

The following articles have been adapted from selected papers
drawn from presentations
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THE RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

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Comparative Risks from Different Energy Systems: Evolution of the Methods of Studies

by L.D. Hamilton

This conference has emphasized the difficulty in estimating energy-entailed health risks engendered by the differences between *objective* measures of risk — the estimated real or actual risk of a process — and the *subjective* perception of these risks. The subjective perception of risk colours the thinking of most decision-makers (including governments and their regulatory bodies), likewise the public. This confusion (apart from any fundamental psychological processes that may be involved) stems from the following:

1. Assessment of the health effects from different energy systems has had only brief and sparsely supported investigations to date, e.g., only seven years ago did government agencies in the USA evince interest in assessing their health effects. Less than seven years ago was there funding for work at Brookhaven.
2. Assessment differs from research. Assessing the health and environmental costs of energy production and use requires scrutiny of various diverse areas of research. Research is needed to define the pollutants emitted by various stages in various fuel cycles, including end-use, and to trace, then quantitate their chemical transformations, transport, including chemical and biological conversion through air, water, food, finally reaching man and important animals and crops.

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RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Epidemiological studies on man and economic species are needed to assess pollution damage. Also needed are laboratories to elucidate the biochemical mechanisms of biological damage. From these epidemiological and mechanisms studies one needs to derive mechanistically reasonable dose-effect relationships so as to provide assessors with usable dose-response damage functions. Health and environmental assessment thus distills the gist of knowledge quantitatively along with estimates of uncertainty and spotlighting areas of ignorance of the health and environmental cost of energy production and use.

Assessment by itself is not research but rather a state-of-the-art distillation of information of known and unknowns in release of pollutants from fuel cycles, their transport and transformation through air, water, food, etc. to man, and thus to their potential damage to health and environment — which leads to the third difficulty.

3. In assessment, because of the state of knowledge (or lack of it), one frequently must make do with, say, only 60 per cent certainty rather than with the 95 per cent significance level that one strives to achieve from research. This statistical gap itself attracts special interest groups, e.g., governments and utilities, who, from the earliest beginnings of comparative assessments, have for one or another reason exploited confusion between reality and perception of risk.

The difficulty encountered between understanding the reality of risk and perception of risk, reflects the short time serious studies have been going on, the sparseness of support, the slow realization that assessment must cope with uncertainty, and that special-interest groups have been bold in taking advantage of this befogged problem. The skewed comprehensions of risk by the public are due far more to inadequacy in availability of disinterested comparisons of health effects, and to their inadequate dissemination to the public than to any intrinsic psychological quirks. What I wrote in 1972 alas still holds true today:

“Much has been made of the public’s perception of damage. This has been especially true in the tremendous increases in the public’s appreciation of the biological costs associated with the production and utilization of energy and especially with the radiation hazard. While there is little evidence that this new awareness has diminished the demand for energy, it is increasing the difficulty government agencies and utilities are finding in approving and constructing facilities to meet this demand. The problem is that the public has not been given clear-cut estimates of risk and that information about biological damage is not readily available. Where quantitative information is on hand, albeit with considerably reservation — as recently holds true for radiation — there has been inadequate dissemination and understanding of the information.” Ref. [1].

STAGE I: IN COMPARING HEALTH DAMAGE FROM DIFFERENT ENERGY SYSTEMS

In developing methods to compare health damage of different energy systems, Stage I was the partial fuel-cycle approach: health damages of electrical power generation were compared with other risks. Radiation and assessment of risks from a nuclear power plant provide the classical example of this approach. Radiation unlike many of the environ-

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 1. Estimated Annual Tissue Absorbed Dose from Natural Sources in "Normal" Areas. Ref. [2]

Source of irradiation	Annual tissue absorbed dose (mrad)			
	Gonads	Lung	Bone-lining cells	Red bone marrow
External irradiation				
Cosmic rays ionizing component	28	28	28	28
neutron component	0.35	0.35	0.35	0.35
Terrestrial radiation (γ)	32	32	32	32
Internal irradiation				
Cosmogenic radionuclides				
³ H(β)	0.001	0.001	0.001	0.001
⁷ Be(γ)	—	0.002	—	—
¹⁴ C(β)	0.5	0.6	2.0	2.2
²² Na(β+γ)	0.02	0.02	0.02	0.02
Primordial radionuclides				
⁴⁰ K(β+γ)	15	17	15	27
⁸⁷ Rb(β)	0.8	0.4	0.9	0.4
²³⁸ U- ²³⁴ U(α)	0.04	0.04	0.3	0.07
²³⁰ Th(α)	0.004	0.04	0.8	0.05
²²⁶ Ra- ²¹⁴ Po(α)	0.03	0.03	0.7	0.1
²¹⁰ Pb- ²¹⁰ Po(α+β)	0.6	0.3	3.4	0.9
²²² Rn- ²¹⁴ Po(α) inhalation	0.2	30	0.3	0.3
²³² Th(α)	0.004	0.04	0.7	0.04
²²⁸ Ra- ²⁰⁸ Tl(α)	0.06	0.06	1.1	0.2
²²⁰ Rn- ²⁰⁸ Tl(α) inhalation	0.008	4	0.1	0.1
Total (rounded)	78	110	86	92
Fraction of absorbed doses delivered by alpha particles or neutrons (%)				
	1.2	31	8.5	2.1

mental pollutants can be accurately measured down to very low doses. The background of radiation from natural sources (Table 1) must be taken into consideration in assessing the risk of damage to be accrued from an additional source. One such additional world-wide source of radiation has been fall-out from nuclear tests. The doses to be accumulated by the tissues at risk from all nuclear tests carried out before 1971 to the year 2000 — the dose commitments — can be compared with those from natural background. They are roughly equivalent to the radiation given by two years' natural background.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 2. Probable Dose from Nuclear Power Station (Ref. [3])

	mrem/year
At boundary of station	5
Population within 6 miles of station	0.6
Eating fish and shellfish 50 g/day caught at exit of condenser discharge canal	2.5
Swimming 3 h/day 120 day/yr in effluent condenser discharge canal	0.03
Swimming 3 h/day 120 day/yr at shore areas either side nuclear power station	0.006
Average Natural Background in USA	105

Note: Probable whole-body doses from a nuclear power station doses calculated for Millstone Point, Connecticut — a station containing two reactors about 1300 MW electrical — on the north shore of Long Island Sound Doses at the boundary of the station and to the population within 6 miles are doses accumulated by individuals remaining for 24 hours/day and 365 days/year at the boundary or in the open within 6 miles of the station

Table 2 gives the probable whole-body dose from a typical nuclear power plant on a sea shore. In the United States the upper limit of the dose on the boundary of nuclear power stations is now limited to at most 5 mrem/year*. Since nuclear power stations are point-sources of radiation, this limits the dose to the population within 6 miles of nuclear power plants to ~0.6 mrem/year and, within 50 miles to ~0.01 mrem/year.

Assuming that the linear relationship between dose of radiation and tumor induction observed at high doses and high-dose rates is extrapolatable down to the very low doses that would be given at low-dose rates by nuclear power plants, one can calculate the number of cancers expected in a population after radiation exposure. These risk estimates can then be used for comparisons with other causes of death. Table 3 gives the risk of death per year for an individual for accidental death, 57 out of 100 000; death in a motor vehicle, 26.7 out of 100 000; death from heart disease, 364 out of 100 000; death from cancer, 157 out of 100 000, death from all spontaneous leukaemias, 67 out of 1 000 000**.

As seen from this Table, autos are 1000 times each year riskier than death from leukaemia under the uppermost-limits of radiation after 35 years' exposure from the nuclear power station. After 35 years exposure, the risk of death from leukaemia under the average

* Appendix 1 to Chapter 10 of the Code of Federal Regulation, Part 50, covering licensing of nuclear power plants

** These figures are from the 1970 "Statistical Abstract of the United States of America".

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 3. Risk of Death per Year for an Individual (Ref. [3])

		Risk	Probability
Accidental Death	57	out of 100 000	0.00057
Death in Motor Vehicle	26.7	out of 100 000	0.00027
Death from Heart Disease	364	out of 100 000	0.00364
Death from Cancer	157	out of 100 000	0.00157
Death from all Spontaneous Leukaemias	67	out of 1 million	0.000067
Leukaemia after 1000 mrem WBR*	1	out of 1 million	0.000001
Leukaemia after natural rad. (35 years)	3.7	out of 1 million	0.0000037
Leukaemia after power reactors (35 years at boundary site eat fish and swimming)	2.6	out of 10 million	0.00000026
Leukaemia after power reactors (35 years — general population within 6 miles of site)	2.1	out of 100 million	0.000000021

Note: Risk of death per year for an individual in the United States (data from 1970 "Statistical Abstract of the United States") Risk of leukaemia, given below the line, represents an additional risk of death to be added to that from all spontaneous leukaemias. Similarly, risk of death from all additional cancers that would be about 5 times of cancer 157 out of 100 000

* Whole-body radiation.

