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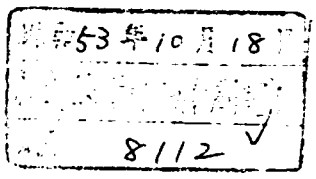
SOURCES AND EFFECTS OF IONIZING RADIATION

United Nations Scientific Committee
on the Effects of Atomic Radiation

1977 report to the General Assembly, with annexes



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ANNEX A

Concepts and quantities in the assessment of human exposures

CONTENTS

Introduction

1. The purpose of this Annex is to define the concepts and quantities that are used by the Committee in collating and reporting data on human radiation exposures and to qualify their use. These concepts and quantities can be unequivocally defined, but a number of basic biological assumptions have to be made to justify their use for particular purposes. While some of these assumptions are presented in this Annex, a more extensive discussion, which a fuller understanding of the qualifications requires, can be found in Annexes G, H, I and J, which deal with the biological effects of radiation.

2. The concepts and quantities discussed in this Annex are designed for the purpose of dealing with low doses of radiation, as usually encountered in occupational and population exposures.

I. BASIC CONCEPTS

3. The mean absorbed dose in various human organs or tissues is the physical quantity usually taken as a basis for radiation risk estimates, and it is derived by averaging the absorbed dose over the mass of the organ or tissue under consideration. The absorbed dose[1] is defined as the mean energy imparted per unit mass at the point of interest (3). In this report, the word "dose", unless specifically qualified, refers to the mean absorbed dose over an organ or tissue.

4. The use of absorbed dose in tissues as a basis for risk assessments makes it necessary to present separately

[1] The SI unit of absorbed dose is the joule per kilogram, the special name for the unit being the gray (1 Gy = 1 J/kg). In the present document use is made of the previous special unit for absorbed dose, the rad (1 rad = 0.01 Gy).

those components of the absorbed dose which are due to high-LET[2] radiation, with specification of radiation type. For low-LET contributions to the absorbed dose, which comprise the most common types of human exposures, it is assumed that the influence of radiation quality and dose rate is negligible.

5. The relations between doses in tissues and risks of deleterious effects are complex; they are presented in detail and discussed in Annexes G, H, I and J. If the effects in laboratory experiments under controlled conditions, as reported in Annexes H, I and J, are plotted against doses, many different types of curves are obtained, some linear and some curvilinear. A significant consideration is that in heterogeneous human populations of all ages and states of health and subject to a number of insults acting with radiation, there may be especially sensitive individuals, in which case a threshold may be less probable. In research it is appropriate to analyse dose-effect curves and factors relating to them in detail in order to gain an insight into the mechanisms involved. For purposes of making risk estimates in humans, however, a different approach has been employed. As described in Annex G, most of the effects of radiation in human beings have been observed at doses (some tens or hundreds of rads) that are considerably higher than those near natural background levels. To estimate risks at low doses the incidence of effects at high dose levels is divided by the dose to give an incidence rate per unit dose, which presupposes that there is proportionality between dose and effect down to the lowest doses. This assumption will be made for present purposes, recognizing that it is also possible that there could be a much lower effect, and perhaps none, at low doses. These considerations indicate that the rates of incidence calculated in these Annexes from observations at high doses may overestimate the effects of low doses in the millirad range.

6. In the present state of knowledge, the possibility that the dose-effect curve has a zero slope at these low doses cannot be ruled out. However, at doses around natural background it is assumed that an approximate proportionality exists between dose increments and corresponding increments of risk, provided that the increments are small. The additional assumption is made that the severity of each type of effect is independent of dose at low doses.

7. In the case of inhomogeneous dose distributions within an organ or tissue, the linear assumption implies that the inhomogeneity does not affect the relationship between risk and absorbed dose, provided that local doses are in the range in which proportionality applies and are small enough not to cause significant cell sterilization. If the latter condition is not met, cell sterilization could reduce the risk per unit dose, and assessments based on the mean absorbed dose over the entire organ or tissue might tend to overestimate the risk.

