Progress Report no. 10.,
COO-3428-5, 1974.
- (28) a)
Mancuso, T.F.,
Sanders, B.S.,
Brotsky, A. Feasibility Study of the Corre-
lation of Lifetime Health and
Mortality Experience of AEC and
AEC Contractor Employees with
Occupational Radiation Exposure.
Progress Report No. 1-5,
NYO-3394-2, NYO-3395-5,
NYO-3394-6, NYO-3394-9 and
NYO-3394-11.
- b)
Manusco, T.F.,
Sanders, B.S. Study of the Lifetime Health
and Mortality Experience of
Employees of AEC Contractors.
Progress Report No. 6-9,
NYO-3394-12, NYO-3394-13,
COO-3428-2 and COO-3428-3.
- (29) Duncan, K.P.,
Howell, R.W. Health of Workers in the United
Kingdom Atomic Energy Authority.
Health Physics 19, 285-291 (1970).
- (30) Sternglass, E. *Esquire*, September 1969.
- (31) Sternglass, E. Infant Mortality and Nuclear
Tests.
Bull. of the Atomic Scientists 25,
18, (April 1969).

- (32) Tompkins, E.A. Infant mortality around three nuclear reactors.
In: *Proceedings of the Sixth Berkeley Symposium on mathematical statistics and probability*, Vol. VI, Univ. of Cal. Press, 1972, p 279-290.
- (33) Harley, J.H. Comments on fallout correlations made by Dr. Ernest Sternglass, HASL-124, Oct. 1969.
- (34) Nilson, A.,
Walinder, G. Strontium-90 dosages and infant mortality.
Bulletin of Atomic Scientists 26, 40 (1970).
- (35) Stewart, A. The pitfalls of extrapolation.
New Scientist 3, 181 (1969).
- (36) Gopal-Ayengar, A.R.
et al. Evaluation of long-term effects of high background radiation on selected population groups on the Kerala coast.
A/Conf. 49/P/535, Volume 11, UN & IAEA, 1972.
- (37) Gonzales, D.E.
et al. Temporary body burdens in dose-effect studies of the Brazilian areas of high natural radioactivity.
In: *Symposium of population exposures*, Health Physics Society, Knoxville, Tenn., CONF-741018, (1974), p. 61.
- (38) Frigerio, N.A.,
Eckerman, K.F.,
Stowe, R.S. Carcinogenic hazard from low-level, low-rate radiation.
ANL/ES-26, Part 1, Sept. 1973.
- (39) Tamplin, A.R.,
Gofman, J.W. Population control through nuclear pollution.
Nelson, Hall, Chicago, 1971.
- (40) Argonne National Laboratory. Conference on the estimation of low-level radiation effects in human populations.
Report ANL-7811, 1970.

- (41) Radiosensitivity and spatial distribution of dose. ICRP (International Commission on Radiological Protection.) Publication No. 14, Pergamon Press, Oxford 1969.
- (42) Report of the United Nations Scientific Committee on the Effects of Atomic Radiation, UNSCEAR, Supplement No. 14 (A/5814) United Nations, New York, 1964.
- (43) Review of the current state of radiation protection philosophy, NCRP Report No. 43 (1975).
- (44) Dolphin, G.W.,
Marley, W.G. Risk Evaluation in Relation to the Protection of the Public in the Event of Accidents at Nuclear Installations. IAEA-SM-117/23, Vienna, 1969.
- (45) Gofman, J.W.,
Gofman, J.D.,
Tamplin, A.R.,
Kovich, E. Radiation as an Environmental Hazard. Paper presented at the 1971 Symposium on Fundamental Cancer Research, Houston, Texas, 1971.
- (46) Stewart, A. Low Dose Radiation Cancers in Man. *Advances in Cancer Research* 14, Academic Press, 1971.
- (47) MacMahon, B. Prenatal X-ray Exposure and Childhood Cancer. *J. Nat. Cancer Inst.* 28, 1173-91 (1962).
- (48) Miller, R.W. Delayed Radiation Effects in Atomic Bomb Survivors. *Science* 166, 569-574 (1969).
- (49) Jablon, S.,
Kato, H. Childhood Cancer in Relation of Prenatal Exposure to Atomic Bomb Radiation. *Lancet* 1970, II, 1000.

- 94 -

Blank

CHAPTER 3
LITERATURE SURVEY:
HEALTH EFFECTS OF CHEMICAL AIR POLLUTANTS

3.1 INTRODUCTION

The purpose of this chapter is to:

- Identify the types and quantities of air pollutants emitted by combustion of fossil fuels.
- Summarize the research work performed to define the health effects associated with these pollutants.
- Report on methods used to estimate air pollution health effects.

In this chapter is considered only work which reports air pollution health effects on humans. A considerable amount of work has been performed on different kind of test animals, but we will not attempt to include this. Mainly information which could be used in the "health effects estimates" methods described in the last section of the chapter is presented.

3.2 AIR POLLUTION FROM FOSSIL FUEL COMBUSTION

The types and quantities of chemical air pollutants emitted from fossil fuel power plants depend upon many factors: fuel characteristics, air-fuel ratio, type of combustion equipment, pressure, temperature, and turbulence of the air-fuel mixture; fuel additives and others (1, 2, 3, 4, 5).

3.2.1 Particulate Matter

During the combustion of fossil fuel some particulates may be emitted. These may originate from two sources - the ash content of the fuel and chemical and physical processes occurring in the combustion process. The chemical nature of the emitted particulates depends largely upon the source of the fuel.

3.2.2 Oxides of Sulfur

Sulfur is a natural impurity in fossil fuels. If it is not somehow removed before combustion then it is also oxidized. Thus sulfur dioxide and some sulfur trioxide are emitted into the atmosphere. These components later form acid mists and sulfate compounds.

3.2.3 Oxides of Nitrogen

Nitrogen from the fuel and the air combine with oxygen at high temperatures to form various oxides of nitrogen. The main constituents are nitric oxide and nitrogen dioxide. These components enter into atmospheric reactions and form nitrate and nitrite compounds. They have also been identified as key components in chemical reactions responsible for "photo-chemical smog".

3.2.4 Carbon Monoxide

This air pollutant is formed in the combustion process because of incomplete reaction between the carbon in the fuel and oxygen in the air. High air-fuel ratios used in fossil fuel power stations keep the amount of carbon monoxide formed small relative to other chemical components in the exhaust gases.

3.2.5 Hydrocarbons

Hydrocarbons are emitted as a result of incomplete combustion or the occurrence of chemical reactions. It is not possible to predict the type or quantity emitted. Often partial oxidation products such as aldehydes may also be formed. Some of the constituents, such as benzo(a)pyrene, have well-known, cancer-causing effects.

3.2.6 Heavy Metals

The toxicity of heavy metals is an established problem. Some fossil fuels contain heavy metals and hence may emit them in the combustion gases. Sometimes metallic compounds are intentionally added to liquid fuels to improve their combustion characteristics. For example, additives containing barium, zinc, manganese, iron and magnesium compounds are used to reduce the visible amount of smoke from oil-fired combustion systems (6). Other metals that are not used as additives, but are often naturally found in fuels include: mercury, cadmium, vanadium, beryllium, and chromium.

3.2.7 Ozone

Ozone is not normally considered a significant pollutant from fossil fuel power stations. However, it should be

considered because it may originate from two sources. Firstly it can be formed in the electrostatic precipitators, especially if they are not operating properly. Secondly recent research has established that ozone may be formed within the plume (7).

3.2.8 Radioactivity

Some fossil fuels do contain natural long-lived radioactive components. The nature and amount of the radioactivity depends completely upon the fuel and its source (8, 9).

3.2.9 Carbon Dioxide

Carbon dioxide is a major component in the combustion of any fossil fuel. It is essential to the biosphere and is not considered a direct health hazard at the concentrations found in the atmosphere. Carbon dioxide emissions may contribute to a global problem since an increase of the carbon dioxide concentration could modify the earth's heat balance (10). This could cause climatic changes and thus indirectly affect the health and welfare of people.

3.3 POLLUTANT EMISSION RATES

The rate at which pollutants are emitted from a power plant can vary. It is a difficult factor to determine. While some pollutant emission rates can be adequately estimated from fuel properties, most must be determined by actual measurements, which may entail difficult sampling and analysis problems. Hence the variation in reported results may be due to differences in processes or sampling and analysis procedures. The data presented here are in the units reported by the original investigators.

The U.S. Environmental Protection Agency (EPA) has compiled air pollution emission factors for various industrial operations (11). Table 3.1 reports data applicable to fossil fuel combustion. These "best estimate" emission factors are based upon evaluation of the data reported by a number of investigators. The factors indicate the amount of pollutant formed per amount of fuel consumed, and assume that no control equipment is in use. Hence the magnitude of these numbers must be reduced in an amount corresponding to the efficiency of the control equipment used.

There is very little data available on the emissions of heavy metals and carcinogenic compounds such as benzo(a)pyrene. Table 3.2 summarizes what has been reported on this (12 - 25).

Several investigators have calculated the annual air pollution emissions for 1000 MW gas and oil-fired power plants. These results are given in Table 3.3. In some instances the same base was used in the calculations. Note the assumptions listed on the table. Investigators have not been able to calculate the emission rates for heavy metals and benzo(a)pyrene because adequate emission data is not available. The emissions for these constituents depend upon the properties of the fuel and the operating conditions of the boilers.

TABLE 3.1. EMISSION FACTORS FOR GAS
AND OIL-FIRED POWER PLANTS
(After Reference 11)

POLLUTANT	GAS-FIRED	OIL-FIRED
	kg/10 ⁶ m ³	kg/10 ³ liters
PARTICULATES	240	1
SULFUR DIOXIDE ^a	9.6	19S
SULFUR TRIOXIDE ^a	NR	0.25S
CARBON MONOXIDE	270	0.4
HYDROCARBONS	16	0.25
OXIDES OF NITROGEN	9600	12.6
ALDEHYDES	NR	0.12

^a S equals percent by weight sulfur in the oil. Sulfur content of the gas assumed to be 2000 grains/10⁶ ft³ (4600 g/10⁶ m³).

NR not reported.

TABLE 3.2. AIR POLLUTION EMISSION FACTORS
FOR FOSSIL FUEL POWER PLANTS

POLLUTANT	EMISSION FACTOR		REFERENCE
	GAS-FIRED	OIL-FIRED	
BARIUM	NA	Less than 1% of fly ash by weight as BaO.	25
IRON	NA	50 mg per pound of fuel	17
MANGANESE	NA	Fly ash contain less than 1% by weight MnO ₂ .	25
NICKEL	NA	Fly ash contains 1.8 - 13.2% NiO.	19
VANADIUM	NA	Fly ash contains 2.7 - 63% V ₂ O ₅ .	21
ZINC	NA	Fly ash contains less than 1% as ZnO.	25
Benzo(a)pyrene	50 µg/10 ⁶ BTU	90 µg/10 ⁶ BTU	23

NA = not applicable.

TABLE 3.3. AIR POLLUTANT EMISSIONS
FROM A TYPICAL 1000 MW POWER PLANT

ANNUAL CHEMICAL RELEASE (10^6 lbs)
ANNUAL RADIOACTIVITY RELEASE (Curies)

POLLUTANT	Gas-Fired Plant			Oil-Fired Plant		
	Ref ^{a,c} 3	Ref ^d 5	Ref 26	Ref ^{b,c} 3	Ref 5	Ref ^e 26
Particulate Matter	1.02	1.02	1.04	1.6	1.6	5.88
Oxides of Sulfur	0.027	0.027	0.0278	116.0	116.0	123
Oxides of Nitrogen	26.6	26.6	27.1	47.8	47.8	54.2
Carbon Monoxide	N	N	NR	0.0184	0.0184	0.0208
Hydrocarbons	N	N	NR	1.47	1.4700	1.17
Aldehydes	NR	0.068	NR	NR	0.2580	NR
Radium-226	NA	NA	NA	} 5×10^{-4} }	} 5×10^{-4} }	6×10^{-4}
Thorium-228	NA	NA	NA			1.3×10^{-3}
Heavy Metals	NA	NA	NA	NR	NR	NR
Benzo(a)pyrene	NR	NR	NR	NR	NR	NR

N = negligible

NR = not reported

NA = not applicable

a burning 68×10^9 SCF/year

b burning 460×10^6 gallons/year.
Assuming 1.6% sulfur content
and 0.05% ash content.

c based on normal heat rates,
load factors and fuel
properties.

d mentions that some radon-222
is present in natural gas.

e assuming 1.5% sulfur fuel.

3.4 HEALTH EFFECTS STUDIES

Ambient air quality standards have been established in several countries for a number of pollutants and the prospects are that the list will continue to grow in the future. These standards and the resulting efforts to limit air pollution from various sources are largely based upon protecting human health. However, the knowledge about human responses to various chemical pollutants has been elusive. There are very few "facts" that are actually facts because their meaning is unclear. For example, it may be true that low concentrations of sulfur dioxide together with ammonium sulfate particles tend to increase breathing resistance in test animals. But does this necessarily infer that somehow sulfur dioxide and/or sulfate particles are harmful to humans and should therefore be controlled? Many investigators have tried to draw conclusions by reviewing the research work of many others. This has resulted in several extensive literature reviews (27 through 45). Their conclusions have often differed. Thus, for example, Battigelli (37) concludes: "If urban pollution has a measurable effect on the health and disease of exposed populations, as it appears to have on the basis of available information, this phenomenon does not appear to involve sulfur dioxide in its mechanism". This is quite contrary to the conclusions presented by the U. S. Environmental Protection Agency (38): "We have examined the impact of community differences in exposure to these pollutants (sulfur dioxide, particulate matter and sulfates) on chronic respiratory disease in adults, acute lower respiratory disease in children, acute respiratory illness in families, aggravation of symptoms in subjects with pre-existing asthma and cardiopulmonary disease, and lung function of school children. These health indicators were selected because past studies by many investigators indicated that the frequency of these responses in a community was affected by sulfur oxides and particulates. Our studies more than substantiate these findings".

3.4.1 Types of Effect Studies

As shown in Figure 3.1, air pollutants can affect the health of individuals over a broad spectrum of biological and physiological responses. However, the specific role and magnitude are often difficult to define. There are many other determinants of death and disease which cannot be adequately measured. This has impaired, but not stopped, efforts to identify and quantify air pollution health effects. The information on such effects has come from a number of different sources:

- studies of occupational exposure and/or industrial accidents
- animal exposure studies
- controlled exposure effects on human volunteers
- air pollution episodes
- planned epidemiological studies

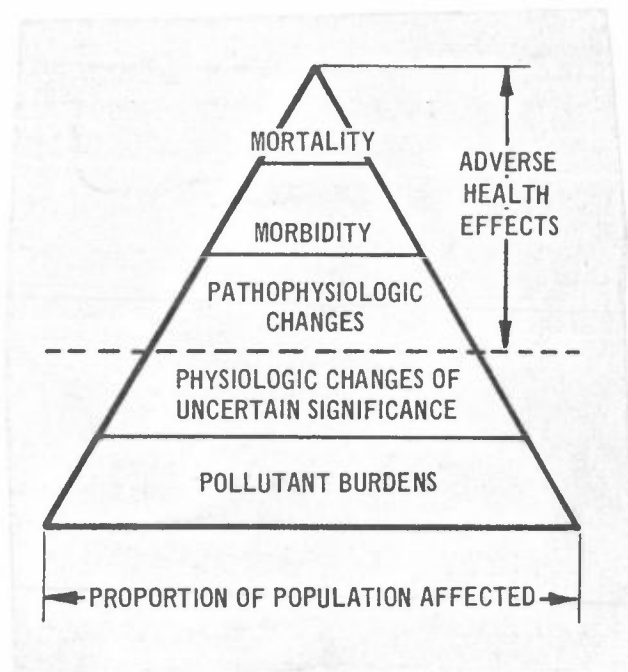


Figure 3.1. Spectrum of Biological Response to Pollutant Exposure (38).

