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Basic Concepts of Radiation Protection[1]

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INTRODUCTION

Soon after the discovery of X radiation and the phenomena of radioactivity, it was recognized that ionizing radiation can cause deleterious effects in living cells and tissues. On the basis of these observations some radiologists proposed first, unofficial guidelines for the limitation of radiation exposure from X rays and radium-226. However, in the year 1928 the International Congress on Radiology founded an International Commission on Radiological Protection, the so-called ICRP. Since this time this international commission has worked out and published recommendations on all basic and practical aspects of radiation protection. These recommendations of the ICRP formed the basis for most radiation protection regulations, which were issued by international bodies or by national authorities. Owing to these circumstances relative uniform standards for radiation protection are in use all over the world.

It is the aim of radiation protection to protect men against the toxic influences of ionizing radiation from man-made sources. The most important radiation effects are the possible induction or promotion of cancer in irradiated tissues and the hereditary effects, which can occur in consequence of an irradiation of the gonads.

The same types of effects can be produced by mutagenic chemicals. Consequently it seems urgently necessary to use the same concepts for protection of human beings against radiation and mutagenic chemicals. To reach this goal in the following paper the basic concepts of radiation protection are outlined, which are recommended by ICRP (1) and which are applied in most international and national regulations for the limitation of radiation exposure to workers and members of the public. Some of these concepts are also valid for the protection against toxic chemicals and can be used as a general, uniform platform for the setting of reasonable standards for environmental quality.

PRIMARY EXPOSURE QUANTITIES: THE DOSE CONCEPT

The primary physical quantity which is used in radiation protection is the "absorbed (radiation) dose." The absorbed dose to a tissue of the human body is defined by the radiation energy imparted to this tissue by ionizing particles divided by the mass of this tissue. It can be also expressed as the time integral over the mean dose rate in this tissue, integrated over the whole exposure period T:

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$$\text{radiation absorbed dose} = \frac{\text{absorbed radiation energy in tissue}}{\text{tissue mass}}$$

$$= \int_0^T (\text{dose rate in tissue})dt.$$

The SI unit of this absorbed dose is 1 gray (Gy) = 1 J/kg. (older unit: 1 rad = 0.01 Gy = 0.01 J/kg).

A congruent quantity for the exposure of a tissue to a toxic chemical substance would be the time integral over the concentration of this substance in the considered tissue, integrated over the whole time period after the uptake in the tissue, until the substance is eliminated or decomposed:

$$\text{"chemical dose"} = \int_0^T \left(\frac{\text{mass of substance in tissue}}{\text{tissue mass}}\right)dt$$

$$= \int_0^T (\text{concentration in tissue})dt.$$

However, expressing the exposure of a tissue by the quantity "dose," we have to recognize that there is a principle difference between radiation and chemical dose. The radiation dose characterizes the energy absorption or the primary, physical action of radiation, which is the first link in the complex reaction chain leading to a biological endpoint. The term chemical dose, however, describes only the existence of a chemical substance in this tissue and not the primary reaction yield of this substance. Thus the term radiation dose involves more information about the possible biological effectiveness than the term "chemical dose" as defined above.

In radiation protection the biological significance of exposure quantities is further improved by the introduction of the quantity "dose equivalent." The dose equivalent is defined as the product of the absorbed dose with a modifying or quality factor, which describes the relative biological effectiveness (RBE) of the used type of radiation relative to 200-kV X rays:

$$\text{dose equivalent } (H) = \text{quality factor } (Q) \times \text{absorbed dose } (D).$$

The SI unit of this quantity is 1 Sievert (Sv) = 1 J/kg. (old unit: 1 rem = 0.01 Sv). All dose limits in radiation protection are expressed in terms of dose equivalent.

Several authors have proposed to introduce this concept of dose equivalent in chemical protection; it enables to sum up the effect-weighted exposures from different chemicals and types of radiation (see (2)).

DOSE–EFFECT RELATIONSHIPS

The assessment of dose limits for individuals requires informations about the relationship between dose or intake, respectively, and the corresponding deleterious health effects. However, the action of radiation or chemicals in tissue comprehends a complex biological reaction chain. The mechanisms and yields involved in this reaction chain are mostly unknown.

