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NOTE

Throughout the present report and the annexes thereto, references to the annexes are indicated by a letter followed by a number: the letter denotes the relevant annex and the number the paragraph therein. Within each annex, references to its scientific bibliography are indicated by numbers.

Symbols of United Nations documents are composed of capital letters combined with figures. Mention of such a symbol indicates a reference to a United Nations document.

ANNEX H

COMPARISON OF DOSES AND ESTIMATES OF RISK

CONTENTS

I. Introduction

1. Much information has been reported since 1958 on dose levels and effects of radiation in animals and man but there are still many gaps in knowledge. It is not possible to determine with desired precision the effect on the world population of the doses from natural radiation (annex E), medical and occupational exposure (annex G) or from fall-out from weapon testing (annex F, part III).

2. Accurate assessment of the late effects of low dose exposure requires a full knowledge of the relevant effects, of the tissue or tissues involved and of the dose-response relationships. At the present time there is a lack of information with regard to each of these factors and also, of course, uncertainty regarding future levels of radiation. In the present annex various of the problems will be reviewed. The first part of the annex deals with the general problems involved in making risk estimates, and presents a method for comparing the risks from various sources of exposure. The second part discusses the problems associated with the assessment of biologically significant doses, estimates of which are presented in the third part. Finally, genetic and somatic risks from the various sources of man's exposure are compared.

II. Problems associated with risk estimation

3. Knowledge of the late effects of radiation comes from clinical and experimental data at much higher levels of dose (and often of dose-rate) than those of natural radiation, fall-out and many types of medical exposure.[1-4] The types of effect, or their dose-response relationships, will not necessarily be the same at low as at high dose levels.

4. The estimation of risk at low dose levels requires answers to three basic problems:

(*a*) The effects to be considered;

(*b*) The critical tissue for each of these effects;

(*c*) The function of dose, dose-rate and dose distribution to be taken as the relevant exposure parameter for each of these effects.

EFFECTS AND RELEVANT TISSUES

5. Both genetic and somatic effects have to be considered. The genetic effect is the production of mutations and the critical organs are the gonads. Possible late somatic effects which have given rise to most concern are the induction of leukaemia, malignant bone tumours, and the reduction of life expectancy. The significant tissue for the induction of leukaemia is generally considered to be the active bone marrow. With bone tumours, early pre-malignant changes have been reported[5] in the connective tissue lining endosteal surfaces or trabeculae and in the loose connective tissue in bone marrow spaces between trabeculae; these are therefore probably the tissues of importance. There is as yet no information concerning tissues important in the reduction of life expectancy. Therefore no estimates will be made of the risk associated with this effect. Although limited exposure data are available for other organs such as thyroid, gastro-intestinal tract and lung, these tissues will

not be used for risk estimates because of uncertainties in dose-effect and dosimetric parameters. More information, including the specification of the relevant effect, would be required for any careful assessment of risk to foetal tissue.

Dose-effect relationship

6. There are two considerations concerning the exposure parameter to be used: (*a*) what is theoretically justifiable? (*b*) what is practicable? For genetic effects experimental data justify the use of a linear dose relationship at low doses and dose-rates. No such generalization can be made about late somatic effects of radiation. In radiation carcinogenesis at high dose levels many different mechanisms may play a part, including various kinds of interactions between damaged cells and tissues, effects of vascular and hormonal changes, as well as specific radiation-induced changes in cells. Also there may be several different ways in which the same macroscopic effect can be brought about, so that the equivalence of macroscopic effect does not imply equivalence of primary mechanism. Carcinogenicity, at these high levels of radiation dose, could only be described by a very complex function of dose and other exposure factors.

7. One would expect, however, that the mechanisms of production of any late effects are simpler at lower doses because interactions between damaged cells, as well as general systemic effects of radiation, will play a smaller part. Although the possible importance of subtle generalized changes in tissue cannot be ruled out, it is likely that, if serious late effects can arise at these low dose levels, they will result predominantly from specific changes induced in individual cells.