These approaches all have something to contribute to defining air pollution health effects. The industrial toxicology studies provide cases with rather well-defined exposure and effects data. Animal exposure studies provide results on both the toxicology and the possible mechanisms for the observed adverse health effects. However, it is usually quite difficult to infer directly from this information what the consequences might be for humans. Understandably, data from human volunteers is limited, but has provided more specific information about how air pollutants can impair the body functions, such as breathing. Air pollution episodes have shown that high levels of air pollution for rather short periods of time (a few days or less) can, in combination with other factors cause increased mortality and morbidity. Unfortunately in most of these episodes the amount and quality of the available air quality data is poor. Planned epidemiological studies have been performed in only a few cases. In these studies the effects of air pollution on health must be identified in the midst of and separated from many other factors. But in essence this is exactly the knowledge which is needed for the present assessment, and only the results of epidemiological studies will be examined here. There may also be many synergistic effects which can only be detected by observing humans in their living environments.

3.4.2 Epidemiologic Studies

Most of the evidence of the harmful effects of community air pollution has come from epidemiological observations of an association of disease with certain geographic localities. Thus, the fact that men between the ages of 45 and 54 living in Britain have five times the death rate from emphysema, chronic bronchitis and bronchiectasis, and twice the lung cancer death rate of their American counterparts, has been

attributed to the effect of higher levels of air pollution in Britain (32). Striking geographic differences in the prevalence of disease could be caused by the geographic distribution of individuals who have the characteristics that are the true causal factors. Age, religion, race, occupation and social class are important characteristics of the individual which also may vary according to geography. To deal with these complicating factors, MacMahon, Pugh and Ipsen (46) recommended five criteria which strengthen the conclusions about environmental factors and disease:

- (1) High frequency rates must be observed in all ethnic groups inhabiting the affected area.
- (2) High frequency rates will not be observed in persons of similar ethnic groups inhabiting other areas. Analysis by age, sex, social class and personal and occupational characteristics should also show that the affected area has a higher rate for each of these sub-classes.
- (3) Healthy persons entering the area will become ill with a frequency similar to that of the indigenous inhabitants.
- (4) Inhabitants who have left the area will not show high rates, and if they were affected at the time of emigration, they will show signs of improvement or recovery.
- (5) Species other than man inhabiting the same area will show similar manifestations.

Table 3.4 summarizes the results of most epidemiological studies. Most of them fulfill only the first two of the criteria. A number of the studies did not isolate the effects of important factors such as smoking.

Table 3.4. Summary Table of Epidemiological Studies.

Health Index	Suspended particulate matter, $\mu\text{g}/\text{m}^3$.	Smoke or black suspended matter (B.S.M.) $\mu\text{g}/\text{m}^3$.	(In brackets) or COH dustfall tons/ mi^2 -mo.	SO_2 $\mu\text{g}/\text{m}^3$ daily average bracketed values are sulfation rates in $\text{mg}/100 \text{ cm}^2$ -day.	Findings	Reference*
<u>A. Acute episodes - Mortality:</u>						
1. London, winter 1958-59.		rise of 200		rise of 75	Correlation of daily mortality (all causes) and daily bronchitis mortality with B.S.M. Significant positive association of SO_2 and deaths.	52
London, winter 1958-59		1000		715	Increase in mortality (all causes).	54
London, winter 1958-59		750		715		53
2. London, fog episodes, 1954, 1955, 1956, 1957		2000		1200	Bronchitis death rate increased, other causes rise also.	49, 50
London, fog episode 1962		2000		1145	Death rates of elderly rose; greater proportionate rise in bronchitis deaths.	51
3. New York, November, 1953			(5)	2460	Excess deaths all age groups; lag effect noted.	59
4. New York, December, 1962			(6)	2145	No excess deaths	NL
New York, December, 1962			(6)	2145	Excess deaths in 45-64 age group and over 65 age group.	55
5. New York, January, 1963			(6)	1715	Peak in death rate superimposed on high deaths due to influenza.	55
6. Detroit, September, 1952	>200 3 days			2860 (max)	Excess infant mortality and cancer mortality.	NL
7. Osaka, December, 1962	1000 (sic)			285	Sixty excess deaths	NL
<u>B. Acute episodes - Morbidity:</u>						
1. New York, November 1953			(3)	715	Increased upper respiratory infection and cardiac disease morbidity.	NL
2. New York, November, 1966					Increased emergency visits for bronchitis and asthma; analysis complicated by Thanksgiving holiday.	NL
<u>C. Long-term air pollution - Day to day variations in mortality and morbidity:</u>						
1. London, winters 1958-59, 1959-60.		>500		>400	Increases in mortality and morbidity appear positively associated with fluctuations in air pollution.	54
2. New York City 1962-64					Some periods of high air pollution appear to be related to excess mortality; relationship not clear during other periods of high air pollution.	55
3. New York City, housing project.					Cough symptoms related to rise in air pollution in general; eye irritation related to rise in SO_2 .	NL

* NL = Reference not listed in this report.

Table 3.4. Summary Table of Epidemiological Studies. (Continued).

Health Index	Suspended particulate matter, $\mu\text{g}/\text{m}^3$.	Smoke or black suspended matter (B.S.M.) $\mu\text{g}/\text{m}^3$.	(In brackets) or COH dustfall tons/ mi^2 -mo.	SO_2 $\mu\text{g}/\text{m}^3$ daily average bracketed values are sulfation rates in $\text{mg}/100 \text{ cm}^2$ -day.	Findings	Reference*
<u>D. Long-term air pollution - Geographic variations in mortality:</u>						
1. U.K., 214 areas, 1955-59		200		200	Excess bronchitis mortality. Social index also important. Pollutant concentrations not measured in same period as mortality.	NL
2. U.K., 1950-54, lung cancer; 1950-53 bronchitis, pneumonia; 1954-57 sampling network data.	n.a.			n.a.	Lung cancer mortality, bronchitis, and pneumonia mortality in males and bronchitis in females, strongly correlated with smoke density. Cancer of stomach and intestine correlates for some areas.	66-70
U.K., 1950-54 lung cancer; 1950-53 bronchitis, pneumonia; 1954-57 sampling network data					Smoke, occupational, and population parameters relate to lung cancer mortality. Vanadium contributes to lung, female cancer, pneumonia mortalities. Pollutant measurements do not parallel time period of mortality studies.	NL
3. Eston, England		160		115	Positive association between the degree of air pollution and the incidence of both bronchitis and lung cancer.	NL
4. Salford, U.K., 3 wards, August, 1957, through		"High": winter 680, summer 270 "Low": winter 450, summer 170		"High" winter 750, summer 270 "Low" winter 360, summer 150	High rate of bronchitis and lung cancer mortality. Death from all causes increased in "high" compared to "low" pollution wards.	NL
5. Buffalo, N.Y., areas July, 1961-June, 1963	100			(SO_2 0.45)	Positive correlation of pollutant levels with chronic respiratory disease deaths, and deaths from all causes. Mortality rates for gastric cancer 2 times higher in high particulate areas as compared to low particulate area. Air pollution levels did not relate to deaths due to cancer of bronchus, trachea or lung. Mortality effects also seen in area with $80 \mu\text{g}/\text{m}^3$ to $100 \mu\text{g}/\text{m}^3$.	71-73
6. Nashville, Tenn., 1940-60			(>1.1)	30	"High" pollution related to increased respiratory disease mortality. Lung and bronchial cancer, bronchitis, and emphysema mortality not clearly related.	74-75

* NL = Reference not listed in this report.

Table 3.4. Summary Table of Epidemiological Studies. (Continued).

Health Index	Suspended particulate matter, $\mu\text{g}/\text{m}^3$.	Smoke or black suspended matter (B.S.M.) $\mu\text{g}/\text{m}^3$.	(In brackets) or COH dustfall tons/ mi^2 -mo.	SO_2 $\mu\text{g}/\text{m}^3$ daily average bracketed values are sulfation rates in $\text{mg}/100 \text{ cm}^2$ -day.	Findings	Reference*
<u>E. Long-term air pollution - Geographic variations in morbidity:</u>						
1. Genoa, Italy					Frequency of bronchitis significantly associated with SO_2 , not with suspended matter or dustfall.	76
2. Japan		n.a.		n.a.	Gradient of respiratory disease morbidity from industrial to rural sites.	NL
3. U.K.-U.S. Comparison	U.S. 100	U.K. 200		U.S. 12-30 U.K. 60-300	Increase of smoke concentration from $100 \mu\text{g}/\text{m}^3$ to $200 \mu\text{g}/\text{m}^3$ and equivalent SO_2 increase leads to higher risk to older men of poorer lung function and increased chronic respiratory disease.	77
(Levels not directly comparable.)						
4. U.K.			(11.6) (1 yr. av.)	(SO_2 1.3)	Differences in area respiratory morbidities attributed by authors to difference in air pollution.	78
5. Canada, 4 cites	Inadeq.			Inadeq.	Increased severe bronchitis, poor lung function performance in "dirty" cities versus "clean" cities.	NL
6. Berlin, N. Hampshire, Chilliwack, B.C.			34.9	(SO_2 0.4) (SO_2 0.05)	No observable differences.	NL
7. Nashville, Tenn.					Cardiovascular disease directly correlated with aerometric data. No increased respiratory disease morbidity.	79
<u>F. Long-term air pollution - Incapacity for work:</u>						
1. U.S.	100-190				No correlation of absence in excess of 7 days with suspended particles. Significant correlation of absence with "suspended particulate sulfate".	NL
2. Salford, U.K.		>1000 (2 consecutive days)			Bronchitis caused absence greater than expected by factor of 2 in four of five episodes.	NL
3. Greater London, U.K.		100-200		150-250	Lowest bronchitis inception rates.	NL
		400		400	Highest bronchitis inception rates. Significant correlation of bronchitis incapacity with smoke and SO_2 for all age groups, and men 35-54 and 55-59.	
4. New York, N.Y.			(2.7) (7 yr. mean).	540 (7 yr. mean).	No strong relation between respiratory illness absence and COH or SO_2 .	NL

* NL = Reference not listed in this report.

Table 3.4. Summary Table of Epidemiological Studies. (Continued).

Health Index	suspended particulate matter, $\mu\text{g}/\text{m}^3$	Smoke or black suspended matter (B.S.M.) $\mu\text{g}/\text{m}^3$	(In brackets) or COH dustfall tons/ mi^2 -mo.	SO_2 $\mu\text{g}/\text{m}^3$ daily average bracketed values are sulfation rates in $\text{mg}/100 \text{ cm}^2$	Findings	Reference*
<u>G. Studies of children:</u>						
1. U.K., 1946-61	>130			>130	Increased frequency and severity of lower respiratory diseases in school children.	81
2. Sheffield, U.K., 1963-65	100			>120	Increased association of "respiratory infections" for school children age 5-6.	NL
3. Ferrara district, Italy, 1959-64	n.a.			n.a.	No correlation of onset of acute respiratory illness to changing pollutant levels except in poorest socio-economic area.	NL
4. Japan			60	(SO_2 0.02)	Total vital capacity of children in polluted and non-polluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	NL
5. Japan					Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	NL
6. Irkutsk, U.S.S.R., time period not definite	n.a.			n.a.	Possible relation of respiratory disease and residence in area of pollution.	NL
<u>H. Studies of pulmonary function:</u>						
1. U.K. - U.S. Comparison					FEV _{1.0} performance of U.K. workers decreased compared to U.S. counterparts.	NL
2. Japan					Total vital capacity of children in polluted and nonpolluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	NL
3. Japan					Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	NL
4. Pennsylvania, 2 communities					Possible relation of dustfall and SO_2 differences in average and specific airway resistance of subjects in the two communities.	87
5. Sheffield, U.K.		100		275	Reduced FEV _{0.75} and FVC in areas of highest pollution. Possible persistence of respiratory function deterioration related to residence in area of high pollution.	NL

* NL = Reference not listed in this report.

Table 3.4. Summary Table of Epidemiological Studies. (Continued).

Health Index	Suspended particulate matter, $\mu\text{g}/\text{m}^3$.	Smoke or black suspended matter (B.S.M.) $\mu\text{g}/\text{m}^3$.	(In brackets) or COH dustfall tons/ mi^2 -mo.	SO_2 $\mu\text{g}/\text{m}^3$ daily average bracketed values are sulfation rates in $\text{mg}/100 \text{ cm}^2$ -day.	Findings	Reference*
<u>I. Studies of panels of bronchitic patients:</u>						
1. London, November, 1955-May, 1956	300			>600	Acute worsening of symptoms in bronchitis patients.	88
2. U.K., October, 1962-April, 1963	400			460	Possible increase in respiratory disease attack rates.	NL
3. Rotterdam, Netherlands		n.a.		n.a.	No indication of residence effect on bronchitis symptoms.	NL
4. London		140 $\mu\text{g}/\text{m}^3$ declining to 60 $\mu\text{g}/\text{m}^3$		200 $\mu\text{g}/\text{m}^3$ declining to 160 $\mu\text{g}/\text{m}^3$	Decrease in morning sputum volume with decreasing air pollution levels in bronchitis patients under observation during 6 years.	NL

* NL = Reference not listed in this report.

3.4.3 Acute Effects

3.4.3.1 Mortality

In cities, such as London, New York, Chicago, Detroit and others, it has been possible to observe deviations from the moving averages of deaths during various seasons, and to relate such deviations to the coincident levels of air pollution for such periods. A number of investigations have analyzed and compared the various London episodes. The report by Brassler et al. (47) appears to cover all the episodes and to present a detailed analysis of each of these episodes, pointing out the importance of the duration of the maximum values. More recently, Joosting (48) has examined the relationship between the duration of maximum values of sulfur dioxide and smoke during air pollution episodes, as well as the differential relationship between sulfur dioxide and smoke levels and the resulting mortality.

Gore and Shaddick (49), and Burgess and Shaddick (50) reviewed acute "fog" episodes, which occurred in London in 1954, 1955, 1956 and 1957, and plotted excess mortality, above a moving average, versus the mean daily readings at seven stations for smoke and sulfur dioxide. The resulting curves had similar shapes, but excess deaths began to appear shortly before the onset of the "fog" episodes. Scott (51) observed a similar relationship for similar periods of "fog", with "effective pollutant" levels at the seven stations in London of $2000 \mu\text{g}/\text{m}^3$ for smoke and $0.4 \text{ ppm} (\approx 1150 \mu\text{g}/\text{m}^3)$ for sulfur dioxide. There was a particularly sharp impact on the elderly and the greatest proportionate rise, for cause of death, in bronchitics.

Martin and Bradley (52) correlated daily mortality and daily bronchitis mortality with mean daily black suspended matter in London for the winter of 1958-1959. They found a significant positive association between mean daily sulfur dioxide

levels and deaths. Lawther (53) examined these mortality statistics and estimated the mortality "effect" to be associated with exposures to $750 \mu\text{g}/\text{m}^3$ for smoke and $715 \mu\text{g}/\text{m}^3$ for sulfur dioxide. Joosting (48) states that the sulfur dioxide concentrations, above which significant correlation occurs with death and disease, were $400 \mu\text{g}/\text{m}^3$ to $500 \mu\text{g}/\text{m}^3$ when there is a co-current high soot content. In a review of the London episodes Martin (54) concluded: "From the data it would be difficult to fix any threshold value below which levels of air pollution might be regarded as safe".

Greenburg et al. studied the 1962 air pollution episode in New York City, but did not discern any excess mortality. McCarroll and Bradley (55), reviewing episodes in New York City in November and December 1962, January and February 1963, and February and March 1964, compared 24-hour average levels of various pollutants with mortality figures, employing daily deviations from a 15-day moving average. Excess deaths, on December 1, 1962, followed a daily average sulfur dioxide concentration of $2060 \mu\text{g}/\text{m}^3$ and "smoke shade" in excess of 6 coh units (coh = coefficient of haze), during a period of atmospheric inversion and low wind speed. The increased death rates were shared by the 45 to 64 age group, as well as the age group over 65. In a later episode (January 7, 1963), associated with an sulfur dioxide concentration of $1715 \mu\text{g}/\text{m}^3$ and a smoke shade at 6 coh units, there was a peak of death rate apparently superimposed upon an elevated death rate average due to the presence of influenza virus in the community.