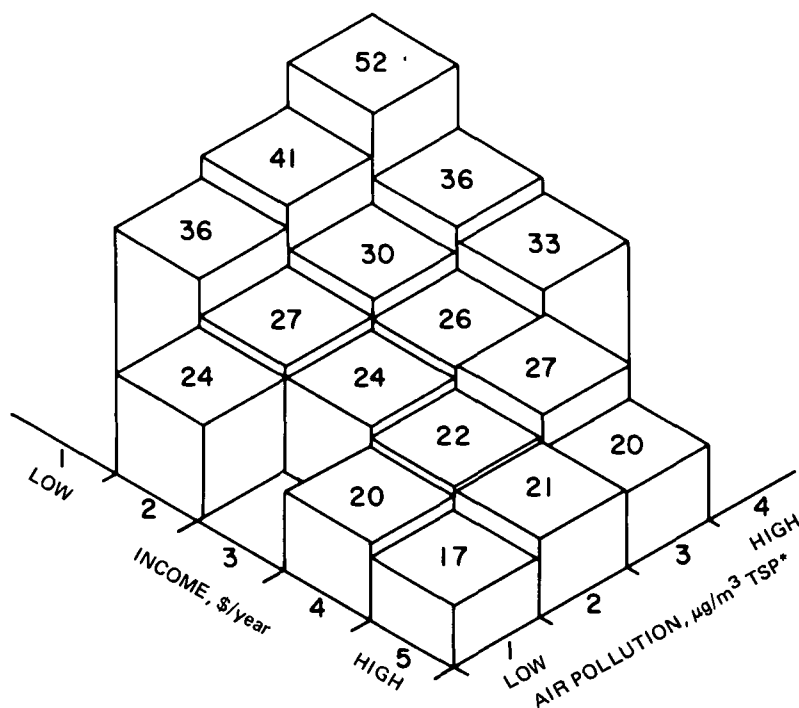
conditions of the population within six miles of the plant, i.e. the population at the greatest risk, is one ten-thousandth that of being slain by auto, i.e. after 35 years one would have had 350 000 times greater chance of being killed in a motor vehicle than of developing leukaemia. Since the total induction of all tumors from radiation is ~5 times that of the incidence of leukaemia, the total risk of dying from cancer at those doses is then, still infinitesimal. Moreover, even taking into account that the foetus may be approximately ten times more susceptible to the oncogenic effects of radiation than the average member of the population, one appreciates that the foetus normally remains in utero for less than one year. Thus, the risk to the foetus will be ~1/3 that calculated for the general population after a 35-year exposure.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

STAGE II: THE FUEL CYCLE APPROACH STANDARDIZED TO A 1000-MW(e) POWER-PLANT YEAR

The next development in methods for comparing the health effects of different energy sources was the fuel-cycle approach standardized to a standardized unit of production or production rate, e.g., a 1000-MW(e) power-plant year.

Most steps in energy generation and use entail unique biomedical, environmental, and other costs, some direct (e.g., risks of injury or death in mining), some indirect (e.g., release of pollutants into air, water, those into food chains, etc.). For comparisons of the health effects of technologies producing the same form of energy, e.g., electricity, a standardized unit of production can be used. When comparisons must extend across technologies producing different energy forms, e.g. coal-electric vs. coal-gasification vs. coal-liquefaction, the quantitative comparison is not always easy. Indeed there may not be a totally satisfactory basis. Streams of electricity, gas, or oil with the same energy content are not really equal: they are used by the consumer with different efficiencies and often for different purposes. Again, this difficulty can be largely overcome by examining



* TSP = Total suspended particulates.

Figure 1. Mortality rate deaths/1000/yr males aged 50-69 Ref. [6].

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

different technological choices with the framework of the entire energy system meeting all demands.

In analyzing the health effects of the fossil-fuel cycle the greatest uncertainty was in the relationship between air pollution from fossil-fuel combustion and ill health Ref. [4]. The Biomedical and Environmental Assessment Division (BEAD) at Brookhaven set about assessing the health damage from air pollution from fossil-fuel combustion in two ways (Ref. [5]). First we completely re-analyzed data in the literature on the association between air pollution and ill health. Secondly we organized the raw mortality, pollution, and economic data de novo and made an independent quantitative analysis of the linkage between fossil-fuel combustion, air pollution, and ill health.

Re-analysis of the Winkelstein Data

Winkelstein and associates at the State University of New York at Buffalo, later at the University of California at Berkeley, extensively studied the effects of air pollution, TSP (total suspended particulates), on mortality in Buffalo during 1959–1961 Ref. [6]. They restricted their study to white males and females aged over fifty. Each census tract (an area of about ten city blocks) in Buffalo was put into one of 20 groups on the basis of the average pollution level and the median family income of that tract. Education level, occupation, and housing were examined as possible contributors to damage. For each economic level, areas with more pollution had noticeably higher mortality (Fig.1).

Winkelstein felt that the data were not good enough to justify more elaborate analysis. Decision-makers need assessment now; delayed decision can be costly; decision often cannot be delayed. For assessment purposes now the Biomedical and Environmental Assessment Division nevertheless carried out a more elaborate analysis of the Buffalo data to validate specific comparisons of the results from several air-pollution studies Ref. [7]. Statistical analysis using multiple regression was applied. This required that each of the groups of census tracts had its income and pollution level specified.

The Brookhaven group used the midpoint of the intervals defining the pollution and income groupings as the pollution and income measurements. Indirect age adjustments (Table 4) within Winkelstein's broad age-groupings were done to rule out that the observed damages were due to differences in age structure. The analysis thereupon revealed that mortality rates were more closely correlated with both air-pollution level and economic level than could be attributed to chance. Increased median income went with lower mortality rates (Table 4). The fitted equation was:

$$\text{SMR} = 0.51 + (0.6 \pm 0.08) E + (0.007 \pm 0.001) P$$

where:

SMR = the ratio of the observed death rate to the death rate which would have occurred if the average United States pattern held;

E = 10 000/family income; and

P = the total suspended particulates in $\mu\text{g}/\text{m}^3$.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 4. Indirectly Adjusted Mortality Ratios from Winkelstein Showing Increasing Ratios with Higher Pollution and Decreasing Ratios with Higher Income in Both White Males and Females (Ref. [7])

Groupings of Increasing Median Family Income	Particulate Pollution Level — Increasing Pollution			
	— Buffalo, White Males aged 50–69 (1959–61)			
	(1)	(2)	(3)	(4)
(1)	—	1.57	1.67	2.11
(2)	1.03	1.15	1.28	1.53
(3)	—	1.04	1.17	1.43
(4)	0.89	0.98	1.19	—
(5)	0.79	0.94	0.94	—
	— Buffalo, White Females aged 50–69 (1959–61)			
(1)	—	1.13	1.40	2.34
(2)	0.92	0.97	1.29	1.49
(3)	—	0.98	1.24	1.39
(4)	1.04	0.84	1.15	—
(5)	0.88	0.92	0.69	—

Since the base mortality rate in the Buffalo data was 200 deaths per 10^4 (males and females aged 50 to 69), the effect of an increase in TSP of $1 \mu\text{g}/\text{m}^3$ on total mortality in this group is $200 \times 0.007 = 1.4$ deaths per 10^4 . The 95 per cent confidence intervals of this estimate are 1.0 to 1.8. A non-linear model was also used to analyse the Buffalo data Ref. [7].

Re-analysis of the Lave-Seskin Data

Lave and Seskin, economists at Carnegie-Mellon University, used multiple regression to study the association of mortality rates in large cities in the United States Standard Metropolitan Statistical Areas (SMSAs) and pollution levels, age distribution, and socio-economic variables. Their cross-section factors were optimal. Using just the average sulfate increment, the damage associated with the incremental effect of additional sulfate air pollution was estimated as:

$$\text{MR} = 3.3 (\Delta \text{ave. SO}_4)$$

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

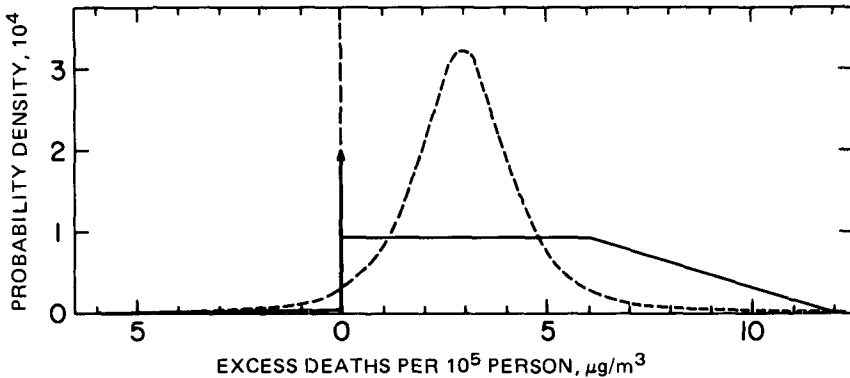


Figure 2 BEAD probability density functions representing the uncertainty in the slope of the assumed linear damage function. The solid curve is the subjective distribution. The dashed curve is the classical estimate (student's t distribution with three degrees of freedom) obtained by treating the results of four regression studies as independent observations of the same slope Ref. [8].

where:

MR = the mortality rate; and
 $\Delta_{ave.}$ = change from average level.

The effect of sulfates used, then, was:

3.3 deaths/year/ 10^5 per $\mu\text{g}/\text{m}^3$ increase in sulfates (Ref. [7]).

BEAD Probabilistic Analysis

To mass all uncertainties underlying the quantitative relationship between operation of a coal-fired power plant and health damage, Morgan and Morris made a comprehensive probabilistic analysis Ref. [8]. Sulfur air pollution transport, dispersion, and impact were modelled from a 1000-MW(e) coal power plant to the population within an 80-km radius. A gaussian plume dispersion model with linear sulfur chemistry was used with a linear health-damage function. Important features of the model involving uncertainty were: fraction sulfur emitted as SO_4 , SO_2 loss rate, SO_4 loss rate, SO_2 to SO_4 conversion rate, health-damage function, the meteorological model. The uncertainty in each of these variables was characterized by a probability density function based on available scientific judgement. This provides not only the "estimate" but the probability that a variable's actual value may be given as higher or lower than the "estimate". Essentially, these distributions define the odds a knowledgeable scientist might set if asked to bet on the outcome of a series of definite experiments which would, at some future time, determine the true value of the variables. These probability density functions (pdfs) were then combined in a simulation analysis to produce estimates of population exposure, premature deaths (Fig.2), and person-years lost.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

There are several advantages in characterizing uncertainty as pdfs based on best scientific judgement over the use of classical statistical methods on available data:

1. Often the best data come from studies having — often unavoidably — serious flaws, e.g., design problems, unaccounted-for variables, etc. Classical statistical methods cannot clearly interpret results from such studies.
2. Available data are often based on a subset of the total possible universe that may not be representative, e.g., epidemiological studies on certain population subgroups, air chemistry studies on days when the plane can fly, etc.
3. The need to combine results from various kinds of studies, e.g., taking laboratory studies as well as those conducted in the “real world” into account. Extrapolation from laboratory results demands more than classical statistics can give.

What important uncertainties were not quantified? The model itself and the form of the parameters were assumed to be known. Both may be quite different from that assumed. This was not explicitly dealt with and adds an uncertainty beyond that characterized. For example, a linear damage function is assumed and no uncertainty due to possible alternative damage function shape is considered.