[2] LET (linear energy transfer) is used in the unrestricted sense as equivalent to the linear collision stopping power (3). Low-LET radiation, in this report, means radiation for which the linear collision stopping power in water is less than 3.5 keV/μm.

II. EXPOSURE OF POPULATIONS

8. When population groups are exposed to radiation from several sources, it is often interesting to compare the exposures and, under some assumptions, to assess the relative importance of the potential hazards due to each source. If the population groups were homogeneously and simultaneously exposed to radiation from these sources, the dose due to each source would give useful information about the relative importance of the source. In the more general case, however, the size of the exposed population and the distribution of doses over this population vary for different sources. Furthermore, the distribution of the doses in time is an important factor to be considered since it may also vary for different sources.

A. THE COLLECTIVE DOSE AND DOSE COMMITMENT

9. Practices involving radiation sources can give rise to a distribution of dose rates in the exposed population. No single quantity can represent adequately the distribution in all possible situations. In some cases, however, a useful quantity for this purpose is the collective dose rate. The collective dose rate $\dot{S}$ in a population from a practice or source is defined by the expression

$$\dot{S} = \int_0^\infty \dot{D}\, N_{\dot{D}}(\dot{D})\, d\dot{D}$$

where $N_{\dot{D}}(\dot{D})$ is the population spectrum in dose rate, $N_{\dot{D}}(\dot{D})\, d\dot{D}$ being the number of individuals receiving a dose rate in a specified organ or tissue, due to a source, in the range $\dot{D}$ to $\dot{D} + d\dot{D}$. Integrals of this type are usually referred to as "weighted products", the collective dose rate from a source being therefore the weighted product of dose rate due to the source and number of individuals in the exposed population. The collective dose rate is thus not a dose rate in the sense of absorbed dose rate as defined in ICRU report 19 (3). The actual population exposed should be specified whenever possible.

10. The collective dose in a population over a specified period of time is defined as the time integral of the collective dose rate. The collective dose is an extensive quantity that can apply to one person, to a population group or to the whole world population. It is expressed in man rad. The period of integration depends upon the purpose of the assessment. If the period of integration is short, the population may be assumed to be composed of the same individuals during the whole period. Conceptually, however, the population for which the collective dose is assessed could comprise successive generations.

11. The collective dose in a defined group of individuals can also be assessed as the weighted product of individual dose and number of individuals:

$$S = \int_0^\infty D\, N_D(D)\, dD$$

where $N_D\,(D)\,dD$ is the number of individuals receiving a dose, in a specified organ or tissue, in the range D to $D + dD$. The defined group may comprise individuals who live at different times or individuals living in a given year, depending upon the purpose of the assessment.

12. The average dose to an individual in the defined exposed group due to a source, called the *per caput* dose $\bar{D}$ from that source, can be calculated as

$$\bar{D} = \int_0^\infty D\,N_D(D)\,dD \Big/ \int_0^\infty N_D(D)\,dD$$

and since $\int_0^\infty N_D\,dD = N$, the total number of individuals in the defined group, it follows that $S = \bar{D}N$.

13. The collective dose and the collective dose rate are particularly useful quantities in source-related assessments. The collective dose rate from a source k, $\dot{S}_k$, is obtained by including in the population under consideration all individuals simultaneously being exposed by the source k. As the value of $\dot{S}_k$ remains unchanged if the population is made arbitrarily larger than the actual exposed group by adding unexposed individuals, it is convenient for the purpose of assessing the collective dose rate from a source to define the population as the world population. This specification is not necessary when the exposed group is small and well defined in a way that every exposed person can be accounted for.