8. If it is assumed that even the smallest dose entails a finite probability of effect, can any statement be made about the shape of the dose-response curve near the origin? For certain radiobiological effects which have a non-linear relationship at high dose levels (e.g. certain types of chromosomal change induced by radiation), it is probable that the slope of the dose-effect curve near the origin is linear. However, the range of effective linearity may be very limited. Formally, if the dose function which determines the incidence of the effect includes a linear dose term, however small, it is this term which will be controlling at the lowest doses. The assumption of a linear dose-effect relationship normally implies that mean accumulated dose, i.e., cell-rad, within the tissue of interest can be taken as the significant dose parameter for calculation of incidence of late effects. Protraction of exposure and non-uniformity of dose within the tissue of interest can be ignored. However, if a non-linear dose relationship holds, a mean dose cannot be used to estimate the incidence of effects. Also with a non-linear dose-effect relationship, the dose-rate might be an important factor.

9. The assessment of risk of specific late somatic effects on the basis of a given dose-effect relationship must take into account the way in which injury is distributed over time. In the time-incidence curve the important parameters will be the shortest latent period L before any effect is manifest and the subsequent shape of the curve. For example, figure 1 shows the time-incidence curve deduced from data on the incidence of leukaemia after radiation therapy for ankylosing spondylitis.[6]

10. If the probability of the induction of a specific late effect (figure 2a) has fallen to zero after a given dose of

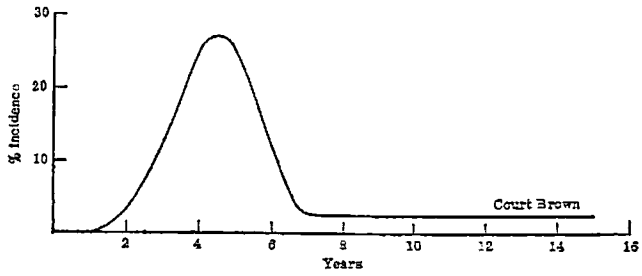

Figure 1. Time incidence curve of leukaemia

radiation in a time which is short compared with the mean life span of an individual (curve 1), a linear dose-effect relationship implies that the area under the curve, i.e., the total incidence of injury, is proportional to dose. If with a reduction in dose there is an increase in the length of the shortest latent period or a change in the shape of the time-incidence curve (curve 2) so that the probability of induction of the effect has not dropped to zero at the end of life, any statement about the dose-effect relationship—linear or otherwise—cannot be made without consideration of life expectancy.

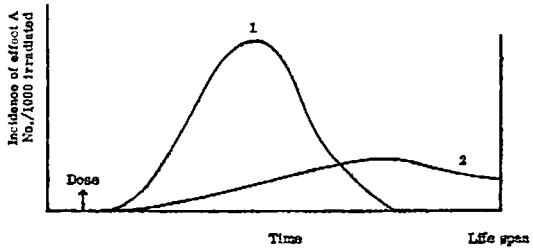

*Figure 2*a (see para. 10)

11. Similarly if, whatever the dose, the effect has a finite probability to the end of life (figure 2b), any simple statement concerning linear relationship can only be made if the shortest latent period does not change with the dose, and if the probability of injury at any later time is proportional to the dose.

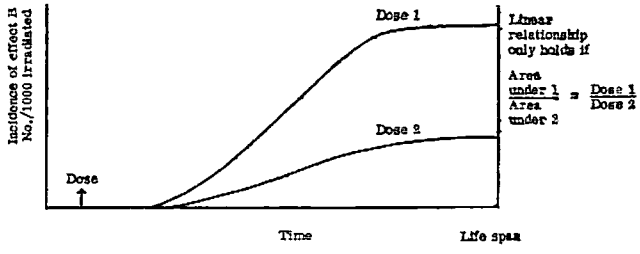

*Figure 2*b (see para. 11)

12. In the present state of knowledge, mean tissue dose is the only parameter that can be used to estimate risks in populations. If the dose-effect relationship is non-linear the use of a per capita mean tissue dose will be inapplicable, and individual dose and dose distribution would need to be considered; this would be a much more difficult task.