A study of the relationship between photochemical oxidant episodes and mortality rates was made in Southern California (56). The number of deaths per day for Los Angeles County were related to temperature and oxidant concentration. Daily mortalities were above the average when temperatures and oxidant levels were high. However, daily mortality decreased when the temperatures dropped while elevated oxidant levels persisted.

Massey et al. (57) compared daily mortality in two areas of Los Angeles County. These areas were selected for both similarities in temperature and differences in air pollution levels. The variables used were daily maximum and mean oxidant levels, and sulfur dioxide and carbon monoxide concentrations. The mean number of daily deaths in the low pollution area was subtracted from the mean number of deaths in the high pollution area and the differences were examined by correlation and regression analyses. No significant correlations between mortality differences and differences in pollutant levels were observed.

Hodgson (58) analyzed the November 1962 and May 1965 episodes in the eastern states of the U.S.A. Using statistical methods he concluded that increased mortality rates are best correlated with air pollution over the same time period (a few days) without any lag period before the episodes.

3.4.3.2 Morbidity

The acute air pollution episodes have resulted in substantial increases in illness. During the November 1953 episode, New York City hospitals indicated a rise in visits for upper respiratory infections and cardiac diseases in both children and adults. The "smoke shade" reading was close to 3 coh units and the sulfur dioxide concentrations were reaching $715 \mu\text{g}/\text{m}^3$ when hospital admissions clearly rose (59).

During the 1966 New York City episode there was a rise in the number of such visits on the third day of the episode among patients over 45 years of age. Unfortunately this episode occurred during the Thanksgiving holiday, which greatly complicated the evaluation of the emergency clinic visits (60).

In the investigation of the London episode of December 1952, information on illnesses was collected from many different sources. The data analysis demonstrated a real and important increase in morbidity. However, the increase was not as large proportionately as the increase in deaths, and the effects rise was not so marked in the early days of the episode. In a number of other severe London episodes the increase in morbidity put a considerable strain on the health services (27).

Sterling et al. (61, 62) tried to relate increased demand for hospital services in Los Angeles to oxidant levels during the period of March 17 to October 26, 1971. Diagnoses were grouped according to "highly relevant", "relevant", and "irrelevant" disorders. Classified as "highly relevant" were allergic disorders, inflammatory diseases of the eye, acute upper respiratory infections, influenza, and bronchitis. "Relevant" disorders were considered to include diseases of the heart, rheumatic fever, vascular diseases, and other diseases of the respiratory system. All other illnesses were classified as "irrelevant". The authors found that the mean number of admissions varied by the day of the week. They were higher in the first-half than in the second-half of the week. They also observed: 1) oxidant precursors were relatively low on Sundays, 2) both oxidant and ozone levels were low on Saturdays and Sundays, and 3) nitric oxide was relatively low on Wednesdays. After correcting for the day of the week with respect to variations in pollutant levels and admission frequencies, "highly relevant" and "relevant" illnesses were found to show significant correlation with oxidant, carbon monoxide, and ozone levels. No significant correlations between hospital admissions and air temperature were found.

Schoettlin and Landau (63) undertook a study to determine, whether Los Angeles "smog" periods aggravated asthma. The study showed that the peak period for asthma attacks was between midnight and 6:00 a.m., while the maximum oxidant levels were recorded between 10:00 a.m. and 4:00 p.m. They thought that a delay in response to the inciting agent might explain the early morning period for such asthma attacks. The correlation coefficient, however, between the number of attacks per day and the maximum oxidant reading was only 0.37.

Several studies have been conducted to determine if air pollution aggravates the condition of subjects suffering from chronic bronchitis and/or emphysema. Motley et al. (64) used a group of 66 volunteers, 46 of whom had pulmonary emphysema. During smog episodes, the group of pulmonary emphysema patients which breathed purified air showed an improvement in breathing capacity, while there was no measurable difference among the volunteers who did not have emphysema.

Wayne et al. (65) studied the athletic performances in 21 competitive meets of student cross-country track runners at San Marino High School, Los Angeles County, from 1959 to 1964. A significant correlation was observed between oxidant levels and the percent of team members whose performance decreased compared to their performance in the immediately previous home meet. They speculated that ozone might have a direct effect on oxygen utilization or that there might be detrimental effects of discomfort from eye and respiratory irritation. An interesting point was that there did not seem to be any concentration threshold value, below which the team performance was not affected.

3.4.4 Chronic Effects

Air pollution episodes represent exposures to relatively massive, overwhelming and unusual pollutant levels. While health effects data for these situations reflect the possibly greater share of the effects, the long term - low exposure effects remains less obvious. In a dose-response situation, only the upper limits are represented by the episode statistics.

3.4.4.1 Geographic Variations in Mortality

Stocks et al. (66 - 70) have related atmospheric pollution in urban and rural localities with mortality due to lung cancer, bronchitis, and pneumonia. Lung cancer mortality was found to be strongly correlated with "smoke density", and social differences in the populations concerned only partially explained this correlation. Bronchitis and pneumonia for males and bronchitis for females similarly showed strong correlations with smoke density in the atmosphere.

Lave and Freeburg related mortality and air quality data for 117 cities in the U.S.A. (Summary of their work and our comments about it are included in the Appendix of this report.)

A number of special studies has been conducted specifically for the purpose of collecting data to find correlations between mortality and air pollution. In a group of studies Winkelstein et al. (71 - 73) analyzed pollution effects in Buffalo, New York. During July 1961, at 21 air sampling stations in and around the city of Buffalo daily values for suspended particulates, dustfall, and oxides of sulfur were measured. Mortality figures for the same area were collected from the period 1959 till 1961, with the 1960 census data providing the population basis. Annual death rate per 100,000

population for 1) all causes of death; 2) chronic respiratory disease; 3) malignant neoplasms of the bronchus, trachea and lung; and 4) gastric carcinoma were related to air pollution and socioeconomic levels of the deceased. A definite correlation between suspended particulate concentration and death rate among white males between the ages of 50 and 69 was found. The death rate in the $135 \mu\text{g}/\text{m}^3$ area was twice that in the area with only $80 \mu\text{g}/\text{m}^3$. Suspended particulate levels, however, did not appear related to deaths from cancer of the bronchus, trachea, or lung. This study did not take into account the smoking habits of the deceased.

The Nashville Air Pollution Study by Zeidberg et al. (74, 75) used sulfation and dustfall data from 123 stations, and sulfur dioxide concentrations and soiling indices from 36 stations. The statistically significant mortality increases were those for all respiratory diseases related to sulfation and soiling. Lung and bronchial cancer mortality, and bronchitis and emphysema mortality were not clearly related to pollutant concentrations.

3.4.4.2 Geographic Variations in Morbidity

Study of records of illness rather than mortality should be more fruitful in defining subtle chronic effects. Morbidity is an earlier and more sensitive index of deviation from normal health. Morbidity data are not routinely available and hence such information can be obtained only from specially planned studies. Morbidity studies of adults involving long-term exposures are frequently not as useful as desired due to the presence of complicating factors such as occupational differences and smoking. Accordingly, several investigators have suggested that it would be better to conduct such studies using children and/or housewives as the subjects.

A study was conducted by Petrilli et al. (76) in Genoa, Italy in which the subjects were women over 65 years of age, all nonsmokers who had lived for a long period in the same area, and who had no industrial work experience. Social and economic factors were also considered. There was a significant correlation between the frequency of bronchitis with mean annual sulfur dioxide level ($r = 0.98$).

Holland et al. (77, 78) studied the prevalence of chronic respiratory disease symptoms and performance of pulmonary function tests in a comparative study of outdoor telephone workmen in urban London, in rural England, and on the east and west coasts of the U.S.A. Persistent cough, phlegm, and chest illness episodes and increased sputum volume were all significantly more frequent in men (between the ages of 50 and 59) in the London urban area than in rural England. Pulmonary function was poorer when the levels of smoke and sulfur dioxide were higher.

In the Nashville air pollution study, Zeidburg et al. (79) reviewed total morbidity in relation to air pollution measurements. Significant correlations of total morbidity with levels of soiling index and sulfur dioxide were observed for individuals over 55 and in the middle socioeconomic class. There was no statistical evidence specifically of increased respiratory disease morbidity in relation to air pollution.

A general health survey was conducted in Los Angeles, and San Francisco and other parts of California in 1956 (80). The survey revealed that asthma, cough, and nose and throat complaints were somewhat more frequent in Los Angeles, Orange, and San Diego counties than in the San Francisco Bay area. Bronchitis was reported by an equal number of sampled persons in the Los Angeles area and in the rest of the state.

3.4.5 Studies on Children

The relationships of respiratory infections to long-term residence in specific localities have been studied in England by Douglas and Waller (81). The health histories of 3866 children born during the first week of March 1946 were followed until 1961, when the children were 15 years of age. The study concluded that lower respiratory tract infections were related to the amount of air pollution, but not those of the upper respiratory tract. Frequency and severity of lower respiratory tract infections increased with the dose affecting both boys and girls, but with no differences detectable between children of middle class and working class families.

Shy et al. (82, 83) studied the effects of community exposure to nitrogen dioxide in four residential areas in greater Chattanooga, Tennessee. One of the areas studied had high nitrogen dioxide and low particulate matter exposure, while another was high in particulate matter exposure and low in nitrogen dioxide exposure. The two other areas served as "clean" control areas. The ventilatory performance of the lungs of second-grade school children in the high nitrogen dioxide exposure area was significantly lower than of the children in the control areas. Illness incidence rates for each family segment in the high nitrogen dioxide area were consistently and significantly higher than incidence rates in the two control areas.

Pearlman et al. (84) later made a retrospective study of acute lower respiratory illness among infants and first and second graders living in three of the Chattanooga neighborhoods used in the study of Shy et al. He used questionnaires to collect information covering the period of July 1966 through June 1969. Responses were validated by checking

physicians' and hospital records. Pearlman concluded that excess acute respiratory illness may be found among children living in areas polluted with nitrogen dioxide.

The World Health Organization has sponsored a research program to study air pollution health effects (85). Participating countries included the Netherlands, Czechoslovakia, Denmark, and Romania. The data analysis has been complicated by differences in field procedures, air sampling methods, exposure levels, and socioeconomic conditions. The study concluded that it would be more fruitful to investigate the air pollution health effects within a relatively smaller region.

The U.S. Environmental Protection Agency is conducting comprehensive health studies in several urban areas in the U.S.A. The program is called Community Health Environmental Surveillance Study (CHESS). In the lower respiratory disease studies conducted in the Salt Lake Basin and Rocky Mountain CHESS areas, three findings were consistently observed (38). First, for all combinations of disease and numbers of illness episodes, no significant association between total lower respiratory disease and pollution was found for children whose parents had been residents of the communities in these study areas for less than 3 years. Second, for single and repeated episodes of croup and repeated episodes of any lower respiratory disease, families of children who had lived 3 or more years in "high" exposure communities, reported more illness for children of all ages (from 0 to 12 years) than did their counterparts in the less polluted communities. Third, for single and repeated illness episodes for all residence durations, there were no associations of pollution exposure with pneumonia or the number of hospitalizations for total lower respiratory

diseases. The only inconsistencies noted were that for children, who had lived 3 or more years in their community, both single and repeated episodes of bronchitis and single episodes of any lower respiratory disease were significantly associated with pollution exposure in the Salt Lake Basin, whereas these associations were not found in the Rocky Mountain study area. Further tests indicated that the lung ventilatory function of elementary school children was diminished in areas of elevated exposure of sulfur oxides. Comparison of results from New York and Cincinnati CHES areas suggested that concentrations of suspended sulfates of about 10 to 13 $\mu\text{g}/\text{m}^3$ might be responsible for reduced ventilatory function.

McMillan et al. (86) measured twice monthly for 11 months the ventilatory performance of two groups of elementary school children living in the Los Angeles basin. One group of 50 children lived in an area subjected to seasonally high photochemical oxidant concentrations. The second group of 28 children lived in a less polluted area. During the study, no correlations were found between changes in photochemical oxidant pollution and ventilatory performance of the lung. It should be noted that the number of children in this study is comparatively small.

3.4.6 Studies of Pulmonary Function

In a comparison by Prindle et al. (87) of pulmonary function and other parameters in two Pennsylvania communities with widely different air pollution levels, average airway resistance and specific airway resistance were measured in persons 30 years of age and older. These measurements were then correlated to the pollution levels in the communities. However, smoking and occupation factors were not accounted for in this study.

Holland et al. (77, 78) found poorer pulmonary function when the levels of smoke and sulfur dioxide were higher (see section 3.4.4.2).

3.4.7 Studies on Special Panels

Studies of selected panels of subjects help to identify what changes in daily air quality aggravate the symptoms of certain groups of disease patients.

Lawther (88) related several episodes of acute urban air pollution to deteriorating health in a group of bronchitis patients. Daily records indicated an acute worsening of symptoms in significant numbers of the group if the air pollution became higher than $300 \mu\text{g}/\text{m}^3$ "smoke" and $600 \mu\text{g}/\text{m}^3$ sulfur dioxide (daily means).

Asthma panels were also used in the Salt Lake Basin and New York CHESS areas (38). Daily asthma attack rates were more consistently correlated with colder outdoor temperature than with any measured pollutant concentrations. However, when the data were grouped in two ranges of minimum temperature: 30 to 50 F, and greater than 50 F, the results indicated that asthma attacks were most closely related to stepwise changes in levels of suspended sulfates. Virtually no relationship between sulfur dioxide and attack rates appeared.

The study in the New York CHESS area used a panel of cardiopulmonary patients. The pattern of daily aggravation of symptoms in cardiopulmonary subjects was similar to that of asthma patients with respect to temperature and pollutants. Elevated suspended sulfates were the only pollutants consistently associated with aggravation symptoms. Daily sulfur dioxide and total suspended particulate concentrations were not associated with the aggravation of symptoms in the "heart and lung" panel.

3.5 HEALTH EFFECTS ESTIMATES

Several investigators have attempted to quantify the relationship between health and air pollution. Most of this work has pertained to estimates of mortality due to some aspect of air pollution. Only recently have there been efforts to do this also for morbidity. Both types of estimates to date have been seriously hampered by the lack of adequate data.

3.5.1 Mortality Estimates

Winkelstein (72) analyzed mortality in men (50 to 69 years of age) that were exposed to different levels of air pollution. Hamilton and Morris (89) used the same data and multiple regression analysis to obtain the following prediction equation:

$$Y = 33.97 + 0.15 P - 0.0034 I$$

where

Y = mortality rate, deaths per 1000 annually
for men ages 50 to 69.

P = annual average concentration of suspended
particulate matter, $\mu\text{g}/\text{m}^3$.

I = annual average family income, dollars.

According to this relationship an increase of 1 $\mu\text{g}/\text{m}^3$ of suspended particulate matter will result in an increase in annual mortality rate of 0.15 deaths per thousand people in this age group.

Hickey et al. (90, 91, 92) used statistical analysis techniques to estimate pollution-related mortality rates. They found sulfur dioxide to be the best single predictor for the mortality from cancer of the digestive organs:

$$M = 16.470 + 8.734 \ln c(\text{SO}_2)$$

where:

$$\begin{aligned} M &= \text{deaths per 100 000 population} \\ c(\text{SO}_2) &= \text{sulfur dioxide concentration} \\ &\quad (\mu\text{g}/\text{m}^3). \end{aligned}$$

However, this equation is able to explain only 38 per cent of the variance. Hickey et al. also used multiple linear regression to develop predictor equations for other cancers and heart diseases, the results of which are presented in Table 3.5. Note that these equations do not account for such factors as smoking habits, occupation, socioeconomic status, etc.

Schwing and McDonald (93) performed multiple linear regression analyses for white male mortality rates, using the data of Duffy and Carroll (94). Air pollution exposures were estimated from emission inventory data. Forty-one different regression equations were developed which lead to the conclusions that:

- gaseous chemical species may have substantially lower association with mortality than the solid particulate species;
- increased concentration of sulfur compounds are associated with a general increase in the total white male mortality rate;
- increased consumption of cigarettes is also associated with increases in the total white male mortality rate;
- associations for nitrogen compounds, hydrocarbons, and ionizing radiation are dependent on statistical method used and data stratification.