The Definition of the Health-Damage Function

The health-damage function used links annual average sulfate exposure to increased annual mortality rate. It does not represent the acute effects of episodes but the long-term impact on the population of a continuing environmental exposure. Although the sequence of events leading to this impact on the population is unknown, long-term exposure to air pollution, particularly in childhood, presumably increases susceptibility to respiratory infection. A history of repeated respiratory infection, possibly coupled with continued air pollution exposure, increases the prevalence of chronic respiratory disease. This leads to more deaths from a broad range of cardio-pulmonary diseases. Thus increase in air-pollution exposure degrades population health; this is eventually reflected in mortality rate. Deaths attributable to an air-pollution exposure in a given year do not necessarily occur that same year, but are distributed over the lifetime of the exposed population. We cannot yet estimate how these deaths are distributed in time. Mortality estimates not only represent premature deaths, but years of decreased respiratory function, perhaps disability before death. Since not all induced respiratory diseases may result in premature death, the annual incidence of new-disease cases is undoubtedly higher than the annual number of deaths. Since the health-damage function is based on annual mortality rates, each death attributed to air pollution represents at least one year of life lost. Reasonable estimates of the age distribution of the deaths leads to the conclusion that 5 to 15 years lost per attributed death are likely.

Under steady-state conditions, the deaths occurring over future years attributable to pollution exposure this year equals the number of deaths occurring this year, due to the summated pollution exposure of all previous years. Based partly on this, a linear health-damage function was drawn from cross-sectional studies as a simplified way to estimate

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

effects of alternative energy strategies. By this simplified linear damage function, incremental sulfate exposure this year inevitably increases health damage and premature deaths. The incremental health damage is proportional to incremental sulfate exposure and is independent of the total sulfate exposure under the linear assumption. These estimated premature deaths may be compared with total deaths annually in the exposed population; this imparts perspective to the estimates. This fraction might be taken as a rough estimate of the eventual contribution to mortality of a continuing air-pollution exposure of that level; it is not the fraction of total deaths attributable to this level of exposure occurring in the same year.

It seems doubtful that the damage function is truly linear with no threshold over the entire range of exposure. More likely, at low absolute levels of exposure, the health impact of a unit increase in sulfate is reduced. There may be a threshold below which there is no detectable health damage. The data on which the dose-response function is based are from urban areas with generally high background sulfate levels but, significantly, the linear function is consistent with data from urban and rural areas with high- and low-pollution levels. Use of a linear function to estimate effects of small changes in sulfate levels in areas with high background levels seems reasonable. Estimates of the effects of substantial changes in background levels, or of small changes in areas with low initial background levels, increase the uncertainty in estimation of damage.

The health damage function described by Morgan and Morris ranges from 0 to 12 deaths/10⁵ per year per $\mu\text{g}/\text{m}^3$ sulfate, with a median value of 3.7 (95 per cent confidence interval 0–11.5). These estimates were derived by analysis of data principally from correlation studies of the type conducted by Lave and Seskin, and are beset by methodological and data problems discussed in detail elsewhere. Standing alone, these studies are inadequate to ascribe the observed effect to sulfate air pollution. In concert with toxicological and epidemiological studies, however, they provide a useful means of estimating the magnitude of the damage.

BEAD's Own Original Analysis of Air Pollution and Ill-health

At the same time as our re-analysis of literature data on the relation between air pollution and ill-health, we developed our own data base for this purpose. This includes the total mortality records in the United States for the years 1969, 1970, and 1971 from the National Center for Health Statistics. This file has about 2 million deaths a year; one is analysing 6 million deaths, totalling the entire 3141 counties of the United States, in contrast to the much smaller number of deaths in the Standard Metropolitan Statistical Areas (SMSA's) in the Lave and Seskin study. Moreover — again in contrast to the SMSA studies — our data include the entire urban and rural portions of the United States. We have also used the 1970 Census Data as a source of some socio-economic variables, especially income. By including three years' total US mortality, and studying people exposed to a wider range of air pollution, one can be that much more confident of the significance of the effects observed.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

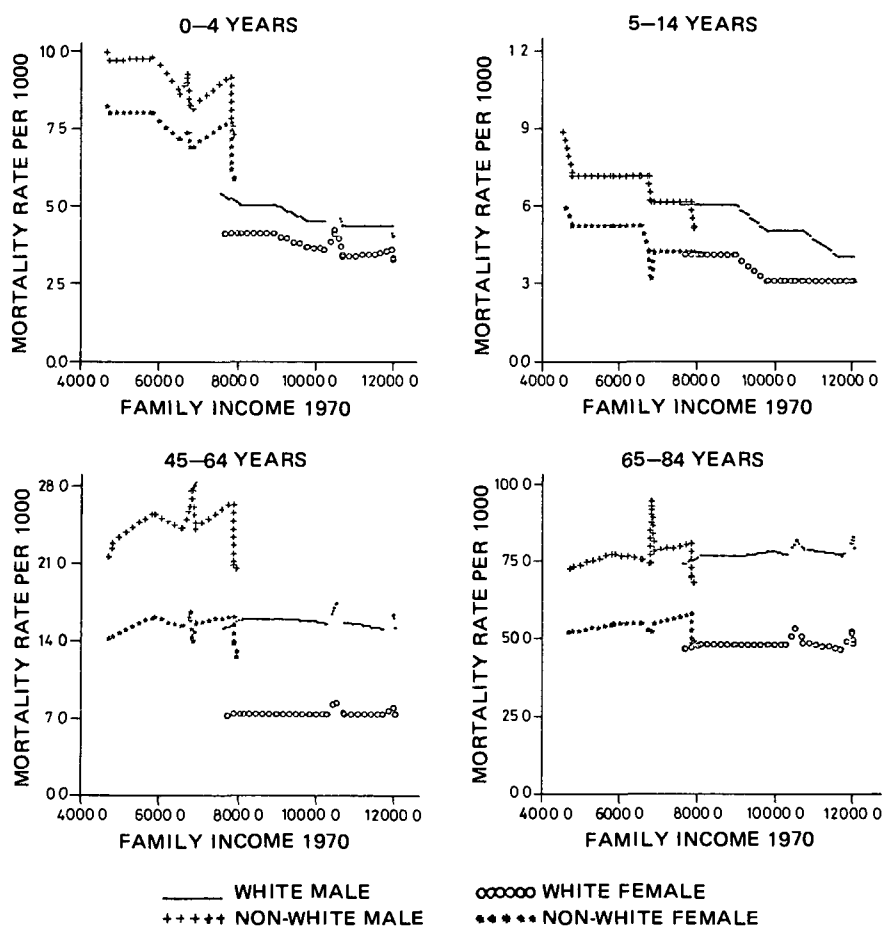


Figure 3. Sex-, race-, age-specific mortality rates and family income, 1970.

Our analysis, using these data on the relationship between air pollution and health effects, has proceeded in three stages.

— **Stage I:** all 3141 counties in the United States were aggregated into 192 groups based on 17 levels of income of the 1970 census and 21 levels of pollution expressed as emissions per square mile. Income variables were represented by race-specific, median family income data from the 1970 census. Ideally, in relating air pollution to health effects, one would like to know the actual dose to which the population was exposed. Unfortunately, the dose of air pollution to the population is not available; Lave and Seskin and other studies have used air quality data (concentration of pollutant/m³) as surrogate for dose.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

The incompleteness of the air quality data reduces their usefulness initially for a nation-wide study of health damage of air pollution. Thus, at this stage in our analysis, we have used estimated emissions as surrogate for dose. This variable varies by five orders of magnitude over urban and rural regions in the United States. Thus, the "pollution" variable was represented by the decimal logarithm of the calculated sulfur emissions (SO_x) in tons per square mile for 1970. This logarithmic transformation of emission has a more normal distribution than the raw estimates and was therefore preferred.

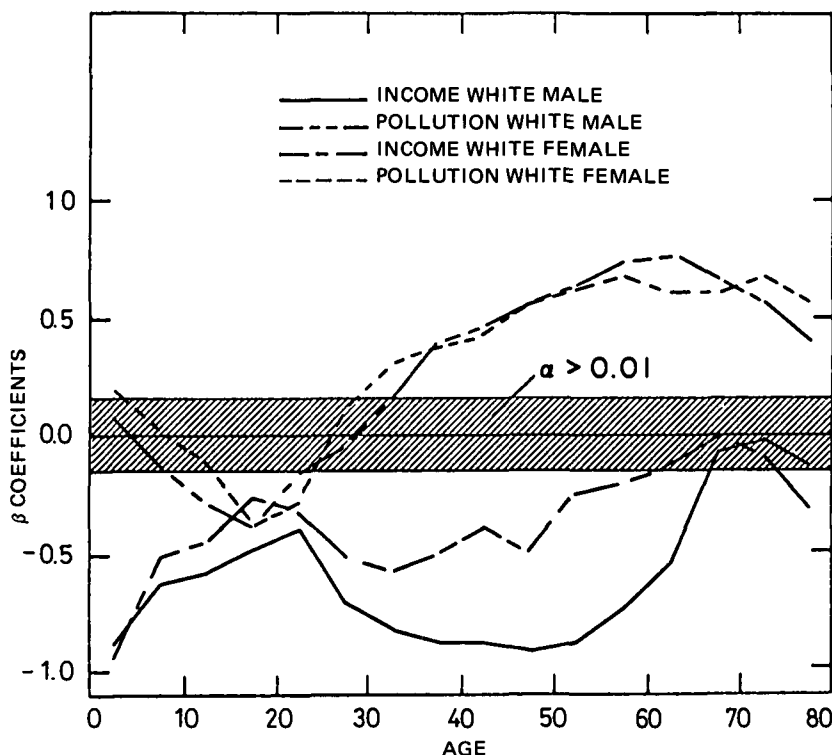
The mortality variable was represented by age-, race-, sex-, and cause-specific mortality rates for 1969–1971. The multivariable statistical techniques (multiple regression and path analysis) used provided distinct estimates of the relationship of income level and pollution to mortality. The effects of pollution and income were observed by age cohorts because it was then possible to compute age-, race-, and sex-specific relationships that address the issue of cost in terms of reduction of lifespan — not simply total attributable deaths Ref. [9].

