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Abstract

In this paper, an attempt is made to review some of the principles that underlie physiological responses following exposure to environmental agents. The review takes the form of the comparison of the health effects from two often competing fuel cycles, nuclear and coal. In addition, efforts will be reviewed to assess the current literature of the health effects of energy systems. This review will not be comprehensive, but will rather indicate briefly those areas where there is general agreement and those where the data are madequate.

Although each individual pollutant, whether it be chemical, biological, radioactive, gaseous or solid, has its own peculiarities which requires individual consideration, there are underlying principles which are useful in evaluating and understanding all of them. It was intended, that, by focussing on these two energy systems, theses principles, their uses and abuses could be illustrated.

The Biological Basis for Standard Setting for Environmental Pollutants: A Critique

1. Introduction

The process of standard setting for environmental pollutants requires that there be some trade-offs between the often conflicting needs of economics, social welfare, preservation of the environment and health. Of these, possibly the most difficult to assess is the last. To begin with, no universally acceptable definition of health exists, nor do we know exactly how to measure health, or deviations from health. Furthermore, as techniques for measurement of chemical pollutants both in the environment and in biological tissues become more refined, more chemical species are detected in human tissues. Whether the mere *presence* of potentially toxic substances is considered hazardous is sometimes controversial, particularly when exposure to larger quantities is known to be toxic, or when effects are known to occur only after many years. For example, analyses of drinking water in the United States have recently revealed the presence of dozens of chemical species, many of which are known to be toxic to animals or humans at higher concentration. The responsible administrator knows that the costs of detecting the source of these pollutants, regulating and restricting their discharge is enormous to say nothing of the industrial costs which may be attendant upon such restrictions. When he turns to the toxicologist or public health specialist for advice, he is likely to be confronted with contradictory, confusing and often incomplete data. What is he to do?

In this paper, we attempt a review of some of the principles that underlie physiological responses following exposure to environmental agents. The review takes the form of a comparison of the health effects from two often competing fuel cycles, nuclear and coal. Although each individual pollutant, whether it be chemical, biological, radioactive, gaseous or solid, has its own peculiarities which requires individual consideration, there are certain underlying principles which are useful in

evaluating and understanding all of them. It was our hope, that, by focusing on these two energy systems, we could highlight and illustrate these principles, their uses and abuses.

Historically, technologies have been allowed to develop solely on the basis of market forces. Since market forces do not include costs for the health effects imposed on the public by such systems (the "externalities"), they could be conveniently ignored. Health effects to industrial employees have never been compensated at their full market value, but would be, in the majority of industrial activities, a small cost of production. Furthermore, even if interest in health costs had been keener, information simply was not available with which to make accurate assessment of these costs. Such evaluation requires a fairly sophisticated society with an enormous information system of vital statistics, medical skills and investigative ability. Unfortunately, necessary information and knowledge with which to make these judgements is still incomplete, but has been markedly increased only during recent decades. Furthermore, the development and availability of the computer enormously enhanced the ability to manage such vast quantities of data. These capabilities have only recently become available.

Secondly, although we have managed to get along without consideration of health or other environmental costs in the past, we cannot any longer ignore the consequences of such a policy. Trial and error alone simply will no longer do. One wonders if, had the health costs of automobile transportation been anticipated, would we have developed such a technology in the way that we have. No longer are we willing to introduce new chemical or pharmacologic agents without some consideration of possible toxic effects and prior testing. The requirements that environmental impact statements be written for Federal projects in the U.S. institutionalized the requirements for some prior thought of potential environmental consequences and alternatives of new systems. Although it has not been proven that such forethought will have benefits, that is at least the reasonable hope.

Lastly, consideration of the health and environmental effects on energy systems is urged upon us by recognition of the enormous

increase in world energy production likely in the near future. Assuming a world population of 12 billion and a per capita consumption of 5 kW, energy requirements could increase almost 10-fold in the next 100 years. Clearly, energy options must be developed in such a way as to optimize health and environmental costs as well as the economic costs of such development.

In this paper, we will review efforts to assess the current literature of the health effects of energy systems. This review will not be comprehensive, but will rather indicate briefly those areas where there is general agreement and those where the data are inadequate, with references to the literature for those who wish to pursue the issues. The literature is not extensive. Charpentier [1,2], in reviewing 159 energy models, found only 13 which incorporated some estimate of environmental damages and only two which contained a health effect evaluation.

Comprehensive study of health effects of an energy system involves several elements. The first is an estimate of the emissions or releases from various portions of the fuel cycle as shown in Fig. 1. This is followed by some estimate of the resulting dose to the population, and is in turn followed by an estimate of the health effects which result from such exposures. The first of these steps is methodologically the simplest. Within the second stage, dispersion, enormous problems are encountered. When emissions are to air, as from a stack, the variety of meteorological conditions that may occur between release and exposure, particularly when the exposed population is at some great distance, make modeling very difficult. Furthermore, during transportation interactions among chemical species may occur that are not well understood. Photochemical smog is an example of these. Emissions may also occur into waterways, resulting in contamination of drinking water, or of the food fish taken from that water. When that water is used for irrigation of agricultural land, pollutants may find their way into the human food chains. These food chains may be highly complex and unusual concentrations may occur unexpectedly. The very high concentraion of fallout radiocesium on arctic lichens and the further concentration of cesium within the tissues of reindeer for whom the lichens are a dietary

mainstay led to relatively high body burden among Laplanders for whom, in turn, the reindeer is an important nutritional source.

Estimating damage functions is possibly the most difficult of the functions shown in Fig. 1. Because an understanding of the relationship and uncertainties in environmental health depends so strongly on damage functions, the next section deals in some length with the subject of establishing dose response relationships in human populations.