14. In some cases, the world population may be conveniently subdivided into several groups labelled by $j = 1, 2, 3, \ldots$, the collective dose rate then being calculated as

$$\dot{S} = \sum_j \int_0^\infty \dot{D}\,N_{j,\dot{D}}(\dot{D})\,d\dot{D} = \sum_j \dot{S}_j$$

where $N_{j\dot{D}}\,(\dot{D})$ is the population spectrum in dose rate of group j, and $\dot{S}_j$ is the contribution of group j to the collective dose rate. The collective dose rate from source k can be assessed as

$$\dot{S}_k = \sum_j \bar{\dot{D}}_{k,j}\,N_j$$

where $\bar{\dot{D}}_{kj}$ is the *per caput* dose rate contributed by source k in group j, and N_j is the number of individuals in group j. All these quantities are functions of time.

15. In order to have a measure of the total exposure of the population caused by a source, the collective dose commitment is used. The collective dose commitment S_k^c due to a given event, decision or finite practice k is defined as the infinite time-integral of the collective dose rate, $\dot{S}_k(t)$, caused by that event, decision or finite practice:

$$S_k^c = \int_0^\infty \dot{S}_k(t)\,dt$$

As discussed above, the collective dose rate $\dot{S}_k(t)$ at a given time can be assessed by integration or summation over the world population.

16. As in previous reports, the Committee has also found it convenient to define a *per caput* quantity for the purpose of assessing the total exposure resulting from a given event, decision or finite practice. This quantity is the dose commitment D_k^c, defined as the infinite time-integral of the *per caput* dose rate due to event, decision or finite practice k:

$$D_k^c = \int_0^\infty \bar{\dot{D}}_k(t)\,dt$$

where the *per caput* dose rate $\bar{\dot{D}}_k(t)$ is the quotient of the collective dose rate and the population size at time t, i.e., $\bar{\dot{D}}_k(t) = \dot{S}_k(t)/N(t)$. If the population size remains unchanged over the time period contributing to the integral, we have $S_k^c = D_k^c N$.

1. Use of the collective dose and collective dose commitment in detriment assessments

17. Under the assumptions given in paragraphs 5 and 6, the collective dose can be used in assessments of the relative detriments from several radiation sources. ICRP has introduced the concept of "detriment" as a quantitative measure of the expected harm in a group of people as a result of a given radiation exposure (4). The detriment is defined as the mathematical expectation of harm, taking into account the probability of occurrence of each type of deleterious effect and the severity of the effect. If the group is composed of N persons and if p_i is the probability of incurring the effect i, the severity of which is expressed by a factor g_i, the detriment G is

$$G = N \sum_i p_i g_i$$

18. Considering only individual exposures to the same type of radiation, and making the assumption that the probability of each effect i attributable to the source under consideration is directly proportional to the dose from that source ($p_i = r_i D$, where r_i is the risk factor) and the further assumption that the severity of the effects is independent of their frequency in the dose range of interest, the health detriment in a homogeneously exposed group becomes $G = ND\Sigma_i r_i g_i$. In the general case where there is a distribution of doses over the world population, the detriment to health due to source k is given by

$$G_k = \int_0^\infty \sum_i (D_k r_i g_i)\,N_{D_k}(D_k)\,dD_k = S_k \sum_i r_i g_i$$

since the sum $\Sigma_i r_i g_i$ is independent of the dose distribution under the assumptions made.

19. The validity of this expression depends therefore on the assumed linearity, with no threshold, between dose and risk, as has been discussed in paragraph 5. The Committee has stressed that this is a cautious assumption, the validity of which has not yet been established. The risk factors r_i may vary both with the previously accumulated dose to which the dose D from the source under consideration is added and also with the dose rate and radiation quality. It cannot be assumed that the values of r_i which can be derived from observations at high doses and dose rates also apply to small dose increments at low dose rates.

20. However, for practices resulting in small dose increments to doses of similar order of magnitude, e.g.,

small dose increments above the natural background, as well as for the background doses as such, and provided there is no large dose-rate dependence, it may be assumed that, in the first approximation, one and the same value of $\Sigma_i r_i g_i$ would apply. In these conditions it would be possible to assess the *relative* detriment from two practices as the ratio of their collective doses: $G_1/G_2 \simeq S_1/S_2$.