TABLE 3.5. REGRESSION EQUATIONS RELATING MORTALITY AND AIR POLLUTION (91)

Annual mortality rate per 10 ⁵ population	Regression coefficients for $\ln c$ (Chemical) predictors										% of variance "explained" ($R^2 \times 100$)
	Constant	NO ₂	SO ₂	Cd	Cu	SO ₄ ²⁻	Ti	As	Water hardness		
Total cancer ^a	-151.33	+40.180	+18.843	-10.120	-	-	-	-	-	-	55.4
Breast cancer, based on total populations ^b	-28.00	+5.523	+2.018	-0.992	-1.510	-	-	-	-	-	58.0
Stomach cancer ^b	-25.36	+4.117	+2.506	-1.433	-	-	-	-	-	-	49.5
Lung cancer ^b	-28.09	+4.986	-	-	-2.197	+5.462	-2.103	-0.6255	-	-	73.2
Diseases of the heart ^a	-258.4	+89.001	+51.616	-28.368	-	-	-	-	-29.964	-	56.1
Arteriosclerotic heart disease ^b	-254.9	+71.912	+41.336	-24.225	-	-	-	-	-23.140	-	47.4

^aMortality rate data were means of annual data for 1961-1964.

^bMortality rate data were means of annual data for 1959-1961.

NOTE: The number in this table fit in the equational form

$$M = A + B \ln c \text{ (chemical)}$$

where

A = constant

B = regression coefficient

$\ln c$ = natural log of pollutant concentration

Carnow and Meier (95) related pulmonary cancer death rates to cigarette smoking and the amount of benzo(a)pyrene in the atmosphere. The regressions equation has the standard form:

$$Y = C_0 + C_1X_1 + C_2X_2$$

where

Y = age-sex-specific pulmonary cancer rate

X₁ = cigarette consumption per capita, in thousands of cigarettes per year per person over 15 years of age

X₂ = benzo(a)pyrene (BaP) concentration in the atmosphere, in $\mu\text{g}/1000 \text{ m}^3$

C₀, C₁, C₂ = regression coefficients.

The regressions coefficients for the various age-sex-specific categories are given in Table 3.6. Carnow and Meier admit that benzo(a)pyrene is really just one of many relevant air pollution factors. Thus, although other chemical species are likely to be involved with air pollution health effects, in their equation they are apparently accounted for in the regression coefficient for BaP.

Hausknecht (96) used the data of Conley (28) to develop an expression for the mortality risk due to sulfur dioxide and particulate matter:

$$M = 400 (\bar{S}\bar{P}t)^{\frac{1}{2}}$$

where

M = excess mortality per 10 million people

S = sulfur dioxide concentration, in ppm

P = suspended particulate concentration, in g/m^3

t = exposure time, in years

The bars indicate mean values over the time t.

TABLE 3.6. REGRESSION ON LUNG CANCER DEATH RATES IN THE UNITED STATES PER ONE MILLION POPULATION (95)

Age Group	Average Death Rate	Constant (C ₀)	Tobacco Sales (C ₁)	BaP Concentration, (C ₂) μg/1,000 cu m
Male-white				
Age-adjusted	867.4	460.7	11.8	47.5
35-44	101.6	86.5	0.3	4.6
45-54	497.8	125.2	3.0	18.7
55-64	1,405.8	894.4	14.4	70.5
65-74	2,064.7	835.4	35.3	152.6
Male-nonwhite				
Age-adjusted	844.6	184.2	16.5	133.1
35-44	132.3	22.8	1.6	46.4
45-54	606.0	245.9	7.8	97.7
55-64	1,367.6	141.6	34.0	179.0
65-74	1,722.4	162.6	38.8	318.8
Female-white				
Age-adjusted	129.5	92.1	1.0	7.1
35-44	32.9	12.7	0.8	-1.1
45-54	86.5	36.4	1.1	14.2
55-64	163.1	113.5	2.0	-6.2
65-74	255.2	184.7	1.7	16.3
Female-nonwhite				
Age-adjusted	156.3	151.5	0.1	1.2
35-44	34.31	66.0	-1.0	-1.1
45-54	85.8	32.4	-0.4	46.6
55-64	184.9	-375.0	17.3	44.5
65-74	381.7	1,286.4	-19.2	-246.1

Tobacco sales (average 28.8) expressed as dollars spent per capita by individuals 15 years of age or older. Average BaP concentration was 1.38μg/1,000 cu m.

Note: The numbers in the table fit in the equational form:

$$Y = C_0 + C_1X_1 + C_2X_2$$

Lave and Freeburg (97) have used statistical methods to estimate mortality rates as a function of several parameters. A detailed discussion of their work is presented in the Appendix of this report.

Lindberg (98) compared weekly means of air pollutant concentrations with total and disease-specific mortality in Oslo for the winter period of 7 years (mid October through mid March 1958/59 - 1964/65). Linear regression analysis between total mortality in Oslo (Y) and the weekly mean SO₂-concentration (X in µg/m³) gave the expression:

$$Y = 94 + 0,0031 X$$

The correlation coefficient was 0.25.

Lindberg also found significant correlation (0.24) between SO₂-concentrations and deaths caused by arteriosclerosis and degenerative heart disease, but not between SO₂ and other disease-specific deaths (bronchitis, pneumonia). There were no significant correlation between mortality and smoke-concentrations, or between mortality and concentrations of polynuclear hydrocarbons.

3.5.2 Morbidity Estimates

Jaksch and Stoevner (99) examined the use of medical services in the Kaiser hospital in Portland, Oregon, in relation to ambient air quality. They concluded that the quantity of out-patient medical services consumed per contact with the medical system was only minimally affected by air quality. However, they believed that air pollution may increase the number of contacts with the medical system.

Carnow et al. (100) proposed a procedure for evaluating the health impact of fossil fuel combustion by power plants, as shown in Figure 3.2. They chose to estimate the health effects for three high risk groups: 1) children five years old or under, 2) people with asthma, and 3) males ages 55 and older with chronic bronchitis. Data from CHES (38) and other sources were used to obtain the relationship shown in Figures 3.3, 3.4, and 3.5. These figures, in conjunction with exposure estimates and total population at risk, can be used to estimate health effects.

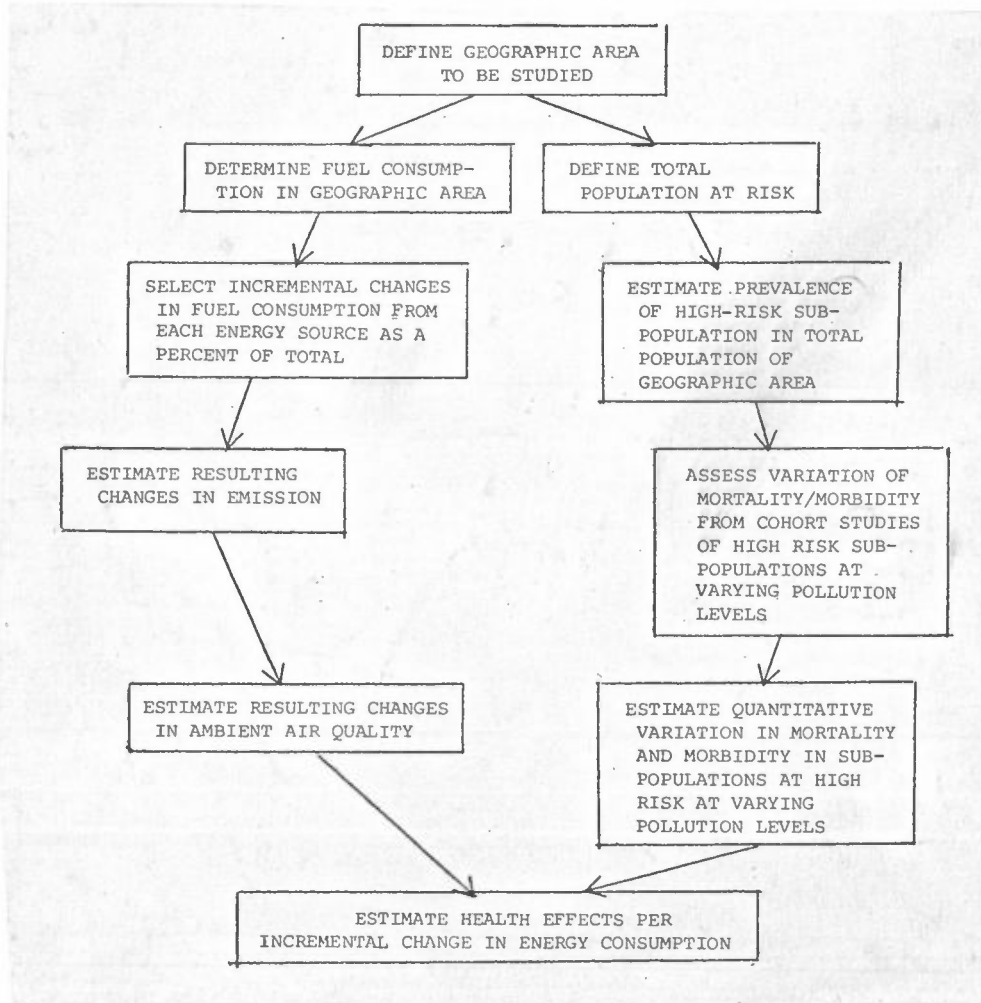


Figure 3.2. Model for Quantitating and predicting Health Impact of Energy Consumption (100).

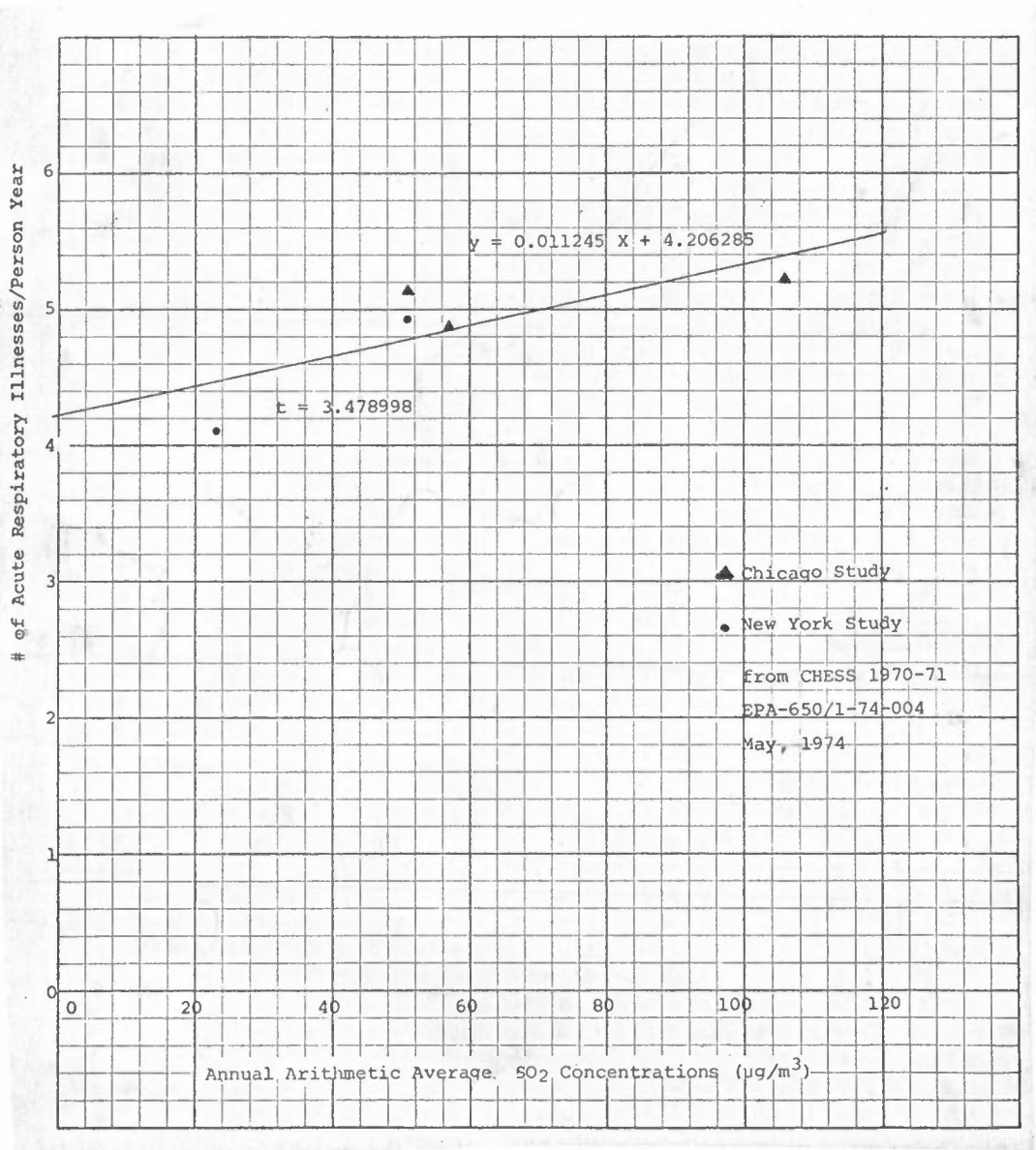
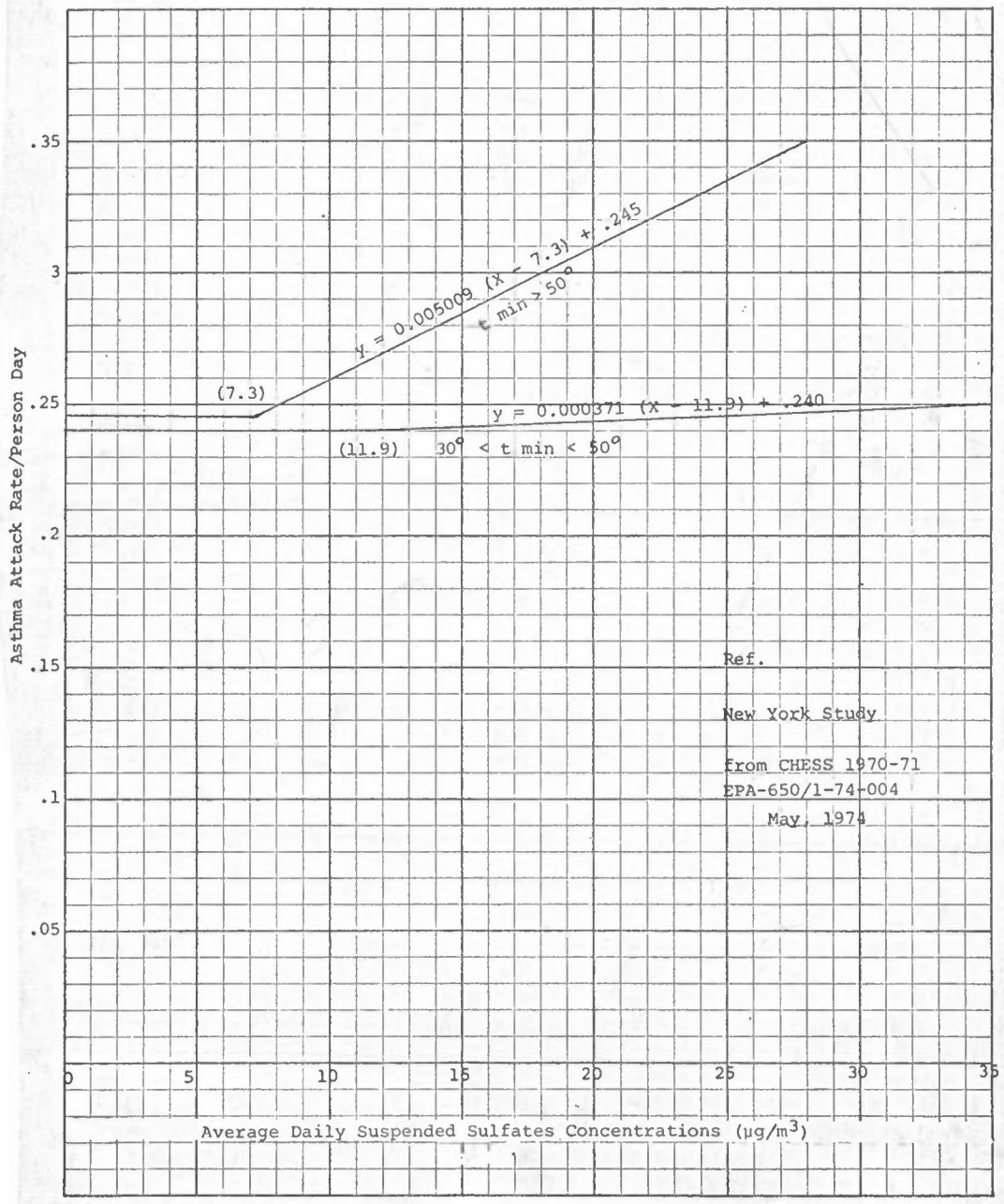


Figure 3.3. Frequency of Acute Respiratory Disease in Children 5 Years Old or Younger with Respect to Annual SO₂ Concentrations (100).