The last factor shown in Fig. 1 is trauma or accidents in which injuries occur due to falls, burns, explosions etc. Note that in both the case of trauma and chemical emissions, persons occupationally involved are likely to have the greatest exposure to risk.

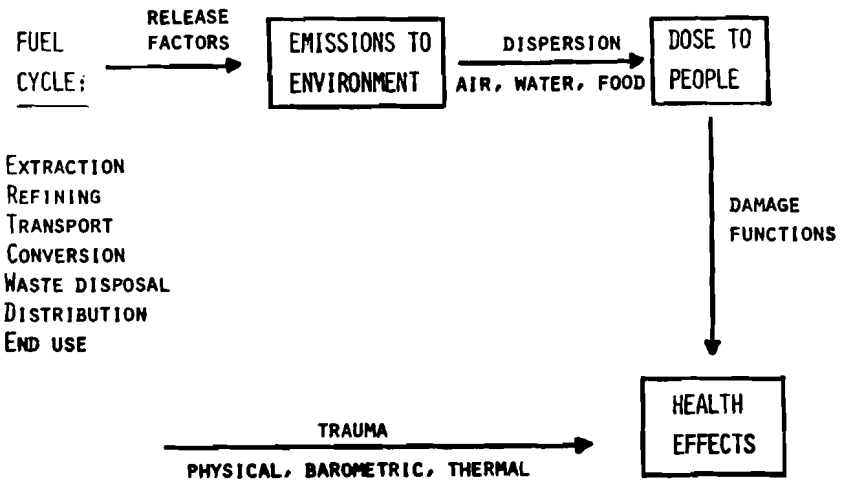

Fig. 1: Elements of Health Effects Estimation

2. Establishing Causal Relationships

Evidence of causality in disease induction derives from a number of sources including animal experimentation and human experiences, both of which are essential. Human observations include accidental exposures, therapeutic exposures, industrial or occupational experiences, and epidemiological studies. The reader should be aware of certain principles which constrain epidemiologic investigation.

2.1 Long latent period

The phrase "latent period" refers to the interval between exposure and appearance of disease. The fact that this interval may be decades enormously complicates the life of the epidemiologist. Examples of such latency are the cancers appearing in uranium miners 20 years following initial exposure, or lung cancers now appearing in asbestos workers whose industrial exposures occurred during World War II. Still another example which might not have been uncovered but for the unusual character of the response is the recent discovery of rare vaginal cancers among young women whose exposures to diethylstilbestrol occurred during intrauterine life some 20 years earlier, a drug with which their mothers had been treated during pregnancy.

Rarely are records or memories adequate to document such remote exposures. Furthermore, because of the great mobility of people, it cannot be assumed that persons residing in a certain community under study had lived there throughout the period of interest. Nor, with rapidly changing industrial practices, can it be assumed that current conditions of exposure had existed during earlier decades. One may then be currently observing effects which are totally unrelated to present environmental conditions but rather to conditions of a distant past for which no records exist. Shocking as it may seem, interest in the environment is very recent and observations of environmental indices frequently do not exist beyond a very few years.

2.2 Non-specificity response

Those diseases which occur in response to environmental exposures, unlike many infectious diseases, are in no way character-

istic, i.e. they are clinically indistinguishable from those which occur spontaneously. The leukemia which occurs in atom bomb survivors, for example, is in no known way different from leukemia which occurs in non-radiation exposed persons. To detect such effects, then, requires comparison of the frequency of disease in exposed populations with the frequency of disease in non-exposed populations. The difficulty here is that, for unknown reasons, the frequency of disease varies remarkably from one community to another, even when the populations appear to be quite similar in all known respects and where proper adjustments for age, sex, and race have been made. The problem posed for the epidemiologist then is to find comparable populations which differ only in respect to their exposure. In fact, he cannot often be certain that differences in disease are the result of exposure to a suspect agent or to some unsuspected factor.

It is of considerable benefit in this respect if the disease entity is rare or unusual. For example, the angiosarcoma of the liver now known to occur among persons working with vinyl chloride first came under suspicion when only three cases were found in a small industrial plant. The physician was aware that the probability of a cluster of three cases in such a small population on the basis of chance alone was extremely small. One of the consequences of this situation, of greater interest to lawyers than to epidemiologists, is that one can never be certain in the individual case whether the outcome is the result of a known exposure or might have occurred anyway. All that the epidemiologist can offer is some estimate of the probability that such an exposure was responsible. Legal requirements of proof have been considerably broadened in such cases.

2.3 Knowledge of dose

In order to make some estimate of the effect of exposure to an environmental agent, it is not only necessary to know *whether* such exposure has taken place, but also necessary is some estimate of the amount of exposure. In the matter of environmental pollutants, it is rare that one is comparing an exposed population with a non-exposed population, but rather it is a matter of com-

paring a more heavily exposed population with a less exposed population, and so the need to estimate dose. A number of obstacles stand in the way of dose-estimation. Some of them are as follows.

Our measures of ambient concentration may not reflect exposure to the individual. A useful example is community air pollution measurements which are typically measured at one or two monitoring stations. Such crude measurements may be totally inaccurate measures of dose for those who are indoors, or for those in distant suburban surroundings or for the traffic policeman standing on a heavily polluted intersection.

Furthermore, we cannot be certain whether it is the peak concentration reached that is important, the average daily dose, average yearly dose or the geometric mean dose. (This leads to the question of dose-rate which will be addressed below in a consideration of radiation effects.) Even this ignores the problem of the faulty measurement of the agent at interest. There is some evidence that the technology for ambient sulfate measurements was badly in error until recently, throwing older measurements into some disrepute. Recalling the above paragraphs on latent periods, it is the measurement of dose 20 years age rather than current measurements that are needed to explain today's morbidity when considering chronic disease induction.

