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Approaches for Protection Standards for Ionizing Radiation and Combustion Pollutants

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The question "can the approach used for radiation protection standards, i.e., to extrapolate dose–response relationships to low doses, be applied to combustion pollutants?" provided a basis for discussion. The linear, nonthreshold model postulated by ICRP and UNSCEAR for late effects of ionizing radiation is described and discussed. The utility and problems of applying this model to the effects of air pollutants constitute the focus of this paper. The conclusion is that, in the absence of evidence to the contrary, one should assume the same type of dose–effect relation for chemical air pollutants as for ionizing radiation.

We will deal in this paper with the first question posed in the report from the international symposium on air pollution and health effects (*1*), "Can the approach used for radiation protection standards, i.e., to extrapolate dose–response relationships to low doses (for which no epidemiological evidence exists), be applied to combustion pollutants? If so, for which pollutants and effects would such an approach appear justified?"

It will be assumed that the approach used for radiation protection standards is that used by the International Commission on Radiological Protection (ICRP). The ICRP defines the purpose of radiation protection as being to safeguard from injury individuals, their progeny and mankind as a whole and at the same time to make possible activities that involve exposure of people to radiation. Many international organizations accept the responsibility of protecting people from chemical pollutants but it is difficult to find one that, along with this, accepts the responsibility of facilitating technological activities.

The chief pollutants released by combustion of fossil fuels are: oxides of carbon; oxides of nitrogen; oxides of sulfur; polyaromatic hydrocarbons and trace elements such as arsenic, chromium, lead, nickel, and zinc.

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Radiation protection is for reducing both somatic effects (delayed cancer) and hereditary effects (mutations in progeny). Of the pollutants listed above, oxides of nitrogen, polyaromatic hydrocarbons, arsenic, chromium, and nickel, are possibly carcinogenic, but little is known about the mutagenic action of any of the pollutants on higher plants and animals (*2*).

The ICRP classifies effects in another way as: "stochastic" effects, for which the probability of the occurrence rather than the severity varies with dose and for which there is no dose without an effect (threshold); and "nonstochastic" effects, for which the severity of the effect is proportional to dose and for which there may be no effect in the lowest range of doses (threshold).

For nonradioactive pollutants arising from the combustion of fossil fuels the only stochastic effect to be considered here will be cancer (usually of the respiratory tract) and the dose–response relations for cancer incidence will be considered for radiation and for other pollutants.

In its evolution, the ICRP promulgated recommendations for maximum permissible doses of radiation received in, first, a day, then in a week, and finally in a year. This meant that the Commission considered all doses additive regardless of when and at what rate they were received. It was realized that this procedure was valid only if the plot of response against dose yielded a straight line passing through the origin, often referred to as the

"linear, nonthreshold model." These assumptions about the dose–effect relation were adopted for practical reasons and because usually the conservatism of the concept provided extra protection (*3*). When the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) began estimating the risks of stochastic effects of radiation exposure in human beings, it decided to express the results as cases of harm per million persons per year (or per lifetime) per unit radiation dose (*4*). This was arrived at by observing the incidence rate in populations exposed to a few tens or hundreds of rads of radiation and dividing the excess rate of incidence by the dose. Such a procedure is valid only under the same assumptions about the dose–effect relation that ICRP has made and these were adopted by UNSCEAR only after debate lasting many sessions. The linear nonthreshold model merits some comment.

Threshold

A threshold can be accepted a priori only if detailed knowledge of the mechanism of carcinogenesis justifies it. Most mathematical relations between cancer incidence I and dose D indicate that for $D > 0$, $I > 0$. Experimental results with animals show thresholds in some cases, not in others.

"Practical thresholds" may arise because of the long latent period with low doses or from the uncertainty of detecting a rise in a fluctuating background rate. (The latter can also be a reason for not detecting a threshold.)

The lack of a threshold observed in some animal experiments may result from the radiation not initiating cancer but accelerating a naturally occurring process.

Linearity

Many shapes of dose–incidence curves have resulted from the data obtained in both animal research and from epidemiological studies. In some cases, the confidence limits of the data are such that a constant proportionality cannot be ruled out. In dose–incidence plots the commonest deviation from a straight line is a curve with increasing slope up to a maximum after which the incidence declines with dose due to cell sterilization or killing. If this is a "true" picture of the relation, a linear relation will overestimate the rate of incidence at low doses by a factor that has been estimated by UNSCEAR as 2–4 (*5*).