A striking feature of the analysis of the relationship of family income to mortality Ref. [10] is seen in Fig.3. The average family income in the 3141 counties for non-whites is less than the average given for whites. This is why we have concentrated in deriving our damage function from data on whites only. One cannot include the non-whites: the impact of income on non-white mortality is overwhelming. In the 0–4, 5–14, as well as 45–54 age groups there is a striking effect of family income on mortality rate with a notable sex difference in all age groups applicable to non-whites and whites. In the 65–84 age group, as one might expect, the effect of income has levelled off, although the differences in rates between males and females are still apparent.

Figure 4 shows the relationship of income and pollution to mortality for white males and females at all ages. The X-axis contains midpoints of five-year age cohorts. The beta coefficient values for income and pollution as predictors of mortality are graphed on the Y-axis. The beta coefficients — a standardized regression coefficient in a multiple regression equation — quantify the strength of the relation between mortality at each age and the indicated variable (income or pollution). A positive coefficient indicates that increase in the variable goes with increased mortality at that age, while a negative coefficient indicates that increase in the variable goes with decreased mortality at the age. Values near 1, or –1, typically represent high association (the beta coefficient may exceed 1 in absolute value, so that beta coefficients should not be interpreted as correlation coefficients). The cross-hatched area indicates where the coefficient is not statistically different from zero Ref. [11].

Income generally tends to be negatively associated with mortality with increasing age. This is particularly evident for white males, in whom income maintains — and in fact increases — protection against mortality with advancing age. Pollution for both sexes becomes more strongly associated with mortality as age advances. The only exception is the 0–4 cohort where a positive association with pollution is suspected. This finding suggests that pollution may be especially damaging to infants, as measured by their mortality, and deaths of the very young. Research is needed.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES



Note: Points plotted at the mid point of each 5 year age interval.

Figure 4. The combined effects of income and pollution by age for white males and females, total mortality.

Comparison of our calculated excess deaths with other research shows striking similarities Ref. [10]. Winkelstein et al. Ref. [6] attributed approximately 14 deaths/100 000/ $\mu\text{g TSP}^*/\text{m}^3$. Lave and Seskin Ref. [12] calculated an order of magnitude lower estimates of 0.9 deaths/100 000/ $\mu\text{g TSP}^*/\text{m}^3$ for white males aged 55–74. Since both estimates are based on a measure of TSP rather than SO_4 , certain transformations of our data were necessary for comparison. The issue of pollution equivalence was a difficult problem. Recent studies have estimated that the SO_4 part of TSP varies by a wide range Refs. [13, 14]. By assuming 25 to 80 per cent to be a reasonable conversion range, we can convert our excess death estimates to figures comparable with the Winkelstein and Lave and Seskin numbers. By a completely different approach, Morgan et al. Ref. [8] also calculated excess deaths, however, since their estimates are based on an SO_4 measure of pollution, no modification beyond differentiating between the relative proportion of SO_2 and SO_4 in SO_x was necessary. A modified version of the Finch and Morris Ref. [7] comparison of excess deaths is found in Table 5.

* Total suspended particulates

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 5. Comparison of Attributable Pollution Death Estimates by Various Researchers

Researchers	Excess Deaths (death per $10^5/\mu\text{g}/\text{m}^3$ pollution type)	95% Confidence Limit	
		Minimum	Maximum
Winkelstein (1967)*			
white males and females aged 50–69 $\mu\text{g}/\text{TSP}/\text{m}^3$	14.00	10.00	18.00
Bozzo et al.			
white males and females aged 50–69			
Assumption: 25% SO_4/TSP	2.29	0.91	3.67
80% SO_4/TSP	7.34	2.91	11.76
Lave and Seskin (1977)**			
white males and females aged 45–64 $\mu\text{g}/\text{TSP}/\text{m}^3$	0.90	0.40	1.40
Lave and Seskin (1977)			
white males aged 45–64 ($\mu\text{g SO}_4/\text{m}^3$)	4.41	–5.80	14.60
white females aged 45–64 ($\mu\text{g SO}_4/\text{m}^3$)	8.10	2.20	14.00
Bozzo et al.			
white males and females aged 45–64			
Assumption: 25% SO_4/TSP	1.68	0.68	2.69
80% SO_4/TSP	5.38	2.16	8.59
Lave and Seskin (1977)			
All ages, white male	4.80	–0.50	10.10
All ages, white female	9.35	4.50	14.20
Morgan et al. (1977)			
all ages ($\mu\text{g SO}_4/\text{m}^3$)	3.71***	0.00	11.47
Bozzo et al. (1977)			
all ages ($\mu\text{g SO}_4/\text{m}^3$)	3.56	1.16	5.96

* As calculated by Finch and Morris from Winkelstein's data.

** Personal communication to S. Finch (in Finch and Morris Ref.[7]).

*** Median value.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

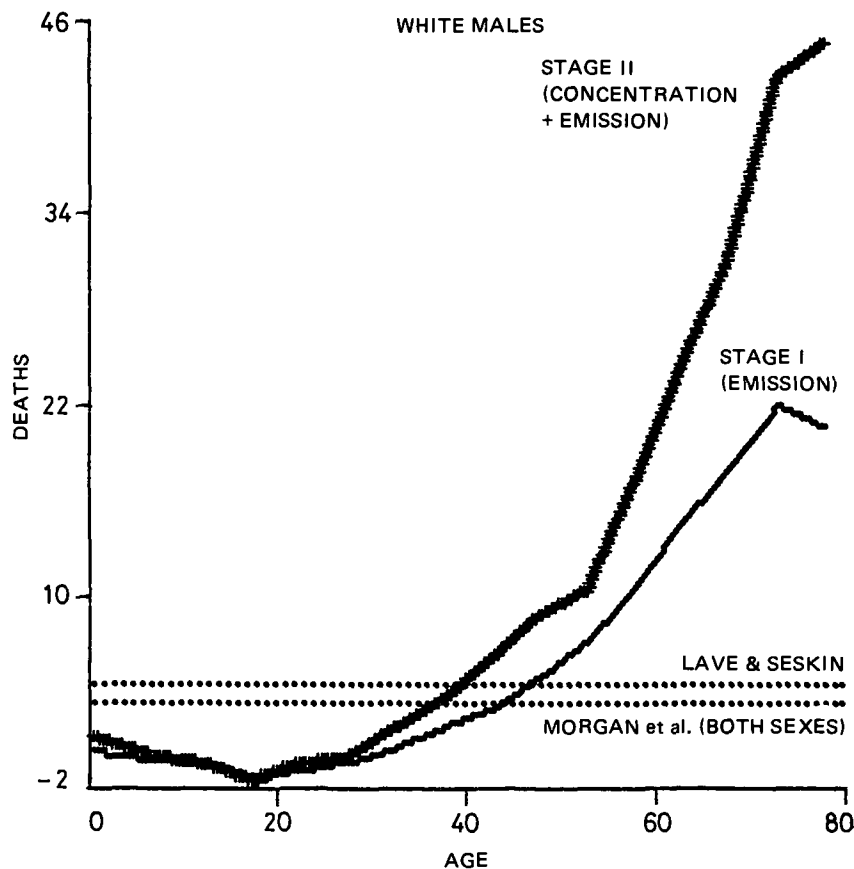


Figure 5. Total deaths associated with SO_4 exposure (deaths/100 000/ $\mu\text{g}/\text{SO}_4$ /year), US 1969–71 white males. [Stage I: Analysis based on emissions data. Stage II. Analysis based on air quality and related emissions data. Note the average values given by Lave and Seskin and the results of the probabilistic study from Morgan et al. for both sexes at all ages.]

— **Stage II:** Since emissions are not an ideal criterion of air quality in Stage II of our analysis, we have used the air-quality data available in 1970 from the 248 EPA** air-quality measurements for three pollutant species: SO_2 , SO_4 , and total suspended particulates (TSP). As we have already noted, these 248 measuring stations fall far short of covering all 3141 counties in the United States — the county was the unit of analysis in Stage I of our analysis; and while major urban areas are monitored by one or more measuring stations, rural areas are sketchily monitored. In an attempt to surmount this problem, i.e. that only 221 counties had one or more EPA air-quality measuring stations in 1970, we have

** Environmental Protection Agency.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

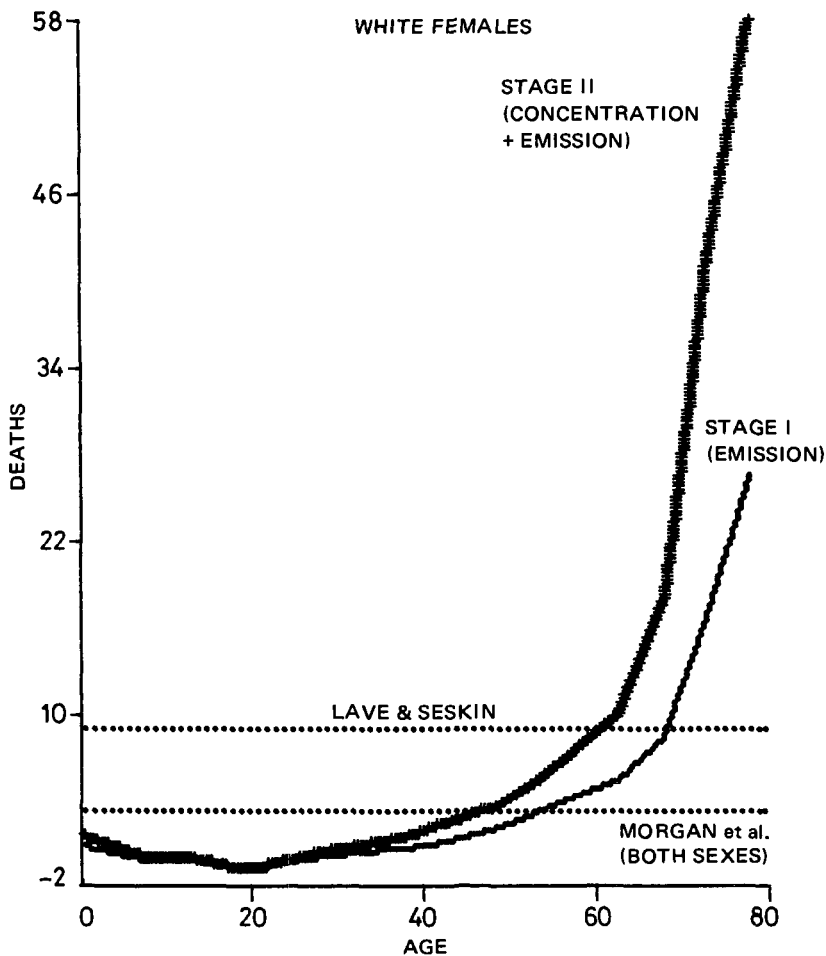


Figure 6. Total deaths associated with SO_4 exposure (deaths/100 000/ $\mu\text{g}/\text{SO}_4$ /year), US 1969–71 females. Stage I, Stage II: Ibid. Fig. 5. Note the average values given by Lave and Seskin and the results of the probabilistic study from Morgan et al. for both sexes at all ages.

assumed that each EPA measuring station provides good estimates of the pollution level for all those counties which have similar emission and socio-economic characteristics. For this purpose we have used the original 192 groups in the first stage of our analysis to aggregate our data to give a population size large enough to calculate statistically significant mortality rates Ref. [15].