2.4 Synergistic effects

Scientists generally prefer to examine effects of single agents on health parameters. To do so is difficult enough without attempting to unravel the enormously more complicated problems of experimental design when two interacting agents are under study, yet there is fairly good evidence that such synergistic or additive effects do occur (a synergistic effect is one in which the total effect is greater than would be expected on the basis of a summation of two independent effects). In the absence of good evidence of health effects of either SO_2 or particulates, considerable suspicion has fallen upon some interactions between these two elements of air pollution. Better examples of synergy are the established links between cigarette smoking and both uranium mining and asbestosis. The data appears to show a greater

than additive effect in the induction of lung cancer among cigarette smokers who are engaged in either of those occupations.

These examples of synergy provide warning signals for unexpected effects that might not be suspected on the basis of studies of individual agent alone, but there is still another phenomenon that further compounds the complexity of the problem and that is the possibility of interference or protective effect of one agent against another. The use of a large number of therapeutic agents are based on this principle. Although no example of interference among agents commonly thought of as pollutants is known, there has been little search for such effects which in all likelihood do exist.

3. Genetic Effects

The foregoing discussion has been fairly extensive reflecting both the authors' interest as well as the breadth of our current state of knowledge. The following discussion of genetic effects will be short for the same reason, but the subject cannot be ignored altogether.

Many agents are known to be mutagenic in animal or other test systems, yet none of these has been shown conclusively to have produced genetic effects in humans. The latter is not taken to mean that humans are peculiarly resistant to mutagenic agents, but rather that the demonstration of mutagenesis is peculiarly difficult. Unlike somatic effects, i.e. those wich occur in the animals who are themselves exposed, the manner in which genetic effects might manifest themselves are unknown. Furthermore, since the majority of mutations are of a recessive character and must occur in both mates to appear in one half of the offspring, the probabilities of demonstrating such effects are vanishingly small. There are occasional reports of cytogenetic changes in circulating lymphocytes of persons exposed to suspect mutagens, but whether or not these are viable and transmissible to offspring is not known. Something more will be said about radiation mutagenesis below.

4. In the Matter of Proof

Under these conditions of uncertainty, what guidelines are available to support a contention of a causal relationship between exposure to an environmental agent and some specific human health effects? There are three: First, that observations be replicated under a large number of conditions, thereby minimizing the likelihood that any one observation be the result of unsuspected variables. For example, the appearance of leukemia in human populations exposed to radiation in both Hiroshima and Nagasaki as well as in therapeutically radiated populations provides indisputable evidence of such an effect. Secondly, a graded dose-response relationship adds important weight to such an observation and such is the case for radiation induced leukemia. Whether or not such a relationship is linear or not is another question that needs not to be discussed here, but is not crucial to the usefulness of some form of gradation. Thirdly, supporting animal data adds strong weight to the claim of causality. Here, some judgement is necessary in extrapolating from the animal data to humans. For example, some mouse strains exposed to radiation develop ovarian cancers. Humans exposed to radiation have not been known to develop ovarian cancers; nevertheless, evidence that radiation is carcinogenic in animals strongly supports the evidence that radiation is carcinogenic in humans, even though not in precisely the same way.

5. Damage Functions

Building on the previous general introduction, we will now turn to consider two specific damage functions: those from radiation exposure, and subsequently, from the emissions from the combustion of coal.

In spite of some uncertainties, evaluation of the health effects of the nuclear power industry is a fairly straightforward operation. Simplicity arises from the fact that there is only a single toxic effluent which requires consideration, i.e. radiation, and secondly because the toxicity of radiation exposure is, although by no means complete, probably better

understood than that of any other environmental agent. In turn, reasons for this understanding are multiple, not the least of which is the very large amounts of money that became available for supporting radiation effects research at the end of World War II. Also, radiation is, for the experimentalist, a very easy tool with which to work. Dose can be easily and precisely administered and measured, facilitating animal work. Human studies involving survivors of the atomic bombings and radiation accidents, occupational exposures, and also the thousands of persons treated with radiotherapy permitted thorough analysis of human radiation toxicity. Furthermore, there has been an enormous effort by several organizations, both national and international, to carefully analyse and assess the significance of this data. Their efforts, in which they reach a surprising degree of consensus, simplifies the task greatly. That is not to say that there are no problems. The most frustrating of these problems is the question of the dose-response relationship. For those not familiar with the concept, the latter can be rephrased in the following questions: How much radiation does it take to produce an effect? Does raising or lowering the dose at all levels have a proportional effect, i.e. a linear relationship, or are there deviations from this? What happens if the dose is protracted over a longer time or is fractionated?

For radiation, much of this information is available *at high doses*. By "high" is meant from a few hundred to a few thousand times "background" or natural radiation levels. At those doses, the data is consistent with a number of dose-response models, including a proportional model in which the line passes through the origin. The implication of this interpretation would be that *any* radiation exposure, no matter how slight, would have some small but significant effect. In fact, that is the interpretation that is most widely accepted, not because it better explains or "fits" the data, but because it is considered by most (but not all) to be the most prudent assumption, prudent in the sense that it does not ignore the possibility that low doses *may* produce some effect.

A second assumption underlying the usual assessment of risk from radiation exposure is that dose *rate* has no effect. This is almost certainly an incorrect assumption. The judgement that it is in error arises from widespread animal studies which strongly support the likelihood that dose rate does have an effect [3-5]. Furthermore, evidence that human response to environmental, chemical or nutritional agents is generally influenced by dose rate reinforces the suspicion of this assumption. Consider the nature of a sudden exposure to intense sunlight or alcohol or aspirin. In any of these examples, intemperate exposure can be harmful or lethal whereas the same dose spaced over a longer period of time can be harmless or beneficial.

