Dose Assessment Analogies

by Bo Lindell*

Assessments of radiation detriment are based on the absorbed dose in the organs and tissues of interest, assuming a linear, nonthreshold dose–response relationship for the induction of cancer or genetic effects at low doses. Similar assessments may be made for nonradioactive, mutagenic, or carcinogenic substances, in which case the time integral of the local tissue concentration of the substance may be the quantity which would correspond to the radiation dose. It can be shown that the assessment then involves calculations which are very similar to those made in the assessment of radiation detriment. It is suggested that an attempt should be made to make such assessments for nonradioactive pollutants, in order to provide a more appropriate basis for comparisons with radiation detriments. In the assessment of the detriment from inhaled substances, the overall collective intake commitment is simply the total amount of each substance ever to be inhaled, irrespective of when or where. This quantity might be assumed to be proportional to the total detriment in form of lung cancer.

In assessing radiation detriments, quantitative estimates are based on the assumption of proportionality between the risk of stochastic radiation effects (cancer and genetic harm) and the amount of energy absorbed per unit mass of the organ or tissue of interest, the absorbed dose of radiation. The unit of this quantity is the gray (1 Gy = 1 joule/kg) but used to be the rad (1 rad = 0.01 Gy).

The absorbed dose in a tissue may be caused by irradiation from radiation sources outside the body (external exposure) or by radioactive substances inside the body (internal exposure). Often the irradiation is due to both external and internal exposures, and the absorbed doses caused by each of these exposures must be added to give the total absorbed dose in the organ or tissue of interest.

If the body is exposed to mutagenic or carcinogenic substances, the situation is in many respects similar to the case of internal exposure from radioactive substances, while external exposure has no equivalence.

If an insoluble radioactive substance (j) is inhaled, it will remain in the lungs and irradiate these until it has either decayed completely or has been removed mechanically. If it is ingested, it will irradiate the stomach and intestinal walls while passing through the body.

If a soluble radioactive substance is inhaled or ingested, a certain fraction \( f_i \) and \( f_g \), respectively, is absorbed from the gastrointestinal tract and reaches the blood. The absorbed dose in the various tissues to which the absorbed material is transported will depend upon the chemical and nuclear characteristics of the substance. In some cases, the substance may be chemically decomposed or transformed. For each substance \( j \), the part of the inhaled or ingested quantity \( a_{ij} \) and \( a_{gj} \) which will reach the blood will be \( f_ia_{ij} \) and \( f_ga_{gj} \), respectively. The absorbed dose in any one organ or tissue \( k \) is proportional to the amount which reaches the blood, and we may call the proportionality factor \( g_{jk} \). The absorbed dose from substance \( j \) in organ \( k \) of an individual \( i \) may therefore be calculated as

\[
D_{uk} = g_{uk} (f_{ij}a_{ij} + f_{gj}a_{gj})
\]

The absorbed dose in the lung and in the gastrointestinal tract (or any selected part thereof) respectively may be calculated as

\[
D_{ul} = g_{ul} (1 - f_{ij})a_{ij}
\]

and

\[
D_{uls} = g_{uls} (1 - f_{ij})a_{gj}.
\]

If there is proportionality between the absorbed dose \( D_k \) in organ or tissue \( k \) and the resulting risk \( R_k \)

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( = \( r_k D_k \)), the proportionality factor \( r_k \) may be called the risk factor for organ \( k \). The total risk to the individual \( i \) from a number of substances \( j \) can be assessed as

\[
R_i = \sum_j \left[ \Sigma_r r_{ik} \gamma_{uk} \left( f_{uij} a_{uij} + f_{siu} a_{su} \right) + r_{ij} \gamma_{ij} \left( 1 - f_{uij} \right) a_{uij} + r_{ui} \gamma_{ui} \left( 1 - f_{siu} \right) a_{su} \right]
\]

The risk factors \( r_{ik} \) may differ between individuals due to differences in, for example, age and sex. Also the organ factors \( \gamma_{uk} \) may differ, for the same reasons.