The stochastic effects of air pollution resulting from burning fossil fuels are confined mainly to carcinogenesis (*2*). Because of the route of entry, the cancers produced are mainly in the respiratory tract. The same document shows that, for the past 35 years there has been a rising "background" rate due to cigarette smoking, general air pollution, and other or unknown causes. There is a multiplicity of causes of lung cancer; there are also observed synergisms between several of them such as cigarette smoking, radiation, silicon and asbestos. The exposed population is extremely heterogeneous with respect to age, dietary habits, health and exposure pattern. All these factors render the possibility to observe, or even to predict, a no-effect dose extremely low.

Studies of the correlation between rates of cigarette smoking and incidence of lung cancer have yielded linear plots of effects against doses (*2*). The National Academy of Sciences of the U. S. has concluded from epidemiological studies that each increment in air pollution represented by 1 μg/100 m^3 of benzo[a]pyrene results in a 5% increase in lung cancer of the population at risk (*6*). Examples such as this persuade me that, in the event of any doubt, one should follow the examples of ICRP and UNSCEAR and assume the same type of dose–effect relation for chemical air pollutants as for radiation, i.e., constant proportionality for all doses and no zero-effect dose.

Nonstochastic Effects

It was reported above that these have been defined by ICRP as effects for which the severity varies with the dose, e.g., cataract of the lens of the eye; skin erythema, blistering, and ulceration; and hematological deficiencies due to cell depletion of the bone marrow and impairment of fertility due to gonadal cell injury. All these are "early" effects and they have a "threshold" dose.

The ICRP definition of stochastic effects is difficult to apply to some late effects of radiation. For example, life shortening in experimental animals increases with the size of the dose and has no observable threshold. In dogs the injection of radionuclides such as ^{226}Ra and ^{239}Pu causes bone cancers, and the time to appearance decreases as the dose increases with an apparent threshold due to the limitation imposed by the life-span.

There are also difficulties in applying the ICRP definition of nonstochastic effects to some early effects of chemical pollutants. For example, the effects of air pollution on structures and of SO_2 or ozone on plants are proportional to the total exposure (concentration × time) with no threshold (*7*). Another example is provided by the eye irritation

observed in humans caused by aldehyde vapor in the air; a plot of percentage incidence against concentration gives a straight line with a very low threshold.

Perhaps the most important nonstochastic effect of air pollution on populations is the induction of respiratory damage or failure. Here, the evidence from studies of the effects of single substances on workers in the workplace or on animals in the laboratory is of little use.

For acute episodes, there is evidence that the number of deaths increases with the air concentration. Because of the number and interactions of the causative agents and the great variation in sensitivity of the exposed individuals there would be no justification to assume a threshold. Until there is reliable evidence to the contrary the assumptions of constant proportionality and no zero-effect dose would seem to be the most prudent basis for protective measures.

Conclusions about the long-term effects of persistent exposure to low levels of air pollutants are difficult. In the first place, most epidemiological studies use, as the measure of dose, the concentration in air of some single pollutant averaged over some period of time. It is possible that the dose should be measured in terms of concentration times time for any single pollutant but how the additivity of several can be expressed quantitatively is not clear. There is also the problem of what is the most relevant effect and how it can be expressed quantitatively.

One effect of chronic air pollution seems well established by epidemiological studies: it leads to an increase in the incidence of lung cancer caused by cigarette smoking (*2*).

Finally, some other practices from radiation biology or the nuclear industry should be considered for possible application to nonradioactive pollution:

- Expression of the dose as total energy absorbed in the target organ. For a chemical this would be the time integral of concentration in the organ.
- Multiplying the dose of each different kind of radiation by a coefficient of efficiency (RBE) so that they can be added. A "Radequivalent" has been proposed (but not widely accepted) as a normalizing unit for protection against environmental chemical mutagens.
- UNSCEAR, prodded by Lindell (*8*), adopted the concept of dose commitment, a measure of the harm to all receptors over future time resulting from a unit of practice such as the exploding of a nuclear bomb or the generation of 1 MW of electricity. This concept might be considered for processes releasing chemical pollutants.
- In the nuclear industry it has become the practice to estimate the harm resulting from all steps leading up to the generation of nuclear power, viz., exploration, mining, milling, reactor operation, fuel processing, transportation, waste disposal. A similar approach should be adopted for all steps in the generation of power from fossil fuel.

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