Why then, is such an assumption made? Again, it is a matter of prudence: Since there are no observations on cancer frequency among human populations exposed to high doses at low dose rates (no such populations exist), the high dose rate data, i.e. radiotherapy, atomic bomb survivors is used even though the populations to whom these risk estimates will be applied will be exposed at very low dose rates, thus leading to what is likely to be a gross exaggeration of risk.

Keeping in mind the above assumptions, just what are the risk estimates? Several organizations have assessed the available data and arrived at such estimates: They are, the United Nations Scientific Committee on the Effects of Ionizing Radiation, UNSCEAR, the U.S. National Academy of Sciences, and the International Committee on Radiation Protection, ICRP. The estimates of the National Academy of Sciences (BEIR Report) are as follows [5]:

(i) It is estimated that exposure of the parents to 170 mrems per year (or 5 rems over the 30 years of the usual reproduction period) would cause in the first generation between about 150 and 3,600 serious genetic disabilities per year in the U.S. population, based on 3.6 million births per year.

(ii) It is estimated that the same exposure of the U.S. population as above *could* cause from roughly 3,000 to roughly 15,000 deaths from cancer annually, with 6,000 being the most likely number. (*Could* is used in the preceding sentence because many scientists feel that as a result of the efficiency of the body's repair mechanisms at the very low dose rates involved, the true effects might approach zero production of cancer.)

The above numerical values are in essential agreement with those reported by the ICRP [3] and the UNSCEAR Committee [4]. The latter report stresses that the risk estimates are valid only for the doses at which they have been estimated (high levels), whereas the BEIR report suggests that the values are useful as upper-limit estimates in assessment of effects at low levels. A recent NCRP report [6] discusses this matter critically and concludes that the BEIR values have such a high probability of overestimating the actual risk that they are of only marginal value, if any, for purposes of realistic risk-benefit evaluation. At this time, we judge the consensus to be that the BEIR values are most likely overestimated by a considerable margin, but if used with that understanding, then there are important comparisons that can be made.

One shortcoming of these estimates is that they leave unanswered the question of the latent period between exposure and development of cancer, or to put that same issue differently, the amount of life shortening that will result. There is some evidence that latency may differ for cancer of different organ systems and may also differ for different conditions of exposure. For example, the latency prior to the development of leukemia was shorter for those persons treated radiotherapeutically to the spine for arthritis than for atomic bomb survivors.

6. Assessment of Risk from Nuclear Power

Health risks to both the general public and occupational personnel from the nuclear fuel cycle are considerably better estimated than those from fossil fuel combustion. This is because (a) there is a single causative agent released from the nuclear plant--ionizing radiation--whereas there are literally hundreds of individual species released from fossil fuel combustion; (b) since the first nuclear weapons tests in the 1940s, about a billion dollars have been spent on research on the effects of ionizing radiation; (c) radiation exposures are easily and precisely measured; and (d) there is a great body of knowledge from natural background exposure and from accidental, industrial, and military exposures of populations.

Table 1 shows the UNSCEAR estimates of man rem exposures to the public, both locally and worldwide per MW(e) per year. Typically, a roughly equivalent total exposures will occur to occupational personnel, but are not similarly distributed in the same portions of the fuel cycle. A large source of occupational exposure occurs to the underground uranium miner whose lungs are exposed to the radioactive gas, radon, and to the alpha particles of the radon "daughter products" which may absorb to ambient chest particles and deposit in the bronchi of the miners lung. Lung cancer has been demonstrated in this group, particularly in those who are also cigarette smokers.

Utilizing a "value of life" approach and the radiation risk estimate described above, a dollar cost per man rem can be calculated. For example, assigning a 300,000 dollar value to life and using the risk value of 100 cases per million man rem, simple arithmetic produces a 30 dollar per man rem value.

In addition to risks from radiation exposures discussed above, some specific issues and concerns have been raised which will be touched on briefly here.

6.1 Accidents

The basic document in regard to reactor accidents is the "Rasmussen Report" [8]. It attempts to predict the probabilities and consequences of a total spectrum of conceivable reactor acci-

Table 1: Summary of Collective Doses per Unit Energy Generated*

	Whole body [man rad/MW(e)y]				Referred organ [man rad/MW(e)y]				
A. LOCAL AND REGIONAL CONTRIBUTIONS									
Mining and milling	$3\ 10^{-3}$				Lung	$5\ 10^{-1}$			
Fuel fabrication	$1\ 10^{-4}$								
Reactor operation	PWR	BWR	GCR	HWR		PWR	BWR	GCR	HWR
1. Atmospheric pathways	$1\ 10^{-2}$	$5\ 10^{-1}$	$2\ 10^{-1}$	$6\ 10^{-2}$	Thyroid	$5\ 10^{-3}$	$5\ 10^{-1}$	$5\ 10^{-3}$	$5\ 10^{-3}$
2. Water pathways	$2\ 10^{-2}$	$3\ 10^{-2}$	$2\ 10^{-2}$	$3\ 10^{-2}$	Thyroid	$1\ 10^{-4}$	$1\ 10^{-2}$	$1\ 10^{-4}$	$1\ 10^{-4}$
Fuel reprocessing									
1. Atmospheric pathways	$5\ 10^{-3}$				Thyroid	$3\ 10^{-3}$			
2. Water pathways	$2\ 10^{-2}$				Thyroid	$6\ 10^{-2}$			
Transportation	$3\ 10^{-3}$								
B. GLOBAL CONTRIBUTIONS									
^{3}H	$1\ 10^{-1}$								
^{85}Kr	$2\ 10^{-1}$								
^{14}C	2.5								
^{129}I					Thyroid	$2\ 10^{-2}$			

*From [4].

dents. Critical reviews of this report have been made by the American Physical Society [9] and the Union of Concerned Scientists [10]. The essence of these analyses is that the Rasmussen estimates would have to be low by 3-5 orders of magnitude in order for the risks from catastrophic accidents to be comparable to those from normal operations of the coal, oil or nuclear fuel cycles. It is a matter of conjecture whether the public would accept the probability, although very small, of a single nuclear event causing an immediate loss of hundreds of lives as preferable to or in place of the loss of a large number of lives from fossil combustion occurring in driblets and therefore unnoticed.