Therefore the models for the dose–effect relationship which are used in radiation protection were directly derived from observed health effects in radiation-exposed

persons. In addition the findings from cytological studies and animal experiments were taken into account. The historical philosophy of radiation protection was based on the assumption that for each kind of radiation effect a threshold dose exists, below which the effect will be zero. Some elements of this threshold concepts are still involved in our present radiation protection regulations. However, the extensive, epidemiological studies carried out in the last 20 years have shown that this threshold concept will be indeed correct for acute and noncancerous radiation effects; but with respect to cancer and genetic effects these studies give a strong indication that no real threshold dose exists, below which the probability or risk for the induction of these so-called "stochastic" effects will be really zero (see (3)). This is confirmed by cytological investigations which have shown that the mutagenic action of radiation starts at zero dose.

The dose–risk relationship for cancer induction in humans by radiation shall be demonstrated by two typical examples. Figure 1 shows the observed excess lung cancer risk among uranium miners in the CSSR as function of their α dose to the lung (3, 4). In this case the α irradiation of the lung results from the inhalation of the short-lived decay products of the radioactive noble gas radon-222, which is formed in the mine rocks by decay of radium-226 and reaches the mine air by diffusion. Due to the increasing statistical error at low doses the real lapse of the dose–risk curve in this range cannot be determined. A statistical threshold can be derived, however, the best fit to the data is a proportional increase of the radiation-induced excess cancer risk with the α dose to the lung.

The second example, shown in Fig. 2, gives the excessive incidence of leukemia among the atomic bomb survivors of Hiroshima and Nagasaki as function of their external dose. In Hiroshima this dose resulted mainly by neutron irradiation, whereas in Nagasaki the γ radiation was dominating. There is a characteristic difference of the risk curves observed in both cities, which expresses the higher biological effectiveness of neutrons compared with photons. For the survivors in Hiroshima the curve can be fitted, similar like for the U miners, by a proportional dose–risk relationship. In Nagasaki the additional leukemia risk at low doses was

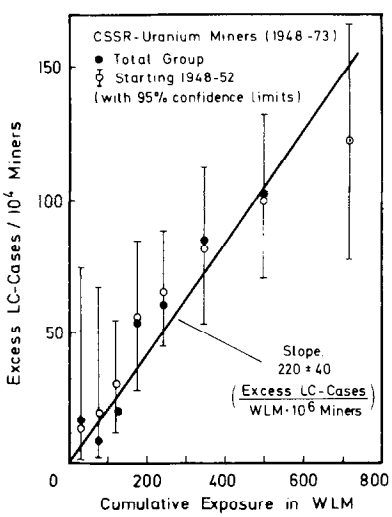

FIG. 1. Excess lung cancer incidence among U miners in the CSSR (1948–73) as function of their exposure to short-lived Rn-decay products.

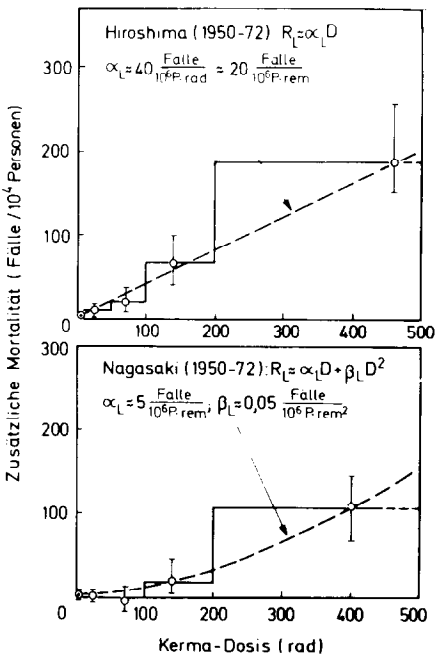

FIG. 2. Excess mortality from leukemia among the atomic bomb survivors in Hiroshima and Nagasaki as function of the kerma dose in tissue.

much smaller. The data can be fitted by a combination of a small linear dose term and a quadratic dose term, leading to a "linear–quadratic" dose–risk relationship: $R(D) \approx aD + bD^2$.