13. So far as an absolute assessment of risk is concerned, that is, an estimate of the actual number of effects from a given radiation exposure, a clear distinction must be made between the genetic and somatic problems. For radiation-induced genetic changes there is good experimental evidence that the dose-effect relationship is linear; the difficulty of making absolute assessments for

a human population lies in lack of knowledge of the slope of the dose-effect curve under various conditions, and uncertainty about the way in which an increased mutation rate will be expressed in a human population.

14. For somatic effects there are no experimental data relevant to the form of the dose-effect curve at low doses and, even at high doses, as indicated in annex D, there are very few reliable dose-response data for late effects. Thus, although the assumption of a linear dose-effect relationship at low doses may be made, there is no means at present of arriving at the actual value of the slope. However, even if adequate dose-response data were available at high doses, any extrapolation to low doses would involve large assumptions on: (*a*) the dose-effect relationship; (*b*) the latent period for manifestation of the effects; and (*c*) dose-rate dependence. For these reasons it is felt that the use of mean dose as the risk parameter can be used only to estimate the comparative risk from various sources, and not absolute risk.

Basis of risk comparison

15. In using the mean dose in calculations of comparative risk of natural radiation, fall-out and medical exposure, it is necessary to take into account that: (*a*) the yearly dose from natural radiation is constant; (*b*) the yearly dose from medical exposure is varying; and (*c*) the yearly dose from fall-out not only varies but the radiation exposure continues long after the event.

16. It is suggested that the basis of comparison should be the number of injuries resulting from procedures carried out during any given period of practice. For natural radiation and medical exposure the period of practice and the period of radiation exposure will coincide. For environmental contamination the period of radiation exposure will greatly exceed the period of practice.

17. This method of treatment permits comparison of total numbers of injuries occurring over all time. To obtain information on the yearly incidence in a population, or on the risk to a given individual, other methods of treatment would be required which, in the present state of knowledge, would involve so many assumptions as to be of little value.

18. The term "dose commitment" is used for the radiation dose resulting from procedures carried out during a given period of practice. For natural background and medical exposure the dose commitment will be the dose actually received during the period of practice. For environmental contamination the dose commitment will be the dose received during the selected period together with that received subsequently as a result of events during the period, i.e., an integration of dose to infinite time.

19. The term "dose commitment" is applied not to individuals but to populations only and represents the mean tissue dose (i.e., the dose to the total pool of specified cells) within the population.

20. Although the dose commitment from environmental radiation due to a given period of practice involves an integration to infinite time, the major fraction of the dose, apart from the contribution from C^{14}, will have been delivered within fifty years. This implies, of course, that a considerable fraction of whatever somatic effects may arise from a given test will have appeared within about fifty years. Any more detailed statement than this on the rate of appearance of somatic effects would require a knowledge of the time-incidence relationships at the relevant levels of dose and dose-rate.

21. In the present annex, dose commitments will be based on world-wide averages of dose from the various sources and, for the comparative risk estimation, the ratio of the respective dose commitments will be used.

Weighting factors for population

22. When considering the genetic effect of uniform irradiation of the population, each increment of time, and therefore of dose to the gonads, contributes an equal number of mutations to the population so long as the age distribution of the population remains the same. However, when only certain individuals in a population are irradiated, as in medical radiology, the gonad doses will have varying importance depending on the age of the individual, as the probable number of children to be born to the individual must be taken into account. The term "genetically significant dose" is defined (G. 9-12) as that dose which, if received by every member of the population, would be expected to produce the same genetic injury to the population as do the actual doses received by the various individuals. This population dose is obtained by weighting the individual gonad doses by a relative child expectancy factor so as to make possible comparisons with doses from sources to which populations are uniformly exposed.[9-12]

23. It is very probably that there is a considerable age dependence in the development of late somatic effects of radiation, but there is at present no information on which to base appropriate weighting factors. In the present calculations it has been assumed that the average latent period, for the somatic effects considered, is short compared with the normal life span, and has not therefore been taken into account.

24. The growth of world population also has to be considered. The expression "cell-rad" implies the product of two terms, one related to numbers of cells, and the other to dose. In the case of the risk comparison between medical exposure and exposure from natural radiation the "cell" term will be identical for each dose commitment, and the comparison can be based solely on the ratio of the doses. However, the dose commitment from nuclear testing will be delivered during a period of time in which the size of population (and thus the number of cells) will increase. Ideally, this increase would have to be taken into account in the calculation of the dose commitment. In view of the uncertainty of the estimates of future world population, this factor has not been taken into account in the comparison of risks.

