REPORT
OF THE
UNITED NATIONS SCIENTIFIC
COMMITTEE
ON THE
EFFECTSOF ATOMICRADIATION

UNITED NATIONS

New York, 1988

NOTE

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.

CONTENTS

CONTENTS (continued)

CONTENTS (continued)

I. INTRODUCTION

1. This is the tenth in a series of substantive reports of the United Nation8 Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1/ to the General Assembly. 2/ The preparation of the present report and it8 scientific annexes took place from the thirty-first to the thirty-seventh sessions of the Committee. The material of the report was developed at annual sessions of the Committee, bared on working paper8 prepared by the secretariat that wore modified and amended from one session to the next according to the Committee's requests. During the period of preparation of the report, which contains seven scientific annexes, another report containing three scientific annexes warn completed at the thirty-fifth session of the Committee. Theme two reports, referred to am the 1986 and 1988 reports, constitute the latest comprehensive assessment by the Committee of the ● ourcea, effects and risks of ionising radiation.

2. The following members of the Committee served am Chairmen, Vice-Chairmen and Rapporteurm, respectively, at the following sessions: thirty-first session, 2. Jaworowski (Poland), D. Beninson (Argentina) end T. Kumatori (Japan)) thirty-eecond and thirty-third sessions: D. Beninson (Argentina), T. Kumatori (Japan) and A. Hidayatalla (Sudan); thirty-fourth and thirty-fifth sessions: T. Aumatori (Japan), A. Kaul (Federal Republic of Germany) and A. Hidayatalla (Sudan); thirty-sixth and thirty-seventh sessions: B. Linde'l (Sweden), K. H. Lokan (Auatralia) and J. Maisin (Belgium). The names of those expert8 who attended the thirty-first to the thirty-seventh sessions of the Committee in an official capacity am representatives or members of national delegations are lirted in appen‐ix I.

3. In approving the present report, and assuming therefore full responsibility for its content, the Committee wishes to acknowledge the help and advice given by a small group of consultants who assisted in the preparation of the text and scientific annexes, upon appointment by the Secretary-General. Their name8 are given in appendix II. They were responsible for the preliminary reviews and evaluation of the technical information received by the Comnittee or available in the open scientific literature, on which rest the final deliberation6 of the Committee. A'ditional assistance and financial support for the preparation of some of the s'entific annexes were offered to the Committee by various international and national organisations. The Committee would like to express its gratitude to theme organizations, which are listed in the relevant annexes.

4. The sessions of the Ccmnittee held during the period under review were attended by representatives of the United Nations Environment Programme (UNEP), the World Health Organisation (WHO), the Food and Agriculture Organisation of the United Nation6 (FAO), the International Atomic Energy Agency (IAEA), the International Commission on Radiological Protection (ICRP) and the International Comnimmion on Radiation Units and M,asurements (ICRU). The Committee wishes to acknowledge their contributions to the discussions.

5. Reports received by the Committee from Member States of the United Nations and members of the specialised agencies and IAEA, as well as from chese agencies themselves, during the period from 19 April 1986 to 17 Juns 1988 are listed in appendix III. Reportm received before 19 April 1966 were listed in previous reports of the Committee to the General Assembly. This information received officially by the Committee was supplemented by, and interpreted with the help of,

many other data available in the current scientific literature or, in a few cases, from unpublished communications by individual scientists.

6. In the following *report* the Committee *summariser* the main conclusions of the specialised studies undertaken, also in the light of previously released substantive documents. The material is presented at the most general level possible, *in* view of the difficult concepts and notation that characterise this field. After a chapter summarising the developments and trends that have become apparent throughout thr years, the highlights and conclusions to be drawn from the most recent studies in the fields of radiation physics and biology are presented. This main text is followed by the supporting scientific annexes, which are written in a format and a language that are essentially aimed at specialists.

7. Following established practice, only the main text of the report is submitted to the General Assembly, while the full report, including the scientific annexes, will be issued as a United Nations sales publication. This practice is intended to achieve wider dissemination of the findings for the benefit Of the international scientific community. The Committee wishes to draw the attention of the General Assembly to the fact that the main text of the report is presented separately from its scientific annexes simply for the *sake* of convenience. It should be understood that the scientific data contained in the annexes are of groat importance because they form the basis for the conalurions of the report.

II. HISTORICAL REVIEW

A. General considerations

8.   Throughout the 33 years of its existence, the Committee has assertively attempted to provide the best possible estimates of :

(a) Doses received by the world's population in the past, and expected to be received in the future, from various natural and man-made sources of radiation)

(b) Risks of induction of various types of harm by radiation, both in the short term and the long term, by individuals directly receiving such doses or by their descendant6 over many generationc.

9.   With the passing of time and the increase in number aud complexity of the reports issued by the Committee, it is becoming increasingly difficult, even for the specialists, to trace back to earlier publications the development of the main ideas underlying the Committee's assessments and how these assessments have changed with time and as a result of increasing scientific knowledge.   It would seem useful, therefore, to make available in compact, summary form the main conclusions reached in the fields mentioned above.   This summary is intended to serve a number of purposes.   First, it will inform the General Assembly about the Committee's work and its findings.   Second, for the Committee's membership, which has been changing gradually over the years, it will form a record of how the Committee's thinking has evolved.   Lastly, it will be placed at the disposal of the international scientific communri t-y, for whom UNSCEAR reports and scientific annexes have become a basic reference.

10.   What follows in this chapter is therefore a summary of the Committee's assessments in the fields of dose estimation (which pertains closely to the subjects of physics) and risk assessment (which involves physical as well as radio-biological and medical considerations).   It aims at giving an account of both the general principles underlying the estimates and the conclusions reached, in a language that is as plain as the complexity of the subjects allows but without much of the discussions supporting the choices made at any particular time.   For this, as well as for other technical and methodological dstails, reference is made to the reports to the General Assembly issued from 1958 to 1986.   A complete list of these publications issued by the Committee appears in footnote 2/ to paragraph 1 of the present report.   Current assessments are examined in more detail in chapter III.

B. Concepts, quantities and units

11.   Radiation is a transport of energy through space.   In traversing material, radiated energy is absorbed.   In the case of ionizing radiation, which is the type of radiation that concerns the Committee, the absorption process consists in the removal of electrons from the atoms, producing ions.   Ionizing radiation may be produced in man-made devices, such as X-ray tubes, or it may come from the disintegration of radioactive nuclides, the phenomenon that is called radioactivity.   While nuclides such as these occur naturally, they may also be produced artificially, as in nuclear reactors.   The two basic quantities in the assessment of radiation levels and effects are the activity of a radioactive material and the radiation dose.   The Committee uses the system of radiation quantities and units adopted in 1980 by ICRU.

1. Activity

12.   The activity of a radioactive material is the number of nuclear disintegrations per unit time.   The unit that the Committee used for this quantity up to and including its 1977 report was the curie (Ci), which is 37 billion ($3.7 \cdot 10^{10}$) disintegrations per second, a number that was originally introduced because it is the approximate activity of 1 gram of radium-226.

13.   The present unit of activity has been given the special name becquerel (Bq). One becquerel is one disintegration per second.

14.   The word radioactivity denotes the phenomenon of radioactive disintegration. It is not a synonym for "activity", nor should it be used to mean "radioactive material".

2. Radiation dose

15.   The term radiation dose can mean several things (e.g. absorbed dose, dose equivalent or effective dose equivalent).   The absorbed dose of radiation is the energy imparted per unit mass of the irradiated material.   Up to and including the 1977 report, the Committee used the rad as the unit of absorbed dose (1 rad = 0.01 joule/kg).   The present unit of absorbed dose is joule/kg, for which the special name gray (Gy) is used.   Thus, 1 rad = 0.01 joule/kg = 0.01 Gy.

16.   Different types of radiation have different relative biological effectiveness (RBE).   The RBE of one type of radiation in relation to a reference type of radiation (usually X or gamma) is the inverse ratio of the absorbed doses of the two radiations needed to cause the same degree of the biological effect for which the RBE is given.

17.   When the first UNSCEAR reports were prepared, ICRP had recommended certain values of RBE for the purposes of radiation protection.   The absorbed doses of various radiations were multiplied by these values to arrive at doses weighted for the purposes of radiation protection (e.g. for comparison with dose limits).   The unit of this weighted absorbed dose was called rem.

18.   The use of the term RBE in two contexts, radiation protection (where it only meant the standard values recommended by ICRP) and in radio-biology (where it meant the most likely value in a given exposure situation for a specified biological effect), caused some problems,   ICRP and ICRU therefore decided to establish a new quantity, the dose equivalent,   This would be the product of the absorbed dose and a so-called quality factor (first denoted QF and later Q), and its unit would be the rem.   The quality factor was given by ICRP as a function of the capacity of each radiation to produce ionization, expressed as the linear energy transfer (LET).   For practical applications, ICRP suggested that it would suffice to use approximations of average values, i.e. one unique value of QF (Q) for each type of radiation.   It suggested values of $Q = 1$ for X-rays, gamma rays and beta particles, $Q = 10$ for fast neutrons (changed to $Q = 20$ in 1985), $Q = 10$ for alpha particles (changed to $Q = 20$ in 1977), and $Q = 20$ for heavy particles,   The Committee has also used these factors but continued to use $Q = 10$ for fast neutrons.

19.   In the UNSCEAR reports, when doses are expressed in rem, the ICRP values of "RBE (protection)", QF or Q have been used in most cases, however, when authors express doses in rem, they may have used the primary, LET-related definition of QF (Q).

20.   When the Committt, began in 1982 to apply the new international unit system and the absorbed dose was given in Gy instead of rad, the new unit for dose equivalent was named the sievert (SV).

21.   In addition to absorbed dose and dose equivalent, there is a third quantity that may be meant when an author speaks of radiation dose, namely, the exposure. Exposure is the total electrical charge of ions of one sign produced in air by electrons liberated by X or gamma rays per unit mass of irradiated air.   Since the exposure is a measure of the ionization that X or gamma radiation would produce in air, it is therefore only applicable for those types of radiation. The unit of exposure is coulomb/ kg, but the old unit, the roentgen (R) is st '11 in use.   One roentgen is equal to 2.58×10^{-4} coulomb/kg.   The word "exposure" is also used in this report in its common meaning of being exposed to something, e.g. a radiation source.

22.   In tnis latter meaning, the exposure to radon decay products can be expressed in two different ways: as the amount of inhaled decay products, taking into account their potential to emit radiation energy, or as the product of the time during which the decay products were inhaled and their concentration in the inhaled air.   The potential alpha energy of the inhaled decay products may simply be expressed in joule (J).   The potential alpha energy concentration in air is expressed in J/m^3 or in the older unit, the working level (WL), where 1 WL = 2.08 10^{-5} J/m^3.   For radon in equilibrium with its decay product, this corresponds to a concentration of 3,700 Bq/m^3.   Exposure to the decay products is customarily expressed in terms of the working level month (WLM) or, as is now also common, Bq h/m^3.

23.   In the 1958 report of the Committee, the word "dose" was used loosely, and the quantity meant had to be inferred from the units used (roentgen, rad or rem). In the UNSCEAR 1962 report, doses were sometimes expressed in rad, sometimes in rem. However, in the next five repnrts, up to and including the 1977 report, the approach was more stringent.   The absorbed dose w s used consistently and the dose equivalent was deliberately avoided.   The main reason for this was that one use of the physical and biological information was to provide a oasis for estimates of RBE and therefore also to evaluate the appropriateness of the recommended values for Q.   To present doses as dose equivalents would have been to beg the issue. Sometimes, however, exposures had to be expressed in roentqan because this was how the original data had been presented.

24.   With the 1982 UNSCEAR report, the practice changed.   The Ccmmittee had gradually become more concerned with risk estimates and was not satisfied with merely reporting levels of abborbed dose.   One reason for this was the growing evidence that radon daughter products caused lung cancer and that these daughter products wet-c present in high concentrations in dwellings.   Previously, dosa contributions from types of radiation with RBEs other than unity had not been considered important and the presentation of absorbed doses was thought to be sufficient..   Now, the situation was different.   While it was recognized that the dose equivalent was a quantity designed for radiation protection and that the Q values recommended by ICRP might. differ from the true values of RBE, the dose equivalent was still believed to give a better indication of risk than the absorbed dose.

3. Development of dosimetric concepts

25.   Paragraphs 25-41 review historical development of other concepts and quantities used by the Committee.   When the 1958 UNSCEAR report was issued, two biological effects were prominentt leukaemia and hereditary harm.   For that reason, priority was given to calculating dose in the red bone marrow and gonads. In the case of dose in the gonads, it was obvious that the dose would be relevant to risk assessment only if it were calculated for individuals young enough to expect children.   In the case of dose in the bone marrow, the question arose as to whether the mean dose or the peak dose would be relevant) the ensuing discussion led to the concept of mean marrow dose.

