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CONTENTS

Recessive mutations produce little effect in the first few generations of descendants, but make a contribution to the general pool of genetic damage in subsequent generations. There are also many deleterious conditions that have a substantial incidence in man and which are due to the interaction of genetic and environmental factors. They are known as multifactorial disorders. A general increase in mutations might increase their incidence, although this has not been demonstrated in either man or animals. In assessing the consequences for exposed individuals, the Commission has previously taken account of the hereditary effects that might occur in their children and grandchildren. This left the effects in later generations to be considered as part of the consequences for society. The Commission now attributes the whole detriment to the dose received by the exposed individual, thus avoiding the need for a two-stage assessment.

(89) For low doses and dose rates, the nominal hereditary effect probability coefficient for severe effects (excluding multifactorial effects, see below) over all generations and related to the gonad doses distributed over the whole population is 0.5×10^{-2} Sv^{-1}. About 80% of the effects are due to dominant and X-linked mutations. Of these, about 15% occur in each of the first two generations. No reliable estimate is available for the probability coefficient for the multifactorial conditions, but, weighted for severity, it is probably about 0.5×10^{-2} Sv^{-1}. Because of the different age distribution of a working population, the coefficients for workers are slightly smaller than for the general population (a reduction by about 40%). The Commission considers that the nominal hereditary effect probability coefficients of 1×10^{-2} Sv^{-1} for the whole population and 0.6×10^{-2} Sv^{-1} for workers adequately represent the weighted number of hereditary effects to be expected in all generations (see Table 3). This only includes weighting for severity. With further weighting for years of life lost if the harm occurs (see paragraph 96), the corresponding numbers will be 1.3×10^{-2} Sv^{-1} and 0.8×10^{-2} Sv^{-1} (see Table 4).

Table 3. Nominal probability coefficients for stochastic effects

	Detriment (10^{-2} Sv^{-1})[1]			
Exposed population	Fatal cancer[2]	Non-fatal cancer	Severe hereditary effects	Total
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

[1] Rounded values.
[2] For fatal cancer, the detriment coefficient is equal to the probability coefficient.

3.4.4. *Effects of antenatal exposure*

(90) The effects on the conceptus of exposure to radiation depend on the time of exposure relative to conception. When the number of cells in the conceptus is small and their nature is not yet specialised, the effect of damage to these cells is most likely to take the form of a failure to implant or of an undetectable death of the conceptus. It is thought that any cellular damage at this stage is much more likely to cause the death of the conceptus than to result in stochastic effects expressed in the live-born. Exposure of the embryo in the first three weeks following conception is not likely to result in deterministic or stochastic effects in the live-born child, despite the fact that the central nervous system and the heart are beginning to develop in the third week. During the rest

of the period of major organogenesis, conventionally taken to be from the start of the third week after conception, malformations may be caused in the organ under development at time of exposure. These effects are deterministic in character with a threshold in man, estimated from animal experiments, to be about 0.1 Gy.

(91) Throughout the period from 3 weeks after conception until the end of pregnancy, it is likely that radiation exposure can cause stochastic effects resulting in an increased probability of cancer in the live-born. The available data are not consistent and considerable uncertainty exists. However, the Commission assumes that the nominal fatality probability coefficient is, at most, a few times that for the population as a whole.

(92) Values of intelligence quotient (IQ) lower than expected have been reported in some children exposed in utero at Hiroshima and Nagasaki. There have been two principal quantitative findings. One is the observation of a general downward shift in the distribution of IQ with increasing dose. The Commission assumes that the shift is proportional to dose. Small shifts cannot be clinically identified. A coefficient of about 30 IQ points Sv^{-1} relates to the dose in the fetus in the period from 8 weeks to 15 weeks after conception. A similar, but smaller shift, is detectable following exposure in the period from 16 weeks to 25 weeks. This appears to be a deterministic effect, probably with a threshold determined only by the minimum shift in IQ that can be clinically recognised.