These different dose–effect relationships for leukemia in both cities are confirmed by the observed excess of chromosome aberrations in lymphocytes of these survivors, which is shown in Fig. 3. It should be emphasized that the results of several epidemiological surveys among patients after diagnostic X-ray exposures indicate a statistical significant increase of cancer incidence down to doses in the

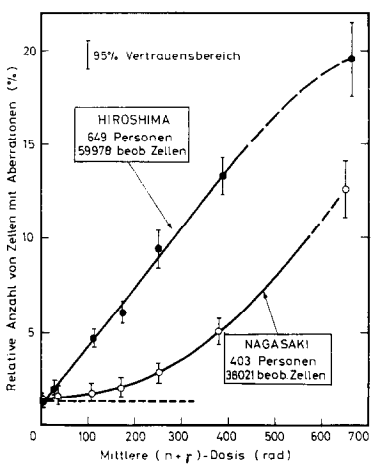

FIG. 3. Observed increase of chromosome aberrations in lymphocytes of atomic bomb survivors in Hiroshima and Nagasaki.

range of several rad (3). Thus, also for an irradiation with so-called low LET radiations like X rays and γ rays and β and electron rays, there is no evidence for the existence of a threshold dose for cancer induction. The animal studies for the genetic effects of radiation lead to the same result.

Summarizing the findings about the induction of cancer and genetic effects by radiation it can be concluded that at the present state of knowledge the best estimate for the dose–risk relationship in the relevant dose range is a proportional relationship for high LET radiation (α rays, neutrons) and a linear–quadratic model for low LET radiation (X and γ rays, β rays). This means that at low doses for all types of radiation a nearly proportional relationship $R = aD$ can be assumed, where the risk coefficient rises with increasing ionization density or LET of the particles along their track in the tissue. This is the so-called linear model without threshold, which is recommended now for purposes of radiation protection by the ICRP.

Applying these models it has to be taken into account that the risk coefficient a for cancer induction by radiation is different for different tissues or organs and depends also on age and sex.

On the basis of the available data the ICRP has recommended for purposes of radiation protection reference values for the risk coefficient of different body tissues. These reference values, which are averaged over age and both sexes, are given in Table 1. For a uniform whole-body irradiation follows a mortality risk by cancer of about $R \approx 10^{-2}$/Sv or 10^{-4}/rem, corresponding to 10^4 or 10^2 cases per 10^6 irradiated persons per Sievert or rem whole-body dose equivalent. The system of dose limits for individuals, which is recommended now by ICRP, is based on these risk values.

THE CONCEPT OF THE EFFECTIVE DOSE

So far only the dose and risk in single tissues was considered. In practice, however, each type of irradiation leads to an exposure of several tissues in the human body. The same situation will be valid for the incorporation of most chemicals. This means that the total risk R to an individual is given by the sum over the risks R_T in the single, exposed tissues T, taking into account the severity of

TABLE 1

REFERENCE VALUES OF RISK COEFFICIENTS AND RISK-WEIGHTING FACTORS FOR DIFFERENT BODY TISSUES RECOMMENDED BY ICRP

Tissue	Risk coefficient ($\times 10^{-4}$ Sv^{-1})	Weighting factor
Gonads[a]	40	0.25
Breast	25	0.15
Red bone marrow	20	0.12
Lung	20	0.12
Thyroid	5	0.03
Bone surfaces	5	0.03
Other tissues	50 (total)	0.30 (total)

Note. See Ref. (1).

[a] Risk of deleterious genetic effects.