21. The use of the collective dose for relative detriment assessments would also be valid in cases where the practices under comparison generate similar dose distributions over the population. Furthermore, if the real dose-risk relationships have an approximate proportional region, the comparison of collective doses would be valid in cases in which all the individual doses fall in the region of proportionality.

22. The evaluation of the relative health detriment resulting from practices which deliver the doses over quite different times can be done, provided that the assumptions of paragraphs 20 or 21 apply, by comparing the collective dose commitments from these practices.

2. Use of the dose commitment in predictions relating to continued practices

23. Both the dose commitment and the collective dose commitment are usually proportional to the size of the originating event. For example, if the event under consideration is the release of a quantity of a radionuclide to the environment, the dose commitment and the collective dose commitment are proportional to the activity released, provided all other influencing factors remain constant.

24. In these conditions, the values of the *per caput* dose rate at given times after the originating event are also proportional to the size of that event. It is therefore possible to define for a type of practice a function $\bar{D}_1(t)$, giving the *per caput* dose rate per unit of originating event (e.g., per unit of activity released) as a function of time elapsed since the event. It is consequently possible to define the dose commitment per unit originating event D_1^c, and the collective dose commitment per unit originating event S_1^c, as

$$D_1^c = \int_0^\infty \bar{D}_1(t)\,dt$$

and

$$S_1^c = \int_0^\infty \bar{D}_1(t)\,N(t)\,dt$$

where $N(t)$ is the population size. If the population size remains constant over the period contributing to the integral, we have $S_1^c = ND_1^c$. In the more general case, the calculation of collective dose commitments often requires the use of a population growth model. For short projections into the future an approximate exponential growth has been assumed in this report. On the other hand, for exposures delivered over very long periods of time, the assessments are based on an assumed upper limit for the world population of 10^{10} individuals.

25. A continued practice causing radiation exposures can be considered as a sequence of originating events discussed in the previous paragraph. The dose commitment per unit practice can be used for predictions of the *per caput* dose rate in the future for the case of a repeated practice at a known and constant rate R. The *per caput* dose rate will increase and for short-lived radionuclides eventually reach a steady value $\bar{D}_\infty$, which can be calculated by integrating the dose rate contributions at an arbitrary time T, when a steady state has been reached:

$$\bar{D}_\infty = \int_{-\infty}^{T} R\,\bar{D}_1(T-t)\,dt = R\int_0^\infty \bar{D}_1(t)\,dt = RD_1^c$$

In the simplified case of a constant population size, we have $S_1^c = ND_1^c$, and therefore $\bar{D}_\infty = (R/N)S_1^c$. In many cases it is possible to make rough projections of the practice rate *per caput* R/N. It is then possible to predict the maximum *per caput* dose rate that will be experienced in the future due to the expanded and continuing practice.

26. For doses delivered over a very long time after a single originating event, it would not be realistic to postulate a continued practice for the long times required for the *per caput* dose rate to approach the steady state value. Again, it is interesting to assess the maximum *per caput* dose rate, $\bar{D}_{max}$, caused in the future by the practice. If the practice continues to a time τ at rate R and then is discontinued, the *per caput* dose rate at any time $T > \tau$ is given by

$$\bar{D}(T) = \int_0^\tau R\,\dot{D}_1(T-t)\,dt = R\int_{T-\tau}^{T} \dot{D}_1(t)\,dt$$

It can be shown that if the function $\bar{D}_1(t)$ has a single maximum at time θ and then declines monotonically, the maximum *per caput* dose rate occurs at a time T_m such that $\tau < T_m < \tau + \theta$. Since in most practical cases $\tau >> \theta$ (and therefore $T_m \simeq \tau$), the maximum *per caput* dose rate is approximately given by

$$\bar{D}_{max} \simeq R\int_0^\tau \bar{D}_1(t)\,dt$$

27. The integral $\int_0^\tau \bar{D}_1(t)dt = D_1^\tau$ does not relate to the total detriment per unit practice, but it is useful in assessing the maximum *per caput* dose rate that will result from a continuing but finite practice. If the population size remains constant during the time contributing to the integration, it is convenient to express the maximum *per caput* dose rate as a function of the practice rate *per caput* R/N as

$$\bar{D}_{max} = (R/N)\,S_1^\tau$$

where $S_1^\tau = ND_1^\tau$ will be referred to in this report as the incomplete collective dose commitment per unit practice.