(a) The genetically significant dose

26.   It was realized early that for most populations the medical uses *of* X-rays were the main source of man-made exposure.   However, dose distribution within a patient is very uneven, so the dose assessment is not easy.   In addition, the age distribution in exposed patient groups differs from that in the general population.   To solve these problems, the Committee derived the concept of genetically significant dose (GSD), defining it as "the dose which, if received by every member of the population, would be expected to produce the same total genetic injury to the population as do the actual doses received by the various individuals".   On the basis of this definition, the Committee developed a formula and an assessment procedure for estimating the genetically significant dose from various types of X-ray examinations,   This is described in detail in the 1958, 1962 and 1972 reports.

(b) The mean marrow dose

27.   Assuming that the mean dose in the active (red) bone marrow would be the quantity relevant to assessing the leukaemia risk and using information on the distribution of active marrow in the skeleton, this quantity was assessed fot various types of X-ray examinations.   While it was recognized that this would not be the relevant quantity if the dose-response relationship was non-linear or showed a dose threshold, it was equally clear that if the relationship was linear end showed no threshold, yet another quantity, the per caput mean marrow dose in a population would be of interest, and this quantity was assessed in the 1956 UNSCEAR report.

(c) The dose commitment

26.   Nuclear test explosions in the atmosphete introduced time elements that made this source of radiation different from, for exam le, medical exposures, in the sense that the period of practice and the period of exposure were different.   After each nuclear explosion, some long-lived radio-nuclides were released that will persist in the biosphere for many years, causing radiation exposures.   To have presented the annual doses caused by the tests that had been carried out up to the time the 1958 UNSCEAR report was drafted would not have given the full picture: namely, it would not have shown that the contamination was expected to last for a long time, thus committing mankind to exposures in future years.   The situation was described by diagrams in the 1958 report.   These diagrams showed the doses to be expected under various assumptions about the period of future testing.

29. In its 1962 report, the Committee introduced the concept of dose commitment. The dose commitment from one year of practice is the sum of the per caput annual doses inevitably caused by the resulting environmental contamination over future years. It can be shown that the dose commitment from one year of a practice is equal to the highest annual per caput dose in the future, if the practice continues indefinitely at constant rate. This relationship made it possible to assess the future consequences of continuing various practices.

30. In the 1964 UNSCEAR report, the dose commitment was defined as "the integral over infinite time of the average dose rate in a given tissue for the world's population, as the result of a specific practice, e.g. a given series of nuclear explosions. The actual exposures may occur over many years after the practice and may be received by individuals not born at the time of the period of practice". This definition was repeated in subsequent reports and a stricter mathematical presentation was given in 1969 and 1977. It should be mentioned that when the integration of the average dose rates is carried out not to infinity but only to some specified time, one is dealing with truncated dose commitments.

(d) Collective doses and collective dose commitments

31. The use of the dose commitment concept did not carry any implication of assumptions with regard to the dose-response relation at the low dose6 of radiation that were assessed for the environmental contamination; it was merely a mathematical device for adding inevitable dose contributions.

32. Another concept is the collective dose, Assuming a proportionality between dose increments and resulting increments in the risk of harm, the expected number of harmfully affected individual6 would be proportional to the collective dose, since the latter is defined as the product of the number of exposed individual6 and their average radiation dose. Before 1977, the Committee hesitated to assess collective doses, because doing so would have implied an unproven dose-response relation. In its 1977 report, however, the Committee assessed collective absorbed doses from various sources and practices. Where a practice was expected to cause exposures over future years, the collective dose commitment was assessed. This is simply the total collective dose expected from a given practice over all future time.

(e) Transfer coefficients

33. Dose commitments from practices causing environmental contamination are proportional to the amount of the relevant radio-nuclides that have been released into the environment. Thus, the assessment involves the study of a chain of events starting from the primary injection of radioactive material into, for example, the atmosphere and ending with the eventual irradiation of body tissues. This chain of events can be represented schematically:

Inhalation

Input → Atmosphere → Earth's surface 3   Diet → Tissue → Dose
(0)   (1)   (2)   (3)   (4)   (5)

External irradiation
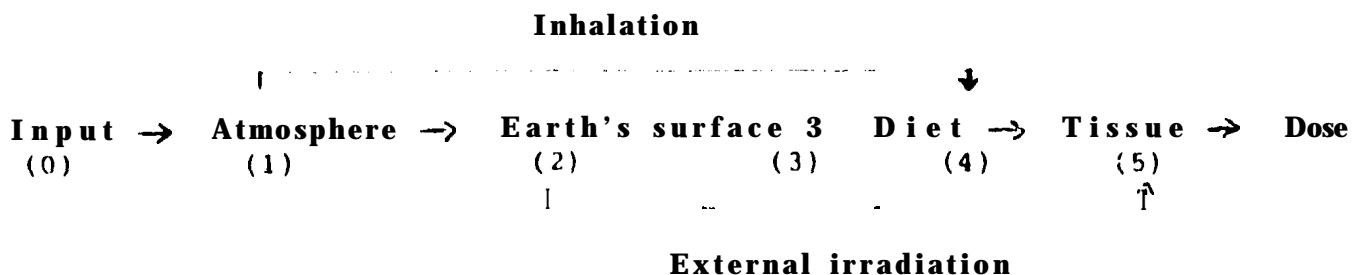
34. Beginning with its 1969 report, the committee has assessed transfer coefficienta, i.e. the quotient6 of the time-integrated quantity (e.g. activity concentration) in each step and the corresponding quantity in a previous step. For example, the transfer coefficient P_{34} is the time-integrated activity concentration in a given tissue divided by the time-integrated concentration of the same nuclide in the diet. The product of all transfer coefficients directly relates the amount of radioactive material injected into the atmosphere to the resulting dose. The mathematical formulation and assessment procedure were described in detail in the 1969 report.

(f) Organs of interest

35. A6 has already been mentioned, in its 1958 report the Committee calculated doses for only two organs: the gonad6 and the active bone marrow. They were the only organs for which some risk estimator had been made at that time. In the 1969 report, the Committee added dose assessments for one more tissue, namely the cells lining bone surfaces. Up to 1972, the dose assessments had thus been made for three organs (gonads, active bone marrow and bone surface cells), although the Committee had in fact made risk estimates for other organs, such as the thyroid (1964 and 1972) and breast and lung (1972). One reason for limiting the number of organ6 was that the dose assessments would become more complicated the more organ6 the Committee included and comparisons between various sources would become very difficult.

36. Nevertheless, in its 1977 report the Committee added still one more organ, the lung, because it had become increasingly evident that the alpha-emitting daughter products of radon in dwelling6 were biologically significant and that radon escaping from uranium mill tailing6 was generating very high long-term commitments,

(g) The effective dose equivalent

37. In 1977, ICRP published a revision (ICRP Publication 26) of it6 general recommendations, in which it suggested that a weighted sum of the radiation dose equivalents in the most radio-sensitive organs and tissues should be the basis for radiation protection assessments. This weighted sum was named the effective dose equivalent. It was to have the same unit as the dose equivalent, i.e. the sievert. The effective dose equivalent is determined using only the organ weighting factor6 recommended by ICRP on the basis of risk assessments. Other types of sums of weighted organ doses, with different weighting factors, must not be called effective dose equivalents,

38. The effective dose equivalent was originally intended to reflect the relative organ risks for an average member of a working population. It gave the same weight to a severe hereditary defect in the exposed individual's first two generations of offspring as to the occurrence of a lethal cancer in that individual. It gave zero weight to curable cancer. The concept was appropriate considering the intended use of the quantity. The same quantity has since found widespread use in the assessment of collective doses to member6 of the public. Here, where its failure to account for the difference between the age distribution of worker6 and that of the public at large and its non-inclusion of curable cancer and hereditary harm in generations beyond the second are known deficiencies, the use of the effective dose equivalent may be questionable. Various corrections to compensate for these limitations have been suggested, but for the purposes of radiation protection, and

considering all other uncertainties, the extensions of tha use of the effective dose equivalent have mostly been accepted,

39. In looking for ways of presenting radiation doses from various sources and practices, UNSCEAR faced problems similar to those faced by ICRP. Particularly *in* the cases of medical exposure and exposure from radon daughter product6 in the lung, different organs receive quite different doses, and the idea of a weighted whole-body dose was attractive, The Committee is wall aware of the fact that the effective dose equivalent ha6 not been designed for its particular purposes, but it has not been able to find an alternative way of expressing radiation exposures by a single number,

40. In the definition of the effective dose equivalent there is an addition of cancer risk and risk of hereditary harm. The risk coefficients for cancer and hereditary harm, as applied to the effective dose equivalent, are clearly identifiable only if all organs receive one and the same dose, In cases where they do not, the effective dose equivalent gives a basis for estimating the total risk but gives no indication of the relative proportions of the cancer risk and the genetic risk (see chap, III, sect, C).

41. The effective dose equivalent was used in the 1982 report and comparison6 were made on the basis of the collective effective dose equivalent commitment. To simplify the presentation of doses and dose comparisons, the Committee has had to resort to more and more complicated term6 and there is, unfortunately, no easy way out of this dilemma.

C. Dose assessments

1. -   -

42. In preparing its first report (1958), the Committee concluded that the three main contributors to radiation doses from natural radiation in soft tissues of the human body were cosmic rays, terrestrial gamma-radiation and potassium-40 within the body itself. When the joint dose contribution of these three sources was assessed in the UNSCEAR report6 of 1958-1977, it varied from 93 to 98 per cent of the total absorbed dose from all natural sources, which was estimated to be about 100 mrad per year. The contribution of the three sources were as follows: about 30 mrad from cosmic rays, 30-50 mrad from terrestrial gamma radiation and 20 mrad from potassium-40 in the body.

43. In all UNSCEAR report6 up to and including that of 1972, doses were assessed for three tissues: gonads, osteocytes and active bone marrow. The per caput dose6 in these tissues were used for dose comparisons in the main text of the reports. The assessed values varied only a little from one report to another, with the exception of an overestimate of the dose from the neutron component of cosmic rays in 1962.

44. In the 1977 report, the lung dose from radon daughter products inhaled indoors was given in the summary tables, but it did not look so conspicuous since it was presented as an absorbed dose. In 1982, however, the effective dose equivalent was calculated for the first time and the significance of this contribution became obvious, since it amounted to about one half of the total, as a world-wide average. The assessed value of the annual effective dose equivalent from natural

radiation source6 wan raised accordingly, to about 2 mSv, i.e. to about twice the value implied in previous UNSCEAR reports, where the lung dose had not been taken into account.

2. Nuclear explosions

45. Most nuclear explosions in the atmosphere occurred before 1963. Their total yields in equivalent amounts of TNT were estimated in the 1964 UNSCEAR report as follows:

Period	Yield
	(Megatons)
1945-1951	0.8
1952-1954	60.0
1955-1956	28.0
1957-1958	85.0
1959-1960	0.0
1961-1962	337.0

These number6 have subsequently been somewhat revised in the light Of more recent information (see pars. 143 and table 5).

46. The atmospheric tests after 1962 were small in comparison with the earlier explosions and they ceased completely after 1980. The many underground explosions carried out in later years have had few environmental consequences. This temporal picture gives an indication of the environmental situation that prevailed when the Committee prepared its various reports,

47. Large explosions in the atmosphere carry most of the radioactive material into the stratosphere, where it remains for some time, the mean retention times being estimated from less than a year to about five years, depending on the altitude and latitude. Fall-out can therefore occur years after an explosion has injected material into the atmosphere. Smaller explosions carry the radioactive material only into the troposphere, and fall-out occur6 within day6 or weeks.

48. When it prepared its 1958 report, the Committee did not yet have sufficient information on the global inventory of long-lived radioactive material6 to be able to formulate the assessment model6 used in later reports. However , the Committee correlated measured fall-out rates and deposits with observed radioactive contamination level6 in vegetation and food. As explained in chapter II, section B, the quantities that were first assessed were the genetically significant dose and the per caput mean marrow dose, because for these the Committee could make risk estimates.

49. In the first four UNSCEAR reports (1958-1966), the Committee described in detail the meteorological processes that deplete the stratospheric inventory of radioactive debris. For man, the highest exposure was found to be due to long-lived radioactive material that causes radiation exposures over many years. The dominant radio-nuclides were strontium-90 (half-life: 28 years), caesium-137 (30 years) and carbon-14 (5,700 years). Some gamma-emitting radio-nucl ides from tropospheric fall-out, e.g. zirconium-95 and ruthenium-106, could also contribute significantly through exposure from the ground deposition.