Note: Temperatures in degree Fahrenheit!

Figure 3.4. Effect of Daily Suspended Sulfate Concentrations on Daily Asthma Rates on Warm Days (100).

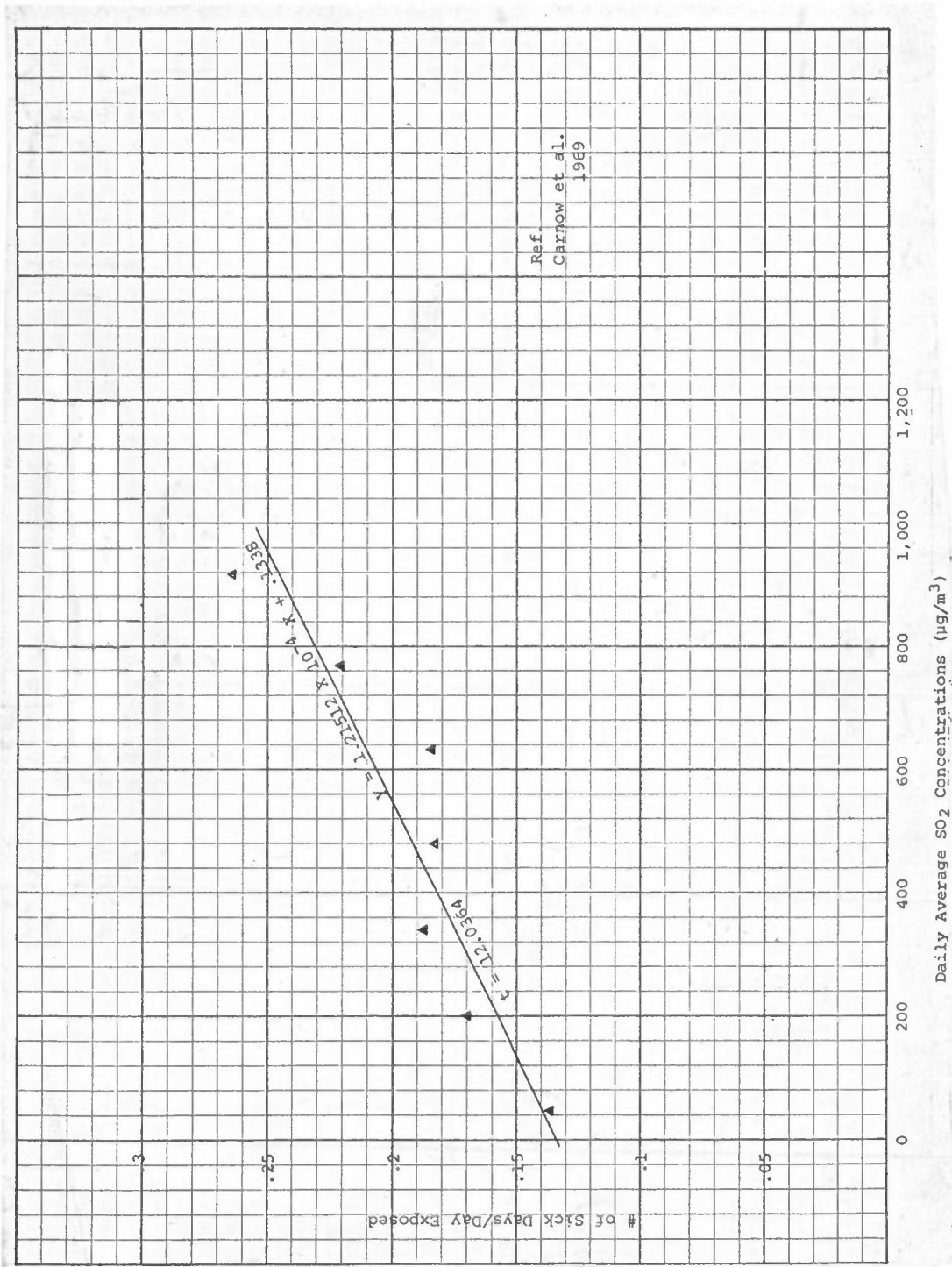


Figure 3.5. Frequency of Sick Days in Males Age 55 and Older with Grade 3 and 4 Bronchitis with Respect to Daily SO₂ Concentrations (100).

REFERENCES

- (1) Atmospheric Emission from Fuel Oil Combustion: An Inventory Guide.
U.S. Department of Health, Education and Welfare, Public Health Service, Publication No. 999-AP-2, Cincinnati, OH, 1962.
- (2) Atmospheric Emissions from Coal Combustion: An Inventory Guide.
U.S. Department of Health, Education and Welfare, Public Health Service, Publication No. 999-AP-24, Cincinnati, OH, 1966.
- (3) Engineering for Resolution of the Energy-Environment Dilemma.
National Academy of Engineering, Washington, DC, 1972, 340 p.
- (4) Air Pollution Aspects of Emission Sources: Electric Power Production.
U.S. Environmental Protection Agency, Pub. No. AP-96, Research Triangle Park, NC, 1971.
- (5) Terril, J.G. Jr., Harward, E.D., Leggett, I.P. Jr. Environmental Aspects of Nuclear and Conventional Power Plant. *Industrial Medicine and Surgery*, 36, 412-419 (1967).
- (6) Effects of Fuel Additives on Air Pollutant Emissions from Distillate-Oil-Fired Furnaces.
U.S. Environmental Protection Agency, Pub. No. AP-87, Research Triangle Park, NC, 1971.
- (7) Davis, D.D., Smith, G., Klauber, G. Trace Gas Analyses of Power Plant Plumes Via Aircraft Measurement: O₃, NO_x, and SO₂ Chemistry. *Science*, 186, 733-736 (1974).

- (8) Eisenbud, M.,
Petrow, H.G. Radioactivity in the Atmospheric
Effluents of Power Plants that
Use Fossil Fuels.
Science, 144, 288-289
(1964).
- (9) Martin, J.E.,
Harward, E.D.,
Oakley, D.T. Radiation Doses from Fossil-Fuel
and Nuclear Power Plants.
In: *Power Generation and Environmental
Change*, D.A. Berkowitz and
A.M. Squires (editors), MIT Press
Cambridge, MA, 1971, p 107-125.
- (10) *Inadvertent Climate Modification.*
MIT Press, Cambridge, Ma, 1971,
308 p.
- (11) Compilation of Air Pollutant
Emission Factors.
U.S. Environmental Protection
Agency, Pub. No. AP-42,
Research Triangle Park,
NC, 1973.
- (12) Preliminary Air Pollution Survey
of Arsenic and Its Compounds.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. APTD 69-26,
Raleigh, NC, 1969.
- (13) Preliminary Air Pollution Survey
of Barium and Its Compounds.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. APTD 69-28,
Raleigh, NC, 1969.
- (14) Preliminary Air Pollution Survey
of Beryllium and Its Compounds.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. APTD 69-29,
Raleigh, NC, 1969.

- (15) Preliminary Air Pollution Survey of Cadmium and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-32, Raleigh, NC, 1969.
- (16) Preliminary Air Pollution Survey of Chromium and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-34, Raleigh, NC, 1969.
- (17) Preliminary Air Pollution Survey of Iron and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-38, Raleigh, NC, 1969.
- (18) Preliminary Air Pollution Survey of Mercury and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-40, Raleigh, NC, 1969.
- (19) Preliminary Air Pollution Survey of Nickel and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-41, Raleigh, NC, 1969.
- (20) Preliminary Air Pollution Survey of Selenium and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-47, Raleigh, NC, 1969.

- (21) Preliminary Air Pollution Survey of Vanadium and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-48, Raleigh, NC, 1969.
- (22) Preliminary Air Pollution Survey of Zinc and Its Compounds. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. APTD 69-49, Raleigh, NC, 1969.
- (23) Sources of Polynuclear Hydrocarbons in the Atmosphere. U.S. Department of Health, Education and Welfare, Public Health Service, Pub. No. 999-AP-33, Durham, NC, 1967.
- (24) Schroeder, H.A. *The Poisons Around Us: Toxic Metals in Food, Air and Water.* Indiana University Press, Bloomington, Indiana, 1974, 144 p.
- (25) Lee, D.H. (editor) *Metallic Contaminants and Human Health.* Academic Press, New York, NY, 1972.
- (26) Hull, A.P. Comparing Effluent Releases from Nuclear and Fossil-Fueled Power Plant. *Nuclear News*, 17, 51-55 (1974).
- (27) Air Quality Criteria for Particulates. Department of Health, Education and Welfare, Public Health Service, Pub. No. AP-49, Washington, D.C., 1969.

- (28) Air Quality Criteria for Sulfur Oxides.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. AP-50,
Washington, D.C., 1969.
- (29) Air Quality Criteria for Photochemical Oxidants.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. AP-63,
Washington, D.C., 1970.
- (30) Air Quality Criteria for Hydrocarbons.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. AP-64,
Washington, D.C., 1970.
- (31) Air Quality Criteria for Nitrogen Oxides.
U.S. Department of Health,
Education and Welfare,
Public Health Service,
Pub. No. AP-84,
Washington, D.C., 1971.
- (32) Anderson, D.O. The Effects of Air Contamination on Health.
Can. Med. Ass. J.,
97, 528-536, 585-593,
802-806 (1967).
- (33) Ferris, B.G. Jr., Environmental Hazards: Effects of Community Air Pollution on the Prevalence of Respiratory Disease.
Whittenberger, J.L. *New England J. Med.*,
275, 1413-1419 (1966).
- (34) Goldsmith, J.R. Effects of Air Pollution on Human Health.
In: *Air Pollution*, Vol. I,
A.C. Stern (editor), 2nd Edition,
Academic Press,
New York, NY, 1968.

- (35) Heimann, H. Status of Air Pollution Health Research.
Arch. Environ. Health,
14, 488-503 (1967).
- (36) Lave, L.B., Air Pollution and Human Health.
Seskin, E.P. *Science*, 169, 723-733 (1970).
- (37) Battigelli, M.C. Sulfur Dioxide and Acute Effects of Air Pollution.
J. Occupational Medicine,
10, 500-511 (1968).
- (38) Health Consequences of Sulfur Oxides: A Report from CHESS, 1970-1971.
U.S. Environmental Protection Agency, Pub. EPA-650/1-74-004, Research Triangle Park, NC, 1974.
- (39) Knelson, J.H., Health Effects Basis for the
French, J.G., U.S. Air Quality Standard for
Shy, C.M. Nitrogen Dioxide.
In: *Colloquium on Nitrogen Oxides*,
VDI-Kommission Reinhaltung der Luft, Düsseldorf, Federal German Republic, 1974.
- (40) Toxicology of Atmospheric Sulfur Dioxide Decay Products.
U.S. Environmental Protection Agency, Pub. No. AP-111, Research Triangle Park, NC, 1972.
- (41) Waldbott, G.L. *Health Effects of Environmental Pollutants*.
C. V. Mosby Company, St. Louis, Missouri, 1973.
- (42) Higgins, I.T.T., Types, Ranges and Methods for
McCarroll, J.R. Classifying Human Pathophysiologic Changes and Responses to Air Pollution.
In: *Development of Air Quality Standards*, A. Atkisson and R.S. Gaines (editors), Charles E. Merrill Publishing Company, Columbus, OH 1970, p 73-96.

- (43) Speizer, F.E. An Epidemiological Appraisal of the Effects of Ambient Air Pollution on Health: Particulates and Oxides of Sulfur. *J. Air Poll. Contr. Ass.*, 19, 647-656 (1969).
- (44) Cassell, E.J. The Health Effects of Air Pollution and Their Implications for Control. *Law and Contemporary Problems*, 33, 197-216 (1968).
- (45) Ayres, S.M., Buehler, M.E. The Effects of Urban Air Pollution on Health. *Clin. Pharmacol. Therap.*, 11, 337-371 (1970).
- (46) MacMahon, B., Pugh, T.F., Ipsen, J. *Epidemiologic Methods*. Little, Brown & Co., Inc. Boston, MA, 1960, p 157.
- (47) Brassler, L.J., Joosting, P.E., von Zuilen, D. Sulfur Dioxide - to What Level is it Acceptable. Research Institute for Public Health Engineering, Report G-300, Delft, The Netherlands.
- (48) Joosting, P.E. Air Pollution Permissibility Standards Approached From the Hygienic Viewpoint. *Ingenieur*, 79, A739-A747 (1967).
- (49) Gore, A.T., Shaddick, C.W. Atmospheric Pollution and Mortality in the County of London. *Brit. J. Prevent. Soc. Med.*, 12, 104-113 (1958).
- (50) Burgess, S.E., Shaddick, C.W. Bronchitis and Air Pollution. *Roy. Soc. Health J.*, 79, 10-24 (1959).
- (51) Scott, J.A. The London Fog of December 1962. *Med. Officer*, 109, 250-252 (1963).
- (52) Martin, A.E., Bradley, W. Mortality, Fog and Atmospheric Pollution. *Monthly Bull. Ministry Health*, 19, 56-59 (1960).

- (53) Lawther, P.J. Compliance with the Clean Air Act: Medical Aspects. *J. Inst. Fuels*, (London), 36, 341-344 (1963).
- (54) Martin, A.E. Mortality and Morbidity Statistics and Air Pollution. *Proc. Roy. Soc. Med.*, 57, 969-975 (1964).
- (55) McCarroll, J., Bradley, W. Excess Mortality as an Indicator of Health Effects of Air Pollution. *Am. J. Public Health*, 56, 1933-1942 (1966).
- (56) Clean Air for California. California Dept. of Public Health, Berkeley, California, Rept 1 (1955), Rept 2 (1956), Rept 3 (1957).
- (57) Massey, F.J., Lanau, E., Deane, M. Air Pollution and Mortality in Two Areas of Los Angeles County. Joint Meeting of the American Statistical Association and the Biometric Society. New York, NY, December 27, 1961.
- (58) Hodgson, T.A. Jr. Short-Term Effects of Air Pollution on Mortality in New York City. *Environ. Sci. & Techn.*, 4, 589-597 (1970).
- (59) Greenburg, L., Jacobs, M.B., Drolette, B.M., Field, F., Braverman, M.M. Report on an Air Pollution Incident in New York City, November 1953. *Public Health Reports*, 77, 7-16, (1962).
- (60) Glasser, M., Greenburg, L., Field, F. Mortality and Morbidity during a Period of High Levels of Air Pollution, New York, November 23-25, 1966. *Arch. Environ. Health*, 15, 684-694, (1967).