(93) The second finding is of a dose-related increase in the frequency of children classified as "severely retarded". The number of cases is small, but the data indicate an excess probability of severe mental retardation of 0.4 at 1 Sv. As shown in Annex B, this finding is consistent with the general shift in IQ distribution with increasing dose. Because of the Gaussian shape of the IQ distribution, the excess number of cases of severe mental retardation will be very small at small IQ shifts, rising steeply only as the shift approaches 30 IQ points. On this basis, a large change in the IQ of an individual can be caused only by a large dose. At doses of the order of 0.1 Sv, no effect would be detectable in the general distribution of IQ, but at somewhat larger doses the effect might be sufficient to show an increase in the number of children classified as severely retarded. The effects at all levels of dose are less marked following exposure in the period from 16 weeks to 25 weeks after conception and have not been observed for other periods. All the observations on IQ and severe mental retardation relate to high dose and high-dose rates and their direct use probably overestimates the risks.

3.5. Tissue Weighting Factors

(94) The tissue weighting factors introduced in Chapter 2 for defining the quantity effective dose were intended to ensure that a weighted tissue equivalent dose would produce broadly the same degree of detriment irrespective of the tissue or organ involved. The Commission has adopted an aggregated representation of detriment for this purpose. It includes four components: the probability of attributable fatal cancer, the weighted probability of attributable non-fatal cancer, the weighted probability of severe hereditary effects and the relative length of life lost. Since effective dose will be used only over ranges where the total probability of attributable death will be small, even the fatal contribution to detriment can be treated as additive when several organs are irradiated. Each consequence can then be weighted by a factor chosen to represent its severity. As in *Publication 26*, death and severe hereditary effects are both given a weighting factor of 1.

(95) Discussions in *Publication 45* (1985) suggest a weight for non-fatal cancers relative to fatal cancers equal to the average lethality fraction of the cancer concerned. A type of cancer that is difficult to cure, and thus has a high lethality fraction and usually a reduced quality of life for the survivors, would have a high weighting factor for the non-fatal events, while an easily cured cancer would have a low weighting factor for the non-fatal events. The weights would then range from about 0.01 for non-fatal skin cancer to about 0.99 for non-fatal leukaemia. The weighting factor to be applied to the fatality coefficient is derived in Annex B. The weighting factors for the severity of hereditary effects is already included in the probability coefficients.

(96) A second weighting is applied to take account of the different mean latency time for different types of cancer. This weighting is simply the relative time lost due to an attributable cancer death or, in the case of non-fatal cancers and hereditary effects, the relative time of impaired life taken for cancers as the same as the time lost by death for the same type of cancer. Finally, the products of the mortality coefficient and the weighting factors for morbidity and time lost are normalised to give a total of unity and thus provide a basis for the tissue weighting factors recommended by the Commission. These tissue weighting factors are provided as rounded values for individual tissues and organs and are given in Table 2 on bases set out in Annex B.

(97) The data in Table 4 are representative of those for a nominal population of equal numbers of men and women. Except for the breast, the differences between the sexes are small. The effect on the tissue weighting factors of combining the data is that some weighting factors are slightly higher and some slightly lower than the values that would relate to men and women separately. The effect of confining the population to workers is to decrease the nominal probability coefficient for workers to 4×10^{-2} Sv^{-1}, but does not significantly change the values of the tissue weighting factors.

Table 4. Nominal probability coefficients for individual tissues and organs[1]

	Probability of fatal cancer (10^{-2} Sv^{-1})		Aggregated detriment[2] (10^{-2} Sv^{-1})	
Tissue or organ	Whole population	Workers	Whole population	Workers
Bladder	0.30	0.24	0.29	0.24
Bone marrow	0.50	0.40	1.04	0.83
Bone surface	0.05	0.04	0.07	0.06
Breast	0.20	0.16	0.36	0.29
Colon	0.85	0.68	1.03	0.82
Liver	0.15	0.12	0.16	0.13
Lung	0.85	0.68	0.80	0.64
Oesophagus	0.30	0.24	0.24	0.19
Ovary	0.10	0.08	0.15	0.12
Skin	0.02	0.02	0.04	0.03
Stomach	1.10	0.88	1.00	0.80
Thyroid	0.08	0.06	0.15	0.12
Remainder	0.50	0.40	0.59	0.47
Total	5.00	4.00	5.92	4.74
	Probability of severe hereditary disorders			
Gonads	1.00	0.6	1.33	0.80
Grand total (rounded)			7.3	5.6

[1] The values relate to a population of equal numbers of both sexes and a wide range of ages.
[2] See paragraphs 95 and 96 and Table B-20 in Annex B.