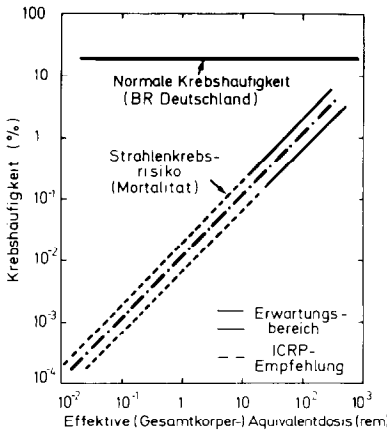

FIG. 4. Expected excess cancer mortality by radiation as function of the effective dose equivalent, assuming a proportional dose–risk relationship; for comparison the normal cancer mortality is given.

each type of effect. Thus on the basis of the linear dose–risk concept the total individual risk from stochastic effects is given by the equation (H_T = dose equivalent in tissue T):

$$R = \sum_T R_T = \sum_T a_T H_T.$$

With reference to this equation a dose quantity which is proportional to this total individual risk can be defined (5). For this purpose the dose equivalent H_T to a tissue has to be multiplied with a weighting factor w_T which is proportional to the risk coefficient a_T of this tissue. The resulting quantity

$$H_E = \sum_T w_T H_T \qquad \text{with} \qquad \sum_T w_T = 1$$

is called the effective dose equivalent. The ICRP recommends the future application of this concept in radiation protection, using the weighting factors w_T in Table 1 which were derived from the given reference values of a_T ($w_T = a_T/\Sigma_T a_T$).

The application of this effective dose concept yields great practical advantages. It enables characterization of an inhomogeneous dose distribution within the human body by a single value of the effective dose, which is proportional to the total stochastic risk of the exposed individual. Applying the values given in Table 1 it results the following relation between effective dose equivalent H_E and the total individual risk R from cancer and genetic effects:

$$R = 1.65 \times 10^{-2} H_E(\text{Sv}) = 1.65 \times 10^{-4} H_E \text{ (rem)}.$$

This relationship is shown graphically in Fig. 4. Consequently, the new, stochastic dose limits recommended by ICRP (1) are given in terms of the effective dose equivalent. For occupational exposure this limit is 0.05 Sv = 5 rem per year.

This concept replaces the previous concept of the critical organ which has been so far used for the evaluation of limits for the activity intake of radionuclides in the human body and which had its roots in the historical threshold concept. Table 2 shows a typical example, the evaluation of the effective dose for inhalation of a $^{239}PuO_2$-aerosol (6). In this case several tissues are exposed, mainly the lung, the

liver, the bone, and bone marrow. Summing up the weighted dose equivalents of these tissues it results an effective dose factor of 1×10^{-4} Sv = 10^{-2} rem/Bq inhaled activity. The intake limit is given by the effective dose limit divided by this dose factor. Thus it follows for occupational exposure to $^{239}PuO_2$ an annual limit of intake (ALI):

$$ALI = \frac{0.05 \text{ Sv}}{1 \times 10^{-4} \text{Sv/Bq}} = 500 \text{ Bq} = 1.35 \times 10^{-8} \text{ Ci}.$$

It should be emphasized that this concept of effective dose depends on the assumption of a linear dose–risk relation for cancer and genetic effects. If this risk concept will be also approximately valid for carcinogenic chemicals it will be reasonable to introduce a similar effective dose quantity for the estimation of the individual risk by these chemicals.

LIMITATION OF DOSE AND RISK

The described risk concept of radiation protection, assuming for stochastic effects a proportional risk increase with dose without a threshold, has important consequences for the setting of dose limits.

First of all it follows that a dose limit cannot be considered as boundary between safe and unsafe. These limits have to be regarded as the lower boundary for an unacceptable exposure region. Consequently, in addition to the observance of these limits it should be taken care to keep the exposure As Low As Reasonably Achievable, taking into account social and economic factors. This is the so-called ALARA principle of radiation protection.

The second problem arising from the risk concept is the consequence that dose limits should be derived from limits of risk, which seem to be acceptable in comparison with other similar types of risk.

It is obvious that these limits have to be different for occupational exposure and for the exposure of members of the public by the release of radioactive material in the environment. For example the ICRP derives the annual dose limit for workers of 0.05 Sv = 5 rem by comparing the expected, calculated radiation risk with the

TABLE 2

COMMITTED DOSE EQUIVALENT TO TISSUES AND THE EFFECTIVE DOSE EQUIVALENT PER UNIT INHALATION INTAKE OF ^{239}Pu-OXIDE

Tissue (T)	Weighting factor w_T	Dose equivalent H_T ($\times 10^{-4}$ Sv/Bq)	Weighted dose equivalent $w_T H_T$ ($\times 10^{-4}$ Sv/Bq)
Lungs	0.12	3.2	0.42
Bone surfaces	0.03	9.7	0.29
Liver	0.06	2.1	0.12
Red bone marrow	0.12	0.77	0.10
Gonads	0.25	0.12	0.03
Other tissues	0.42	0.10	0.04
Effective dose equivalent H_E per Bq intake			1.0

observed occupational risks in other industries having a high standard of safety.