Exactly the same expressions should be valid for carcinogenic substances. It may be said that this is a great oversimplification, but most objections would seem to be equally valid in respect to the assessment of radiation risk. Any substance which is inhaled or ingested will either remain in the lungs, pass through the gastrointestinal tract or be partially absorbed into the blood and transported to various tissues. The quantity that would correspond to the absorbed dose of radiation as the basis for risk assessment would be the time integral of the local tissue concentration of the carcinogenic substance (which may not be identical to the substance inhaled or ingested).

For illustration, let us consider the simple case when the lungs are the critical organ and inhalation is the only mode of entry of the substance into the body. The risk of lung cancer to individual \( i \) would then simply be

\[
R_{ui} = \sum_j r_{uij} \gamma_{ui} a_{uij}
\]

The use of one and the same risk factor \( r_{uij} \) for all substances \( j \) is possible only if the organ factors \( \gamma_{uij} \) for the lungs will transform the intakes \( a_{uij} \) to “doses” which are additive. This is the case for radioactive substances if the organ factors include the weighting for different relative biological effectiveness (RBE) between, for example, \( \alpha \)- and \( \beta \)-emitting radionuclides. It would also be true for carcinogenic substances if the concentration time integrals were weighted for the relative biological effectiveness of the different carcinogenic substances, e.g., by expressing the “dose” in rad-equivalents as suggested by Ehrenberg (1, 2).

In both cases, the organ factors \( \gamma_{uij} \) would need to include a consideration of the mean retention time in the lungs, and it would need to be decided whether to assess the maximum dose or the dose averaged over the whole of the lungs. This choice is arbitrary but will influence the subsequent choice of value for the risk factor.

If the assessment is made for just one substance \( j \), the weighting for RBE is not necessary if the risk factor \( r_{uij} \) has been determined for that particular substance. The risk to individual \( i \) may be assessed as

\[
R_{ui} = r_{ui} \gamma_{ui} a_{uij}
\]

In assessments of radiation risks, the absorbed dose is usually calculated and the risk is assessed as

\[
R_{ui} = r_{ui} D_{uij}
\]

However, the risk may equally well be determined from the inhaled amount \( a_{uij} \) and can be expressed as

\[
R_{ui} = h_{uij} a_{uij}
\]

(with \( h_{ui} = r_{ui} \gamma_{ui} \)). This is in fact what is usually done in the case of exposure to radon daughter products, when the amount inhaled rather than the actual absorbed dose is used as the basis for the risk assessment. In practice, this method is preferable in any assessment of lung cancer risk from carcinogenic substances. The calculation of a tissue “dose” is needed only when there is both ingestion and inhalation and may even then be avoided by appropriate weighting of the intakes through the two modes of entry.

If the risk is assessed directly from the intake, the problem is reduced to the assessment of the intake. The intake \( a(\tau) \) accumulated over the time \( \tau \) can be calculated from the concentration \( c(t) \) of the substance in the inhaled air:

\[
a(\tau) = \alpha \int_0^{\tau} c(t) \, dt = \alpha I_c(\tau)
\]

In this expression \( \alpha \) is a proportionality factor which measures the amount of air inhaled per unit time. The air concentration integral \( I_c(\tau) \) is therefore an indirect measure of the “dose” and of the risk. In radiation risk assessments of exposure to radon daughter products, the concentration of these in the air is often given in terms of “working levels” (WL) and the concentration integral in terms of “working level months” (WLM). For carcinogenic substances, the unit of the concentration integral would be kg-sec/m³, or appropriate denominations thereof.

Risk assessments may serve two purposes: to give a measure of the risk to the individual at a given location and to indicate the total detriment from a given practice, e.g., per unit practice such as the production of 1 MW-year of electric energy.
For the first purpose, the concentration time integral over a lifetime or over each year, in air at the point of interest, will suffice. For the second purpose, however, a collective quantity needs to be calculated. The detriment from a given practice may be defined as the mathematical expectation of harm after some suitable weighting for the severity of the effect in different types of harm. If there is only one type of harm, as would be the case if we were only interested in the risk of lung cancer from exposure of the lungs, the detriment is simply the mathematical expectation of the number of cases of lung cancer (if we neglect the different severity in effect due to different age with different loss of years).