(98) If the equivalent dose is fairly uniform over the whole body, it is possible to obtain the probability of fatal cancer associated with that effective dose from the nominal fatality probability coefficient. If the distribution of equivalent dose is non-uniform, this use of the nominal coefficient will be less accurate because the tissue weighting factors include allowances for non-fatal and hereditary conditions. For example, the contribution of fatalities from the equivalent dose in the lung will be underestimated by about 25%, and the contribution from the skin and thyroid will be overestimated by a factor of about 3. If the tissue equivalent doses are known, the nominal fatality probability coefficients for the individual tissues and organ can be used, but the difference between the two methods will not be significant because the individual tissue coefficients are not known with sufficient accuracy. The necessary data for both methods are provided in Table 4. As an approximation for a wide range of distributions of equivalent dose, the non-fatal somatic detriment adds about 20–30% to the fatal detriment.

4. THE CONCEPTUAL FRAMEWORK OF RADIOLOGICAL PROTECTION

Chapter 4 deals with the general policy of radiological protection. It introduces the idea of source-related and individual-related assessments. It outlines the basic system of protection for occupational, medical, and public exposures and distinguishes between a "practice", which causes exposures to radiation, and "intervention", which decreases exposures.

4.1. The Basic Framework

(99) Everyone in the world is exposed to radiation from natural and artificial sources. Any realistic system of radiological protection must therefore have a clearly defined scope if it is not to apply to the whole of mankind's activities. It also has to cover, in a consistent way, a very wide range of circumstances.

(100) The basic framework of radiological protection necessarily has to include social as well as scientific judgements, because the primary aim of radiological protection is to provide an appropriate standard of protection for man without unduly limiting the beneficial practices giving rise to radiation exposure. Furthermore, it must be presumed that even small radiation doses may produce some deleterious health effects. Since there are thresholds for deterministic effects, it is possible to avoid them by restricting the doses to individuals. On the other hand, stochastic effects cannot be completely avoided because no threshold can be invoked for them. The Commission's basic framework is intended to prevent the occurrence of deterministic effects, by keeping doses below the relevant thresholds, and to ensure that all reasonable steps are taken to reduce the induction of stochastic effects.

(101) Most decisions about human activities are based on an implicit form of balancing benefits against costs and disadvantages, leading to the conclusion that a particular course of action or practice either is, or is not, worthwhile. Less commonly, it is also recognised that the conduct of a practice should be adjusted to maximise the net benefit to the individual or to society. This is not a simple process because the objectives