50.   Because **it was interested in the radiation dose in active bone** marrow **and in osteocytes, the Committee initially made its** most **thorough dose calculations for strontium-90.   Eventually, however, caesium-137 turned out to cause higher doses because of its double exposure** modes;   by **external gamma-radiation from ground deposition and by internal exposure after intake with food.   The exposures** from **caesium-137 could be verified using direct measurements of the body content, but this was** more **difficult for strontium-90.**

51.   **With its 1962 report, the Committee applied the concept of dose** commitment. **This made it possible to assess the impact of tests carried out** in **a particular year or of all the tests up to the time of a report.   In such** assessments, **however, the contribution from** carbon- 4 **turned out to be high, because of its long half -life.   Models for estimating the dose commitment from carbon-14 were developed in the 1962 and 1964 reports,**

52.   **In 1964, attention was drawn to the high individual doses caused by enhanced concentrations of caesium-137 in** some **food chains, in particular the lichen-reindeer chain.   This was further discussed in the 1966 report, where it was reported that levels** of **caesium-137 in reindeer** meat **had in** some **cases reached 100** nCi/kg (3,700 Bq/kg) **and, in freshwater fish, 10** nCi/kg.

53.   **In the 1969 report, the mathematical formalism of all calculations was reviewed and the concepts of transfer chains and** transfer **coefficients were introduced.   By the time the 1972 report was prepared, the fall-out rate had decreased substantially,** most **of the testing having ceased in 1962.   Better** estimates **could therefore be made of some transfer coefficients, which resulted in somewhat** lower **dose** estimates.

54.   **In 1977, for the first time, collective dose commitments to most soft tissues of the body from the nuclear test explosions before 1976 were estimated and found to be between 400 and 800 million man rad without the full carbon-14 contribution and** about **twice as great with the full carbon-14 commitment.   For comparison, in the 1977 report the annual collective dose to the world population from natural** sources of radiation **was estimated to be about 300 million man rad.**

55.   **In** the **1982 report, essentially the same basic information was reviewed.   The dose** assessment **models were then described in** a **special annex, which also listed conversion coefficients, symbols and units,   This time the effective dose equivalent was calculated.   According to the 1982 assessment, the collective dose contributions from the major radio-nuclides were as follows:**

Radio-nuclide	collective effective dose equivalent commitment (10^6 man Sv)	
	External	Internal
Strontium-90		0.5
Zirconium-95	0.6	
Ruthenium-106	0.2	0.1
Caesium-137	1.5	0.7
Others, except carbon-1.4	0.2	0.7
Subtotal	2.5	2.0
Total	4.5	

56. One of the main problems in estimating future collective doses is that assumptions have to be made about the size of the population. In deriving estimates in the 1982 report, the Committee assumed a world population of $4 \cdot 10^9$ persons when calculating collective doses from radio-nuclides with half-lives of 10-30 years. The dose commitment from these and from shorter-lived radio-nuclides was estimated to be about 1 mbv. In calculating the collective dose from carbon-14, the Committee used a world population of $4 \cdot 10^9$ in its 1977 assessment, but a projected population of $10 \cdot 10^9$ in its 1982 assessment. The latter assumption made the estimated collective effective dose equivalent commitment. from carbon-14 as high as 26 million man Sv.

3 . Nuclear power production

57. In 1970, the world-wide total installed capacity for generating electric energy in nuclear reactors was about 20 GW. Over the next 10 years, nuclear electric generation increased by more than 10 GW installed capacity per year, to reach 144 GW in 1981. This rapid introduction of nuclear power on a large scale warranted assessments by the Committee starting with its 1972 report. Facing a situation similar to that which it had faced with the nuclear explosions, the Committee realised its assessment of future doses would depend on the assumptions it made about the continuation and extension of the practice of nuclear energy generation. It is interesting to note that, at that time, the projections for expansion which the Committee quoted were an order of magnitude higher than turned out to be the case.

58. Thus, in addition to assessing dose commitments and collective dose commitments per year of practice at the current rate, the Committee therefore also estimated these quantities per unit of electric energy produced, i.e. per MW year. The main contributions to the collective dose commitment were believed to come from

global contamination by tritium and krypton-85 released during the reprocessing of spent fuel and from local exposures near the power stations, The total was assessed at about 0.4 man rad/MW year. This value, however, was not used in the summary tables or in the main text of the report. Instead, there was an estimate of the annual per caput dose to the world population if nuclear power production would be maintained at the level expected for the year 2000 (an installed capacity of 4,300 GW electric power). This annual doss was estimated to be about 0.2 per cent of the dose from natural sources of radiation.

59.   In the 1977 report, there was a more systematic approach to assessing the collective dose commitments per unit of electric energy produced for each step of the nuclear fuel cycle (mining, milling, fuel fabrication, reactor operation and fuel reprocessing), including occupational exposures. The estimates made in the 1977 report were substantially higher than those made in the 1972 report, because more data became available and a fuller treatment was possible, Occupational exposure was estimated to contribute nearly 4 man rad/MW year and exposure of the public between 1.5 and 3.8 man rad/MW year to various tissues. The highest. single contribution was again found to come from global distribution due to reprocessing. In the Committee's opinion, these values may be somewhat pessimistic, because the prior experience of reprocessing and research and devslopmont -- two contributors that were together assessed to cause between 4 and 6 man rad/MW year - may not be able to indicate future experience. The Committee faced a special problem in dealing with the exposures from radon released from uranium mill tailings. This source would cause lung doses that would not be high for any one individual, but the long time period over which radon might emanate from the tailings (determined by the physical half-life of thorium-230) could make the collective dose commitment quite high.

60.   The problem posed by radon was recognized more clearly in the 1982 report, where the effective dose equivalent was calculated, The various steps in the fuel cycle were together estimated to cause 5.7 man Sv/GW year (0.57 man rem/MW year), excluding global distribution. About 2 man Sv/GW year were estimated to be caused by global distribution from tritium and krypton-85. Occupational exposure was estimated to contribute somewhat less than 30 man Sv/GW year. The total estimate was therefore about 35 man Sv/GW year (3.5 man rem/MW year), somewhat lower than the 1977 estimate.

61.   In addition, however, the Committee expected a contribution from the very long-lived radio-nuclides carbon-14 (half-life 5,700 years) and iodine-129 $(1.6 \ 10^7$ years): from radon emanation primarily controlled by thorium-230 $(8 \ 10^4$ years): end from long-lived actinides leaking from high-level waste repositories. With the exception of carbon-14, these nuclides were not expected to cause any significant cumulative collective dose over any 1,000-year period (carbon-14, however, would give 10 man Sv/GW year during the first 100 years). However , after 1 million years, assuming a world population of 10^{10} persons, the collective dose from the long-lived radio-nuclides was estimated at about 3,400 man Sv/GW year:

Radon from mill tailing6	2 800
Uranium fron mill tailing6	460
Carbon-14	110
High-level waste	30
Iodine-129	28

The corresponding doses to any one individual over a lifetime would be negligible, e.g. compared to the doses from natural background radiation, the large numbers being due merely to the long time periods. It is not a scientific question as to what extent exposures over such time periods are relevant in decision-making.

62. Using the concept of incomplete (truncated) dose commitment and assuming future annual nuclear energy generation of 10,000 GW years, the Committee finally projected the annual per caput effective dose equivalent to be 25 microsievert i.e. about 1 per cent of the annual dose from natural background radiation*

4. Medical exposures

63. In 1957, when it was preparing the 1958 report, the Committee issued an important statement: "It appears most important ... that medical irradiations of any form should be restricted to those which are of value and importance, either in investigation or treatment, so that irradiation of the population may be minimixed without any impairment of the efficient medical use of radiation". The statement also solicited further information on medical exposures, which were recognized to constitute a substantial proportion of the total radiation received by mankind.

64. In the 1958 report, the Committee gave priority to the assessment of genetically significant dose. It was realized that the highest genetically significant doses were caused by diagnostic X-ray exposures, which, at that time, were frequently carried out with fluoroscopy rather than with radiography. Diagnostic procedures were classified into 23 types, and the exposure data for these were presented for a few countries, permitting comparisons of doses between the various procedures. In addition, crude estimates were made of the per caput mean marrow dose from these procedures. More than 80 per cent of the genetically significant dose was found to be contributed by only six or seven procedures, which together made up only about 10 per cent of all procedures. The data indicated that it might be possible to reduce the doses considerably, simply by careful attention to techniques. The total genetically significant dose from X-ray procedures ranged from 17 to 150 mrem per year in the various national estimates.

65. In the 1966 report, the Committee continued its review of the national data that had been submitted. Detailed data were available from 12 countries. The results were similar to those in the 1958 report. The values of the genetically significant doses now assessed ranged from 7 to 58 mrem per year. Ways of reducing patient doses were discussed and the most effective protective measures were listed, such as the use of the smallest possible radiation field and the reduction of fluoroscopy time. This, in effect, was a protection recommendation, released before ICRP had issued any special recommendations on the protection of patients.

66. Medical exposures were next reviewed in the 1972 report. The emphasis was still on the genetically significant dose, and the values now assessed ranged from 5 to 75 mrad per year, although the number of X-ray examinations was reported to have increased by between 2 and 6 per cent per year. The Committee felt that, finally, enough information was available from industrialized countries to provide a basis for attempting to eliminate unnecessary exposures, It noted that a large proportion of the world population did not have easy access to modern X-ray facilities and the health benefits they would provide.

67.   In the 1977 report, the Committee discussed the problems of comparing doses from exposures to sources as diverse as natural radiation, nuclear explosions, nuclear power production and medical exposures, With regard to the latter, the organ dose6 caused by diagnostic radiology range from a few millirad to a few tens of red and are usually delivered at high dose rates, The dose distribution is uneven, both within the body and in the population. Moreover, the emphasis that had so far been put on the genetically significant dose might have hidden the possibility of substantial exposures of other organs, so the Committee extended its assessments to include organs other than the gonads and the active bone marrow,

68.   In its attempts to find bases for dose comparisons, the Committee looked *for,* but failed to find, a satisfactory way of combining doses to various organs into some weighted whole-body dose that would be of relevance in cancer risk assessments.   As a compromise, in the 1982 report, ths Committee decided to aasess the effective dose equivalent, which, in spite of its shortcomings, was best suited to its purposes.

69.   The 1982 assessment confirmed that medical exposures constitute the largest man-made contribution to radiation doses received by the population and that, in some industrialised countries, this contribution approaches the dose received from natural sources.   However, the Committee reminded the reader that medical exposureu differ from other man-made exposures in that the practice directly benefits those who arc exposed.   The yearly number of diagnostic X-ray examinations was now found to vary between 300 and 900 examinations per year and per thousand inhabitants in industrialised countries, excluding mass surveys and dental examinations.   X-ray oxaminations contribute the major portion of the collective effective dose equivalent from medical procedures) radiation therapy and nuclear medicine contribute only a minor portion.

70.   The Committee expressed disappointment that very little information was available for the two thirds of the world's population who live in countries where radiological examinations are an order of magnitude less frequent than in the more developed countries.   For developed countries, the Committee estimated the annual collective effective dose equivalent from medical procedures at about 1,000 man Sv per million of population, i.e. about 50 per cent of the exposure from natural sources.

5.   Occupational exposures

71.   The Committee discussed occupational exposures in its 1958, 1972, 1977 and 1982 reports and pointed out repeatedly that the data that had been submitted were, for a number of reasons, difficult to analyse.   The doses reported are those measured by personal dosimeters, and the quantity measured depends on both the type of dosimeter and on its calibration.   These recorded dose6 depend on the location of the dosimeter on the body, and it. must be assumed that they approximate a uniform whole-body dose.   The number of persons occupationally exposed is not the same as the number of person6 monitored, the difference depending on national requirements for radiation monitoxinq.   The objective of most monitoring programmes is not. t.o provide data for purposes such a6 those of the Committee, but to check that authorized dose limit6 are not exceeded.   So-called investigation levels are usually applied, below which doses are ignored or recorded as zero.   Little information is therefore available for t.he low--dose region.

72. The treatment of the subject in the 1958 report was brief. The number of workers in the medical field in countries that had submitted data was estimated to be between 0.2 and 0.7 per thousand of the total population. The treatment of occupation&l exposures in the 1962 report was also brief. The number of dental workers was found to be about twice the number of medical workers, while the number of persons occupationally exposed in industries or in research was substantially lower. The contribution of occupational exposures to the annual genetically significant dose was estimated at 0.2-0.5 mrem.