Of these original 192 groups, 92 county groups contained at least one EPA measuring station. In 44 of these 92 county groups, 50 per cent or more of the population reside in

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

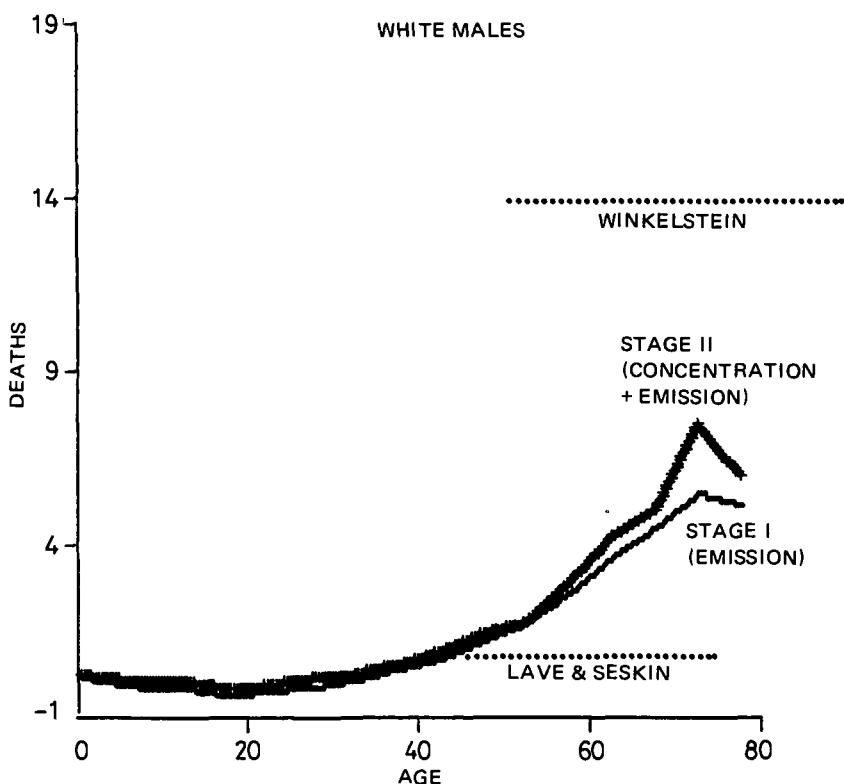


Figure 7. Total deaths associated with total suspended particulates exposure (deaths/100 000/ $\mu\text{g}/\text{TSP}/\text{year}$), US 1969–1971 white males. Stage I, Stage II: Ibid. Fig. 5. For comparison note values given by Winkelstein for age 50–69 and Lave and Seskin for age 45–64.

counties containing one or more measuring station. Unfortunately, these 44 groups are more representative of polluted urban areas in the US and caution must be exercised when extrapolating these results to the more sparsely represented rural populations. Nevertheless, as will be seen from Figures 5–7, there is, in general, good agreement between the age-specific damage function derived from the emission-mortality analysis from stage I of our analysis and that obtained by using EPA measuring stations and grouping emissions in counties without measuring stations with these, stage II of our analysis. The data agree reasonably with those derived by and from Lave and Seskin, Morgan et al., and Mendelsohn and Orcutt Ref. [16] for SO_4 , and with Winkelstein and Lave and Seskin for TSP.

Mendelsohn and Orcutt Ref. [16] give an age-specific damage function for white males 45–64 of 16.7 per 10^5 per $\mu\text{g} \text{SO}_4/\text{m}^3$. The corresponding damage functions from

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

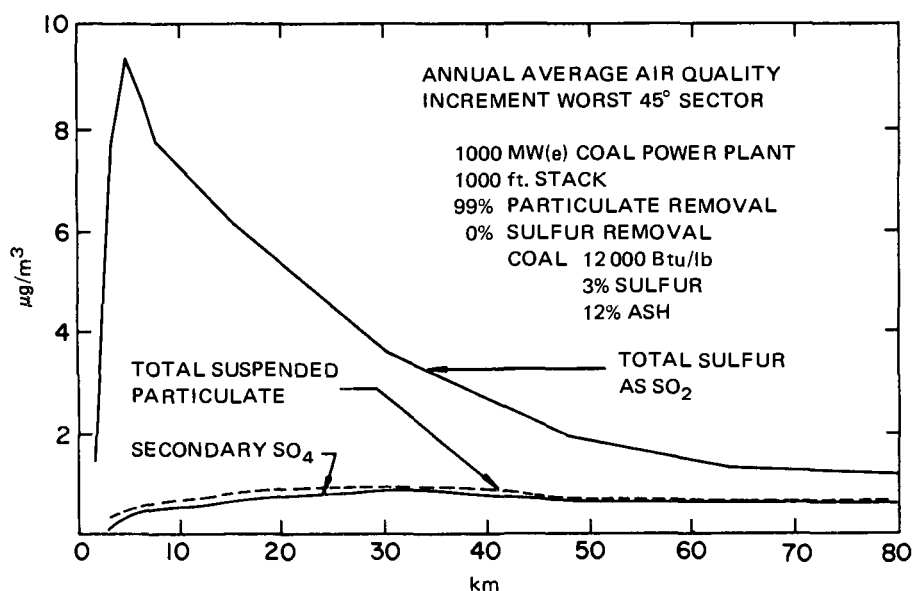


Figure 8. Distribution of SO_2 , total suspended particulates, and secondary SO_4 in the 45° worst sector within an 80-km radius of a standard 1000-MW(e) fossil-fueled electrical power plant using a wind-rose dispersion model with a constant SO_2 - SO_4 conversion rate Ref. [17].

Stage I and Stage II of our analyses are 9.1 and 14.4 respectively. Similarly for white males aged 65 and more their figure is 82.2; our corresponding values from Stage I and Stage II are 20.1 and 38.1. Their damage function for females 45–64 is 8.0 per 10^5 per $\mu\text{g SO}_4/\text{m}^3$; the corresponding values from Stage I and Stage II of our analyses are 3.7 and 6.2. Similarly for white females aged 65 and more their figure is 56.9; our corresponding values from Stage I and Stage II are 16.7 and 37.6. These comparisons confirm the striking similarity of our calculated age-specific damage functions with those derived from other epidemiological studies. The difference in the damage function for SO_4 exposure derived from Stage I and Stage II in age groups over 60 may be related to differences in the size of the populations included in each stage, and the under-representation of the rural population in Stage II.

BEAD's Standard 1000-MW(e) Plant

It is sited on a plain, and air pollution emission rates are determined assuming plant, fuel characteristics, and emission-control devices. Initially Ref. [17] we used a distribution of wind velocity and atmospheric stability representing a composite of atmospheric conditions around nuclear power plants Ref. [18] and then meteorological data from the Pittsburgh International Airport Ref. [8]. Initially population distributions in the 80-km radius

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

**Table 6. Excess Mortality due to Air Pollution Exposure from 1000-MW(e)
Fossil Fuel Power Plant within 80 km (305-m stack height, 65% capacity factor)**

	Number of annual excess deaths			
	Multi-city Studies ¹			Buffalo ¹
	Lower 10%	Median	Upper 10%	Linear
Eastern high-sulfur coal				
(2.9×10^7 J/kg coal, 3% sulfur)				
(1.25×10^4 Btu/lb)*				
No sulfur removal				
3×10^6 people within 80 km	0	17	87	113
0.7×10^6 people within 80 km	0	4	20	27
90% sulfur removal				
3×10^6 people	0	1.7	9	11
0.7×10^6 people	0	0.4	2	2.7
Eastern low-sulfur coal				
(2.9×10^7 J/kg, 0.4% sulfur)				
(1.2×10^4 Btu/lb)				
No sulfur removal				
3×10^6 people	0	2.3	11	16
0.7×10^6 people	0	0.5	2.7	3.5
Montana coal				
(2.1×10^7 J/kg, 0.8% sulfur)				
(8.6×10^3 Btu/lb)				
No sulfur removal				
3×10^6 people	0	6.3	32	42
0.7×10^6 people	0	1.5	7.4	9.5
High-sulfur oil				
(4.6×10^7 J/kg, 2.5% sulfur)				
(2×10^4 Btu/lb)				
No sulfur removal				
3×10^6 people	0	9	45	61
0.7×10^7 people	0	2.1	10.6	14
Low-sulfur oil				
(4.6×10^7 J/kg, 0.2% sulfur)				
(2×10^4 Btu/lb)				
No sulfur removal				
3×10^6 people	0	0.7	3.6	4.8
0.7×10^7 people	0	0.2	0.9	1.1

¹ Derived from re-analysis of data of Lave and Winkelstein.