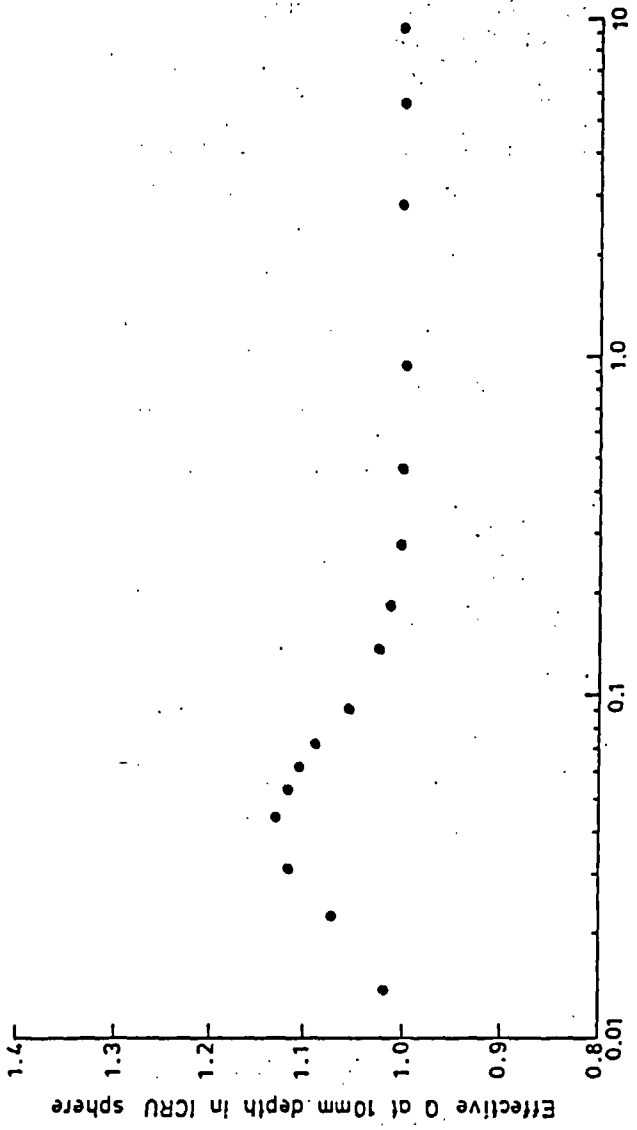

Fig. A-2. Effective Q, ($\bar{Q}$) as a function of photon energy. Reference: Drexler *et al.* (1990).

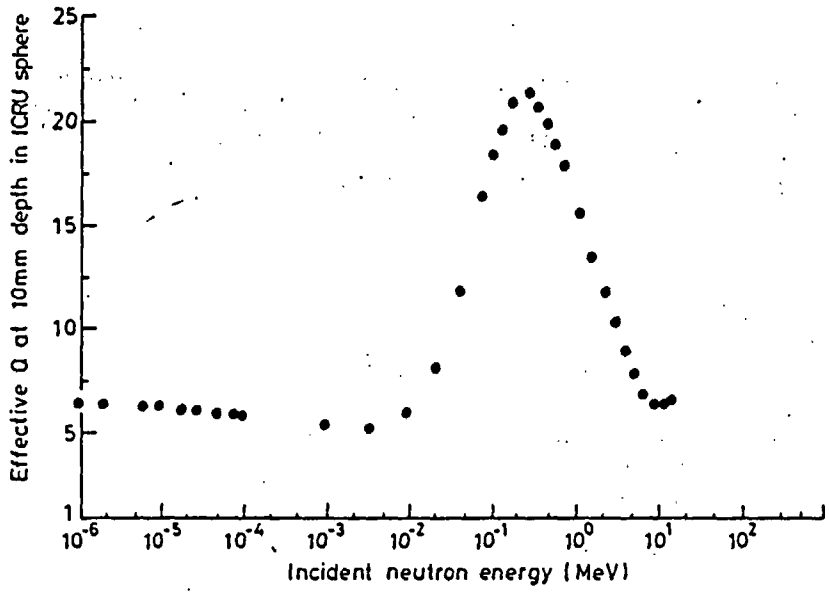

Fig. A-3. Effective Q, ($\bar{Q}$) as a function of neutron energy. Reference: Leuthold (1990).

A.10. Tissue Weighting Factors and Effective Dose

(A17) The relationship between the probability of stochastic effects and equivalen dose is found also to vary with the organ or tissue irradiated. It is, therefore, appropriat to define a further quantity, derived from equivalent dose, to indicate the combination o different doses to several different tissues in a way which is likely to correlate well witl the total of the stochastic effects. The factor by which the equivalent dose in tissue o organ T is weighted is called the tissue weighting factor, w_T. The values of w_T are chose so that a uniform equivalent dose over the whole body gives an effective dose numeri cally equal to that uniform equivalent dose. The sum of the tissue weighting factors i then unity. This weighted equivalent dose (a doubly weighted absorbed dose) ha previously been called the effective dose equivalent but this name is unnecessaril complicated, especially in more complex combinations such as collective committe effective dose equivalent. The Commission has now decided to use the simpler nam effective dose, E. The unit of effective dose is J kg^{-1}, with the special name sievert (Sv).