73. At the t'me of the 1972 report, there was still very little published data on occupational exposures. The number of workers in the medical field could now be narrowed down to 0.3-0.5 per thousand in the countries for which data were available and the total number of persons reported as occupationally exposed was 1-2 per thousand of the total population. The mean recorded dose for most workers exposed to radiation was found to be between 0.2 and 0.6 rad per year, but mean doses as high as 2.7 rad wet-e reported from some industrial radiography workers. The annual dose to crews of supersonic aircrrft was assessed to be about 1 rem. Occupational exposures in the nuclear power industry were expressed per unit electric energy produced and were calculated to be 2.3 ma.1 rad/MW year (1.6 man rad from fuel reprocessing and 0.7 fror reactor operation).

74. In the 1977 report, an annex was devoted to occupational exposures. For the first time, the Committee systematically reviewed the purposes and methods of assessment. It was found that the distribution of doses within the exposed occupational groups was mostly log-normal and on this basis a reference dose distribution was defined. To avoid the problems of determining the actual number of workers exposed and therefore, also, average doses, the Committee emphasized collective doses, the values of which would be largely independent of the administrative requirements on the degree of monitoring. The Committee a160 calculated the fraction of the collective dose accounted for by annual individual doses exceeding 1.5 rad. The submitted data were analysed on this basis. For most occupations, the mean dose was 0.1-1.0 rad per year. A detailed mathematical description of the log-normal distribution and of the reference distribution was given. The collective dose from each step of the nuclear fuel cycle was calculated, with the doses from all steps adding up to about 4 man rad/MW year (see chap. II, sect. C.3). The collective absorbed dose in the lungs of uranium miners wa6 estimated to be 0.1 man rad/MW year and examples of high radon levels in non- uranium mines were reported.

75. In its 1982 report, the Committee continued the analysis on the basis of more data. It noted with satisfaction that its 1977 proposal for methods of analysis had been adopted by several organizations and that the arrangement of submitted data had been influenced by the proposal, thus facilitating the analysis. However, the Committee now found that it6 suggestion of a reference radiation dose distribution had cometimes been misinterpreted, 60 it limited its presentation to the average dose, the collective dose and the fraction of the collective dose exceeding 15 mSv (corresponding to the previous 1.5 rad).

76. For countries with a high standard of medical care, medical worker6 were found to receive a collective dose equivalent. of about. 1 man Sv per million of population. The number of workers in the nuclear industry had increased substantie ly since 1977. Occupational exposures in each step of the nuclear fuel cycle were assessed more fully, indicating that the total collective effective Jose equivalent. might be near 30 man Sv/GW year (3 man rem/MW year). However, half of

this came from fuel reprocessing and nuclear research and it was uncertain whether such high contribution6 should be expected also in the future.   In reactor operstion, the highest exposures were to maintenance workers and radiation protection staff during special maintenance operations.

b. Miscellaneous exposures

77.   In addition to tho main radiation sources discussed thus far, a few other sources were identified by the Committee as far back a6 in the 1958 report.   Then, a8 now, they were referred to as miscellaneous sources. Mentioned in the 1958 report were watches with radio-luminescent paint, television sets that could produce soft X-rays and shoe-fitting equipment that used X-ray fluoroscopy.   None of these sources was eupected to cause a genetically significant dooe exceeding 1 mrem per year, although the shoe-fitting machines could cause high local doses. The 1962 report mentioned enhanced cosmic radiation to passengers in aircraft but considered the dose insignificant. The total genetically significant dose from all miscellaneous source6 was not expected to exceed 2 mrem per year, the largest contributor to which was radioactive watches.

78.   In the 1972 report, a full annex dealt with the miscellaneous sources. Incidents, transportation accidents and loss of radioactive material were mentioned a': additional sources of public exposure. A number of radioactive consumer good6 were also described, such a6 radio-luminescent timepieces and other self-luminous devices, ceramic glazes containing uranium and thoriated electrodes in welding rod6.   Radioactive substances in patient6 released from hospitals, pace-makers with nuclear batteries and demonstration materials in schools were also mentioned. Television sets were again discussed, particularly colour ones, whose cathode-ray tubes operate on higher voltages.   Finally, it was recognized that enhanced levels of natural radiation could cause problems, as, for example, do radioactive building materials.   In later report6 this would become an important topic, no longer treated a6 a miscellaneous source

79.   In the 1977 report, the miscellaneous sources were discussed in an annex dealing with technologically enhanced levels of radiation.   One of the many consumer product,, added to the list was ionization-chamber smoke detectors. However, the discussion centred on enhanced exposures to natural radiation. Enhanced exposure6 to cosmic ray6 in aircraft, including supersonic transports, and in spacecraf t, were discussed in detail.   Another subject was public exposure due to natural radio-nuclides emitted from coal-fired power plants,   A third subject was exposures due to the industrial use of phosphate products containing uranium-238 and radium: in this case, the exposure pathway6 were via phosphate fertilizer: and by the use of waste gypsum a6 a building material,   Normal exposures from radioactive building materials, whether direct (by gamma-radiation) or indirect (by radon daughter products), were dealt with in the discussion on natural sources.

80.   In the 1982 report, miscellaneous sources were again considered together with technologically modified exposures to natural radiation.   Essentially the 6ame consumer products were discussed as in the previous reports.   It was noted that the radium in wrist watches had now almost entirely been replaced by tritium, thereby eliminating the extei nal exposui e and 1 imi t.'i ng the ann al effective dose equivalent to the wearer f rom leakage tritium to less than 1 microsievert.   The average effective dose equivalent. to air passengers passing X-ray fluoroscopic scai ners was

estimated to be much lower still, about 7 nanosievert per scan. Exposures from coal-fired power plants were reassessed and the collective effective dose equivalent commitment was estimated to average 2 man Sv/GW year (this is 50 per cent of the local and regional collective dose from the same energy production in nuclear power stations, see table 6). The 1977 production of phosphate rock was estimated to have resulted in a collective effective dose equivalent commitment of 300,000 man Sv, predominantly from the use of gypsum in dwellings; the total contribution from other uses was thought to be only 6,000 man Sv.

7. Accidents and incidents

81.   The Committee discussed radiation accidents in the 1962, 1972, 1977 and 1982 reports. In 1962, it reviewed the eight major accident8 known to it at the time; these had caused at least four deaths, Seven of the accidents were criticality accidents (five in the United States, one in the Soviet Union and one in Yugoslavia). The eighth accident involved pulsed X-rays from an unshielded electronic tube at a radar station, The course of the accidents and the clinical symptoms of the exposed persons were discussed in some detail.

82.   In the 1972 report, accidents were treated only briefly The Committee noted that about 100 incidents in connection with the transport of radioactive material had been reported throughout the world from 1954 to 1968. There had been 14 accidents involving aircraft carrying nuclear weapons or components of nuclear weapons. Two nuclear submarines had disappeared and a plutonium-238 isotopic generator had burned up in the upper atmosphere. A number of incidents had also been reported wherein radioactive material had been lost or stolen. An analysis of 115 radium incidents occurring from 1966 through 1969 showed that 55 per cent of the incidents were losses. In another study of 299 incidents involving the loss or theft of radium, 66 per cent of the sources were recovered. The same report also briefly discussed occupational accidents, showing that thay had been particularly frequent in X-ray analytical work and in industrial radiography.

83.   In the 1977 report, the Committee for the first time discussed accidents at nuclear power plants. In its review of the collective dose commitments from the various steps in the nuclear fuel cycle, the Committee approached the difficult problem of dose commitments from accidents that had not yet occurred. Any nuclear power programme is also a commitment to a certain accident probability, so in that sense, the Committee said, there was also an accident dose commitment.

84.   In 1982, the Committee observed that there had so far been only two reactor accidents known to have caused measurable irradiation of the public: one at a military plant at Windscale, United Kingdom, in 1957, and one at a nuclear power station at Three Mile Island, Pennsylvania, United States, in 1979. The collective whole-body dose from the latter accident bad been estimated between 16 and 35 man Sv within 50 miles, most of it dus to xenon-133, and about of equal magnitude outside 50 miles. The collective effective dose equivalent from the Windscale accident had been estimated at about 1,300 man Sv, of which almost half was due to iodine isotopes and thyroid irradiation. The Committee decided thnt the probabilistic approaches, which predict the risk from reactor programmes by extrapolating into the future, should not be used as a basis for estimating future components of collective dose commitment.

65. In another part of the 1982 report the Committee reviewed information on occupational accidents, It tabulated those accidents on which it had received data or which had been reported in the open literature. The Committee noted that the serious accidents had occurred early in the development of nuclear technology and that not one serious accident had been reported in reactor operation since the mid-1960s. Radiation accidents *in* other industries had caused *one* death ● ince 1960; this death occurred in 1975 in an irradiation facility with cobalt-60. As had been noted in the earlier reports, industrial radiography seemed to have a special potential for accidents, Some severe injuries had occurred when persons picked up lost radiography sources without being aware of the danger.

D. Risk assessments

1. Hereditary harm

86. The methods used so far to quantify genetic risk can be broadly grouped under two headings : the doubling dose (or relative mutation risk) method and the direct (or absolute mutation risk) method. The doubling dose method aims at expressing the risk in relation to the natural prevalence of genetic diseases in the general population; the direct method aims at expressing absolute risk in terms of expected increases in the prevalence of genetic diseases. Owing to the paucity of direct human data on radiation-induced genetic damage leading to disease states, the rates of induction for the pertinent kinds of genetic damage (mutation and chromosomal aberrations) are based on experimental data in animals. These rates are converted, using a number of assumptions and reduction factors, into the expected number of additional cases of genetic disease in man.

87. To apply the doubling dose method, one needs (a) an estimate of the doubling dose, i.e. the radiation dose that will produce as many mutations as those occurring spontaneously in a given qenerationr (b) information on the prevalence of naturally occurring genetic diseases in the population and the extent to which these are maintained by mutation] and (c) an estimate of the dose received by the population. Over the years the doubling dose estimates have been based on experimental data obtained in mice; the prevalence figures for naturally occurring genetic diseases are those collected in several epidemiological studies. With the doubling dose method, the risk is the product of the prevalence of naturally occur ring genetic diseases, the mutation component, the reciprocal *of* the doubling dose and the dose sustained by the population.

ne. Over the past. three decades, there have been shifts in emphasis in the use of these methods and there have also been a number *of* refinements, as extensively discussed in annex E. The principles that guided UNSCEAR, as well as other scientific bodies, in its early assessments of radiation-induced hereditary risk in the 1950s were those that had emerged from the extensive investigations in Drosophila, the preliminary results in mammals, particularly the mouse, and the sparse human dnt a. Two of these principles were the followinqr (a) mutations, i nduced or spontaneous, are generally harmful! and (b) mutations induced by radiation increase linearly with dose without a threshold.

89. In the light of new data f rom studies on male mice showing that a chronic gamma dose was only about one third as effective as the same dose given at a hiqh dose rate (and even more reduced in female mice), the 1962 report suggested that t he pi eviously used doubling dose of 30 roentqen would probably be too low by a

factor of 3 to 4. With confirmation and extension of these results and other data showing that the interval between irradiation and conception had a dramatic effect on mutation frequency in female mice (all mutations were found in the progeny conceived during the first seven weeks after irradiation), the Committee in 1966 abandoned the doubling dose approach in favour of other methods, two of which will be mentioned here. In one, the estimated rate of induction of dominant visible mutations in mice (range: 10^{-9} to 10^{-7} per locus and rad) was multiplied by the assumed number of loci determining dominant disorders in man (50-500) to obtain the total risk (5 10^{-8} to 5 10^{-5}). In the other, the estimated rate of induction of recessive visible mutations in mice (10^{-7} per locus and rad) was multiplied by the estimated total number of gene loci in man (20,000) to obtain an estimate of total risk from the induction of these point mutations (2 10^{-3}). The risk to first generation offspring was then computed as a fraction (2-5 per cent) of the above figure.

90. In the 1972 report the interest of the Committee in the doubling dose method was revived but was given a low profile. The doubling dose was taken to be 100 rad, and the number of extra cases of severe hereditary diseases per million live births and rad of low-LET radiation was estimated to be about 300 for the irradiation of parental males; of these, 6-15 cases occurred in the first generation and the rest occurred in subsequent generations.

91. By 1977 new data on the natural prevalence of genetic and partially genetic diseases had been obtained. Furthermore, data that had been obtained in the mid-19606 on the induction of dominant mutations having their primary effect in the mouse skeleton had been extended in the mid-1970s, demonstrating transmission. By 1982, new data on the induction of another kind of dominant mutation, namely, those which cause cataracts in the eye of the mouse, became available. All these data allowed the Committee to arrive at direct estimates of genetic risks. It is worth noting that from 1977 onwards both the doubling dose method and the direct method have been used.