* Btu/lb = British thermal unit/pound.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 7. Coal Fuel Cycle Effects Summary (per 1000 MW(e) plant-year, 65% capacity)*

	Deaths	Disease/Injury
Mining¹		
Public	—	—
Workers		
Accidental Injury ²	0.6	42
Occupational Disease	0.02–0.4	0.5–1.0
Processing		
Public		
Workers		
Accidental Injury	0.05	2.9
Occupational Disease	—	—
Transport³		
Public and Workers		
Accidental Injury	0.3–1.3	1.2–5.9
Electricity Generation		
Public		
Air Pollution (50-mile radius) ⁴	0.6 (0–3)	Not estimated
Air Pollution (total US) ⁵	6 (0–30)	Not estimated
Workers		
Accidental Injury ⁶	0.1 (0.02–0.3)	3.3 (2.7–4.0)
TOTAL	7.7–9.1	

¹ Assumes 62% underground, 38% surface mining (the ratio of Appalachian coal production) (Ref. [21a])

² Coal miners accidental (non-fatal) injury (1965–73 men)

Underground mining — 27.6 injuries per 10⁶ tons

Surface mining — 5.2 injuries per 10⁶ tons

$[(27.6 \times 0.62) + (5.2 \times 0.38)] \times 2.2 \times 10^6 = 42$ injuries per plant-year (Ref. [21b]).

³ Assumes rail transport, 300-mile trips. Range is due to different methods of estimation.

⁴ Assumes 3 million people within 50-mile radius, sulfur oxide emission rate of 0.12 lbs SO₂ per 10⁶ Btu input (low-sulfur coal combined with 90% removal of sulfur in flue gas). Results are approximately linear for SO₂ emissions.

⁵ Assumes total effect 10X local effect.

⁶ Estimates from Bertolett and Fox, with Poisson 95% confidence limits

* A 1000 MW(e) power plant operating with an average capacity factor of 65% produces 0.65 GWy, or 2.05 10¹⁶ J, or 1.94 10¹³ Btu in a year.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 8. Nuclear Fuel Cycle Effects Summary (per 1000 MW(e) plant-year, 65% capacity)*

	Deaths	Disease/Injury
Mining		
Public	0.08	0.08
Workers		
Radiation Induced Cancer	0.06	0.03
Non-Radiation Induced		
Occupational Disease	0.07	0.14–2.8**
Occupational Accidents	0.31	11.96
SUBTOTAL	0.52	12.21–14.87
Processing		
Public	0.002	0.002
Workers		
Radiation Induced Cancer	0.034	0.034
Occupational Accidents	0.004	1.3
SUBTOTAL	0.04	1.34
Electricity Generation		
Routine Public	0.017	0.017
Workers		
Radiation Induced Cancer	0.07	0.07
Occupational Accidents	0.013	1.13
Catastrophic Accidents	0.1	—
SUBTOTAL	0.20	1.217
Waste Management		
Public	5.1×10^{-5}	5.1×10^{-5}
Workers	7.45×10^{-4}	7.45×10^{-4}
SUBTOTAL	7.96×10^{-4}	7.96×10^{-4}
Transport		
Routine Public	6.1×10^{-4}	6.1×10^{-4}
Workers		
Radiation Induced Cancer	8.5×10^{-4}	8.5×10^{-4}
Occupational Accidents	0.01	0.1
Catastrophic Accidents		
Cancers	8.3×10^{-5} to 7.1×10^{-4}	8.3×10^{-5} to 7.1×10^{-4}
Prompt Deaths	2.1×10^{-7} to 9.3×10^{-5}	
SUBTOTAL	0.01	0.10

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 8 (cont.)

	Deaths	Disease/Injury
Decommissioning		
Public	5.3×10^{-9}	5.3×10^{-9}
Workers		
Radiation Induced Cancer	4.2×10^{-3}	4.2×10^{-3}
Occupational Accidents	8.0×10^{-4}	0.07
SUBTOTAL	5×10^{-3}	0.07
TOTAL	0.77	14.9–17.6

* A 1000 MW(e) power plant operating with an average capacity factor of 65% produces 0.65 GWy, or 2.05×10^{16} J, or 1.94×10^{13} Btu in a year

** Based on ratio of occupational disease/death in coal miners. Lower estimate is used in total

were taken from average populations around existing nuclear power stations Refs. [17, 18] and then within 80-km of the first three of four plant locations in the Pittsburgh area Ref. [8]. The input components of the air pollution are detailed elsewhere Ref. [8]. A small percentage of the sulfur was assumed to be converted to sulfate in the plant; this was represented as a subjective probability with a median value of 1.5 per cent, and the remainder emitted as SO₂. A wind-rose meteorological dispersion model, coupled with an air chemistry model based on a linear (varying with distance) SO₂-SO₄ conversion rate, was used to determine ground-level exposures within an 80-km radius around the power plant (see Figure 8 for early distribution of SO₂, TSP, and secondary SO₄). Use of a linear (varying with distance) SO₂-SO₄ conversion rate is the only atmospheric chemistry incorporated in the analysis at this time.

Use of only one index of pollution — sulfates — is undoubtedly an oversimple way of indexing the health hazard of air pollution. Finally, because of lack of knowledge of the exposure-response curve, particularly at low levels of air pollution, we have assumed a linear dose-effect relationship as is common in estimating radiation risks. As we are considering the effects of small increments on background levels of air pollution close to clinically effective doses, the error in this assumption, as noted, is likely to be less than that involved in extrapolating from high to low doses of radiation. Even were the exposure-response curve not linear, the levels which we are considering probably do not fall far outside the linear portion of the curve.

Table 6 shows the increased mortality within 80-km to be expected from various technological and population alternatives due to air pollution from a 1000-MW(e) fossil-fuel fired power plant. The nominal plant was assumed to have a 305 m stack, a stack exit diameter of 8.2 m, a stack-gas exit velocity of 16 m/sec, and exit temperature of 135°C. Possible additional effects from other pollutants are not considered, nor are the larger effects beyond the 80-km radius in this Table.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Tables 7 and 8 summarize our current estimates of the health effects on a unit-plant basis, assuming currently mandated environmental controls Ref. [19]. Table 7 shows our current estimates of the health effects of the coal fuel cycle on a unit-plant basis. Most of our attention has been directed to quantifying coal-mining accidents and occupational disease, coal transport accidents, and air pollution from coal combustion. Table 8 shows a summary of the nuclear fuel cycle effects on a unit-plant basis.

Only light-water reactors have been thoroughly evaluated for risk associated with catastrophic accidents. Reactors, however, probably account for most of the potential impact of major nuclear accidents in the uranium fuel cycle since they represent 80 per cent of the facilities and, were an accident to occur, the health impact of an accident at a reactor is likely to be much larger than of an accident at other types of facilities in the uranium fuel cycle.

On an actuarial basis, the Reactor Safety Study Ref. [20] estimated reactor accidents contribute only 0.02 deaths per GWy(e). Since almost all of the health effect calculated from even the worst hypothesized accident is attributable to low doses over a large population, the Reactor Safety Study applied dose and dose-rate factors. Converting back to the upper bound estimate yields 0.1 deaths per GWy(e). The use of this figure in Table 8 goes some way towards meeting the greater uncertainty in the estimates suggested by the Ad Hoc Risk Assessment Review Group, Chairman, H.W. Lewis Ref. [22]. Both tables represent effects that would be expected from good practice and full controls in plants to be built.

STAGE III: MULTIPLYING THE STANDARDIZED 1000-MW(e) POWER-PLANT YEAR (80-km pollution model)

Data similar to those in Tables 7 and 8 from earlier fuel cycle analyses, including oil and gas and reflecting current practice, with public health effects of electricity generation calculated only to populations within 80-km of plants, were used to calculate the total health effects associated with the production of electric power in the USA in 1975 Ref. [23]. These data are summarized in Table 9.

The estimated health effects in 1975 associated with a total fuel cycle standardized to produce 10^{10} kWh electric power (approximately the annual output of two 1000-MW(e) plants) are:

- from coal: deaths 20–300, disabilities 100–300;
- from gas: deaths 0–2, disabilities 20;
- from nuclear: deaths 1–3, disabilities 7–40.

For overall perspective, the approximate annual total deaths in USA $\approx 2 \times 10^6$; the per cent associated with electricity production is 0.1–1. Approximate deaths in USA ages 1–74 is 1.1×10^6 ; the per cent associated with electricity production is 0.2–1.9. One may compare this with ~ 17 per cent of deaths associated with smoking, about ~ 2.5 per cent associated with car accidents half of which were due to drunken drivers, ~ 5 per cent due to iatrogenic lethality.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 9. Estimated Health Effects in 1975 Associated with Production of Electric Power

Fuel	1975 kWh(e) $\times 10^9$ ¹	Equivalent number of 1000-MW(e) plants	Estimated deaths	Estimated disabilities
Coal	844	128	1 900–15 000	25 000–39 000
Oil	292	44	88–4 400	4 000–7 900
Gas	297	45	6	600
Nuclear	168	26	18–42	130–470
Totals	1604	243	2 000–19 000	29 000–48 000

¹ Preliminary.
[Data from Electrical World, 185(6), 54 (1976)]

STAGE IV: HEALTH EFFECTS OF RENEWABLE SOURCES OF ENERGY

The reference energy system framework described above* is widely used for analyses of health damage from conventional energy technologies, but its use is more limited for analysing technologies which have no fuel supply impacts, e.g. some renewable sources of energy such as photovoltaics. To meet this need several have examined the material system impacts of renewable sources of energy Refs. [24–28]. A uniform framework in which to prepare consistent analysis remains to be developed.

Elements that need to be specified for the framework to produce estimates of occupational health and safety costs and environmental residuals production and hence estimates of health damage to the public include: (i) the end-use material demands; (ii) efficiency coefficients for all processes, i.e., the ratio of material output: material input; (iii) standardized labour productivity estimates for all processes, e.g., that amount of labour required to mine a unit of material e.g., 1 ton of copper, (iv) occupational health and safety coefficients by process; and (v) environmental emission coefficients by process. Specification of a network structure and quantities (i) to (v) suffice to generate the occupational health and safety risks and environmental residuals produced in developing a renewable technology.