B. ENVIRONMENTAL MODELS USED IN THE ASSESSMENT OF DOSE COMMITMENTS

28. The chain of events leading from the primary release of radioactive substances to the irradiation of human tissues can be schematically represented by compartment models, in which the rates of transfer of

radioactivity between compartments are specified by constants or by time functions. The use of compartment models, even when very complex, implies considerable simplification of the real transfer processes.

29. Since the dose commitment from a given source is the integral over infinite time of the *per caput* dose rate resulting from the input, steps in the sequence from input to the final dose commitment can be conveniently described in terms of the quotient of the infinite time-integral of the appropriate quantity in step j of the sequence to the infinite time-integral of the appropriate quantity in the preceding step i. These quotients define the *transfer factors* P_{ij} in the pathway from input of radionuclides into the environment to the subsequent radiation dose in man:

$$P_{ij} = \int_{-\infty}^{\infty} M_j(t)\,dt \Big/ \int_{-\infty}^{\infty} M_i(t)\,dt$$

where P_{ij} is the factor relating compartment j and the preceding compartment i, and $M_i(t)$ and $M_j(t)$ are the appropriate quantities (e.g. activity concentrations) in the respective compartments at time t. The factors P_{ij} must be expressed in terms of the dimensions of the two quantities they link.

30. The network of pathways linking the release of radioactive material (input) to the dose commitment consists of pathways in series and in parallel. The total transfer factor of a branch (pathway in series) is the product of the transfer factors involved; the total transfer factor of several branches in parallel is the sum of the transfer factors of the branches. The dose commitment, therefore, can be assessed from the input Y by the relation

$$D^c = Y \sum_{\text{parallel}} \prod_{\text{series}} P$$

Transfer factors, their estimation for a number of nuclides, and the uncertainties involved in this estimation because of the variability of the transfer processes were discussed extensively in the 1969 and 1972 reports of the Committee (10, 11). The essential information from those reports is summarized in the relevant annexes of this report.

III. QUANTITIES USED IN THE ASSESSMENT OF ORGAN DOSES

31. In assembling information on radiation levels, the Committee, as in its previous reports, has selected primary data which can be used for the derivation of absorbed doses in human tissues, collective absorbed doses and absorbed dose commitments. Data which describe conditions of exposure to radiation in a useful way for the Committee's purposes are released activities[3] and activity concentrations, from which internal or external irradiation may be assessed, and quantities specifying ambient radiation levels, from which external irradiation may be assessed.

[3] The SI unit of activity is the second to the power minus one, the special name of the unit being the becquerel (1Bq = 1 s^{-1}). In the present document use is made of the previous special unit for activity, the curie (1 Ci = 3.7 10^{10} Bq).

A. INTERNAL IRRADIATION

32. In the case of internal irradiation, the doses in the organs of interest of a given individual may be estimated from one or more of the following basic determinations:

(a) Measurements of activity concentrations in the environment and in diet components, leading to estimates of the intake and, by the use of appropriate metabolic models, to estimates of the residence of the activity in the tissues of interest;

(b) Assessments of the activity in the body or in the relevant tissues, made on the basis of measurements of radiation emitted from the body or of measurements of activity in tissue samples;

(c) Measurements of the activity of excreta or exhaled air leading, by the use of appropriate metabolic models, to estimates of the activity in the body.