92. In 1977, using a doubling dose of 100 rad, the Committee estimated that, if a population is continuously exposed to low-LET radiation at the rate of 1 rad per generation, there will be a total of about 185 cases of Mendelian, chromosomal and other diseases per million live births at equilibrium, of which about one third would appear in the first generation. The first-generation increase was estimated to be about one third of that at equilibrium.

93. These estimates, as well as those arrived at in the 1982 and 1986 reports, are summarised in table 1; for convenience, they are expressed on a per Sv basis. It can be seen that (a) for dominantly inherited diseases, the estimates have remained essentially unchanged; (b) the estimates for chromosomal diseases have become lower, this being a consequence of having excluded diseases attributable to numerical anomalies (such as Down's syndrome), for which there is still no good evidence of induction by radiation; and (c) while in 1977 and 1982 the Committee had provided estimates of risk for congenital anomalies and other multifactorial diseases using certain assumptions, in 1986, concerned about persistent uncertainties over the assumptions used, it no longer did so.

94. The risk estimates made using direct methods from 1977 up to 1986, are given in table 2; they include risks from (a) the induction of genetic changes having dominant effects in the first-generation progeny (i.e. dominant mutations, as well as recessive mutations, deletions and balanced reciprocal translocations with dominant effects); and (b) unbalanced products of balanced reciprocal translocations, which may lead to congenitally malformed children.

Table 1. **Estimates of the risk of severe genetic disease per million live births in a population exposed to a genetically significant dose equivalent of 1 Sv per generation of low-dose-rate, low-dose irradiation, according to the doubling dose method**

(Based on the 1977, 1962 and 1986 UNSCEAR reports)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per *million* live births	Effect of 1 Sv per generation	
		First generation	Equilibrium
1977			
Autosomal dominant and X-linked	10 000	2 000	10 000
Autosomal recessive	1 100	Relatively slight	Very slow increase
Chromoaomal (due to numerical and structural anomalies)	4 000	3 800	4 000
Congenital anomalies and other multifactorial diseases	43 000) 47 000)	450	4 500
1982			
Autosomal dominant and X-linked	10 000	1 500	10 000
Autoeomal recessive	2 500	Relatively slight	Very slow increase
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3 000	Probably very small	
Congenital anomalies and other multifactorial diseases	43 000) 47 000)	450	4 500
1986			
Autosomal dominant and X-linked	000	1 500	10 000
Autosomal recessive	2 500	5	1 500
Chromosomal			
Due to structural anomalies	400	240	400
Due to numerical anomalies	3 400	Probably very small	
Congenital anomalies and other multifactorial diseases	60 000) 600 000)	Not estimated for reaaons given in para. 166	

Note: The derivation of the above figures is given in annex E; see also para. 93.

T a b l e ?. Estimates of the risk of genetic disease in the first
generation (for a genetically significant dose
equivalent of 1 Sv) *per* million live births following
low-dose-rate, low-dose exposure of the parental
generation according to the direct method

(Based on the 1977, 1482 and 1986 UNSCEAR reports)

Risk associated with	Expected frequency of genetically abnormal children in the first generation per million live births after irradiation of	
	Males	Females
1977		
Induced mutations having dominant effects	2 000	None given
Unbalanced products of induced chromosomal rearrangements	200-1 000	None given
1982		
Induced mutations having dominant effects	1 000-2 000	O-900
Unbalanced products of induced chromoaomal rearrangements	30-1 000	O-300
1986		
Induced mutations having dominant effects	1 000-2 000	O-900
Unbalanced products of induced chromosomal rearrangements	100-1 500	O-500

Note: The derivation of the above figures is given in annex E; see also
paras. 94 and 95.

95. The first of these estimates (item (a) in para. 94) is based on dominant
skeletal and cataract mutations in mice and the second (item (b) in that paragraph)
on primate cytogenetic data. The estimates based on experience in mice do not
include induced genetic changes so severe as to cause death before they can be
detected. It can be seen that the changes in risk estimates from 1977 to 1986 are
relatively small. Furthermore, a comparison of these estimates with those arrived
at using the doubling dose method (table 1) for the first generation reveals that
they are of the same order of magnitude, in spite of the different asaumptions and
reduction factors.

2. Cancer

96. As far back as in the 1958 report, the Committee emphasized that any attempt
to evaluate the biological effects of radiation sources to which the world

population is exposed can produce only tentative estimates, subject to wide margins of uncertainty. Despite these reservations, the report included assessment6 of the annual numbers of leukaemia and bone cancer cases that could result from natural radiation and fall-out. Data relating the incidence of leukaemia to radiation exposure came mostly from the atomic bomb survivors and patients suffering from ankylosing spondylitis.

97. At that time, the Committee estimated the total probability of leukaemia induction over 15 years to be 12 per million population per rem. It noted, however, that in Hiroshima the probability per unit dose decreased markedly with decreasing dose and that the incidence of leukaemia in that city did not appear to be linearly related to dose. The Committee also made what it called a crude estimate of the leukaemia risk to patients suffering from ankylosing spondylitis who had been treated with X-rays. Over 15 years, the risk of induction was estimated to be about 20 per million and rem. Over 35 years, which is the average remaining lifetime of the population and might be the period of risk under conditions of prolonged exposure at lower dose rates, the lifetime risk was assessed to be 52 per million and rem.

98. In discussing the assumed hypothesis of non-threshold linearity between dose and incidence of cancer, the Committee stated in the 1952 report that somatic effects were less likely to occur at low dose rates than at the high dose rates employed in many experiments. The only justifications for applying to low doses the relationships observed at higher dose6 were expediency and the consistency of the assumptions regarding mechanisms in both dose ranges. Nevertheless, the Committee could not say whether, in doing so, it was under- or overstating the risk. For these reasons, it decided not to estimate absolute risks, but rather to present comparative risk estimates for the gonads (genetic effects), the bone marrow and the cells lining bone surfaces, based on the doses and dose commitments to these tissues from natural radiation sources, medical, occupational and miscellaneous exposure, as well as from nuclear testing.

99. Three basic questions needed to be addressed in the estimation of risk at low dose: the type of effect; the critical tissue for each type; and the function of dose, dose rate end dose distribution to be taken as the relevant parameter for each of the effects. For the somatic effects, the critical tissues were taken to be the active bone marrow and the connective tissue lining endosteal surfaces or trabeculae.

100. Although for genetic effects the experimental data justified an assumption of non-threshold linearity at low doses and dose rates, no such assumption could be made for late somatic effects, because tumour induction at high doses was a very complex function of dose and other exposure factors. Nevertheless, it would be expected that, at low dose levels, the mechanisms by which late effects are produced would be much simpler and any effects that could arise would result from specific changes induced in individual cells. For certain effects having a non-linear relationship at high dose levels, it was thought probable that the slope of the dose-effect curve near the origin would be linear. Thus, protraction of exposure and non- uniformity of close distribution could be ignored. The Committee also discussed the importance of taking into account the way an effect manifests itself over time.

101. Referring to the problems of obtaining estimates of absolute risk, the Committee noted, in 1964, that it had earlier confined itself to estimating

comparative **risks except for leukaemia,** After having reviewed the available information, the Committee saw no possibility of changing this procedure in the 1964 report. It immediately went on to state, however, that data published since 1962 had led it to believe that it would be possible, for a few tissues and mainly in the high-dose range, to make estimates of absolute risk that would be valid for the observed range of doses and the given conditions of irradiation. It was considered unlikely that the risk per unit dose at very low doses would be greater than that at higher doses; in fact, at low doses the risk was likely to be much less.

102. By 1964, tentative dose estimates had become available for some Of the survivors from Hiroshima and Nagasaki, and the Committee believed that they were almost certainly not in error by a factor of more than 2 or 3. The new dose estimates made it possible to conclude that the annual incidence of radiation-induced leukaemia was approximately proportional to dose in the range from about 100 to 900 rad, with a proportionality factor between one and two cases per million and rad. The Committee warned that because the Japanese survivors might have been selected by the lethal effects of the irradiation itself, this estimate of risk could only be applied with caution to the general population. The estimate obtained from the atomic bomb survivors was consistent with that determined from subjects who had been irradiated therapeutically for ankylosing spondyli tis, at doses between 300 and 1,500 rad. However, as the latter group was also highly selected, the estimate would apply strictly to spondylitic patients only.

103. New information suggested that for children irradiated in utero, the risk of leukaemia per unit dose could be several times higher than for adults. The doses received had been only a few rad, suggesting that under certain conditions, low doses could induce malignancy. As with the ankylosing spondylitis patients, there was the possibility that the irradiated children might not have been representative of all children.

104. A risk estimate for thyroid cancer was obtained from surveys on the induction of cancer as a result of irradiation of the thyroid region during childhood. In the range 100-300 rad, the Committee estimated the annual risk to be about one per million and rad, over approximately 16 years after irradiation. Once again, the Committee pointed out that the subjects might have been a highly selected group,

105. Irradiation was known to cause other malignancies, including tumours of the bone, liver, skin and lung; however, the information was not considered to be reliable enough for deriving risk estimates. The Committee was not optimistic about being able to obtain such estimates for all, or even many, types of human tissue. Indeed, it concluded that leukaemia might well be the predominant type of mal ignancy produced, and that the overall risk of all malignancies was unlikely to exceed by any large factor that of leukaemia.

106. In 1972, the Committee decided to review again the subject of radiation carcinogenesis in man. The review pointed out that, in order to assess the extent of radiation effects in man, it was essential to obtain empirical information from epidemiological studies. In evaluating such studies it would be necessary to bear in mind a number of inherent difficulties, such as those having to do with the si ze of the population studied, the dosimetry, the latent period, the relation to natural incidence of cancer, mortality versus morbidity statistics, the confounding effects of illness and the infrequency of true, uniform whole-body irradiation. The Committee discussed all of these points in detail and also considered t he

question of absolute and relative risks for the first time. It emphasized that the number of people exposed to substantial doses was so small that the relationship between dose and incidence of malignancies in man could be studied only for the most radio-sensitive tissues.

107. Evidence on the induction of leukaemia indicated that its incidence increased with dose in the range 50-500 rad and that above this range the frequency tended to decrease, possibly owing to the cell-killing effect of high doses. Radiation-induced leukaemias tended to occur most frequently within a few years of exposure: after 25 years the frequency tended to return to normal, by which time some 15-40 cases per million and rad had been observed.

108. Lung cancers appeared to have been induced at Hiroshima by external gamma exposure at doses of some 30-100 rad. The data indicated a risk coefficient of from 10 per million and rad (at 250 rad) to 40 per million and rad (at 30 rad) during the first 25 years after exposure; this risk estimate was supported to some extent by data from patients treated for ankylosing spondylitis. The Committee noted that an estimate of risk could also be derived from data on uranium miners, but that not much reliance could be placed on such an estimate.

109. The Committee assessed the risk of induction for breast cancer among women exposed in Hiroshima as being between 6 and 20 cases per million and rad during the first 20 years after exposure and over a dose of 60-400 rad. These estimates refer to the 1965 dosimetry. For the induction of thyroid cancers an average risk coefficient was obtained of about 40 per million and rad over a dose of 60-400 rad. For all other malignancies, without clearly identifying their specific types, the Committee tentatively put forward a risk estimate for induction of 40 per million and rad over the first 25 years after exposure to 250 rad. For a number of reasons, the Committee considered that these risk coefficients were likely to overestimate the risk of environmental exposures, that is, low-dose exposures from both natural and man-made sources.

110. The 1977 report also contained a major review of radiation carcinogenesis in man. After dealing extensively with the validity of the data on which risk estimates might be based, the Committee presented its estimates of risk coefficients for leukaemia and tumours in a number of organs. It noted that the risk of a malignancy developing at doses of about 100 rad might vary with the LET of the radiation, sometimes with the age and sex of the subject, and probably with the dose rate and the number of fractions with which the dose is deliver-1. In that report the Committee for the first time referred to the induced mortality from leukaemia and other cancers. Previously it had always presented its risk estimates in terms of the incidence of cancer, not in terms of fatality.

111. The thyroid and the breast seemed to have the highest rates of induction, with risk coefficients of around 100 per million and rad. The low mortality rate for radiation-induced thyroid cancers and the moderately low rate for breast cancers were thought to bring the risk of facality to about one tenth and one half of the incidence values, respectively. Lung cancer also had a high induction rate for males over 35, as judged from the experience of uranium miners. The Committee thought that for lung cancer a mean fatality risk coefficient for all ages of 25-50 per million and rad was probable.