III. Problems associated with the estimation of the dose received by body tissues

25. As has been explained above, comparative risk assessments will be made for genetic effects, induction of leukaemia and induction of bone tumours. Radiation doses from the various sources must therefore be calculated for the relevant critical tissues.

26. The estimation of the radiation doses to any tissue must include contributions from external and internal sources. The conversion of exposure dose measured outside the body to absorbed dose in the relevant tissues can be made by calculation, but often only

with major assumptions. Alternatively, measurements may be made on tissue-equivalent "phantoms", but these will also have limitations since phantoms can only approximately simulate man.

27. In determining the contribution from internally deposited radio-nuclides it is necessary to recognize that the mean dose to the relevant tissue will not necessarily be the same as the mean dose to the organ containing that tissue, if dose distribution throughout the organ is not uniform.

28. This problem does not arise with the gonads since it may normally be assumed that the distribution of radio-nuclides is uniform throughout the gonads and the dose in all parts of the gonads, including the germinal cells, will be the same.

29. The dose to bone from bone-seeking radio-nuclides, such as Sr^{90}, may not be uniform. There is the additional complication, in estimating dose to bone surfaces, in that the lack of electron equilibrium near the surface has to be taken into account. With single injections of bone-seeking nuclides the problems of dose estimation may be very severe since there will be "hot spots", i.e., high local concentrations of radio-activity, in areas of bone growth and remodelling. However, with continuous intake of radio-nuclides, non-uniformity will be much less, particularly with beta-emission of relatively long range (e.g. $Sr^{90} + Y^{90}$).

30. For continuous uniform ingestion of radio-active materials during steady bone growth, as in young children, the distribution of activity in a given bone is relatively even. In the adult there will be greater non-uniformity, but there is much still to be learned about the effect of age. The effect of such variations on the dose to bone and bone marrow has been discussed.[7] There is evidence from recent studies of a substantial variation between different bones, but presumably this will become less marked with prolonged ingestion.

31. Another factor affecting the calculation of bone dose from internally deposited radio-nuclides in the adult is the degree of mineralization of bone, which also may change considerably with age. This again is a subject on which much further information is required.

32. In the present calculations the major problem, in determining the internal dose to the bone surface and bone marrow, is the contribution from Sr^{90} derived from fall-out. There is some contribution in natural radiation from the α-emitters, but this dose represents only about 10 per cent of the total from natural sources.

33. Assuming no gross non-uniformity in dose distribution in bone, the Sr^{90} contribution to the mean dose at the surface of bone will be approximately one half of the mean Sr^{90} skeletal dose derived in annex F, part III. With regard to the bone marrow dose from Sr^{90} in the bone, it is shown in annex F, part III, that the mean bone marrow dose within trabeculae will be approximately one-quarter of the mean skeletal dose. These factors have been used in the present calculations.

The problem of RBE (relative biological effectiveness)

34. As has been shown in annexes B and D, the value of the RBE of ionizing radiations of different characteristics, e.g. neutrons and X-rays, depends on the biological effect considered.[8,9] For the assessment of any given biological effect, it is clear that a precise analysis requires an RBE for each of the radiation conditions as well as for each effect under consideration. However, values of RBE that have been obtained experimentally apply only to the conditions under which the measurement was made. At the present time there is no information on the RBE values appropriate to the production of specific late effects in man, and without this information there is no alternative but to use the values adopted by ICRP. The values of RBE quoted by the ICRP, reproduced in annex A, have been chosen as those which are unlikely to be exceeded under conditions of occupational exposure.

Effect of transmutation of C^{14}

35. One further outstanding problem is that associated with the interpretation of the effects of the incorporation of C^{14} into body tissue. Carbon atoms make up about 37 per cent of the deoxyribonucleic acid (DNA) which is an important constituent of chromosomes and is associated with the genes. Hence if a C^{14} atom becomes incorporated into a DNA molecule and later distintegrates, the DNA molecule may be damaged not only by the ionizing beta particle emitted and the recoiling nucleus, but also by the transmutation of the C^{14} atom to N^{14}, a process which might also give rise to a gene mutation (annex B).