Dose limits for members of the public can be recommended in a similar way, taking into account that radiation risks are a very minor fraction of the total number of environmental hazards to which our population is exposed. It seems reasonable therefore to consider the magnitude of radiation risks to the general public in the light of the public acceptance of other risks of everyday life. This acceptance could be motivated by the benefits that would not be received otherwise, or by an implicit judgement that the risk is negligible. On this basis the ICRP comes to the conclusion that a radiation risk from man-made sources (except medical exposure) in the range of 10^{-6} to 10^{-5} per year would be likely to be acceptable to any individual member of the public. This would result in an effective dose limit of about 1 mSv = 100 mrem per year (1), averaged over the whole lifetime.

However, for the assessment of dose limits for individual members of the public another possibility is existing: A comparison with the natural radiation background and its local variation. This method seems to be the best method for judging acceptable radiation risks to the public from man-made sources. The radiation protection regulation in Germany follows this method. The annual dose limit of 0.3 mSv or 30 mrem for whole-body exposure of individuals, which is laid down in this regulation, corresponds with the standard deviation of the natural radiation background. Applying this standard the average per caput-dose of the population will not exceed a few percentage of the natural radiation exposure of the population.

Another possibility for the judgment of the cancer risk by radiation is a comparison with the normal cancer risk in our population. Figure 4 shows the additional risk of cancer by radiation as function of the effective dose equivalent received during the total lifetime on the basis of the linear model. Our mean, natural lifetime exposure is about 0.1 Sv = 10 rem, which gives an additional calculated cancer risk of about 0.1%, compared with an observed cancer risk of nearly 20% in our population. The present system of dose limitation provides that the risk from exposures due to radioactive emissions in the environment will be more than three orders of magnitude lower than the observed, normal cancer risk.

This comparison indicates the necessity to apply the same concepts for the setting of limits for radiation and carcinogenic or mutagenic chemicals. Only by a standardization of these concepts we can reach a uniform basis for the quantification of the term environmental quality.

DETRIMENT AND COLLECTIVE DOSE

In the past protection against radiation and toxic chemicals was mainly engaged with the task to propose reasonable limits for the exposure of individuals and to take care that these limits will not be exceeded. But in addition we have to consider the total detriment to the society resulting from a technology or from a source. With respect to health this detriment is given by the total number of deleterious effects on health which can be expected in the population from the considered source or application. Also if the individual exposure limits are not exceeded it remains a collective risk or detriment due to effects for which no threshold dose exists.

Two examples shall demonstrate the requirement to comprehend this collective risk. Maintenance workers in nuclear power plants have to work sometimes in radiation fields of high intensity. To avoid an excess of individual dose limits their

working time in such areas is restricted. To carry out this work therefore the number of workers is raised, which leads to an increase of the collective detriment or risk. Probably similar situations are occurring in other industries.

The other example concerns the emission of toxic substances from a stack into the atmosphere. By increasing the stack height the maximum dose to individuals in the local environment is reduced; however, the immission area and the total number of exposed persons is enlarged. Thus, depending on the population density distribution and the dose–risk relationship we cannot exclude that the total detriment or the collective risk to the population is enhanced by an increase of the stack height.

These examples indicate that the observance of individual exposure limits is not sufficient for the judgment of the hazard from a radiation or chemical source.

For the assessment of this detriment we are using in radiation protection the quantity "collective dose." This quantity is defined by the sum of the effective dose H_E of all individuals in the considered collective of persons: $H_{E,coll} = \sum_N H_E$. Assuming a proportional relationship between effective dose and cancer and genetic risk (see Fig. 4) this collective dose is proportional to the total detriment G_{coll} or risk of this population group:

$$G_{coll} = \sum_N R = \bar{a} \sum_N H_E = \bar{a} \times H_{E,coll}.$$

The unit of the collective dose equivalent is "man·Sv" or "man·rem".