Using material system impacts Inhaber Refs. [25, 26] made quantitative estimates of worker-days lost (WDL) or public person-days lost (PDL) for six non-conventional energy technologies: methanol, ocean thermal, photovoltaic, solar space-heating, solar thermal electric, and

* Stage II: The Fuel Cycle Approach Standardized to a 1000-MW(e) Power-plant Year.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

wind, and compared them with estimated prepared for five existing technologies: coal, natural gas, nuclear, oil, and hydro. Health risks were quantified by correlating technology specific material labour requirements with occupational health statistics to estimate occupational effects, and by estimating air pollutant emissions and material transport impacts on the public to estimate public health damage. For comparison of effects from new with those from existing technologies, Inhaber claimed that his analysis included not only the primary fuel cycles, but the entire cycles for producing energy, including material gathering, transport, fabrication, construction, operation and maintenance, decommissioning, and disposal.

While this may be so for his analysis of the new technologies, it is immediately apparent that his analyses of existing technologies were necessarily more incomplete. Nevertheless, Inhaber concluded that the risk of human health from non-conventional energy sources could be as high, or even higher, than that from existing conventional sources. The risk from new energy sources related to large labour and material requirements for their construction, and also to their need for energy back-up and storage. Inhaber believed that back-up and storage would be needed to meet electricity demands during bad weather, e.g., no wind, no sunshine. He assumed that coal-fired power plants would provide such back-up, calculated the risks from the back-up technology, and added it to the risk calculated for the new technologies, where in fact the risks from the back-up technology usually accounted for most of the risk.

Not surprisingly, the analysis of Inhaber encountered strong criticism notably from Holdren Refs. [27,28], his associates, and others Ref. [29]. Apart from many mathematical errors and conceptual flaws, e.g. types and quantities of material used, control strategies applied, pollutants emitted, and the incompleteness of the existing energy fuel cycles as compared with those of the new technologies, use of the coal-fuel cycle as required back-up accounted for most of the upper-limit values of total health risks calculated for renewable technologies. A more reasonable view would be that the near-term application of intermittent renewable electricity sources will not be as base-load plants, but rather as intermediate fuel-saving plants that would lower some of the demand for fossil-fuel generated electricity by harnessing wind and sun whenever available. This mode of operation requires neither back-up or storage. Thus the net risk of generating electricity by nonconventional technologies is the risk of building and operating the renewable technologies *minus* the higher risks of supplying and burning the amounts of fossil fuels that are actually conserved by renewable sources replacing fossil fuels.

Holdren's review and re-analysis of Inhaber's data reduces the occupational hazards of all new technologies ~6–50 times, and public health hazards by ~9–900 times Ref. [28] (Table 10). Existing conventional technologies are more damaging than the new unconventional technologies, i.e., the opposite of Inhaber's conclusions. A difficulty with all these comparisons still remains: the incompleteness of analyses of the existing cycles compared with those of the new unconventional technologies. The Biomedical and Environmental Assessment Division (BEAD) at Brookhaven has taken the first steps towards undertaking such comparisons by developing methods for assessing health damage from renewable sources of energy.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 10. Health Risks of Renewable and Existing Energy Technologies*

	Inhaber	Holdren et al.
Nuclear		
WDL	1.7–8.7	3.1–12
PDL	0.3–1.5	0.3–70
Ocean Thermal		
WDL	23–30	2.2–4.6
PDL	0.8–1.4	0.40–0.90
Solar Heating		
WDL	91–100	11–17
PDL	4.6–9.5	2.1–5.5
Methanol		
WDL	220–350	6.3–6.6
PDL	0.05–0.14	0.005–0.015
Solar-Thermal-Electric		
WDL	62–100	7.4–15
PLD	9.4–520	1.0–2.7
Photovoltaic		
WDL	140–190	5.0–14
PDL	10–510	0.9–2.2
Wind		
WDL	220–290	9.7–10
PDL	22–540	0.21–0.58
Oil		
WDL	2–18	3–19
PDL	9–1900	9–1000
Coal		
WDL	18–73	19–43
PDL	20–2000	20–1500

* Figures in worker-days lost (WDL), or public person-days lost (PDL) per megawatt-year of output, rounded to two significant figures.
[Modified from Table 1 5. in Ref. [31]]

STAGE V: HEALTH AND ENVIRONMENTAL RISK ACCOUNTING: USE OF INPUT-OUTPUT ANALYSIS

The risk accounting method of analysis of health and environmental impacts, under development by the Biomedical and Environmental Assessment Division (BEAD) of the National Center for Analysis of Energy Systems, estimates occupational health impacts

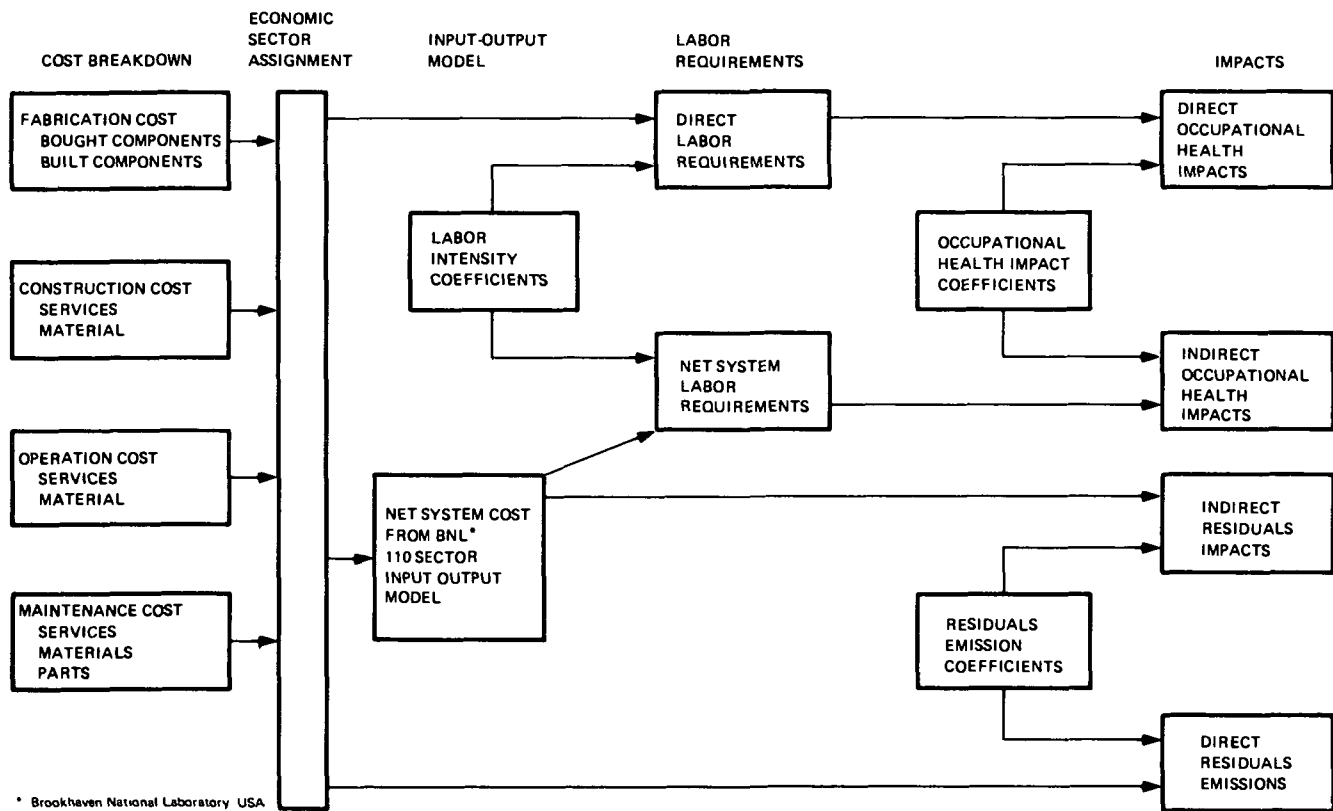


Figure 9 Health and Environmental Impact Analysis Methodology

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

and residuals emissions from fabrication, construction, operation, and maintenance of energy technologies (Fig.9). The method is based on an input-output model of the national economy, capable of tracing flows of dollars through the system and calculating net changes in production induced by demand from energy technologies for materials, goods, and services. This method has several advantages over conventional analysis based on material flows, in that the analysis is complete and not confined only to those materials used in large quantities. Analysis includes system-wide impacts as well as direct impacts of building and operating energy facilities.

An important assumption of the method is that engineering cost estimates are the most accessible, detailed, and accurate information available for most developing energy technologies. Engineering cost estimates are normally made by creating a preliminary design from components of known characteristics — usually components already in production — sufficiently similar to those needed, to provide accurate cost estimates. For many technologies in early stages of development these are the only data available having the detail and rigour required for quantitative health and environmental impact analysis.

Given disaggregation of a developing technology into components of known characteristics, direct health and environmental damages are inferred as the sum of damages from the components required to produce it. The BEAD risk accounting begins by assigning the cost of technology components, such as basic equipment, materials, bought components, construction, operation, maintenance, etc., to the Standard Industrial Classification (SIC) code most appropriate to that component (Table 11). National statistics on labour intensity by industry are used to convert dollar cost to man-hours of labour required. National occupational health statistics per man-hours by industry are used to estimate occupational accidents, illness, and fatalities (Table 12), and national residuals emission data by industry are used to calculate total emissions.

Indirect health and environmental impacts are those produced throughout the economy in response to demands for goods and services from the energy technology. If the technology requires a generator, then the industry producing the generator must buy insulated copper wire, the wire company must buy plastic for insulation, and the plastic company must buy petroleum feed stock, etc. All these industries hire employees who must buy shoes for which someone must raise cattle. Most probably several of these industries also require generators, and so the cycle repeats. Input-output models are specifically designed to describe these interconnections. The input-output model currently used in the BEAD method is a 110-sector model developed by Brookhaven National Laboratory (BNL) (Table 13).