112. The induction of leukaemia, specifically the acute and chronic granulocytic (but not chronic lymphatic) forms, appeared to decrease from about 50 per million

and rad at moderately high doses to about 20 per million and *ad at lower dose levels . The Committee was rather confident that this estimate would include all the cases likely to appear because, with radiation-induced leukaemia, the average interval between exposure and death appeared to be only about 10 years. With other cancers, which have latent periods of 25 years or greater, it was more difficult to estimate the total number of cases likely to be induced.

113. Risk coefficients were also presented for the stomach, liver and large intestine, brain and salivary glands, all of which had values in the region of 10-15 per million and rad; bone, oesophagus, small intestine, bladder, pancreas, rectum and lymphatic tissue, which had values of 2-5 per million and rad; and skin, for which both the risk of induction and the fatality rate were thought to be low.

114. The Committee also considered the question of estimating the total risk for all fatal malignancies from the observation that this might be 4-6 times that for leukaemia alone. At doses of a few rad, at which the lower leukaemia risk coefficient of about 20 per million and rad might apply, the total of all fatal induced malignancies, including leukaemia, could be about 100 per million and rad, while it was assumed to be about 250 per million and rad at high doses. The risk coefficient for non-fatal malignancies was assumed to be about equal to that for the fatal malignancies. The Committee once again pointed out that the estimate for low doses was derived from mortalities induced at doses greater than 100 red. The value appropriate to the dose levels involved in occupational exposure - and even more so in environmental exposures - might be substantially less.

115. It was likely that malignancies might be induced by exposure of the fetus in utero at average doses of 0.2-20 rad from diagnostic X-rays. The induction rate was difficult to determine with any confidence but was estimated to be around 200 per million and rad.

116. In view of the limited amount of new epidemiological evidence available since the 1977 report, and because the dosimetric estimates for the survivors of the atomic bombing of Hiroshima and Nagasaki were in the process of being revised, the Committee decided not to review human carcinogenesis in the 1982 report. However, it said that it did not expect that the revisions would change the previous risk estimates by a factor of more than 2. The Committee's risk estimates up to 1977 for cancer are summarised in table 3, where they are expressed per sievert in order to facilitate comparisons with later estimates.

(Per cent per Sv)

Tissue	Report			
	1958	1964	1972	1977
Bone marrow	0.2-0.5	0.01-0.02 a/	0.15-0.40	0.20-0.50
Breast	–	–	0.06-0.20	0.50
Lung	–	–	0.10-0.40	0.25-0.50
Thyroid	–	0.16	0.40	0.10
Stomach	–	–		0.10-0.15
Liver	–	–		0.10-0.15
Brain	–	–		0.10-0.15
Salivary glands	–	–		(0.10-0.15) b/
Large intestine	–	–		0.10-0.15
Small intestine	–	–		(0.02-0.05)
Bone	–	–	0.40	(0.02-0.05)
Oesophagus	–	–		(0.02-0.05)
Bladder	–	–		(0.02-0.05)
Pancreas	–	–		(0.02-0.05)
Rectum	–	–		(0.02-0.05)
Mucosn of cranial sinuses	–	–		(0.02-0.05)
Lymphatic tissue	–	–		(0.02-0.05)
Skin	–	–		Low
Estimated total				1.0-2.5

a/   Per year.

b/   Numbers within parentheses refer to total incidence, the fatality risk not having been estimated.

3.   <u>Non-stochastic effects</u>

(a)   <u>Irradiation of the adult</u>

117. The Committee considered from time to time the somatic effects of radiation on laboratory animals and human subjects. These effects were first discussed in the 1958 report, which attempted to summarize 60 years of knowledge, at a time when information about radiation lesions and their pathogenesis was still rather scanty. Although the Committee had few details on which to base that discussion, , the general picture that emerged seemed to be consistent, particularly for the effects induced by high doses. The Committee was aware at that time of the main physical factors affecting the induction of these effects, such as dose, dose rate, fractionation and radiation quality, end it also gave an account of the main biological factors, such as species, age, sex and partial-body irradiation.

118. The main radio-biological concepts, such as that of cell **sensitivity** and tissue response, as they manifest themselves in the rate of cell division and differentiation, are to be found in the 1958 report, although the concept of cell lethality could not be quantified because there were no techniques for single-cell culture. The term recovery was also used in a loose sense, without identifying the many underlying mechanisms. The classification of effects between morphological and functional gave rise to some problems, but the Committee identified, even at that early stage, the difficulties in settling the existence of thresholds, particularly with low doses and late effects.

119. Many of the same criteria were used in 1962 in classifying the somatic effects into early and late effects, with the result that effects very different in **nature** from tumours and leukaemia, such as lens **opacification,** induction of sterility or non-specific life shortening, ended up being classified together with them just because they also appeared late. The 1962 report contained no important departures from the gcneralizations described above, particularly with respect to the form of the dose-effect relationships, the uncertainties as to the precise form of these relationships at dases below those tested directly and the pronounced dependence of the effects on the irradiation dose rate.

120. Twenty years elapsed between that report and the next one, released in 1982, when an extensive annex discussed the non-stochastic effects of radiation on normal tissues. The new treatment reflected the impressive advances in the understanding of somatic effects that had taken place during the interim. The very title of the annex implied that there had been a reclassification of the effects into the stochastic and the non-stochastic. To the first class belong those effects for which only the probability of induction is a (linear) function of dose; to the second belong those effects for which severity (as well as probability, for a given severity) is a (sigmoid) function of dose. The report discussed mainly the effects of irradiation of single tissues and organs; it reviewed a large body of human data interpreted in the light of experience gained in experimental animals.

121. The Committee considered the nature of these effects, their pathogenesis as it results from the interplay of cell killing and tissue kinetics, and the quantitative relationships between them and the time of appearance and degree of the non-stochastic clinical damage. The most general conclusions drawn by the Committee pertained to the existence of a dose threshold for the induction of these effects and the variability of this threshold according to the type of effect. The annex also contained a detailed analysis of how the dose threshold for each specific type of effect would be expected to vary as a function of the important radio-biological variables such as radiation quality, dose, dose rate, dose fractionation and protraction.

(b) Pre-natal irradiation

122. The earliest mention that the tissues of the embryo and fetus could be particularly sensitive to the action of radiation and that the exposure of pregnant mothers might cause teratological effects to be induced in the product of conception dates from the first UNSCEAR report (1958). **Also,** the fact that there are critical periods in development, during which some structures may be particularly vulnerable to the specific action of internal or external irradiation, was already **recognized** at that time. Finally, it also discussed the shape of dose-effect relationships for effects in utero, without specifying the nature of the effects or their induction mechanisms, although implying that the relationships would be of the threshold type.

123. The 1962 report reiterated the notion of the special sensitivity of embryonic and fetal structures, pointing out that minor injuries during development could be amplified by the growth of the relevant structures to produce major anomalies. From data on the pre-implanted mouse it was inferred that doses of 0.25 Gy to the embryo could be lethal to 40 per cent of the animals. The Committee also concluded, on the basis of the fairly large set of experimental results then available, that irradiation during major organogenesis would cause developmental malformations and that there was a good correspondence between the malformed structures of animals and man for corresponding stages in development. In man, malformations were found more frequently in the central nervous system, the eye and the skeleton.

124. In the context of a special discussion of the effects of radiation on the nervous system contained in the 1969 report, the Committee paid special attention to the damage caused in the brain structures of the developing mammal. It confirmed that pre-natal irradiation during the time when the relevant structures were undergoing differentiation could produce severe developmental anomalies. Depending on the time of the irradiation, specific anomalies (microcephaly, encephalocoele, hydrocephalus) could be produced in man, probably following threshold-type kinetics as a function of dose. Disorganization of the cortical architecture was described in animals, accompanied by functional impairment in the form of loss of visual, olfactory and distance discrimination. Other learning processes were impaired in animals after doses of 1 Gy os more had been administered during the second or third week of pregnancy in rats; effects of doses below 0.5 Gy were regarded as uncertain. Although changes in conditioned reflexes had been described in animals irradiated near-term with doses as low as 0.01 Gy, the relevance of these effects to risk estimation in man was also doubtful. In man, the Committee recognized small head size and the induction of mental retardation as true effects, but it could not detect any correlation between such morphological and functional abnormalities and structural changes in the central nervous system. The Committee even ventured to derive a risk coefficient for mental retardation in the survivors of Hiroshima and Nagasaki: 1 per thousand and rad for doses over 50 rad delivered at high dose rates.

125. Recogniziag the importance of keeping the effects of radiation on growth and development under observation because of their relevance to the general population and to female workers, the Committee undertook another review of this subject in annex J of its 1977 report. This review centred on experimental animal data, which was the only information available, and on the mechanisms whereby effects are induced in utero: it also described dose-time relationships obtained from the more quantitative data.

126. Annex J of the 1977 report generalized the so-called "periods of maximum sensitivity" of the various anatomical structures, to coincide with the growth spurt: it also generalized across species the notion that lethal effects were typical for the pre-implantation period, teratogenic effects for the major organogenesis period and growth disturbances for the fetal period. An analysis of the dose-effect relationships showed that these were mostly curvilinear. The . Committee confirmed its previous risk assessment for mental retardation and suggested, on the basis of mouse data, that the risk coefficient for the increment of embryonic killing soon after fertilization could be taken at 1 per cent per roentgen.

127. From this review the Committee concluded that although **data in man** On the induction of malformations by radiation were very scarce, the data on other animal species were so unanimous and uniform in indicating a pronounced sensitivity to such effects that the human species could not be regarded as an **exception.** While the Committee found it impossible, given the paucity of human data, to derive reliable, quantitative estimates of risk from pre-natal human irradiation at comparable developmental **stages,** particularly at low doses and dose rates it could on the basis of experimental animal data exclude that the sensitivity of the human species might be a factor of 10 higher than expected.

4. Other **types** of harm

128. At various times and in different reports, the Committee gave special attention to types of harm not easily classifiable into one of those treated above. One such harm is the shortening of life-span, which was said in the 1958 report to result from a number of acute or late radiation-induced changes, both specific, such as leukaemia in radiologists, or pathologically diffused in all organs or tissues. These latter conditions were thought to accelerate the normal aging processes and so were termed non-specific, life-shortening.

129. The Committee carried out a special study of the so-called aging effects of radiation and presented the results in the 1982 report. There seemed to be insufficient grounds to define aging in precise, biological terms, which would allow postulating non-specific effects of radiation at low doses and dose rates that might cause an animal to age prematurely. The Committee therefore focused on the life-shortening action of radiation, an effect that can be more objectively defined. At the doses of greatest interest for practical purposes, that is, those well below the **LD$_{50}$** range and down to the smallest doses and dose rates, evidence showed overwhelmingly that irradiated animals live, on the average, fewer years than non-irradiated controls.

130. This life-shortening effect has precise relationships with dose and time. **A** very large body of evidence in experimental animals allowed the report to conclude that at low to intermediate doses and dose rates, life shortening is essentially due to the induction of malignancies at a rate above the natural rate characteristic of the species investigated. This conclusion applies to experimental animals and, as far as could be judged from limited human experience, also to man.

131. In its 1969 report, the Committee presented a special study of the effects of radiation on the nervous system. That review also covered aspects of morphological and functional disturbances produced by irradiation during the pre-natal stages. Irradiation of the nervous system can cause effects in adults only at high doses, in which case there are profound structural and functional alterations. It was **recognized,** however, that for doses as low as 0.1 Gy or less, reactions of a "physiological nature" could be induced. The most remarkable finding remained the striking difference in sensitivity between the pre- and post-natal stages, the former being much more vulnerable than the latter.