6.2 Plutonium

Of all the radionuclides involved in the nuclear fuel cycle, plutonium has aroused the greatest public concern in regard to potential hazard. A great deal of experimental work has been done over the years on the biological effects of plutonium [11, 12]; but of course as with other toxic substances it is not possible to predict precisely the effects of low levels in the range of exposure that would produce undetectable effects.

Following is a discussion of those factors that tend to cause plutonium-239 to be hazardous, and then of those that tend to reduce its hazard. Plutonium, as any alpha-emitting radionuclide, is very biologically effective in producing cancer when it is located within the body in direct contact with living tissues. When it is inhaled it comes into direct contact with living tissue, and when it enters the blood it is deposited in such tissues as bone, liver, and lymph node; once deposited it remains for a long time during which it irradiates the tissue. Because of its long physical half-life (24,300 years) it must be regarded essentially as a permanent contaminant just as are many other stable idustrial chemicals that pollute the biosphere.

Because alpha radiation will not even penetrate the dead layer of skin, plutonium is not a hazard when it exists outside of the body. Contrary to popular conception, plutonium when swallowed remains essentially outside the body because it is extremely poorly absorbed, does not enter the bloodstream in

significant proportions, and being mixed with intestinal contents does not irradiate the surface of the intestines as it passes through the gastrointestinal tract. Plutonium does not become concentrated in the food chain. These characteristics result in large part from the low solubility of plutonium in water and biological fluids and its tendency to remain fixed in soil.

It appears that inhalation of plutonium is the most hazardous route of exposure. Because plutonium deposited in the lung may be presented as small particles, a question has been raised as to whether a given amount of such radioactivity deposited in the lung would be more hazardous if present as small particles rather then being uniformly deposited. This is presently a matter of controversy. One group of workers [13] claims on the basis of theoretical considerations that small particles would be more hazardous (hot particle theory) and therefore that existing standards, which are based on uniform distribution, should be made more stringent. Other workers and several official groups claim that experimental data support existing concepts and that there is no reason for any drastic change of standards [14-17].

The problem of malevolent use of plutonium cannot be logically assessed; this matter has been discussed by Cohen [18]. It appears that except for an unreasoning widespread public fear, terroristic purposes could be much more readily achieved by using other more easily available chemical or biological agents.

In general it can be stated that plutonium when inhaled is a toxic carcinogen and great care should be taken to prevent its access to the biosphere. Essentially none would be released from normal operation of the nuclear fuel cycle. Estimates of risks from it as a component of nuclear fuel cycles and the experience of the past 30 years indicate that they are lower than from other parts of the cycle and from other fuel systems.

7. Fossil Fuels

The data presented on the health effects associated with fossil fuels suffer from certain limitations and uncertainties. First, genetic effects are not included because our present state of knowledge does not allow even an approximate estimate for such effects. Second, the data do not adequately discriminate between premature deaths that may occur early in life, such as from accidents, and those that may shorten life only slightly, as seen, for example, in increased mortality among persons hospitalized for chronic disease, who already have high mortality rates. Perhaps of greatest importance is the uncertainty about the validity of the upper estimates for the effects on the general public from burning coal and oil. Not only is there the problem of the magnitude of the effect, but lack of knowledge about the causative agents makes it difficult to institute effective control procedures.

The primary data come from epidemiological studies. Major episodes (Meuse, Donora, London, New York City, etc.) clearly showed that air pollution, sufficiently severe, could cause illness and premature death. During the 1950s and 1960s the major issue was whether air pollution in concentrations usually existing over industrial cities would cause adverse health effects. The emphasis shifted next to quantifying pollution relative to effects produced and more recently to the effects of low levels of pollution and the effects of interactions.

From a methodological standpoint epidemiological, animal, and experimental human studies are needed. Epidemiological studies are important in uncovering possible associations that can then be tested under controlled conditions; they are also needed for evaluation of human risks suggested by laboratory experiments. Animal studies are used to determine efficiently the sites of effects, mechanisms, and dose--response relationships, and they are more easily adapted for chronic studies than are human investigations. Because of species differences, controlled studies on humans are needed to establish responses and to determine the influence of disease or of various physiological states on the effects of pollution.

Since all of the human data from which damage functions have been drawn rest on the use of regression analysis, we offer in the Annex some brief comments on that technique so that the reader may be aware of the use and limitations of the method.

8. Air Pollution Damage Functions

In calculating the health effects of energy, it is sometimes possible to rely on actual experience. For example, reliable records of accidents incurred in coal mining exist [21]. These can be used to calculate the expected fatality or injury rate per unit of coal extracted. Other effects, such as radiation hazards, have been statistically estimated from a wealth of data as discussed above. The effects of chemical air pollution, however, have been more elusive. The derivation of quantitative damage functions has been attempted only in the past decade and researchers relied almost exclusively on regression analysis. Furthermore, since reliable morbidity data are difficult to obtain, most studies have restricted themselves to mortality rates. The major studies in this area are summarized here.

Lave and Seskin [22] considered 117 standard metropolitan statistical areas (SMSAs) of the U.S. As dependent variables, they used total, infant, and certain disease-specific mortality rates. The independent variables included the percent non-white population, the population density, the percent of population over 64 years old, and the percent poor population, i.e. families with annual income under $ 3000. The air pollution data consisted of 26 biweekly concentration measurements of suspended particulates and total sulfates. From these, they used as independent variables the minimum, maximum and mean of each group of 26 values. The data were collected for the years 1960 and 1961 and multiple linear regression were performed for each year. The following is an example of their 1960 regression results:

Total mortality rate (per 10,000 people) = 19.607
+ (0.041) mean suspended particulates ($\eta g/m^3$)
+ (0.71) minimum sulfate ($\eta g/m^3$)
+ (0.001) number of persons per square mile
+ (0.0041) percent non-white
+ (0.0687) percent over 65 years.