- (61) Sterling, T.D. Urban Morbidity and Air Pollution:
A First Report.
Arch. Environ. Health,
13, 158-170 (1966).
- (62) Sterling, T.D.,
Pollack, S.V.,
Phair, J.J. Urban Hospital Morbidity and Air
Pollution: A Second Report.
Arch. Environ. Health,
15, 362-374 (1967).
- (63) Schoettlin, C.E.,
Landau, E. Air Pollution and Asthmatic
Attacks in the Los Angeles Area.
Public Health Reports,
76, 545-548 (1961).
- (64) Motley, H.L.,
Smart, R.H.,
Leftwich, C.I. Effect of Polluted Los Angeles
Air (Smog) on Lung Volume
Measurements.
J. Amer. Med. Assoc.,
171, 1469-1477, (1959).
- (65) Wayne, W.S.,
Wehrle, P.F.,
Carroll, R.E. Oxidant Air Pollution and
Athletic Performance.
J. Amer. Med. Assoc.,
199, 901-904 (1967).
- (66) Stocks, P. Statistics of Cancer of the Lung.
J. Faculty Radiologists,
6, 166-173 (1955).
- (67) Stocks, P. Air Pollution and Cancer Mortality
in Liverpool Hospital Region and
North Wales.
Intern. J. Air Poll.,
1, 1-13 (1958).
- (68) Stocks, P. Cancer and Bronchitis Mortality
in Relation to Atmospheric
Deposit and Smoke.
Brit. Med. J., 1, 74-79 (1959).
- (69) Stocks, P.,
Davies, R.I. Epidemiological Evidence from
Chemical and Spectrographic
Analyses that Soil is Concerned
in the Causation of Cancer.
Brit. J. Cancer, 14, 8-22 (1960).

- (70) Stocks, P. On the Relations between Atmospheric Pollution in Urban and Rural Localities and Mortality from Cancer, Bronchitis, and Pneumonia, with Particular Reference to 3.4 Benzopyrene, Beryllium, Molybdenum, Vanadium, and Arsenic.
Brit. J. Cancer, 14, 397-418 (1960).
- (71) Winkelstein, W. The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Men (II. Oxides of Sulfur).
Arch. Environ. Health, 16, 401-405 (1968).
- (72) Winkelstein, W. The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Man.
Arch. Environ. Health, 14, 162-169 (1967).
- (73) Winkelstein, W. The Relationship of Air Pollution to Cancer of the Stomach.
Arch. Environ. Health, 18, 544-547 (1969).
- (74) Zeidburg, L.D., Hagstrom, R.M., Sprague, H.A., Landau, E. Nashville Air Pollution Study. VII. Mortality from Cancer in Relation to Air Pollution.
Arch. Environ. Health, 15, 237-248 (1967).
- (75) Zeidburg, L.D., Horton, R.M., Landau, E. The Nashville Air Pollution Study. V. Mortality from Diseases of the Respiratory System in Relation to Air Pollution.
Arch. Environ. Health, 15, 214-224 (1967).

- (76) Petrilli, F.L.,
Agnese, G.,
Kanitz, S. Epidemiology Studies of Air
Pollution Effects in Genoa,
Italy.
Arch. Environ. Health,
12, 733-740 (1966).
- (77) Holland, W.W.,
Reid, D.D.,
Seltzer, R.,
Stone, R.W. Respiratory Disease in England
and the United States. Studies
of Comparative Prevalence.
Arch. Environ. Health,
10, 338-345 (1965).
- (78) Holland, W.W.,
Reid, D.D. The Urban Factor in Chronic
Bronchitis.
Lancet, 1, 445-448 (1965).
- (79) Zeidburg, L.D.,
Prindle, R.A.,
Landau, E. The Nashville Air Pollution
Study, III. Morbidity in
Relation to Air Pollution.
Am. J. Public Health,
54, 85-97 (1964).
- (80) Hausknecht, R. Air Pollution Effects Reported
by California Residents (from
California Health Survey).
California Department of Public
Health,
Berkeley, California (1960).
- (81) Douglas, J.W.,
Waller, R.E. Air Pollution and Respiratory
Infection in Children.
Brit. J. Prevent. Soc. Med.,
20, 1-8 (1966).
- (82) Shy, C.,
Creason, J.P.,
Pearlman, M.E.,
McClain, K.E.,
Benson, F.B.,
Young, M.M. The Chattanooga School Study:
Effects of Community Exposure
to Nitrogen Dioxide Methods,
Description of Pollutant
Exposure and Results of
Ventilatory Function Testing.
J. Air Poll. Contr. Assoc.,
20, 539-545 (1970).
- (83) Shy, C.,
Creason, J.P.,
Pearlman, M.E.,
McClain, K.E.,
Benson, F.B.,
Young, M.M. The Chattanooga School Study:
Effects of Community Exposure
to Nitrogen Dioxide. II.
Incidence of Acute Respiratory
Illness.
J. Air Poll. Contr. Assoc.,
20, 582-588 (1970).

- (84) Pearlman, M.E.,
Finklea, J.F.,
Creasjón, J.P.,
Shy, C.M.,
Young, M.M.,
Horton, R.J. Nitrogen Dioxide and Lower
Respiratory Illness.
Pediatrics, 47, 391-398 (1971).
- (85) Chronic Respiratory Diseases
in Children in Relation to Air
Pollution.
World Health Organization,
Regional Office for Europe,
Copenhagen, 1974.
- (86) McMillan, R.S.,
Wiseman, D.H.,
Hanes, B.,
Wehrle, P.F. Effects of Oxidant Air Pollution
on Peak Expiratory Flow Rates
in Los Angeles School Children.
Arch. Environ. Health,
18, 94-99 (1969).
- (87) Prindle, R.A.,
Wright, G.W.,
McCaldin, R.O.,
Marcus, S.C.,
Lloyd, T.C.,
Bye, W.E. Comparison of Pulmonary Function
and Other Parameters in Two
Communities with Widely Different
Air Pollution Levels.
Am. J. Public Health,
53, 200-218 (1963).
- (88) Lawther, P.J. Climate, Air Pollution, and
Chronic Bronchitis.
Proc. Roy. Soc. Med.,
51, 262-264 (1958).
- (89) Hamilton, L.D.,
Morris, S.C. Health Effects of Fossil Fuel
Power Plants.
Proceedings of the Eighth
Midyear Topical Symposium of
the Health Physics Society.
Knoxville, Tennessee,
October 21-24, 1974.
- (90) Hickey, R.J. Air Pollution.
In: *Environment: Resources, Pollution &
Society*, W.W. Murdoch (editor),
Sinauer Associates Inc.,
Stamford, CN, 1971.
- (91) Hickey, R.J.,
Boyce, D.E.,
Harner, E.B.,
Clelland, R.C. Ecological Statistical Studies
Concerning Environmental
Pollution and Chronic Disease.
IEEE Trans. Geosci. Electronics,
8, 186-202 (1970).

- (92) Hickey, R.J.,
Schoff, E.P.,
Clelland, R.C. Relationship Between Air
Pollution and Certain Chronic
Disease Death Rates.
Arch. Environ. Health,
15, 728-738 (1967).
- (93) Schwing, R.C.,
McDonald, G.C. Measures of Association of Some
Air Pollutants, Natural Ionizing
Radiation and Cigarette Smoking
with Mortality Rates.
General Motors Research
Laboratories, Report GMR-1573,
Warren, MI, 1974.
- (94) Duffy, E.A.,
Carroll, R.E. United States Metropolitan
Mortality, 1959-1961.
U.S. Public Health Service,
Pub. No. 999-AP-39,
Washington, D.C., 1967.
- (95) Carnow, B.W.,
Meier, P. Air Pollution and Pulmonary
Cancer.
Arch. Environ. Health,
27, 207-218 (1973).
- (96) Hausknecht, D.F. Approximate Mortality Risk from
SO₂ and Particulates.
Public Health Risks of Thermal
Power Plants (Appendix VI),
UCLA School of Engineering and
Applied Science.
Los Angeles, CA, 1972.
- (97) Lave, L.B.,
Freeburg, L.C. Health Effects of Electricity
Generation from Coal, Oil and
Nuclear Fuel.
Nuclear Safety, 14, 409-428 (1973).
- (98) Lindberg, W. Den alminnelige luftforurensning
i Norge.
Røykskaderådet, Oslo 1968.
- (99) Jaksch, J.A.,
Stoever, H.H. Outpatient Medical Costs Related
to Air Pollution in the Portland,
Oregon Area.
U.S. Environmental Protection
Agency, Report EPA-600/5-74-017,
Washington, D.C., 1974.
- (100) Carnow, B.W.,
Wadden, R.,
Scheff, P.,
Musselman, R. Health Effects of Fossil Fuel
Combustion: A Quantitative
Approach.
Report to the Ford Foundation
Energy Policy Project,
Washington, D.C., 1974.

CHAPTER 4
LITERATURE SURVEY:
METHODS OF COMPARING AIR POLLUTION HEALTH EFFECTS
CAUSED BY FOSSIL FUEL AND NUCLEAR POWER PLANTS

4.1 INTRODUCTION

The purpose of this chapter is to identify the different methods used by investigators to compare the air pollution health effects of different types of power plants.

The environmental impact of different types of power generation cannot be compared in an unbiased fashion unless somehow a common denominator can be found. There are many differences in their environmental impacts and these must be converted to a common scale before quantitative comparisons can be made of the total impact. Of course qualitative observations can be made, but these most often just become pseudo-technical expressions of personal opinion. Quantitative comparisons are thus needed. Hub et al. (1) examined this problem and concluded that such quantitative comparisons could not be made at the present time because the present health effects data for chemical pollutants are inadequate. Other researchers attempting to make such quantitative assessments have taken different approaches by considering:

- pollutant emission rates
- specific health effects
- the overall risks
- air quality standards

The following sections identify the authors who have used these approaches. A discussion section is also included which presents some of our viewpoints about these approaches. However, only the work of Lave and Freeburg (2) was studied in detail, with comments presented in the Appendix.

The following is a partial listing of individuals and organizations now working directly on this problem, all of whom were contacted during this project. Some of the findings of their work are reported in this chapter, but in most cases the results of their studies have not reached the point of publication.

Dr. William C. Nelson
Chief, Biometry Branch
U.S. Environmental Protection Agency
Research Triangle Park
North Carolina

Mr. Thomas E. Waddell
Economist
U.S. Environmental Protection Agency
Washington, D.C.

Dr. William Jones
Energy Laboratory
Massachusetts Institute of Technology
Cambridge, Massachusetts

Dr. David Okrent
Engineering Department
University of California at Los Angeles
Los Angeles, California

Dr. R.C. Schwing
General Motors Research Laboratories
Detroit, Michigan

Ford Foundation
Energy Policy Project
Washington, D.C.

Dr. L.D. Hamilton
Brookhaven National Laboratory
Upton, New York

Mr. J. Kumpf
European Office
World Health Organization
Copenhagen, Denmark

Dr. Bertram W. Carnow
School of Public Health
University of Illinois
Chicago, Illinois

Dr. Lester B. Lave
Carnegie-Mellon University
Pittsburgh, Pennsylvania

Dr. K.A. Hub
Argonne National Laboratory
Argonne, Illinois

Dr. D. Hausknecht
Science Applications, Inc.
El Segundo, California

4.2 POLLUTANT EMISSION RATES

Eisenbud and Petrow (3), Martin et al. (4), Jaworowski et al. (5), and Hull (6) have compared pollutant emission rates for various types of power plants. The comparisons hinge on the fact that fossil fuels may contain appreciable quantities of radioactive substances which may be emitted in the combustion gases. Coal and oil contain Radium-226 and Thorium-228, but natural gas contains neither of them. Thus, Eisenbud and Petrow concluded "Our measurements of the natural radioactivity of fossil fuels have thus led us to the conclusion that an electrical generating station that derives its thermal energy from such fuels discharges relatively greater quantities of radioactive substances into the atmosphere than many power plants that derive their heat from nuclear energy."

Jaworowski et al. (5) reported the following regarding ionizing radiation emissions from coal, BWR and PWR power stations:

"... the radiation dose to the general population, being relatively higher in case of fossil fuel power stations and smaller in nuclear ones, are minute enough to be neglected as the health hazard. On the other hand, there is no evidence that non-radioactive pollutants dispersed by conventional power production, can similarly be neglected." (See also Table 4.1.)

Table 4.1. Radiation Dose Rates to Critical Organs from Three Power Stations (5).

Power station	Dose rate, mrem/year	Dose rate per energy output mrem/year/MW
Siekierki fossil fuel 15 km	43 - osteocyte	0.1
Dresden I nuclear/BWR/ exclusion boundary	$7 \cdot 10^{-5}$ - thyroid	10^{-7}
Yankee nuclear/PWR/ exclusion boundary	0.15 - whole body	$2 \cdot 10^{-4}$

4.3 SPECIFIC HEALTH EFFECTS

Work on the comparisons of specific health effects according to the type of power plants is reported in the following section on Overall Risks. Only mortality has been considered as the basis for these assessments.

4.4 OVERALL RISKS

Several investigators have estimated the overall mortality risks associated with electric power production (6, 7, 8, 9). In doing this they have identified the various activities in the overall operation such as mining, fuel preparation, fuel use, pollution exposure and others, and calculated the risk for each. The work of Lave and Freeburg (2) is typical of this approach, with the following order of desirability ranking for power plants:



Lave and Freeburg did not consider gas-fired power plants, and used only sulfur dioxide and particulate matter as the relevant pollutants for the oil- and coal-fired power plants.

Hull (6) reported that: "The risk from the highest radiation levels of a few millirems per year to an individual living adjacent to the boundary of a nuclear reactor site seems trivial in comparison with the many risks seldom taken into consideration by the populace." See Table 4.2 in support of his conclusions.

Table 4.2. Annual Probability of Fatal Injury from Radiation and Other Causes (6).

Type of cause	Individual probability of fatal injury or effect per year of exposure
Radiation at 1 mrem/year*	1×10^{-7}
Natural disasters	2×10^{-6}
Fossil-fueled power plants	4×10^{-6}
Electricity	2×10^{-5}
Firearms	2×10^{-5}
Air pollution†	1×10^{-4}
Smoking ⁺⁺	5×10^{-4}
Automobiles	1×10^{-2}
All diseases	1×10^{-2}

✓ Answer?

*Estimated from ICRP data, which are based on the conservative assumption that effects observed at higher levels (100 rems) are linear with decreasing dose and dose rate.

†Based on entire population exposed 100% of the time.

⁺⁺Based on smoking at a continuous rate.

Starr and Greenfield (7) and Starr (8) have compared the relative risks of electric power production to other daily activities as follows:

- The public-health risk from routine operations of electricity-generating plants using nuclear fuel or oil is in the range of the very low hazards to which the public is exposed by uncontrollable events of nature, such as being struck by lightning or bitten by a venomous animal or insect (about one death per year in a million population).

- Routine operation of a nuclear plant presents a significantly smaller public-health risk than the routine operation of an oil-fired plant, typically by a factor of 10 to 100.
- The public-health risks due to accidental releases from either a nuclear or an oil-fired plant are both of the same magnitude and are about 100,000 times smaller than the risk from routine operation of the plants.

4.5 AIR QUALITY STANDARDS

Standards exist for nuclear and fossil fuel power plant emissions and ambient air quality. Some investigators (6, 9, 10, 11, 12) have tried to compare these technologies by estimating: a) how well each meets its own standards, or b) the contribution each would make to the degradation of ambient air quality. This approach then seeks to use environmental standards as the common denominator for comparisons between the various types of power plants. Starr et al. (9) used this approach in Los Angeles to answer the question: "How many plants of a given type can be operated without reaching a pollutant concentration having public health significance?" Their answers are shown in Table 4.3.

Table 4.3. Tolerable Numbers of Power Plants as Implied by Current Practices in Los Angeles County* (9).

PLANT TYPE	CRITICAL POLLUTANT	TOLERABLE NUMBERS OF 1,000 Mwe PLANTS (EXCLUSIVE OF POLLUTANTS FROM OTHER SOURCES)
Oil	SO ₂	10
Natural gas	NO ₂	23
Nuclear reactor (LWR)	Radio-active gases	160,000

*Based on the following assumptions:

1. *Unspecified mixture of radio-active isotopes released from nuclear plant (Most restrictive assumption based on 1 mrem).*
2. *Compliance with 0.5 percent by weight sulfur content for oil.*
3. *Air volume of Los Angeles County was assumed to be 3,165 km³, which implies a mean inversion height of 300 m.*
4. *Ventilation of this volume requires one day.*
5. *Effluent volume rate for 1,000 MWe reactor is taken as 0.5×10^6 cfm which is an estimated upper limit.*

Terril et al. (10) made comparisons in terms of the volumes of air required to dilute power plant emissions down to the concentrations specified in ambient air quality standards. The results of this work are shown in Table 4.4. Hull (11) has made a similar study, and he reached the same conclusions as Terril et al. (10).