132. The same report contained a separate annex on the induction **of** chromosomal aberrations in human germinal and somatic cell lines. The induction of **chromosomal** aberrations in somatic cells is an interesting effect by virtue of its potential use as an _in vivo_ dosimeter and its biological significance with respect to the

causation Of (Or **correlations with**) induction of **malignancies.** The annex covered
in depth the dose-time relationships for the induction of **chromosoml damage and
the variability of aberrations as a function** *of* other physical and biological
agents. **It concluded that, aside** from its **practical applications** in **biological
dosimetry, chromosomal analysis could be of little use in assessing the risk of
neoplastic, immunological or life-shortening effects of radiation.** **Risk estimates
would continue to** be **baeed on the observed incidences of the specific clinical
conditions as a function of dose, a conclusion that remains** true **to thin day.**

133. The 1972 report contained a special otudy on the effects of radiation on the
immune **response wherein the Committee, mostly** on **the basis of experimental data,
tried to discuss the role the immune** system **plays in the development of early and
late radiation effects, essentially those of the non-stochastic** type. **The study
concluded that the immune system has iarge, built-in safety factors that allow it
to withstand and recover from substantial injury by radiation.** The Committee
reported that at whole-body doses around 9.1 Gy, damage to the immune system **could
be observed but that such damage did not cause** great concern. **Whole-body doses
higher by an order of magnitude could** increase **susceptibility to infection, while
doses of 2 or more Gy could significantly increase the** risk **of mortality from
infection.** For **non-stochastic effects, these conclusions still appear to be valid.**

134. Another special study was carried out **of the possible interaction between
radiation and othar agents that are widely distributed in the en ronment. This
study was also contained in the 1982 report, and the Committee paid particular
attention in it to exposure conditions that affect large numbers of people, thereby
substantially changing average risk coefficients.**

**135. The Committee found that for effects of wide practical significance (induction
of cancer,** genetic **effects or developmental abnormalities), there was little
systematic information to substantiate claims of non-additive interactions** between
radiat ion and ither **agents.** **The theoretical analyses, which** were accompanied by
illustrative examples from **experimental or epidemiological work, treated** this
**matter in all its complexity: the different natures of the interacting agents,
their different mechanism of action, the different** dose levels **and the different**
ways of **administering the doses – all could give rise to a variety of possible
interactions, in the additive, inhibiting or synergistic** sense, but **only one case
of synergism appeared** to be well documented, **that between tobacco** smoke **and radon
decay products in uranium miners.** **This synergism prevents the direct extrapolation
of findings** in the miners to the general population.

III. THE PRESENT SITUATION

136. This chapter describes the Committee's findings and conclusions in its most recent reports. For most subjects the latest account is the one contained in the present (1989) report, but for some subjects thrt are not reported here, e.g.
● xposurss f tom nuclear explosions, the latest account is contained in the 1982 report.

A. Radiation levels and doses

1. Natural sources of radiation 3/

137. The assessment of the radiation doses in humans from natural source8 is Of special importance because natural radiation is by far the largest contributor to the collective dose received by the world population, The natural radiation sources are classified into;

(c) External sources of extraterrestrial origin (that is, cosmic radiation) and radiation of terrestrial origin (that is, the radioactive nuclides present in the crust of the earth, in building materials and in air);

(b) Internal sources, comprising the naturally occurring radio-nuclides that are taken into the human body.

138. Some of the contributions to the total exposure from the nature1 radiation background are quite constant in space and time and practically independent of human practices and activities. This is true, for example, of the doses received from the ingestion of potassium-40, an element that is homeostatically controlled, and also of doses from the inhalation and ingestion of cosmogonic radio-nuclidrs, which are relatively homogeneously distributed over the surface of the globe. Other contributions depend strongly on human activities and practices and are therefore widely variable. The doses from indoor inhalation of radon and thoron decay products are examples: building design, as well as the choice of building materials and of ventilation systems, influences the indoor levels, so that as techniques and practices evolve, the doses received from radon will also change. Between those extreme types of exposure, there are some intermediate types: external doses from cosmic rays, which are affected by human practices and are quits predictable but uncontrollable (except by moving to an area where the dose is lower); doses from the inhalation and ingestion of long-lived nuclides of the uranium-238 and thorium-232 decay series, which make a small contribution to the total dose from natural sources and are relatively constant in space; and doses from external irradiation by terrestrial sources, which are also significantly altered by human activities end practices, especially through indoor exposure.

139. The Committee has reassessed the doses received globally from natural radiation sources (table 4). The mean annual effective dose equivalent is estimated to be 2.4 mSv; it refers to the adult part of the population. Variation around this mean is due mainly to variations in the external exposure to terrestrial sources and in the internal exposure (inhalation) to short-lived decay products of radon isotopes. The external exposures typically vary around the mean by a factor of 1.5 and the internal ones by a factor of 2.5. For both types of exposure, the extreme values vary around the mean by a factor of 100.

Table 4. <u>Annual effective dose equivalent from natural sources</u>

Source of irradiation	Annual effective dose equivalent (mSv)		
	External	Internal	Total
Cosmic rays:			
Directly ioniaing component	0.30		0.30
Neutron component	0.055		0.055
Cosmogenic radio-nuclides		0.015	0.015
Primordial radio-nuclides:			
Potassium-40	0.15	0.18	0.33
Rubidium-87		0.006	0.006
Uranium-238 series:	0.1	1.24	1.34
Uranium-238 to uranium-234		0.005	
Thorium-230		0.007	
Radium-226		0.007	
Radon-222 to polonium-214		1.1	
Lead-210 to polonium-210		0.12	
Thorium-232 series:	0.16	0.18	0.34
Thorium-232		0.003	
Radium-228 to radium-224		0.013	
Radon-220 to tellurium-208		0.16	
Total	0.8	1.6	2.4

140. There are several changes from the estimates given in the 1982 report:

(a) For external exposure to cosmic radiation, the new **estimate** of the annual effective dose equivalent is higher by 50 microsievert, from taking into account the geographical distribution of the world population as a function of altitude as well as the shielding effect of the building materials;

(b) For external exposure to terrestrial sources of radiation, the estimate of the annual effective dose equivalent has been raised by 60 microsievert as a result of a better knowledge of the indoor gamma absorbed doses in air;

(c) The estimates of the annual effective dose equivalents from internal exposure to primordial radio-nuclides have been slightly decreased for the uranium-238 and lead-210 series as well as for the decay products of radon-220, whereas those for the short-lived decay products of radon-222 have been increased by about 300 microsievert on the basis of the results of nation-wide indoor **surveys.**

The net effect of these corrections is a 20 per cent increase in the estimate of . the annual effective dose equivalent from all natural sources of radiation.

141. Table 4 shows the paramount importance of doses from the inhalation of radon-222 and its short-lived decay products. Industrial activities that release materials with enhanced concentrations **of** naturally occurring radio-nuclides do not significantly alter the overall exposure estimates.

2. Nuclear explosions

142. *In* its 1982 report, the Committee assessed the exposures to the world's population from the release to the environment of radioactive material6 produced in nuclear explosions carried out in the atmosphere since 1945. Since no atmospheric nuclear tests have taken place since 1980, the assessment remains complete and valid.

143. The number and yield of atmospheric nuclear explosions are summarized in table 5, which shows that the most test programmes took place during the periods 1957-1958 and 1961-1962. Large-yield explosion6 carry radioactive debris into the stratosphere, from where it is dispersed and deposited around the world (this is known as stratospheric radioactive fall-out). Exposures to population6 are highest in the temperate regions and in the northern hemisphere, where most of the testing occurred. The dose commitment *for* the southern temperate zone is about 70 per cent of that for the northern temperate zone. The radiation doses are due mostly to the ingestion of radio-nuclides that have become incorporated in food6 and to external irradiation from ground deposition.

Table 5. **Number and yield of atmospheric nuclear explosions**

Year	Number	Estimated yield (Mt) Fission	Total
1945-1951	26	0.8	0.8
1952-1954	31	37.0	60.0
1955-1956	44	14.0	31.0
1957-1958	128	40.0	81.0
1959-1960	3	0.1	0.1
1961-1962	128	102.0	340.0
1963	0	0.0	0.0
1964-1969	22	10.6	15.5
1970-1974	34	10.0	12.2
1975	0	0.0	0.0
1976-1980	7	2.9	4.8
1981-1987	0	No further tests	

144. The most significant radio-nuclides contributing to the assessed dose commitment6 *for* various parts of the world from all atmospheric tests carried out 60 far are, in decreasing order of importance; carbon-14, caesium-137, zirconium-95, strontium-90, rubidium-106, cerium-144 and tritium. Residual irradiation from only four of these, carbon-14, caesium-137, strontium-90 and tritium, remain6 to be received by the present and future world population. An additional contribution of about 0.1 per cent of the total effective dose equivalent commitment will be received from plutonium-239, plutonium-240 and americium-241 at very low dose rate6 over thousand6 of years.

145. The collective effective dose equivalent commitment due to all atmospheric nuclear explosions was estimated in the 1982 report to be $3 \cdot 10^7$ man Sv, an estimate that is still valid. This value, which takes into account projected future growth of the population of the world, was found to be equivalent to about four years of exposure to natural sources for the population of the late 1970s, on the basis of an annual per caput exposure to natural sources of 2 mSv and a world population of $4 \cdot 10^9$. Owing to the increase in the world population to about $5 \cdot 10^9$ at the present time and to the revised estimate, 2.4 mSv, for the annual per caput exposure to natural sources, the collective effective dose equivalent commitment due to all atmospheric nuclear explosions is now assessed to be equivalent to about three years of exposure to natural sources for the present population.

3. Nuclear power production 4/

146. The number of nuclear reactors being operated to generate electricity has increased since the 1982 report. At the end of 1987, the 417 reactors operating in 26 countries had an installed capacity of 298 GW. This represents a 100 per cent increase in capacity since the Committee last reported in 1982, when installed capacity was 144 GW. Projections to the year 2000, although still somewhat speculative, amount to around 500 GW, a further growth of 80 per cent from present capacity.

147. The nuclear fuel cycle includes several steps: mining and milling of uranium ores; enrichment of the isotopic content of uranium-235 for some types of reactors: fabrication of fuel elements; production of energy in the reactors; reprocessing (although this is not always under taken) of irradiated fuel and recycling of the fissile and fertile nuclides recovered; transportation of nuclear materials between fuel cycle installations; and, finally, the disposal of radioactive wastes. Although most of the radioactive materials associated with nuclear power production are present in the irradiated fuel, small amounts are released to the environment in effluents at each of the steps in the cycle. Most of these releases are only of local and regional concern, because the radio-nuclides have short half-lives and are limited in their environmental mobility. However, some nuclides, because of their long half-lives or rapid transfer through the environment, may contribute to the irradiation of man on a global scale.

148. For each step in the fuel cycle and its associated release of radioactive materials, the Committee has evaluated the doses to workers within nuclear installations and to members of the public. In its evaluations, four population groups have been considered: those exposed in normal conditions because of their work within the fuel cycle; the population living within about 100 km of the plant; the population within a few thousand kilometres; and, finally, the world population.

149. The concentrations of radio-nuclides in effluents are generally low, and it is hardly feasible and not practicable to monitor members of the population for uptake of radio-nuclides. Instead, environmental modelling has been used by the Committee to estimate doses at long distances from the plant. The transfer of radio-nuclides through environmental media can be predicted from measured values obtained by monitoring foodstuffs and water, and from experimental studies.

150. The starting point for environmental modelling at long distances is data **on** the quantities and composition of radioactive materials emanating from various nuclear installations. This information is usually available to the Committee from those countries having nuclear power programmes and has been collected for the six-year period **1980-1985.** Since the size of a particular stage in the nuclear fuel cycle is proportional to the nuclear generating capacity served by the stage, the releases have been normalized per gigawatt year of generated electric energy, enabling comparisons to be made and to facilitate the use of averages over all plants of a similar conceptual design; the results are not representative of a specific site, but they do give an idea of the impact of each type of facility. Averaging overall energy production and for all plants of a particular type accounts also for releases that **may** arise during maintenance shut-downs, when little or no electricity is generated.

151. To assess the collective doses corresponding to the normalised releases, the Committee had previously specified hypothetical sites with broadly representative characteristics for each stage of the fuel cycle: mining and milling, enrichment and fabrication, reactor operation and reprocessing. The Committee also assumed that the environment receiving the releases from each model facility was a hypothetical environment containing the main features of existing sites, so that the **most** common pathways to man are included. The Committee has used the same models again because it believes they are still adequate for the purpose and because doing so allows the current impact to be compared with the previously assessed impact of 1974-1979.

152. Uranium mines give rise to effluents, which when operating consist mainly of ventilation air in the case of underground mines and of releases into the pit in the case of surface mines. Further effluents are produced during milling operations to extract the uranium. The stockpiles of ore and other extracted materials are the source of airborne emissions when the mine is operating, and this source persists even after the mine has been closed. The tailings that are discharged from the mills also become long-term sources of airborne emissions. The **most** important radio-nuclide in all these airborne releases is radon-222. Using the same general models as in the 1982 report, doses have been assessed both for the operational period and for the long term (10^4 years). Doses from fuel fabrication and transport have also been assessed, but since these are so much **smaller** than the doses from other components **of** the nuclear fuel cycle, they are not considered separately.

153. During operation of nuclear power stations and reprocessing plants, solid wastes are produced and have to be disposed of. **For** purposes of analysis, these wastes have been characterised in **terms** of volumes and activity concentrations of important radio-nuclides per unit energy generated. Two typical disposal facilities of the shallow land burial type were specified and terrestrial dispersion models used to calculate the release rates of radio-nuclides and the resulting effective dose equivalents.