In this equation, only the variables whose regression coefficients were statistically significant at the 0.05 level were included. To understand the relative practical significance of each variable, the authors calculated the elasticity. That is, based on the regression coefficients, they calculated the percent increase in the mean value of mortality rate if the mean of each of the independent variables was increased by 10%. These calculations are presented in Table 2. Thus, understandably, mortality rates are most sensitive to the older segment of the population. Lave and Seskin conclude from these elasticities that mortality would decrease by 4.5% if the level of pollution was decreased by 50%.

Table 2: Elasticities of Air Pollution and Socioeconomic Variables*

Variable	Increase in Mortality Due to 10% Increase
Mean P	0.53%
Min S	0.37%
P/M^2	0.07%
% N-W	0.57%
% 65	6.32%

*From [22].

In another paper, Lave and Seskin [23] considered the effects of some meteorological and home heating variables. The pollution variables remained statistically significant and the

magnitude of the regression coefficients did not change appreciably. The authors also conclude that transformations of the variables did not improve the goodness of fit [24].

Winkelstein et al. [25] studied mortality rates from all causes among white males 50-69 years of age in Erie County, New York. His statistical unit was the census tract (125 in all). He tabulated these death rates for subgroups of this population, based on the socio-economic levels and mean air pollution measurements. The strong associations suggested by these data prompted Hamilton and Morris [26] to compute the following regression equation:

Mortality rate of men 50-69 years per 1000
= 33.97
+ (0.15) mean total suspended particulates ($\eta g/m^3$)
- (0.0034) mean family income ($).

Hickey et al. [27] considered 15 measurements of atmospheric chemicals and mortality rates due to cancer and heart disease. This restricted their sample size to 38 SMSAs. The data were averages for the period 1957-1964. With no adjustments for age and socio-economic status, they obtained regression equations of these disease-specific mortality rates on the logarithms of the pollutant concentrations. The concentrations of SO_2 and NO_2 appeared consistently as significant predictors in these equations.

Carnow and Meier [28] used benzo[a]pyrene as an index of air pollution. Their dependent variables were age-specific death rates due to pulmonary cancer. They compared urban with rural, migrant with nonmigrant and smoking with nonsmoking populations. The independent variables were average cigarette smoking levels and benzo[a]pyrene concentrations in the 48 contiguous states of the U.S. For 19 highly developed countries they calculated regression equations with tobacco sales and consumption of solid fuels as independent variables. They summarized their study with the statement: "A reduction of 60% in urban

air pollution might be expected to reduce the deaths from pulmonary cancer by 20% in all smoking categories."

Schwing and McDonald [29] noted that these and other regression studies suffered from two limitations:

(1) They included a limited number of pollution measurements; and

(2) They used ordinary least squares to estimate the regression coefficients.

In attempting to overcome these limitations, they included "a rather broad (but still incomplete) list of explanatory variables". These consisted of seven chemical pollution measurements, two radiation values, tobacco sales, four weather variables and nine variables describing population and socio-economic distributions. The dependent variables were mortality rates among white males for the 15 leading causes of death, age-stratified total deaths and age-stratified deaths due to lung cancer and heart disease. The sample consisted of 46 SMSAs. In all, they computed 40 regression equations, one for each of the disease and age categories. The authors also calculated elasticities for the pollution, radiation and smoking variables. Although the results were not always consistent, concentration of sulfur compounds and cigarette smoking were generally strongly associated with mortality. Associations with nitrogen compounds, the hydrogen index used, and ionizing radiation were less conclusive.

Later in this paper, we present a summary of calculations of health effects of energy based on these and other damage functions. To illustrate the difference between these functions, we present calculations made by Hamilton and Morris [26] based on the above mentioned equations derived by Winkelstein (W) [25], Schwing and McDonald (S-MC)[29] and Laven and Seskin (L-S)[22]. They considered a 1000 MW(e) coal-fired power plant with a 1000 foot stack, using 3% sulfur coal, 12% ash and 99% particulate removal. In an 80 km radius area with "typical" population distribution (164,000 people), they calculated the

expected ambient concentrations of SO_2 and sulfur particulates. Applying the above equation, Table 3 of expected "excess deaths" was generated.

Table 3: Comparison of "Excess Mortality" Based on Various Damage Functions*

Damage Function	Excess Mortality
Men 50-69 (W)	29.0
Men 50-69 (S-MC)	0.1
Total male (S-MC)	90.0
Total population (L-S)	19.0

*From [26].

It is interesting to note that these estimates are within two orders of magnitude of each other. This is typical of the calculations made by different researcher in this area and reflect the state of our knowledge at the present time.

In 1970, air quality standards for selected pollutants were mandated by the United States Clear Air Amendments. Emphasis was placed on sulfur dioxide because of the evidence that ambient levels were associated with health effects of air pollution disasters. Subsequent studies indicated that sulfur dioxide by itself could not be the primary causative agent and it was postulated that a combination of sulfur dioxide and particulates was responsible [30-32]. More recent evidence suggests that oxidation products of sulfur dioxide (i.e. sulfuric acid and particulate sulfates)--possibly acting synergistically with sulfur dioxide and other pollutants such as nitrates, particles, and ozone--are primarily the causative agents [33-35]. It must be emphasized that although suspended sulfates are now being used as an indicator of health effects and there appear to be correlations between them and such effects, there is no firm evidence as to which substance or substances in polluted air are the

causative agents. Without such knowledge, air pollution control strategy based on reduction of sulfur alone does not have a valid scientific basis.