The BEAD impact analysis assumes that construction and operation of new energy technologies creates a set of final demands for goods and services from the economic system. Even components that must be specially made during construction are assumed to be "purchased" from the existing system. On this assumption, the BNL input-output model generates the total flow of dollars throughout the system generated by fabrication, construction, operation, and maintenance of any energy technology. The total dollar

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 11. SIC* Code Assignments Wind Energy Conversion Systems

Components	Inputs	Preferred SIC Code	SIC Description
Rotor Blade	Fiberglass, epoxy laminates	3728	Aircraft parts and auxiliary equipment, not elsewhere classified Includes propellers, variable and fixed pitch
Rotor Hub	Bearings and structural steel	3568	Mechanical power transmission equipment, not elsewhere classified Includes bearings, couplings, collars and shafts
Transmission Gear	Gearbox, shafts, bearings	3566	Speed changers, industrial high speed drives, and gears
Generator	Generator, switch gear, transformer relays, controls	3621	Motors and generators Includes convertors, generators and invertors
Tower and Foundation	Structural steel, concrete and fabrication	1541	General contractors — industrial buildings and warehouses Includes prefabricated building construction
Controls	Hydraulic and electronic systems	3622	Industrial controls. Includes controls, clutches and switches Engineering, architectural and surveying services
Other	Engineering system integration, testing spare parts and land	8911	Engineering, architectural and surveying services
Operation	Operating personnel, supervisors and overhead	73	Establishments primarily engaged in rendering services, including management and consulting
Maintenance	Routine inspection maintenance, and repair of plant		
Rotor		3728	Aircraft parts and auxiliary equipment not elsewhere classified. Includes propellers, variable and fixed pitch
Power		762	Electrical repair shops, includes electronic and electrical industrial equipment
Tower		176	Roofing and sheet metal work; includes painting and repair
Site Facilities		7349	Cleaning and maintenance service to buildings

* SIC = Standard Industrial Classification

Table 12. Occupational Health Impact Assessment: Wind Energy Conversion Systems

Component	SIC code	Cost	Cost	Labour Intensity	Labour Requirements	Impact Coefficients		Occupational Health Impacts	
		1976 \$/ 1.5 MW rated capacity ¹	10 ⁶ 1976 \$/ 10 ¹² Btu Output ²	1976 \$/ Man-hour	Man-year 10 ¹² Btu ³	Illness μ Injury/ 100 man-year	Fatalities/ 1000 man-year ⁴	Illness μ Injury/ 10 ¹² Btu Output	Fatalities/ 10 ¹² Btu Output
Rotor Blade	3728	72 300	0.141	26.334	2.68	10.7	0.06	0.287	0.161 $\times 10^{-3}$
Rotor Hub	3568	129 700	0.254	27.055	4.69	13.9	0.06	0.652	0.281 $\times 10^{-3}$
Transmission	3566	191 000	0.374	27.055	6.91	15.5	0.06	1.071	0.415 $\times 10^{-3}$
Generator	3621	67 900	0.133	22.298	2.98	11.0	0.06	0.328	0.179 $\times 10^{-3}$
Tower & Foundation	1541	112 000	0.219	22.764	4.81	19.3	0.25	0.928	1.20 $\times 10^{-3}$
Controls	3622	14 400	0.028	22.298	0.628	9.6	0.06	0.0603	0.0377 $\times 10^{-3}$
Other	891	178 000	0.348	16.080	1.08	2.6	0.05	0.0281	0.0540 $\times 10^{-3}$
Operation	73	13 722/yr	0.806	16.804	24.0	4.7	0.05	1.128	0.120 $\times 10^{-3}$
Maintenance									
Rotor	3728	20 400/yr	1.20	26.334	22.8	10.7	0.06	2.44	1.37 $\times 10^{-3}$
Power	762	9 583/yr	0.563	7.612	37.0	6.9	0.05	2.55	1.85 $\times 10^{-3}$
Tower	176	695/yr	0.0408	19.033	1.07	21.4	0.25	0.229	0.268 $\times 10^{-3}$
Building & Site	7349	52/yr	0.00305	16.804	0.0908	7.2	0.05	0.00654	0.00454 $\times 10^{-3}$
TOTAL			4.11		108.7			9.71	0.00702

¹ Cost estimates are in US \$ for a 1.5 MW horizontal axis windmill with all-steel tower and foundation. Design is by Kaman Aerospace Corporation Ref [30]

² Capacity factor assume to be 0.38, which is appropriate for high wind regimes Ref [31], plant life is assumed to be 30 years. Total output is 0.511 $\times 10^{12}$ Btu of electricity.

³ One man-year is 2000 man-hour

⁴ Fatality rates are estimated by major industrial groups Ref [32]

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 13. Sectoral Classification of the BNL Energy Input-Output Model

1. Coal	34. Maintenance and repair construction, residential
2. Crude oil	35. Maintenance and repair construction, all other
Crude gas	
3. Shale Oil	
4. Methane from coal	36. Ordnance and accessories
5. Coal Liquefaction	37. Food and kindred products
6. Refined oil products	38. Tobacco manufacturers
7. Pipeline gas	39. Broad and narrow fabrics, yarn and thread mills
8. Coal combined cycle electric	40. Misc. textile good and floor coverings
9. Other fossil electric	
10. Light-water reactor — electric	41. Apparel
11. High-temperature graphite-moderated reactor — electric	42. Misc. fabricated textile products
Hydroelectric	43. Lumber and wood products, except containers
12. Geothermal	44. Wood containers
Solar Electric	45. Household furniture
Solar Direct	
	46. Other furniture and fixtures
13. Ore reduction feedstocks	47. Paper and allied products except containers and boxes
14. Chemical feedstocks	48. Paperboard containers and boxes
15. Motive power	49. Printing and publishing
16. Process heat	50. Chemicals and selected chemical products
17. Water heat	
18. Space heat	51. Plastics and synthetic materials
19. Air conditioning	52. Drugs, cleaning and toilet preparations
20. Electric power	53. Paints and allied products
	54. Paving mixtures and blocks
21. Livestock and livestock products	55. Asphalt felt and coatings
22. Other agricultural products	
23. Forestry and fishery products	56. Rubber and miscellaneous plastics products
24. Agricultural, forestry and fishery services	57. Leather tanning and industrial leather products
25. Iron and ferroalloys ores mining	58. Footwear and other leather products
	59. Glass and glass products
26. Nonferrous metal ores mining	60. Stone and clay products
27. Stone and clay mining, quarrying	61. Primary iron and steel manufacturing
28. Chemicals and fertilizer mineral mining	62. Primary nonferrous metals manufacturing
	63. Metal containers
29. New construction, residential buildings	64. Heating, plumbing and fabricated structural metal products
30. New construction, non-residential buildings	65. Screw machine prod., bolts, nuts, etc. and metal stampings
31. New construction, public utilities	
32. New construction, highways	
33. New construction, all other	

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 13 (cont.)

66. Other fabricated metal products	86. Professional, scientific and controlling inst and supp.
67. Engines and turbines	87. Optical, ophthalmic and photographic equip and supplies
68. Farm machinery	88. Miscellaneous manufacturing
69. Construction, mining, oil field machinery, equipment	89. Railroads and related services
70. Materials handling machinery and equipment	90. Local, urban and interurban highway pass. trans.
71. Metalworking machinery and equipment	91. Motor freight transportation and warehousing
72. Special industry machinery and equipment	92. Water transportation
73. General industrial machinery and equipment	93. Air transportation
74. Machine shop products	94. Pipeline transportation
75. Office, computing and accounting machines	95. Transportation services
76. Service industry machines	96. Communications except radio and television broadcasting
77. Elec. trans. and dist. eq and elec. industry apparatus	97. Radio and TV broadcasting
78. Household appliances	98. Water and sanitary services
79. Electric lighting and wiring equipment	99. Wholesale and retail trade
80. Radio, television and communications equipment	100. Finance and insurance
81. Electronic components and accessories	101. Real estate and rental
82. Miscellaneous elec. machinery, equipment and supplies	102. Hotels and lodging, pers. and repair serv., except auto repair
83. Motor vehicles and equipment	103. Business services
84. Aircraft and parts	104. Automobile repair and services
85. Other transportation equipment	105. Amusements
	106. Medical, educ. services and non-profit inst.
	107. Federal government enterprises
	108. State and local government enterprises
	109. Business travel, entertainment and gifts
	110. Office supplies.

flow by industry is then converted to estimates of systemwide occupational health and environmental impact as described above.

Tables 14 and 15 show preliminary estimates of the direct and system-wide occupational impacts of a windmill. System-wide impacts are two to three times greater than the direct impacts of fabrication, construction, operation, and maintenance of the plant.

RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

**Table 14. Direct Occupational Impacts of Wind Energy Conversion Systems
(per 10^{12} Btu output)**

Component	BNL I-O Sector	Labour (men/year)	Fatalities (10^{-4} cases)	Illness (10^{-1} cases)	Days lost
Rotor Blade	84	2.68	1.58	1.74	1.01
Rotor Hub	73	4.69	2.77	7.14	3.65
Transmission	73	6.91	4.08	10.5	5.38
Generator	77	3.28	1.94	3.18	4.98
Tower and Foundation	31	5.16	15.7	8.52	5.76
Controls	77	0.69	0.41	0.67	1.05
Other	103	77.4	23.2	37.9	27.0
Operation	103	17.9	5.38	8.79	6.26
Rotor Maintenance	84	0.02	0.01	0.01	0.01
Power Maintenance	102	0.04	0.01	0.02	0.01
Tower Maintenance	35	0.00	0.00	0.00	0.00
Building and Site Maintenance	102	0.00	0.00	0.00	0.00
Total Direct Impacts		118	55.1	78.5	55.1

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RESPECTIVE RISKS OF DIFFERENT ENERGY SOURCES

Table 15. Indirect Occupational Impacts of Wind Energy Conversion Systems
(per 10^{12} Btu output)

Component	BNL I-O Sector	Labour (men/year)	Fatalities (10^{-4} cases)	Illness (10^{-1} cases)	Days lost
Rotor Blade	84	6.19	4.63	5.42	3.48
Rotor Hub	73	10.5	8.67	14.6	9.11
Transmission	73	15.5	12.8	21.6	13.4
Generator	77	6.57	5.18	7.01	7.97
Tower and Foundation	31	10.7	21.5	16.1	11.3
Controls	77	1.38	1.09	1.48	1.68
Other	103	156	95.1	105	73.6
Operation	103	36.2	22.0	24.3	17.0
Rotor Maintenance	84	0.05	0.04	0.05	0.03
Power Maintenance	102	0.05	0.02	0.04	0.02
Tower Maintenance	35	0.00	0.00	0.00	0.00
Building and Site Maintenance	102	0.00	0.00	0.00	0.00
Total Indirect Impacts		243	171	196	138

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