33. The use of these three types of information for the purpose of dose assessments requires the postulation of models to describe the body and tissue radioactive contents as a function of time. For example, for contamination through the diet, the activity in the body or a tissue, as a function of time, is given by

$$Q(t) = \int_0^t I\,C(\tau)\,A\,r(t-\tau)\,d\tau$$

where $Q(t)$ is the activity in the body or tissue of interest at time t,
I is the diet consumption per unit time,
$C(\tau)$ is the activity concentration in the diet at time τ,
A is the fractional absorption into blood of the ingested activity, and
$r(t)$ is the fraction of the activity absorbed into blood that is retained in the body or the tissue under consideration at a time t after a single uptake.

The basic data for this equation are the diet concentrations as a function of time. Similar formulations can be derived to make use of basic information of types *(b)* and *(c)* referred to in paragraph 32.

34. For the purpose of assessing dose commitments, the population weighted average of $Q(t)$ is needed. The parameters of the expression given in paragraph 33, as well as the function $r(t)$, are usually functions of age, and this must be taken into account in the derivation of the *per caput* value $\bar{Q}(t)$.

35. The dose commitment is calculated as

$$D^c = K \int_0^{\infty} \bar{Q}(t)\,dt$$

where K is a conversion factor appropriate to the population group. The conversion factors involved in the calculation of absorbed dose from $\int_0^{\infty} Q(t)dt$ do not necessarily relate to the same tissue or organ for which $Q(t)$ is given. For example, the dose in the red bone marrow and the endosteal cells may be caused by irradiation from radionuclides in bone. Also, different

parts of one and the same organ or tissue may receive different doses, but as already mentioned, it is the *mean* dose in the specified organs and tissues which is of interest to the Committee.

B. EXTERNAL IRRADIATION

36. The assessment of organ doses from external irradiation requires, in principle, the knowledge of the energy and angular differential distribution of the fluence rate of each component of the radiation field. These differential distributions can be obtained by spectrometric measurements, but in general only calculated values, for cases where the source distributions are known, are available.

37. Most reported field measurements provide values of integral quantities, usually describing the field in the absence of an exposed person—in "receptor-free" conditions. These quantities, if specified unambiguously, can be used for the assessment of organ doses of a person introduced in the radiation field, provided some information is available on the characteristics of the field. The present report uses two quantities to describe receptor-free irradiation situations: the absorbed dose rate in air $\dot{D}_a$ and the absorbed dose index rate $\dot{D}_I$.

38. The absorbed dose rate in air $\dot{D}_a$ is used in this report to describe environmental exposure situations resulting from gamma-emitting nuclides, and it is unambiguously specified when it may be assumed that full secondary electron equilibrium exists in air. In this condition, the absorbed dose rate in air can be derived from the exposure rate $\dot{X}$ as $\dot{D}_a = a\dot{X}$, where the value of the conversion factor a is 0.869 rad/R. The absorbed dose rate in air will usually be expressed in microrad per hour.

39. For describing receptor-free irradiation situations due to cosmic radiation it is convenient to select a quantity which, for the purpose of comparison, could equally be used at any altitude and in space. The absorbed dose rate can be used for this purpose but its magnitude depends upon the geometry of the material in which it is determined. Selection of a specified geometry for such a determination provides an unambiguous value for the absorbed dose rate. Such a specified geometry is contained in the definition of the absorbed dose index rate $\dot{D}_I$ which is used in this report to describe receptor-free environmental radiation levels due to cosmic rays. The absorbed dose index rate is defined as the maximum absorbed dose rate that would occur in a 30-cm diameter tissue-equivalent sphere if the sphere were located with its centre at the point of interest (3). The $\dot{D}_I$ also will usually be expressed in microrad per hour.

40. From the environmental quantities, the absorbed doses in a number of human organs of interest will be assessed, usually as annual doses or as dose commitments per unit practice. These assessments necessitate a number of assumptions, both about factors which influence the depth dose calculations and on the periods of time during which persons are exposed to the various radiation fields.