The major categories of health effects associated with air pollution are (a) chronic respiratory disease; (b) symptoms of aggravated heart-lung disease; (c) asthma attacks; (d) children's respiratory disease; and (e) premature death. It would be most useful to understand the quantitative relationships between exposures to specific agents and these health effects in order to know how much investment is justified for control measures, to know which chemical effluents to control, and to make comparisons with biological costs of nuclear power.

In a recent report of the National Academy of Sciences-National Academy of Engineering-National Research Council [36], illustrative calculations were made of the health effects associated with sulfur oxide emissions for representative power plants in the Northeast. The results are presented in Table 4. They were derived from models that related ambient levels to emissions including factors for conversion of SO_2 to sulfates; health effects from ambient levels were calculated by using dose-response curves from epidemiological data from studies of the Environmental Protection Agency (EPA). It must be emphasized that the numerical estimates of Table 4 are controversial, relying on limited information and numerous arbitrary assumptions, and cannot be regarded as proven results. A critique in the same document from which Table 4 was derived [36, Chapter 4] suggests that the estimates could be low by a factor of two or high by a factor of ten. What can be concluded from Table 4 with reasonable assurance is that the effects listed are produced at detectable levels by factors associated with air pollution, with power plants most likely making a significant contribution. It should also be noted that a cost-benefit assessment of the data in Table 4 indicates that the economic impact of the nonlethal effects is much greater than of the premature deaths.

Table 4: Health Effects Associated with Sulfur Oxide Emissions*

	Remote Location	Urban Location
Case of chronic respiratory disease	25,600	75,000
Person-days of aggravated heart-lung disease symptoms	265,000	755,000
Asthma attacks	53,000	156,000
Cases of children's respiratory disease	6,200	18,400
Premature deaths	14	42

*Source: [36, Chapter 13]. Illustrative calculations based on distributive models, postulated conversions of SO_2 to SO_4, and EPA epidemiological data for representative power plants in the Northeast emitting $96.5 \cdot 10^6$ pounds of sulfur per year--equivalent to a 620 MW(e) plant.

9. Health Effects from Electricity Generation

Several reports have been published that contain estimates of the health effects associated with electricity production [7, 21, 37-42]. Tables 5 and 6 summarize the available estimates for each phase of the fuel cycle for each of the four fuels: coal, oil, natural gas, and nuclear. By and large, the estimates relate to contemporary technology and existing circumstances. In each case the data have been adjusted to represent the number of premature deaths or occupational impairments produced per year by processes associated with a 1000 MW(e) power plant, which is roughly that required for a population of 1,000,000 people. The values given represent the lowest and highest from the cited references. The references should be consulted for an understanding of the methodology and detailed assumptions; limitations have been discussed in the previous section.

Consider first from Table 5 the effects on workers. For coal-fired plants the values range from 0.5 premature deaths per year and for the other fuel sources they range somewhat lower, from 0.06 to 1.3. Most of these effects are due to accidents in coal mines, to conditions that cause black lung disease, or to activities in oil refineries, uranium mining, and nuclear fuel reprocessing.

Consider now from Table 5 the effects on the general population. It has been estimated that the transport of coal required for a year's operation of a 1000 MW(e) plant is responsible for 0.6 to 1.3 premature deaths by accidents at railroad crossings; no estimates are available for truck or barge transport. The comparative values for the other fuel systems are insignificant.

The data so far discussed have a reasonable statistical base of past operation and are to that extent reliable. The number of premature deaths among the public from power plant operation (conversion or generation of electricity) results primarily from dissemination of air pollutants and, as discussed earlier, these effects are a matter of great uncertainty. The upper-limit estimates for coal and oil are about 100 premature deaths per year compared with 1 or less for natural gas and nuclear.

Table 6 presents data on the number of nonfatal occupational injuries per year associated with the operation of a 1000 MW(e) power plant. These have been defined as injuries serious enough to cause loss of working time for several days or more. These effects are roughly the same for coal and oil, ranging from about 12 to 100 cases per year, and somewhat lower for natural gas and nuclear. Most of these effects are associated with mining, well digging, coal transport, oil refining, and nuclear reprocessing.

Table 5: Premature Deaths per Year Associated with Operation of a 1000 MW(e) Power Plant (values are lowest and highest estimates from cited references)[a]

	Coal	Oil	Natural Gas	Nuclear
Occupational				
Extraction				
Accident	0.45-0.99[21,37,39,40,42]	0.06-0.21[37-40,42]	0.021-0.21[37-40,42]	0.05-0.2[7,37,39,40,42]
Disease	0-3.5 [39]	-	-	0.002-0.1[7,39,41,42]
Transport				
Accident	0.055-0.4 [37,39,40,42]	0.03-0.1[37-39,42]	0.02-0.024[37,39,40,42]	0.002[37,40,42]
Processing				
Accident	0.02-0.04[39,40]	0.04-1[37-40,42]	0.006-0.01[37,39,40,42]	0.003-0.2[7,37,39,40,42]
Disease	-	-	-	0.013-0.33[7,39,41,42]
Conversion				
Accident	0.01-0.03[37-40,42]	0.01-0.037[37-40,42]	0.01-0.037[37-40,42]	0.01[37,39,40,42]
Disease	-	-	-	0.024[7]
Subtotals				
Accident	0.54-1.5	0.14-1.3	0.057-0.28	0.065-0.41
Disease	0-3.5	-	-	0.039-0.45
Total	0.54-5.0	0.14-1.3	0.057-0.28	0.10-0.86
General Public				
Transport	0.55-1.3[21,37,39,42]	-	-	-
Processing	1-10[39]	-	-	-
Conversion	0.067-100[21,39]	1-100[39]	-	0.01-0.16[b] [7,37,39,41,42]
Total	1.6-111	1-100	-	0.01-0.16
Total Occupational and Public	2-116	1.1-101	0.057-0.28	0.11-1.0

[a]Note: Dashes indicate no data found; effects, if any, are presumably too low to be observed; and no theoretic1 basis for prediction. From [43].