In the evaluation of the collective dose from a source which is emitting radioactive material in the environment all relevant pathways of the radionuclides to man are taken into account (7). In the case of long-lived radionuclides, like ^{239}Pu, the dose is integrated over several generations to obtain the real dose commitment to the population. As an example in Table 3 the estimated mean collective effective dose equivalent is given, to which the population might be

TABLE 3

ESTIMATED COLLECTIVE DOSE TO THE POPULATION BY NUCLEAR ENERGY PER GW·YEAR-PRODUCED ELECTRIC ENERGY

Step in the nuclear fuel cycle	Collective effective dose (man·Sv)
Uranium mining and milling	4
Fuel fabrication	<1
Reactor operation	20–30
Fuel reprocessing	20–50
Transportation	<0.1
Waste disposal	<1
Research and development	5–10
Total fuel cycle	50–100

Note. From data in Ref. (3).

committed by the different steps of the nuclear fuel cycle (the values are given per GW·year-produced electric energy).

Taking into account a world population of 5×10^9 this collective dose corresponds a mean per caput-dose of about $1-2 \times 10^{-8}$ Sv/GW·year. Thus an installed nuclear power of 1000 $GW_e \cdot y$ would lead to a mean individual dose of about $1-2 \times 10^{-5}$ Sv = 1–2 mrem per year averaged over the world population, which is about 1% of the normal natural exposure of the population.

The described concept of collective dose should be considered as an attempt to quantify the collective exposure and detriment from a source and to optimize the protection measures. In addition this quantity can be used as an index for the comparison of hazards from different sources and for the judgment of different technological alternatives.

FINAL REMARKS

I have tried to summarize the basic concepts of radiation protection, which are now recommended by the ICRP (1). They are the result of knowledge, experiences, and reflections which were obtained in radiation protection during the last decades. These concepts form the basis for the system of dose limitation in radiation protection, whose main objectives can be summarized in the following three principles:

1. the principle of justification, which means that no practice shall be adopted unless its introduction produces a positive net benefit;
2. the principle that certain dose limits for individuals shall not be exceeded;
3. the principle of optimization, to keep the exposure levels as low as reasonably achievable, social and economic factors being taken into account (ALARA principle).

I believe that these principles characterize also quite well the objectives for the protection against chemicals. Although the concepts of radiation protection cannot be considered as the endpoint of wisdom, some of them might be applicable also for the protection against carcinogenic or mutagenic chemicals.

In any case we should coordinate the protection on all fields and we should try to standardize the concepts of dose limitation for radiation and chemicals to avoid wrong decisions, to find out the best alternatives, and last but not least for the purpose of saving money.

REFERENCES

1. *Recommendations of the International Commission on Radiological Protection* (1977). ICRP Publ. 26. Pergamon, Elmsford, N.Y.
2. *Radiological Protection; Proc. of the First European Symposium on Radiation Equivalence (Paris, 1976)*, Commission of the European Communities, Report EUR 5725e, Luxemburg, 1977.
3. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (1977). *Sources and Effects of Ionizing Radiation*. United Nations, New York.
4. Ševc, J., Kunz, E., and Plaček, V., (1976). Lung cancer in uranium miners and long-term exposure to radon daughter products. *Health Phys.* **30**, 433–437.
5. Jacobi, W. (1975). The concept of the effective dose: a proposal for the combination of organ doses. *J. Radiat. Environ. Biophys.* **12**, 101–109.
6. *Limits for Intakes of Radionuclides by Workers* (1979). ICRP Publ. 30. Pergamon, Elmsford, N.Y.
7. *Radionuclide Release into the Environment: Assessment of Doses to Men.* (1979). ICRP Publ. 29. Pergamon, Elmsford, N.Y.
8. Otake, M., (1979). The nonlinear relationship of radiation dose to chromosome aberrations among atomic bomb survivors, Hiroshima and Nagasaki. Report RERF Tr 19–78.