36. Estimates of the magnitude of the transmutation effect vary from one tenth to many times the effect due to ionization[10–13] and more experimental data are needed before a reliable assessment of this effect can be made.

IV. Comparison of doses

37. With the reservations outlined in the previous sections and in the relevant sections of the other annexes, the doses to present and future generations are summarized in the following paragraphs.

Doses from natural radiation

38. Natural radiation includes cosmic rays, radiations from radio-active nuclides in the earth and in building materials, and radiations from internal radio-activity. The yearly population doses to gonads and bone marrow are given in table I. These represent only average values of natural radiation; they do not reflect the large variations throughout the world.

Doses from medical exposure

39. Table I also gives representative values of the yearly dose due to medical exposure. During the next decade the availability of X-ray facilities will be much greater throughout the world, and information will be required regarding the doses to the larger numbers of people being examined or treated. This expansion cannot be predicted, nor can the possible development of more conservative procedures. For the purpose of this annex, the doses to the population will be assumed to be constant. Although much smaller, the doses from occupational exposure and miscellaneous sources of radiation are included in table I.

Doses from fall-out

40. The world average doses (weighted for population distribution) resulting from fall-out do not include the doses from local fall-out within the first few hundred miles from megaton surface nuclear explosions.

41. The estimation of the dose from current fall-out is possible with some accuracy on the basis of observed data. However, when one attempts to predict levels of activity on the ground or in foodstuffs due to past and future testing, and to derive the ensuing doses, the unknowns make any estimate extremely difficult.

42. In the case of external exposure and internal exposure from substances with rapid turnover, the dose-commitment from a given period of practice can be calculated as the dose actually received up to the present date plus the dose to be expected in the future. For isotopes, such as Sr^{90}, with slow turnover, the same calculation is more difficult. As has been shown in annex F, part III, the dose-commitment from Sr^{90} can be derived from an integral of the environmental contamination

$$D_\infty = k \int_{t_0}^{\infty} c\,(t)\,dt$$

If experimental data are available for the period t_0 to t_1, the dose commitment can be written

$$D_\infty = k \left(\int_{t_0}^{t_1} c\,(t)\,dt + \int_{t_1}^{\infty} c\,(t)\,dt \right)$$

where the first integral can be evaluated from the measured values of c(t) and the second integral has to be derived from a predicted future environmental contamination. It should be realized that the dose actually received during the period t_0 to t_1 is only part of the first integral (F III, 67).

43. The dose-commitments given in table II have been calculated on the following assumptions with regard to testing conditions:

(a) *Testing up to the end of 1960.* The dose-commitments can partly be derived from experimental data (cf. para. 42). The future doses have been calculated on the basis of a total atmospheric injection of 6.6 Mc Sr^{90} and 2.2×10^{28} atoms of C^{14}.

(b) *Future testing.* As a model to be used for the calculation of the doses from possible future testing, it has been assumed that the yearly rate would involve the injection of 1 Mc Sr^{90} and 10^{28} atoms of C^{14} into the atmosphere;

(c) *Testing during the period 1954 to 1961 inclusive.* Since the experimental data do not permit an assessment of the atmospheric injection of Sr^{90} and C^{14} during 1961, it has been assumed for the purpose of the dose estimates that the total injection during the period 1954-1961 (8 years) was $(6.6 + 1) = 7.6$ Mc Sr^{90} and $(2.2 + 1)\ 10^{28} = 3.2 \times 10^{28}$ atoms of C^{14}. It is shown in annex F, part I, that this is the most reasonable estimate that the Committee can at present venture to make.

V. Comparative genetic and somatic risk estimates

44. The period of practice for which the dose commitments have been calculated is the period 1954-1961 (eight years). It has been assumed that all the weapon tests were carried out during this period and none previously. This period has been used because it is difficult to analyse the measurements of fall-out to determine the actual doses likely to be received from any one series of nuclear testing, but only the doses arising from the total testing so far carried out.