If the individual risk is $R_i$, the detriment would be

$$W = \sum_{i=1}^{N} R_i$$

which may also be written

$$W = \sum_{n} \overline{(R_i)}_n N_n$$

if $\overline{(R_i)}_n$ is the average risk to the $N_n$ individuals in each population subgroup $n$ with assessable exposure. The detriment may therefore be written

$$\sum_{n} (h_i a_i) n N_n = \sum_{n} [h_i \alpha_i \int_{0}^{\infty} I_{cn}(\infty)] n N_n$$

If we assume that $h$ and $\alpha$ are independent of age (a crude approximation), the detriment may be written

$$W = h\alpha \sum_{n} I_{cn}(\infty) N_n$$

The concentration time integral $I_{cn}(\infty)$ may be interpreted in two ways. Assuming that the unit practice causes an air pollution $c(t)$, with different values $c_n(t)$ for each subgroup $n$, the concentration time integral $I_{cn}(\tau)$ would only apply directly to those individuals who existed in the subgroup at time $t = 0$ and who would still be members of the subgroup at time $t = \tau$. If the maximum value of $I_{cn}(\tau) = I_{cn}(\infty)$ is reached before there is any change in the individual composition of the subgroup, then the total detriment is

$$W = h\alpha \sum_{n} I_{cn}(\infty) N_n$$

If, however, the pollution lasts long enough so that individual members of the subgroup die and are replaced by new individuals, the individual risks would be

$$R_i = h\alpha \int_{t_{bi}}^{t_{ai}} C_i(t) dt$$

where $t_{bi}$ is the time of birth and $t_{ai}$ is the time of death of the $i$th individual (latency periods of cancer induction being neglected). In order to calculate the total detriment, all these individual risks must be added within each subgroup $n$, over infinite time (or, in practice, over the time period of pollution). However, the same result will be obtained if the concentration integral over infinite time is calculated for each subgroup $n$, without any consideration of individuals, and the total detriment is assessed as

$$W = h\alpha \sum_{n} I_{cn}(\infty) N_n$$

the implication being a stable population of $N^n$ individuals in each subgroup $n$ over all future years. This latter condition is not likely to be valid if the pollution is local, but may be approximately true for global contamination.

The terms $\alpha I_{cn}(\infty) N_n$ may be called the collective intake commitments for the various subgroups $n$, and $\alpha \sum_n I_{cn}(\infty) N_n$ is the total collective intake commitment, the unit of which is simply the mass unit.

The first conclusion—-as would be expected—is that the total detriment of the practice is proportional to the total amount of the carcinogenic substance which will ever be inhaled by humans, irrespective of when or where.

This may seem to be a rather obvious outcome of an unnecessarily involved calculation. The valuable conclusion, however, is that the case of exposure to carcinogenic substances and the assumptions needed for detriment assessments are not basically different from the situation and assumptions when radiation detriment is assessed. In fact, if one follows the necessary chain of calculations, step by step, they prove to be virtually identical in the two cases as far as formulation is concerned. It is therefore strongly recommended that those who assess risk from chemical pollutants should familiarize themselves with the assumptions and methods used in radiation detriment assessments, particularly with respect to assessment of “dose commitments” and of the total “collective dose” per unit practice (3, 4). The quantification of radiation risks and the account of total future pollution has made the radiation detriments obvious to the public but has caused a lack of balance in the appreciation of other risks. There is little excuse for not attempting to make similar assessments for nonradioactive pollutants. This is in line with a recommendation from a group of scientists associated with the United Nations Scientific Committee on the Effects of Atomic Radiation, in a contribution to the 1972 UN Conference on the Human Environment (5).
REFERENCES

1. Ehrenberg, L., and Osterman-Golkar, S. Reaction kinetics of chemical pollutants as a basis of risk estimates in terms of radequivalence. First European Symposium of Rad-equivalence (Euratom) 1976.

2. Ehrenberg, L. Methods of comparing effects of radiation and chemicals. Position paper for IAEA Consultant Meeting, Brighton, October 21–23, 1976.

3. International Commission on Radiological Protection. Recommendations of the ICRP. Publ. 26, Pergamon Press, Oxford, 1977.

4. United Nations Scientific Committee on the Effects of Atomic Radiation. Report to the United Nations General Assembly, 1977.

5. Butler, G. C. et al. Assessment and control of environmental contamination—experience with artificial radioactivity. Biol. Conserv. 4: 177 (1972).