41. When the receptor is located in the region of interest, the procedure of assessing organ dose rates from absorbed dose rates in air involves the following considerations: Absorbed dose rates in air are multiplied by the s-factor[4] to transform them to tissue dose rate. The change from receptor-free conditions to the situation where a person is present in the radiation field will influence the field at the surface of the body. Back-scattered radiation may increase the dose rate at the surface, but the body will also act as a shield and reduce the dose rate. The influence of body shielding will depend upon the location of the organ of interest, and the energy and angular distribution of the radiation.

42. Except for the rare case where the radiation field is monodirectional and the irradiated person is not moving, the "depth" of an organ would be the weighted average of the distance in the body that would have to be penetrated by rays entering from different directions. The difference in absorbed dose rate at the surface of the body when the radiation is monodirectional and exposing the surface under consideration, and when the field is isotropic, may be substantial. The ratio of the "monodirectional dose rate" to the "isotropic dose rate" at any depth, when the fields produce one and the same absorbed dose rate in air under receptor-free conditions, may be referred to as the isotropy factor k at that depth. The isotropy factor cannot exceed 2 for points near the surface of the body, and it approaches 1 near the centre of the body and at all locations if the absorbed dose rate is constant throughout the body.

43. The assessment of organ doses requires therefore that the absorbed dose in air (after having been transformed to tissue dose by multiplication by the s-factor) be then multiplied by the depth transmission factor d, the backscatter factor b and a shielding factor, which would be the inverse of the isotropy factor $1/k$. The absorbed dose rate in an organ would be

$$\dot{D}_{org} = sbdk^{-1}\dot{D}_a = sg\dot{D}_a$$

where the product of the factors b, d and k^{-1} could be called the geometry factor g.

44. In the 1972 report, the Committee referred to gonad dose calculations by Bennett (2), indicating that $sg = 0.82$ for external outdoor exposure (2π geometry), and to gonad dose measurements by Spiers and Overton (9), indicating that $g = 0.63$ ($sg = 0.69$) for exposure indoors. By transport calculations, O'Brien and Sanna (6) have assessed absorbed doses in several organs (in the MIRD phantom (8)) per unit exposure, as a function of the energy of gamma rays. Combining these calculations with the environmental gamma-ray exposure spectrum in energy (1), they have calculated organ absorbed doses per unit exposure for environmental conditions. From these calculations sg for gonads can be estimated to be about 0.8 in males and about 0.55 in females. As the mathematical phantom, however, uses the Reference Man mass (5) in all calculations, the factor for female gonads is somewhat underestimated. O'Brien (7) has

[4] The s-factor is the ratio of the mass energy absorption coefficients for tissue and air. A value of $s = 1.10$ is correct within ± 1 per cent for photon energies between 0.1 and 4 MeV for soft tissues.

recently calculated the organ doses for a female phantom. From these calculations the value of *sg* for female gonads is estimated to be about 0.6.

45. If the occupancy factor (i.e., the fraction of the year during which a person is actually exposed to a given source) is q, the annual gonad dose, assuming constant environmental irradiation levels, can be derived from the absorbed dose rate in air as

$$D = c_\gamma q \dot{D}_a$$

where c_γ = 7.2 mrad μrad^{-1} h for outdoor exposures and 6.0 mrad μrad^{-1} h for indoor exposure, using the values of *sg* given in the 1972 report. The calculations of O'Brien and Sanna (6) give a value of *sg* equal to about 0.7 for the ratio between the average absorbed dose in the body and the absorbed dose in air, and show that the doses in other organs of interest to the Committee are very nearly equal to the gonad dose. It will be assumed that the equation given above applies also to organs such as the breast, the lung and the red bone marrow.

46. In the case of exposures to cosmic radiation, it will be assumed that the absorbed dose index rate represents sufficiently well the relevant organ dose rates in an individual introduced at the location of consideration. If the occupancy factor for that location is q, the annual organ dose can be derived from the absorbed dose index rate, assuming constant environmental levels, as

$$D = c_c q \dot{D}_I$$

where D is the annual organ dose, and c_c is 8.76 mrad μrad^{-1} h.

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