Table 4.4. Dilution Air Required to Meet Concentration Standards for Various Power-Plant Pollutants (10).

Type of plant	Critical pollutant	Exposure vector	Concentration standards†	Discharge quantities per Mw(e)-year	Yearly volume of air required for dilution, m ³ /Mw(e)
Coal	SO ₂	Air-SO ₃ -lungs Air-lungs	0.3 ppm	306 × 10 ³ lb	1.77 × 10 ¹¹
	Fly ash ²²⁶ Ra	Air-lungs	1.0 × 10 ⁻¹³ μc/cm ³	17.2 μc	1.72 × 10 ⁸
	²²⁸ Ra	Air-lungs	3.0 × 10 ⁻¹³ μc/cm ³	10.8 μc	3.6 × 10 ⁷
Oil	SO ₂	Air-lungs	0.3 ppm	116 × 10 ³ lb	6.75 × 10 ¹⁰
	NO ₂	Air-lungs	2 ppm	47 × 10 ³ lb	5.77 × 10 ⁹
		Air-O ₃ -smog-irritants of lungs and eyes	Unknown		
	Fly ash ²²⁶ Ra	Air-lungs	1.0 × 10 ⁻¹³ μc/cm ³	0.15 μc	1.5 × 10 ⁶
	²²⁸ Ra	Air-lungs	3.0 × 10 ⁻¹³ μc/cm ³	0.35 μc	1.2 × 10 ⁶
Gas	SO ₂	Air-lungs	0.3 ppm	0.027 × 10 ³ lb	1.5 × 10 ⁷
	NO ₂	Air-lungs	2 ppm	26.6 × 10 ³ lb	3.22 × 10 ⁹
	Particulates - random daughters	Air-lungs	Unknown	Unknown	
Nuclear	Radioactive noble gases ⁸⁵ Kr + ¹³³ Xe	External	1 × 10 ⁻⁷ μc/cm ³	5.7 × 10 ³ μc	5.7 × 10 ⁴ (Shippingport 5 - year average)
				9.5 × 10 ³ μc	9.5 × 10 ⁴ (Yankee 1965)
	¹³¹ I	Air-lungs-thyroid	1 × 10 ⁻¹⁰ μc/cm ³	No detectable levels reported in available literature	No detectable levels reported in available literature
		Air-grass-milk-thyroid	1.6 × 10 ⁻¹³ μc/cm ³		

† In the case of radioactive materials, they are based on AEC regulatory concentration standards (10CFR20), and in the case of chemical pollutants from combustion of fossil fuel, they are based on recommended permissible concentrations in the available literature.

Schikarski et al. (12) compared coal-fired power plants with nuclear power plants by calculating the dispersion of pollutants and comparing the resulting concentrations to the allowable ambient standards. On this basis, they concluded that nuclear energy systems are "better" than coal by a factor of 180.

4.6 DISCUSSION

4.6.1 Pollutant Emission Rates

When comparing radioactivity from fossil fuel stations with nuclear stations problems arise. The forms of the emitted radioactivity are different. For example, radium and thorium from fossil fuel plants are in particulate form, while the radioactivity from nuclear stations is mostly associated with gaseous emissions. In addition, the respective radioactive species have vastly different half-lives. It is likely that the health impact of these species is also quite different. The gaseous radionuclides would contribute mainly to the whole-body exposure whereas the particulate forms would cause problems if lodged in the respiratory system from where soluble fractions could pass to other parts of the body and cause adverse effects. Thus, although total radioactivity emissions can be quantitatively compared, the comparisons could be made more refined by extending the analysis to consider the health effects of exposures to various forms of radioactivity. Even then this method of comparison cannot be considered as adequate. Fossil fuel power plants emit a number of chemical species for which there are no counterparts in the emissions from nuclear power plants. Nevertheless, as indicated in Chapter 3, these pollutants do have adverse health effects and they therefore cannot be ignored.

4.6.2 Specific Health Effects

The health effects associated with exposures to radiation and chemical air pollutants have been discussed in Chapters 2 and 3, respectively. Relatively few attempts have been made to compare these effects. There are three effect categories in which this could be done:

- total mortality
- cancer
- genetic changes

The pollutants causing these effects are of course not the same, but this does not matter. However, it is the total health impact that should be compared; not just one or other of these effects. Some types of health effects may not apply to all the power generation alternatives. For example, there may be morbidity effects associated with certain types of fossil fuel power plants, which do not exist for nuclear power plants and vice versa.

Producing estimates of total mortality, cancer and genetic changes for comparison is hampered by a common problem: lack of dose-effect data at the appropriate level of exposure. Two diverse interpretations have been advanced:

- One possible attitude is that the lack of data by itself may be taken as an indication that the effect is so insignificant that it would be unreasonable to expect to be able to observe it. No exposure - effect value is assigned.
- Another attitude is that since nothing is known definitely about the importance of the effects, the exposure - effect relationship is assigned the highest value that can within reason be assigned to it, in order to be on the safe side.

Too often the first attitude has been applied to the delayed health effects from fossil fuel power plants, while the second attitude has been applied to the nuclear power plants. A comparison made on such diverse bases is clearly invalid.

Evaluating the specific health effects and then calculating the total health impact would definitely be the best approach. However, more work must be done to define a specific method for making such evaluations; in addition, more quantitative information is needed regarding:

- long term exposure to low level radiation
- effect of trace metals emitted by fossil fuel power plants
- synergistic effects of various pollutants
- long term effects of chemical air pollution

4.6.3 Overall Risks

There is some risk in everything we do. However, all actions do not contain the same risk. Most people have at least a simple concept of risk. For example they understand that some occupations are more dangerous than others. Also they know that accidents can happen, so they often buy insurance for their lives, houses, cars and other things. The need for and cost of this insurance is based upon the probability of the event occurring.

The concept of risk may also be applied to the production of electric power. Adverse affects on the population at risk may occur due to accidents or from normal operations. Under normal operations various pollutants are emitted, thereby exposing the surrounding population to possible health effects.

The work done on the overall risks had considered only mortality risks. This underestimates the total risk to the population, because additional relevant factors, such as morbidity effects and probable genetic changes caused by pollution should be included.

The public may not respond to such risk analyses even if they understand them. There are a number of discouraging examples where the public has chosen to assume risks and ignore the facts. Drinking and traffic accidents, and smoking and cancer are just two such examples. Research and statistics have documented these problems; yet liquor and cigarette sales have not decreased. Thus telling someone that "Decision A" is clearly a thousand times less risky than "Decision B" will not necessarily convince them. They may still prefer "Decision B". There are obviously other "human" factors that enter into the decision-making process.

4.6.4 Air Quality Standards

The wide divergence exhibited by the results of the various assessment approaches warrants a look at the standards used to make these calculations. At least part of the problem here can be clarified by considering what the standards are and how they were developed. This important point is often not considered. Firstly, the various standards (see Figure 4.1) are not set by the same organization and hence it is not likely that the same development criteria and procedures are used. This in itself is conducive to dissimilar results. Secondly, the public has had a great concern over the use of nuclear technology. For most people their experience and understanding of nuclear energy relates to the destructive nature of bombs and missiles. This concern has supported the

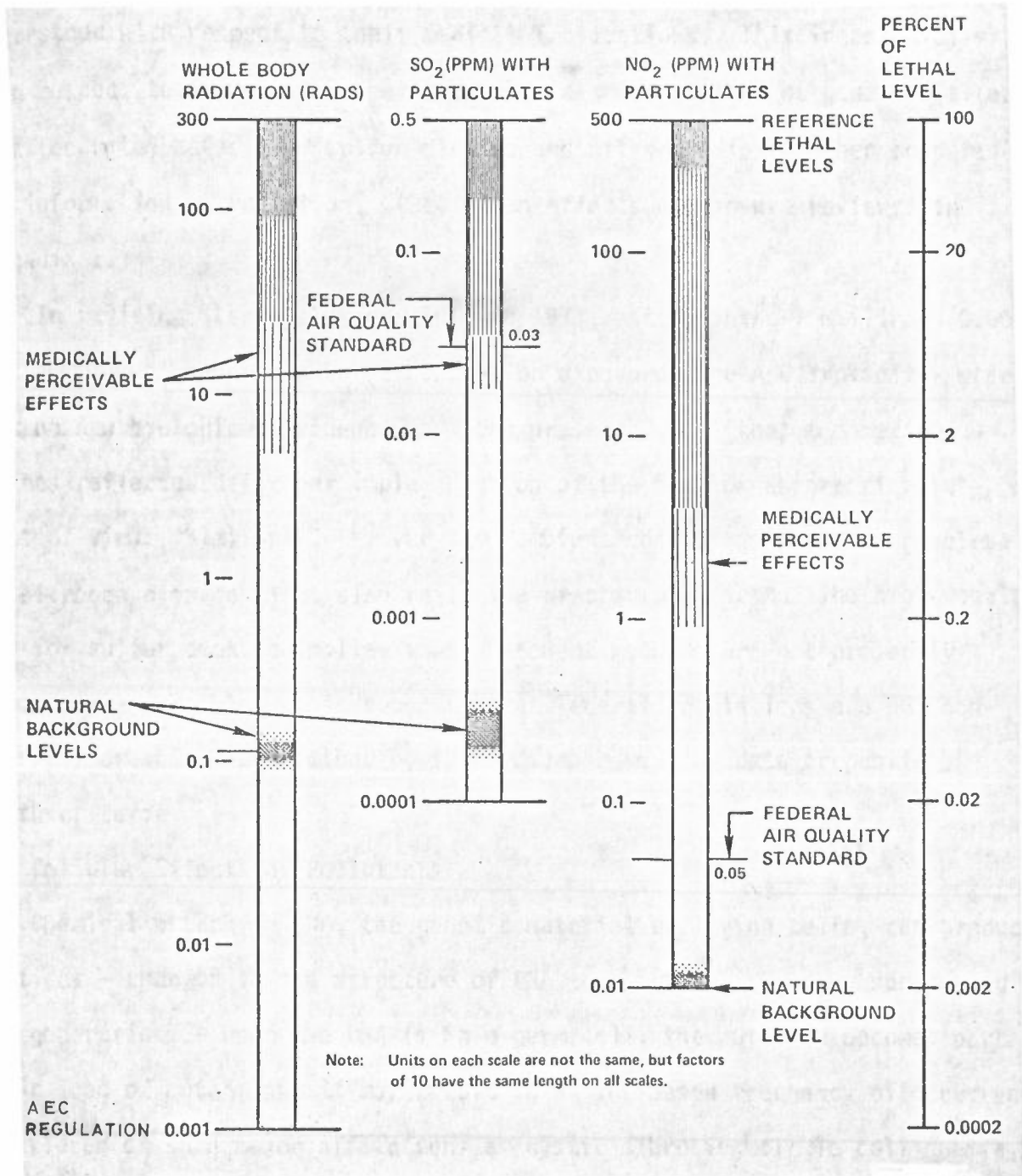


Figure 4.1. Observed Pollutant Effects on Physiological Function of Humans (14).

idea that if nuclear power plants are to be used they must meet very strict requirements. Thirdly, fossil fuel technology was in use a long time before anyone decided that its environmental aspects needed to be regulated. Since the technology was in wide spread use, the basis for regulation has been centered more around "what is technically and economically feasible?". The health effects are still not well known and this consideration has received less attention. Of course over the last few years this has begun to change. People are now also concerned about how pollution from fossil fuel power plants affects their health. Standards have been made more strict, but they are continuously being challenged by the energy companies and electric utilities. Lave (13) believes that using these standards as a basis of comparison is a use for which they were not intended. We agree with this point.

4.7 CONCLUSIONS

The "Specific Health Effects" approach is the best one for comparing the nuclear and fossil fuel power plants. The other three approaches previously identified all have serious deficiencies:

- The "Pollutant Emission Rate" approach can only compare radioactive emissions and it must therefore neglect many important pollutants emitted by fossil fuel power plants.
- The "Overall Risk" approach is deficient at present because it considers only mortality risks. However, if specific mortality causes, morbidity and genetic changes were included, then this approach could be used in conjunction with the "Specific Health Effects" approach.

- The "Air Quality Standard" approach neglects the fact that the bases for the nuclear and fossil fuel related pollutants are very different.

Hence the relation between pollution level and the standard cannot be expected to represent the relationship between pollution level and health effects.

Before the "Specific Health Effects" approach can be used two tasks must be accomplished. Firstly, a common calculation method must be selected to determine what pollutant dose a population receives from nuclear or fossil fuel power plants. Secondly, additional information is needed to describe the dose-effect relationship for the different pollutants involved. This latter requirement must also consider the possibility of synergistic effects.

REFERENCES

- (1) Hub, K.A., et al. Social Costs for Alternative Means of Electrical Power Generation for 1980 and 1990. Argonne National Laboratory Report ANL-8093 V-3, Argonne, Illinois, 1973.
- (2) Lave, L.B., Freeburg, L.C. Health Effects of Electricity Generation from Coal, Oil, and Nuclear Fuel. *Nuclear Safety*, 14, 409-428 (1973).
- (3) Eisenbud, M., Petrow, H.G. Radioactivity in the Atmospheric Effluents of Power Plants That Use Fossil Fuels. *Science*, 144, 288-289 (1969).
- (4) Martin, J.E., Harward, E.D., Oakley, D.T. Comparison of Radioactivity from Fossil Fuel and Nuclear Plants. In: *Hearings on Effects of Producing Electric Power*, Vol. 1, 91st U.S. Congress, 1st Session, Joint Committee on Atomic Energy. Superintendent of Documents, U.S. Government Printing Office Washington, D.C. 1969, p. 773-809.
- (5) Jaworowski, Z. et al. Radiation Hazards to Population from Conventional and Nuclear Production. In: *Symposium on Environmental Surveillance Around Nuclear Installations*, Vol. 1, International Atomic Energy Agency, IAEA-SM-180/20, Warsaw, Poland, 1973, p. 403-412.
- (6) Hull, A.P. Radiation in Perspective: Some Comparisons of the Environmental Risks from Nuclear- and Fossil-Fueled Power Plants. *Nuclear Safety*, 12, 185-196 (1971).

- (7) Starr, C.,
Greenfield, M.A. Public Health Risks of Thermal
Power Plants.
Nuclear Safety, 14, 267-274, (1973).
- (8) Starr, C. Social Benefits Versus
Technological Risk.
Science, 165, 1232-1238 (1969).
- (9) Starr, C.,
Greenfield, M.A.,
Hausknecht, D.F. A Comparison of Public Health
Risks: Nuclear on Oil-Fired
Power Plants.
Nuclear News, 15, 37-45, (1972).
- (10) Terril, J.G.jr.,
Harward, E.D.,
Leggett, I.P.jr. Environmental Aspects of
Nuclear and Conventional
Power Plants.
Ind. Med. Surg., 36,
412-419, (1967).
- (11) Hull, A. P. Comparing Effluent Releases
from Nuclear and Fossil-Fueled
Power Plants.
Nuclear News, 17, 51-55, (1974).
- (12) Schikarski, W.,
Jansen, P.,
Jordon, S. An Approach to Comparing Air
Pollution from Fossil-Fuel
and Nuclear Power Stations.
In: *Symposium on Environmental
Aspects of Nuclear Power Stations*,
International Atomic Energy
Agency, IAEA-SM-146/57,
New York 1970.
- (13) Lave, L. B. Personal communication,
April 1975.
- (14) Starr, C.,
Greenfield, M.A.,
Hausknecht, D.F. Public Health Risks of Thermal
Power Plants.
School of Engineering and
Applied Science,
University of California,
UCLA-ENG-7242, Los Angeles,
California 1972.