45. The dose commitments to all generations due to nuclear weapon testing during this period are given in table III for the genetic risk and for the selected somatic risks (induction of leukaemia or bone tumours). These dose commitments are compared to the dose commitments from natural radiation and medical and occupational exposure during the same period. For these two latter sources, the dose commitment for any one year is equal to the dose actually experienced during the same year.

46. It can be seen from table III that, for the period chosen (i.e., 1954-1961), the comparative genetic risk from fall-out is about one-tenth of that from natural sources. The genetic risk from medical exposures* is about one-third of that due to natural sources. Fall-out contributes to the dose commitment for the induction of leukaemia and bone tumours between one-quarter and one-sixth of that from natural sources.

47. If no further testing is carried out, the relative importance of the dose commitment due to previous tests will decrease in comparison with the accumulated doses from natural sources and medical exposure. The figures in table III indicate that the whole series of tests during 1954-1961 will give a dose commitment corresponding to about one to one and a half years of exposure from natural radiation.

48. In the event of continued testing at a constant rate of injection, equilibrium conditions would obtain in about 100 years, except for C^{14}. For this nuclide equilibrium conditions would imply many thousands of years of testing. The dose commitment from one year of injection is numerically equal to the yearly dose under equilibrium conditions at the same rate of injection. The dose commitments per year of testing at the assumed rate are compared with the dose commitments due to one year of natural irradiation and medical and occupational exposure in table III, columns 5-7.

49. If the dose commitment is derived from an integration of future doses to infinite time, the dose from C^{14} will be found to contribute more than 60 per cent of the total dose from fall-out. If, however, the integration is only carried out to the year 2000, the contribution from C^{14} is only about 5 per cent of the dose from fall-out.

Summary

50. The assumption of a linear dose-effect relationship and the use of the mean tissue dose have been used to estimate the comparative risk of the doses from the various sources of radiation to which the population is exposed, but there are insufficient data to make absolute risk estimates at the present time.

* See table I, footnote.

Table I. Average yearly dose to the population

	Genetically significant dose (mrem)	*Bone marrow dose (mrem)*	*Cells lining bone surfaces (mrem)*
Natural radio-activity.....	125	122	130
Medical exposure[a]			
Diagnostic.....	30	50-100	?
Therapeutic.....	5	?	?
Occupational and miscellaneous exposure.....	~2	?	?

[a] Based on the values reported in annex G.

TABLE II. DOSE COMMITMENT FROM NUCLEAR TESTING

Organ	Contribution	Tests 1954–1961: Dose commitment (mrem)	Tests 1954–1961: Fraction of dose commitment reached by 2000	Future tests: Dose commitment per year of testing (mrem)
Gonads	All sources but C^{14}	41	0.97	7
	C^{14}	70	0.10	22
	TOTAL	111	0.42	29
Cells lining bone surfaces	All sources but C^{14}	128	0.94	20
	C^{14}	116	0.10	37
	TOTAL	244	0.54	57
Bone marrow	All sources but C^{14}	84	0.94	13
	C^{14}	70	0.10	22
	TOTAL	154	0.56	35

TABLE III. COMPARISON OF RISK[a]
(*Dose commitment to all generations*)

	Tests 1954–1961: Dose commitment (mrem)						Future tests: Dose commitment per year of testing (mrem)					
	Gonads		Cells lining bone surfaces		Bone marrow		Gonads		Cells lining bone surfaces		Bone marrow	
Natural sources	1,000	(1.00)	1,040	(1.00)	1,000	(1.00)	125	(1.00)	130	(1.00)	125	(1.00)
Medical and occupational[b]	300	(0.30)	?		400–800	(0.4–0.8)	37	(0.30)	?		50–100	(0.4–0.8)
Fall-out:												
All but C^{14}	41	(0.04)	128	(0.12)	84	(0.08)	7	(0.06)	20	(0.15)	13	(0.10)
C^{14}	70	(0.07)	116	(0.11)	70	(0.07)	22	(0.18)	37	(0.28)	22	(0.18)
Total fall-out	111	(0.11)	244	(0.23)	154	(0.15)	29	(0.23)	57	(0.43)	35	(0.28)

[a] Figures in parentheses indicate contribution relative to natural sources.

[b] See table I, footnote.

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