[b]For processing and conversion.

Table 6: Occupational Injuries per Year Associated with Operation of a 1000 MW(e) Power Plant (values are lowest and highest from cited references)*

Occupational Injuries	Coal	Oil	Natural Gas	Nuclear
Extraction				
Accident	22-49[37,39,40,42]	7.5-21[37-40,42]	2.5-21[37-40,42]	1.8-10.0[37,39,40,42]
Disease	0.6-48[21,39]	-	-	-
Transport				
Accident	0.33-23[37,39,40,42]	1.1-9[37-39,42]	1.2-1.3[37-39,42]	0.045-0.14[37,40-42]
Processing				
Accident	2.6-3[39,40]	3-62[37-40,42]	0.05-0.56[37-39,42]	0.6-1.5[37,39,40,42]
Conversion				
Accident	0.9-1.5[37-40,42]	0.6-1.5[37-40,42]	0.6-1.5[37-40,42]	1.3[37,39,40,42]
Totals				
Accident	26-77	12-94	4-24	4-13
Disease	0.6-48	-	-	-

*From [43].

10. Areas in Need of Further Research

Several issues and controversies regarding health effects of energy need to be settled. On the biological side, the mechanisms involved in the effects of radiation are poorly understood and those of chemical pollution even more so. This is reflected in the controversy of threshold versus linear extrapolation theories alluded to earlier. Further light may be shed on the problem if scenarios are constructed where each theory is adopted in turn. On the one hand (threshold), some deleterious effects on health may be neglected while on the other (linear) the use of energy may be needlessly restricted. Balancing these two types of "errors" at our present incomplete state of knowledge is a pragmatic issue which needs to be resolved.

Another pragmatic issue is to find a common index for various health effects which may be quantitatively and qualitatively different. On the one hand, chemical versus radiation effects and on the other normal operations versus accidental effects. This involves comparison of short-term somatic effects with long-term genetic effects. It also involves effects on which varying magnitudes of data are available and some on which no data are available (and hopefully never will be).

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Regression Analysis

Regression analysis is a statistical technique used to study the relationship between a criterion (or dependent) variable and a set of explanatory (or independent) variables. For example, the dependent variable may be mortality rate and the independent variables may be various measurements relating to air pollution, health conditions and socio-economic status of a given population.

Denoting the dependent variable by Y and the independent variables by $X_1,\ldots,X_p$, we can postulate the functional relationship:

$$Y = f(X_1,\ldots,X_p) \quad .$$

The major statistical problem is how to best estimate the parameters of this function. To this end, a number of measurements from different regions (cross-sectional) or at different time points (longitudinal) must be obtained. Then the parameters can be estimated from the data using, e.g. the least squares or maximum likelihood methods.

Some assumptions are usually made:

(i) independence, i.e. a data point does not affect, and is not affected by any other data point;

(ii) homoscedasticity, i.e. the variance of the distribution of Y at a given combination of values of $X_1,\ldots,X_p$ is the same as that at any other combination.

Another assumption is often made in order to be able to test hypotheses about the parameters of the regression function f; namely:

(iii) normality, i.e. the distribution of Y at any combination of values of $X_1,\ldots,X_p$ is normal (or Gaussian).

To proceed with the analysis, the form of the function f must be specified. The one used in most applications is the linear function, i.e.

$$f(X_1,\ldots,X_p) = \alpha + \beta_1 X_1 + \ldots + \beta_p X_p \quad ,$$

where $\alpha,\beta_1,\ldots,\beta_p$ are unknown parameters to be estimated from the data. The main advantage of this function is its simplicity; Y changes by an amount β_1 if the value of X_1 is increased one unit (provided $X_2,\ldots,X_p$ are not changed). The estimates of the parameters are easily obtained by the method of least squares. Indeed, several efficient packaged computer programs exist for this purpose [19]. Furthermore, the least squares estimates are optimal under assumptions (i) and (ii) [20].

The disadvantage of the linear model is that it may not provide an adequate description of the underlying functional relationship. If a nonlinear regression function is assumed, however, complex iterative procedures must be used to estimate the parameters. When the number of independent variables is large, the calculations become difficult, even with the aid of a large computer. This leads many researchers to using linear functions as approximations. The degree of approximation can be improved by limiting the range of variables, limiting the diversity (or span) of the population, or making transformations of the variables.

Although the regression curve may be non-linear over a wide range of the variables, it is frequently possible to consider only limited ranges over which the curve can be approximated reasonably well by a straight line. Similarly, if the population (or time span) under study is restricted, a linear regression may prove an adequate model. Finally, transformation of some or all the variables can produce (at least approximately) linearity. For example, suppose that the regression of Y on one

independent variable X is

$$Y = e^{\alpha+\beta x} \quad .$$

Then,

$$\log Y = \alpha + \beta x \quad ,$$

which is a linear regression equation of log Y on X. Quadratic or higher order powers of the independent variables can also provide an approximation of the non-linear regression curve while keeping the model linear in the parameters.

At this point some words of caution about interpreting the results of regression analysis may be useful. In using data not collected from a planned experiment, it is rarely possible to control for, or include measurements on all of the factors involved. Therefore, based on the regression results alone, it is not possible to infer causal relationship. The variables measured often act as surrogates of the underlying, unmeasured, causal factors. Furthermore, intercorrelations, i.e. collinearities among the independent variables often make it difficult to quantify the effect of an individual independent variable on the dependent variable. Thus, although regression analysis is a powerful predictive tool, it must be used only with caution as a normative explanatory technique.