(A18) The effective dose, E, is the sum of the weighted equivalent doses in all th tissues and organs of the body. It is given by the expression:

$$E = \sum_T w_T \cdot H_T$$

where H_T is the equivalent dose in tissue or organ T and w_T is the weighting factor fo tissue T.

Evidently:

$$E = \sum_R w_R \sum_T w_T \cdot D_{T,R} = \sum_T w_T \sum_R w_R \cdot D_{T,R}$$

where $D_{T,R}$ is the mean absorbed dose in tissue or organ T delivered by radiation R. In both expressions the radiation is that incident on the body or emitted by a source within the body. The two forms of summation are clearly identical.

(A19) The recommended values for tissue weighting factors are given in Table A-3.

Table A-3. Tissue weighting factors[1]

Tissue or organ	Tissue weighting factor, w_T
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05[2,3]

[1] The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

[2] For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific w_T or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

[3] In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

A.11. Committed Tissue or Organ Equivalent Dose

(A20) Exposure to a radiation field of penetrating, externally applied radiation results in the simultaneous deposition of energy in a tissue. Tissue irradiation from incorporated radionuclides, however, is spread out in time, energy deposition occurring as the radionuclide decays. The time distribution of energy deposition will vary with the physicochemical form of radionuclide, and its subsequent biokinetic behaviour. To take account of this time distribution, the Commission recommends the use of committed equivalent dose which is the time integral over time τ of the equivalent-dose rate in a particular tissue that will be received by an individual following an intake of radioactive material.

When the period of integration τ is not given, a period of 50 years is implied for adults or a period of 70 years for children.

(A21) The committed equivalent dose is defined by:

$$H_T(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_T(t)\mathrm{d}t$$

for a single intake of activity at time t_0 where $\dot{H}_T(t)$ is the relevant equivalent-dose rate in an organ or tissue T at time t and τ is the time period over which the integration is performed. In specifying $H_T(\tau)$, τ is given in years.

A.12. Committed Effective Dose

(A22) If the committed organ or tissue equivalent doses resulting from an intake are multiplied by the appropriate weighting factors, w_T, and then summed, the result will be the committed effective dose.

$$E(\tau) = \sum_T w_T \cdot H_T(\tau)$$

In specifying $E(\tau)$, τ is given in the number of years over which the integration is made. The dose commitment ($H_{c,T}$ or E_c) is a calculational tool. It can be assessed for a critical group as well as for the whole world population. It is defined as the infinite time integral of the per caput dose rate ($\dot{H}_T$ or $\dot{E}$) due to a specified event, such as a unit of practice (e.g. a year of practice):

$$H_{c,T} = \int_0^\infty \dot{H}_T(t)\,\mathrm{d}t$$

or

$$E_c = \int_0^\infty \dot{E}(t)\,\mathrm{d}t$$

In the case of an indefinite practice at a constant rate, the maximum annual per caput dose rate ($\dot{H}_T$ or $\dot{E}$) in the future for the specified population will be equal to the dose commitment of one year of practice, irrespective of changes in the population size. If the practice is continued only over a time period, τ, the maximum future annual per caput dose will be equal to the corresponding *truncated* dose commitment, defined as

$$H_{c,T}(\tau) = \int_0^\tau \dot{H}_T(t)\,\mathrm{d}t$$

or

$$E_c(\tau) = \int_0^\tau \dot{E}(t)\,\mathrm{d}t.$$

A.13. Activity

(A23) The activity, A, of an amount of radioactive nuclide in a particular energy state at a given time is the quotient of $\mathrm{d}N$ by $\mathrm{d}t$, where $\mathrm{d}N$ is the expectation value of the number of spontaneous nuclear transitions from that energy state in the time interval $\mathrm{d}t$.