APPENDIX
AN EVALUATION OF THE STUDY
"HEALTH EFFECTS OF ELECTRICITY GENERATION FROM COAL,
OIL AND NUCLEAR FUEL"
BY LAVE AND FREEBURG

A.1 BACKGROUND

What is the optimal amount of air pollution? This may seem like a rather surprising question since no one likes air pollution. But to an economist this is not only a reasonable question, it is "the" question. Dr. Lester B. Lave is an economist. He wants to find out if the benefits derived from abating air pollution outweigh the costs. The cost of such control action may be calculated, but the benefits are hard to define and quantify. It was this problem which caused Dr. Lave and his co-workers to begin studying the relationship of air pollution and its health effects. They have written a number of technical papers describing this research work (References 1 - 8).

Lave and Freeburg (1) developed the following regression relationship which explains 83 percent of the total variation:

$$\begin{aligned} MR_i = & 19.607 + 0.041 \text{ mean } P_i + 0.071 \text{ min } S_i \\ & + 0.001 P/M_i^2 + 0.041\%NW_i + 0.687\%_{\geq 65}_i + e_i \end{aligned}$$

where

- MR_i = total mortality rate (per 10,000 people) in city i
- mean P_i = arithmetic mean of suspended particulate readings in city i
- min S_i = smallest biweekly sulfate reading in city i (X 10)
- P/M_i^2 = population density in city i
- $\% NW_i$ = proportion of the population which is nonwhite in city i (X 10)
- $\%_{\geq 65}_i$ = proportion of the population 65 and older in city i (X 10)
- e_i = error term for variation in the mortality rate not explained by the equation

They used this expression to estimate how changes in mortality rates might be caused by the different variables of the equation. This sensitivity analysis showed that a 10 percent increase in air pollution (sulfates and particulate matter) would result in 0.90 percent increase in the mortality rate.

This section reviews and evaluates the work performed by Lave and Freeburg (1) in developing the above regression relationship. This work is of particular interest because it is one of the few cases where air pollution health effects have been put in quantifiable terms. This review is done with the thought that the results of the method itself might be applied to Norwegian conditions. Unless otherwise indicated, our comments pertain only to the work in Reference 1. Further work by Lave and co-workers and other researchers has been cited to provide a more complete evaluation.

A.2 STATISTICAL APPROACH

At the present time adequate theoretical models describing the air pollution and health effects relationships do not exist. Statistical analysis methods have been used because they can:

- indicate overall relationships that may not otherwise be noticeable
- provide some estimate of the dose-effect relationship in context of other parameters
- indicate areas for more specific research

However, there is quite a difference between having data from a planned experiment and merely analyzing a set of data collected from various sources well after the event. Lave and Freeburg had no choice, but to do the latter and they had to accept problems associated with:

- inadequate sample size
- non-representative samples
- lack of controls
- presence of interaction and confounding factors
- measurements with poor precision
- other unknowns

The statistical model used by Lave and Freeburg is a straightforward one. It can be expressed as the functional relationship:

$$\text{Health Effect} = F(P_1, P_2, \dots, P_n, e)$$

where the Health Effect is the dependent variable, the P's are independent variables which affect health, and "e" is an error term. Virtually all of the work relating pollution to health effects has assumed a linear relationship. Lave (5) did

consider other functional forms, such as log linear and quadratic, but concluded that assumption of linearity was as good as any. In the absence of any specific knowledge about the dose-response relationships this approach seems acceptable. It does not, however, provide for any estimates of interaction effects. A much quoted synergism is the sulfur dioxide and particulate matter phenomenon (9). There may of course be a number of such interactions which we do not know about now.

The model and least squares analysis method used by Lave and Freeburg are well-established and therefore not subject to very much criticism. However, we should point out that McDonald and Schwing (10) have suggested that the Ridge Regression method of Hoerl and Kennard (11, 12) should be used to estimate the linear regression coefficients. They used this method for a study which estimated the effect of hydrocarbons, oxides of nitrogen and sulfur oxides on overall mortality. In some instances the regression coefficients differed considerably from those obtained from ordinary least squares analysis. Reinke (13) recognized this as a possible problem and also suggested that the methods of Hoerl and Kennard should be used for air pollution work. We feel that the data used by Lave and Freeburg does not warrant this additional degree of statistical sophistication. However, the Ridge Regression technique definitely should be considered for any future work.

A.3 PARAMETERS USED

The state of ones health depends on many variables. Some of these variables are known, but the nature of others remains unknown. A further complication is that whatever variables do determine health, they do include both present and past conditions. Some variables often mentioned include:

- inherited characteristics
- personal habits such as smoking and exercising
- general physical condition
- diet
- living conditions
- occupation
- ambient air quality
- climate
- health care
- age
- sex
- race

Some of these variables may not be the causal variables at all, but rather indices of other variables which are directly important. Hence, if a variable is found to be statistically significant, there is no assurance that the variable enters directly into the mechanism for health effects.

A.3.1 Health Effects

Health effects may be short-term (acute) or long-term (chronic). The effects may range from sensory irritation to death. Lave and Freeburg (1) have considered only mortality, although Lave has said that he would have preferred to use morbidity data (5). While death is a very well-defined event, the conditions leading to death are often very complicated. Often the "immediate cause of death" will have little to do with the initial disease leading to death.

In essence then, to use only mortality data is to tell only part of the story. There are many morbidity effects which should also be evaluated. Lave understood this when he stated: "The optimal investigation would be one on morbidity rates for various respiratory and similar directly related diseases" (5).

A.3.2 Socioeconomic

Lave and Freeburg used three different socioeconomic parameters: population density, percent non-white, and percent over 65 years of age. These and other parameters have been used by a number of researchers performing similar statistical analyses. Of course any number of other factors could also be included: educational status, occupation, economic status and housing conditions, to name a few. Lave and co-workers did consider these in some of their other work, but concluded that they did not appreciably alter the estimates for the coefficients of the air pollution parameters (3, 4).

A.3.3 Air Pollution

There are many pollutants which may cause health effects. However, Lave and Freeburg have considered only two general categories: particulate matter and sulfur compounds. They have expressed each of these in three different ways: maximum, mean, and minimum concentrations. These choices were necessary for Lave and Freeburg, because no other data existed for other pollutants of possible relevance. However, this condition has changed since 1960 and now more data exists for many cities in America. Certainly any future work should consider using more specifically defined air pollution parameters. The following could be considered:

- chemical composition of particulate matter, i.e., sulfates, nitrates, heavy metals, benzo(a)pyrene
- oxides of nitrogen
- ozone

A.3.4 Other Variables

It may be impossible to identify all of the variables which affect human health. This poses a problem in that no matter how many variables are included, still an important factor may be left out. A good correlation does not prove causality. For example a parameter that is included in the regression analysis may be highly correlated with a "causing variable" not included in the analysis. Thus although a low correlation may indicate that important factors are not being considered, a high correlation does not necessarily mean that the correct ones have been included.

A.4 DATA USED

A statistical analysis cannot be any better than the data used. Lave and Freeburg used the data which was available to them and it varied both in quantity and quality. All of the data was taken for reasons other than the use Lave and Freeburg had in mind. Thus the eventual success of Lave and Freeburg's work is related directly to this problem. There is no doubt that the correlations would have been better if there had been greater accuracy in the data used.

A.4.1 Health Data

Lave and Freeburg used the mortality data reported in Reference 14. These data gave the best correlations with the independent parameters using "total mortality" as the dependent health parameter. Lave also ran regression analyses for disease-specific mortalities, such as cancer, bronchitis, asthma, pneumonia, influenza, hypertension, and heart-diseases (5). He was less successful with these which may have been due to the errors in reporting the causes of death.

Even using total mortality data has its problems. People migrate in response to health problems and also take steps to lessen their exposure to air pollution. They might move to less polluted areas or install air cleaning devices in their homes.

While the total death rate has the virtue of being quite accurate, it has the disadvantage of being so aggregate that many important specifics cannot be considered. An analysis of age specific death rates and specific causes would be of interest. The use of total mortality data does not give allowance for either of these.

A.4.2 Socioeconomic Data

Lave and Freeburg obtained their socioeconomic data from Reference 15. Such variables are reported by the census taken decennially, i.e. 1950, 1960, etc. There is not a more detailed source of data available. However, if this information is to be extrapolated to other years then errors may occur. For example, population changes are to some extent dependent upon employment conditions. Lave and Freeburg avoided this problem by using 1960 data. They therefore did not have to make any estimates. Lave and Seskin (2) did, however, have this problem for some of their work.

A.4.3 Air Pollution Data

Lave and Freeburg used air quality data which they obtained from the U.S. government. While most of the data was for the year 1960, a part of it was taken in 1959. This was done whenever insufficient data existed for 1960 or considerable problems had occurred in taking measurements at a station during 1960. The present reviewers did not obtain and study the original data.

Obtaining reliable air quality data is always a problem. The data used by Lave and Freeburg was obtained at only one measuring station in each city, forcing an assumption that the air quality was the same throughout the city. Logic and experience show that this is not the case. Furthermore, in performing their regression analyses, Lave and Freeburg used minimum sulfate concentration. Since this was a biweekly measurement, they had at most 26 measurements, from which they selected the minimum value. Such a procedure uses a single sample, taken over a 24-hour period, to estimate the minimum concentration of sulfates present during the year. As will be discussed later, the situation is actually made even worse, because the statistical analysis method used current air quality as a measure of past exposure! As Lave and Freeburg themselves admit, all of this adds up to a rather "heroic" assumption.

A final comment should be made regarding the measurement methods and the location of the air monitoring stations in the cities. The pollution parameter Lave and Freeburg call "sulfates" is actually a measure of the amount of oxides of sulfur, mainly sulfur dioxide, present in the air. It should not be confused with the particulate sulfates which would be determined by performing a chemical analysis of the suspended particulate matter collected. During the late 1950's and the early 1960's measurements for sulfur dioxide were just being implemented. There were a number of different measurement methods and many people were having sampling and analysis problems. Many of these problems have now been solved with improved technology and standardization, but the uncertainties of the 1960 data used by Lave and Freeburg still remain.

The criteria for locating air monitoring stations have not been well-defined. Many stations were located where people thought they might find air pollution. Others were located

where it was most convenient to service and keep the vandals from destroying them. It is doubtful that any of the stations were located with the intent of providing data for air pollution health effects studies.

A.5 RESULTS

The regression relationship developed by Lave and Freeburg (1) does a remarkable job of explaining the variation in mortality. Lave and Seskin (3) also included the effect of occupation in the analysis and were then able to explain 92 percent of the variation. However, even in this work the relative importance of the air pollution variables remained the same.

Neither Lave and Freeburg's equation, nor all of the other work performed by Lave and his co-workers (References 1 - 8) prove that suspended particulate matter and sulfates are causality variables to mortality. Lave notes that this does not matter because it may still show the nature of the relationship, if it is casual. If the relationship between these variables and the "true causality variables" remained unchanged then this equation could still be useable. This, of course, is what Lave and Freeburg assumed, but proving the above "if" is just another uncertainty.

It is interesting to note that Lave and Freeburg did not find any significant interaction effect between particulates and sulfates. Yet there have been more than a dozen reports on such synergism, that is, the effect of sulfur dioxide increasing by three to four fold in the presence of particulate matter (9). But this paradox is the nature of such statistical analyses. This effect may be true and have been confounded with other variables, or the effect may occur with respect to morbidity, but not mortality, or it may be that another set of data might show such an interaction term to be significant.

When examining the results, we must once again remind ourselves of the assumptions Lave and Freeburg either chose or were forced to accept. Their correlations relate total mortality to a number of variables. The statistical model, however, assumes that the variables are measured without error. As previously discussed, this is a rather poor assumption for much of the data collected and used in this work. The mortality rates that Lave and Freeburg used undoubtedly reflected also chronic effects. If exposure to a certain pollutant may cause cancer, it will still take a long time for the cancer to develop. The latency period may be tens of years, and the risk of cancer from earlier exposure is not removed by moving a person to a pollutant-free location. This means that cancer mortality rates may be related to exposures to the pollutants many years earlier, rather than the pollution situation at the time of death. Hence, the air pollution data was assumed to be representative of a long term exposure level. Other researchers have attempted to avoid this problem by using exposure data that is calculated from emission inventories by using dispersion models (10, 17).

A.6 APPLICATIONS

In applying Lave and Freeburg's work to other countries and situations, both the method and the results must be considered. The general method used by Lave and Freeburg could be adapted to Norwegian conditions. The main problems would be the same ones faced by Lave and Freeburg. Does the proper data exist and how reliable is it? We have not investigated either of these points. We should also mention that there is no reason why radiation parameters could not be added as relevant variables. This is feasible at least from a statistical analysis point of view.

It is, however, doubtful that the specific expression developed by Lave and Freeburg can be directly applied in Norway. Due to the homogeneity of the Norwegian population, the "percent non-white" parameter would probably not be a significant one. But this may not be a true causal variable anyway. Freeman (16) has shown that non-whites reside in city areas with higher pollution levels than those where whites reside. Thus, in the absence of adequate air quality data, the socioeconomic variables (such as race and income) tend to become more significant. This could mean that the association of air pollution and health effects is being underestimated.

REFERENCES

- (1) Lave, L.B.,
Freeburg, L.C. Health Effects of Electricity
Generation from Coal, Oil and
Nuclear Fuel.
Nuclear Safety, 14, 409-429 (1973).
- (2) Lave, L.B.,
Seskin, E.P. An Analysis of the Association
Between U. S. Mortality and
Air Pollution.
J. Am. Stat. Assoc., 68, 284-290
(1973).
- (3) Lave, L.B.,
Seskin, E.P. Health and Air Pollution: The
Effect of Occupation Mix.
Swedish J. Econ., 73, 76-95 (1971).
- (4) Lave, L.B. Air Pollution, Climate, and Home
Heating: Their Effects on U. S.
Mortality Rates.
Am. J. of Publ. Health,
62, 909-916 (1972).
- (5) Lave, L.B. Air Pollution Damage: Some
Difficulties in Estimating the
Value of Abatement.
In: *Environmental Quality Analysis*,
A. Kneese and B. Bower (editors).
Johns Hopkins University Press
Baltimore, MD 1972, p. 213-242.
- (6) Lave, L.B.,
Seskin, E.P. Air Pollution and Human Health.
Science, 169, 723-733 (1970).
- (7) Lave, L.B.,
Economic Implications of Trace
Contaminants in the Air.
65th Annual Meeting of American
Institute of Chemical Engineers,
New York, November 1972.
- (8) Lave, L.B.,
Seskin, E.P. Does Air Pollution Shorten Lives?
Proceedings of the Second
Research Conference of the
Inter-University Committee on
Urban Economics, University of
Chicago, September 10-11, 1970.

- (9) Air Quality Criteria for Sulfur Oxides.
U.S. Department of Health,
Education and Welfare,
Publication AP-50,
Washington, D.C. 1969.
- (10) McDonald, G.C.,
Schwing, R.C. Instabilities of Regression
Estimates Relating Air
Pollution to Mortality.
Technometrics, 15, 463-481 (1973).
- (11) Hoerl, A.E.,
Kennard, R.W. Ridge Regression: Biased
Estimation for Non-orthogonal
Problems.
Technometrics, 12, 55-67 (1970).
- (12) Hoerl, A.E.,
Kennard, R.W. Ridge Regression: Applications
to Nonorthogonal Problems.
Technometrics, 12, 69-82 (1970).
- (13) Reinke, W.A. Multivariate and Dynamic Air
Pollution Models.
Arch. Environ. Health, 18, 481-484
(1969).
- (14) Vital Statistics of the United
States, 1960.
U.S. Department of Health,
Education, and Welfare.
U.S. Government Printing
Office 1973, Washington, D.C.
- (15) Country and City Data Book.
U.S. Bureau of Census,
U.S. Government Printing
Office 1972, Washington, D.C.
- (16) Freeman III, A.M. Distribution of Environmental
Quality.
In: *Environmental Quality Analysis*,
A. Kneese and B Bower (editors).
Johns Hopkins University Press
Baltimore, MD 1972, p. 243-280.
- (17) Health Consequences of Sulfur
Oxides: A Report from CHESS,
1970-1971.
U.S. Environmental Protection
Agency, Research Triangle
Park, N.C., May 1974.