154. The only operating commercial fuel reprocessing plants are at Sellafield in the United Kingdom and at Cap de la Hague and Marcoule in France. In its 1982 report, the Committee assessed the impact of reprocessing using a notional plant representative of plants that would be reprocessing oxide fuel in the future. At present the throughput of fuel at the three reprocessing plants represents an energy output equivalent to about 5 per cent of that generated by nuclear power. The Committee has therefore decided to assess the impact of the actual reported

discharges from these commercial reprocessing plants and weight the resulting collective doses by the fraction of fuel reprocessed to obtain values of exposure per GW year generated.

155. Calculations of collective dose to the world'8 population and various subgroups require assumptions to be made about the size of these populations, their dietary and other habits, and agricultural and fishing practices. The broadly representative values of these parameters previously used by the Committee have burn retained to evaluate the radiological impact of each stage of the fuel cycle.

156. The estimates of collective effective dose equivalent to local and regional populations and to the global population from widely dispersed radio-nuclidea are given in table 6. Occupational exposures per GW year are approximately three times those received by the local and regional population.

Table 6. Collective dose per unit practice of nuclear power generation

(Man Sv per GW a)

	Over next 100 year8	Over all time
Mill tailing8 (radcn), long term	1.5	150 a/
Globally dispersed nuclides and waste	6.0	60
Local and regional exposures	4.0	4
Occupational exposures	12.0	12
Total	24.0	230

a/   Over 10,000 years.

157. Estimates of dose to the public have been reduced, partly because discharges to the environment from reactor8 have generally decreased and also the estimate for carbon-14, which account8 for half the public exposure from routine reactor releases, is much lower than the estimate in the 1982 report due to new, lower measured value8 of carbon-14 releases from heavy-water reactors.

156. The annual exposure received by the world's population from the release of radio-nuclides that become globally dispersed is currently much less than that received by local and regional populations. Only if the current level8 of discharge of these radio-nuclides continued and all fuel from all reactors were reprocessed could the global component of the annual collective effective dose equivalent eventually equal the local and regional components.

159. The collective and per caput doses *from* nuclear power production may be compared to the dose8 to the world population from natural sources of radiation. The more immediately delivered component of the normalized collective effective dose equivalent commitment ha8 been estimated to be 4 man Sv per GW a from radio-nuclides in the effluents of nuclear fuel cycle installations. For the present annual nuclear power production of about 180 GW year, the annual collective

dose is assessed to be 760 **man Sv.** Dividing by **the** world population of 5 10^9
gives an annual per caput **dose estimate of 0.15 microsievert. The doses are around**
0.01 per cent of the collective and per caput doses from natural background sources.

4. Medical exposures 5/

160. Good data on the frequency of examinations and absorbed doses from medical
examinations **come** mainly from the developed countries, which comprise less than
25 per cent of the world's population. There are fragmentary data on examination
rates or number of diagnostic units and little or no data on absorbed doses for
approximately another 25 per cent of the population, For 50 per cent of the
world's population there are no data at all. For this reason, the Committee has
developed a modelling approach based upon the good correlation that exists in **most**
countries between population per physician (about which there is **more** information)
and the medical uses of radiation.

161. Access of populations in the world to radio-diagnosis is very uneven: one
X-ray machine is shared by fewer than 2,000 people in **some** countries and by
l00,000-600,000 people in other countries. The frequency of procedures is also
very uneven: 15-20 procedures per year are carried out per 1,000 population in
some countries and l,OOO-2,000 procedures per year in others. At the present time,
there are about 5 10^9 people in the world, and **some** estimates are that more than
three quarters of the world's population have no chance of receiving any
radiological examination, regardless of what disease they have.

162. While absorbed dose data exist for many standard radiographic and nuclear
medicine procedures, information now available suggests that the previous absorbed
dose estimates for the world population **may** be somewhat low. An important reason
for this is the widespread use of fluoroscopy in developing countries. There are
also large numbers of malfunctioning machines, which produce high doses. Neither
of these factors was widely appreciated in the past.

163. The collective effective dose equivalent **from** diagnostic X-ray procedures is
far greater than that from dental or diagnostic nuclear medicine examinations. The
per caput annual effective dose equivalent is likely to be no lower than 0.4 **mSv**
(the Committee's previous estimate) and **may** be as high as 1.0 **mSv.** Similarly, the
annual genetically significant dose **may** range from 0.1 to 0.3 **mSv.** However,
considering the age structure of the population, the effective dose equivalent **may**
overestimate the detriment. This would be particularly true in countries where the
older portion of the population receives **most of** the medical irradiation.

164. The world-wide collective effective dose equivalent is estimated to be between
2 and 5 10^6 **man Sv.** Of this, **90-95** per cent is attributable to diagnostic X-ray
procedures. Dental radiography, nuclear medicine and radiation therapy (ignoring
target doses) together contribute only 5-10 per cent of the collective dose. In
developed countries, the contribution to the collective effective dose equivalent
is about 0.001 man Sv per examination.

165. There are many possibilities for reducing dose without jeopardizing **the**
benefits of the radiological practices. In the developed countries, it **may** be
possible to reduce the per caput effective dose equivalent by half. In the less
developed countries, the use of radiography rather than fluoroscopy, appropriate
collimation, proper film developing, as well as the calibration and maintenance of

equipment, would reduce the dose per examination; however, the feasibility and costs of these measures are not known. The genetically significant dose can be significantly reduced through the use of gonadal shielding, a practical, low cost method. Still, the collective effective dose equivalent may increase as X-ray examinations become more widely available in a number of countries, and such an increase may in fact be appropriate.

166. The frequency and total use of medical irradiation is expected to increase over the next several decades because of the aging of the world's population, the growth of this population, and urbanization in the developing countries. By the year 2000, the collective dose will probably have increased by 50 per cent, and by 2025 it may have more than doubled.

5. Occupational exposures 6/

167. Two categories of 'workers are exposed to radiation: workers in the nuclear industry and in the medical field, where radiation sources are managed, and workers in occupations where higher background radiation levels are encountered (air crews and non-uranium miners are examples), The Committee gave a full assessment of occupational exposures in its 1982 report. Updated estimates of exposures to workers in nuclear fuel cycle activities (average annual doses in the range of 3 to 8 mSv for reactor operation, and a collective dose of 12 man Sv for each GW year of electric energy generated, in total for all work in the whole nuclear fuel cycle, see table 6) and to medical personnel (average annual doses in the range of 0.3 to 3 mSv, and a collective dose of about 1 man Sv per million of population, see also para. 166; in developed countries an average occupational dose of about 1 microsievert per examination) are included along with exposures of the general public in the respective annexes dealing with these subjects.

168. Exposures of radiation workers are subject to detailed regulatory control in all countries and in the majority of cases the doses are but a small fraction of established limits, partly as a result of the current emphasis on optimising radiation protection. The collective effective dose equivalent commitment per unit of electricity generated to workers in all nuclear fuel cycle installations is estimated to have changed little from the commitment previously estimated by the Committee, but such stability is only to be expected if reductions in exposures are balanced by the greater numbers of workers employed in the expanding industry.

169. Occupational exposure from medical practices includes the contributions from diagnostic X-ray procedures, dental radiography, nuclear medicine and radiation therapy. The average annual collective effective dose equivalent from occupational exposures in these practices is about 1 man Sv per 10^6 population. In spite of the increase in the medical uses of radiation in most countries, the limited trend data indicate that both individual and collective annual occupational doses are decreasing by 10-20 per cent every decade. For developed countries, the average occupational exposure is about 1 microsievert per examination.

6. Miscellaneous exposures

170. Exposures from miscellaneous sources of radiation are evaluated by the Committee whenever warranted by new information or new developments. The latest assessment, in the 1982 report, dealt with various consumer devices that contain radioactive materials and with electronic and electrical equipment that emit X-rays. Individual exposures to these various sources were generally very small. The Committee believes that assessment to be still valid and feels that no new evaluation is required.

7. Accidents

171. With the large size of the nuclear industry in some countries and the large number of radiation sources used for industrial and medical purposes, accidents are bound to happen. The accidents that have occurred have generally been criticality and other industrial accidents that exposed one or a few workers; transport accidents, including also accidents involving satellites, aircraft and submarines; losses or thefts of radiation sources; and reactor accidents.

172. Three reactor accidents have caused measurable exposures of the public: Windscale in 1957, Three Mile Island in 1979, and Chernobyl in 1986. The Chernobyl nuclear reactor accident was a significant event and is discussed in detail in two annexes (annex D, "Exposures from the Chernobyl accident", and annex G, "Early effects in man of high doses of radiation").

173. In all, six notable accidents have occurred since 1982, when the Committee last dealt with this subject:

1983: Constituyentes, Argentina. An accidental prompt critical excursion occurred during a configuration change in a critical assembly, resulting in the death of an operator, who was only 3-4 metres away. The dose to the victim was estimated to be 5-20 Gy from gamma rays and 14-17 Gy from neutrons.

1983: Ciudad Juarex, Mexico. An improperly disposed of cobalt-60 source found its way into a scrap metal shipment, contaminating the delivery truck, the roadsides and the processed steel into which the scrap was incorporated. Some 300-500 individuals were exposed, 10 to doses of I-3 Gy. There were no deaths.

1984: Mohammedia, Morocco. A source of iridium-192 used to make radiographs of welds at a construction site became detached from the take-up line to its shielded container. The source dropped to the ground and was noticed by a passer-by, who took it home. Eight persons, an entire family, died from the radiation over-exposure with doses of 8-25 Gy.

1986: Texas, United States. An accident at a linear accelerator caused two deaths from over-exposure.

1986: Chernobyl, Soviet Union. The accident at the nuclear power station resulted in two immediate deaths of reactor operating personnel from the explosion. About 145 firemen and emergency workers suffered acute radiation sickness: 28 of them died during the three months following

the accident. There were 30 deaths in all; one worker died **from** mechanical injury and one **from** burns. Local residents, none **of** whom received high exposures, were evacuated. The widespread dispersion of the released materials caused low exposures, primarily to **populations** of the western part of the Soviet Union and other European countries.

1987: Goiania, Brazil. A **caesium-137** source was dismantled in a residential area causing **some** 240 people to become contaminated. Fifty-four of them were hospitalixed and four died.

8. The Chernobvl accident 7/

174. The accident at the Chernobyl nuclear reactor in the Soviet Union, which occurred on 26 April 1986, caused extensive contamination in the local area and resulted in radioactive material becoming widely dispersed and deposited in European countries and throughout the northern hemisphere. The extent to which such a wide region could be affected by an event of this type was unanticipated. Intensive monitoring was undertaken to evaluate the radiation levels.

175. It was apparent soon after the arrest of releases from the reactor that the radiological impact of the accident, from the point of view of individual risk, would be insignificant outside a limited region within the Soviet Union, either because contamination levels were generally low or because remedial actions to ban the consumption of particularly contaminated foodstuffs prevented high exposures.

176. The accident at the Chernobyl reactor occurred in the course of a low-power engineering test, during which safety systems had been switched off. The uncontrollable instabilities that developed caused explosions and fire, which damaged the reactor and allowed radioactive gases and particles to be released into the environment. The fire was extinguished and the reactor core sealed off by the tenth day after the accident.

177. The death toll within three months from the accident was 30 members of the reactor's operating staff and the fire-fighting crew. Two died immediately, 28 died from radiation injury. Radiation doses to the local population were well below the doses that could cause immediate effects. Local residents were evacuated from a 30 **km** exclusion **zone** surrounding the reactor. Agricultural activities were halted and a large-scale decontamination effort has been undertaken.

178. The initial release of radioactive materials from the accident spread with winds, in a northerly directjon. Subsequent releases dispersed towards the west and south-west and in other directions as well. Deposition onto the ground was governed primarily **by** rainfall, which occurred sporadically at the time in Europe. The deposition pattern and the associated transfer of radio-nuclides to foods and irradiation of individuals was very inhomogeneous, necessitating a regional approach for dose calculations.

179. Measurements since the accident have shown that the radio-nuclides contributing most significantly to doses are iodine-131, caesium-134 and **caesium-137** mainly by external irradiation from deposited material and by ingestion of contaminated foods. The Committee's dose assessment takes **most** account for these important radio-nuclides and pathways.

180. Detailed information was available to the Committee to calculate first-year radiation doses in the Soviet Union and all European countries. To extend these results and to estimate the projected doses from deposited materials, wider regions were evaluated. Since there is insignificant inter-hemispheric mixing of material released into the troposphere, southern hemisphere countries could only have been affected through imported food; this possibility is accounted for in the assessment by considering total food production as well as local consumption in northern hemisphere countries.

181. The input values for the calculation made full use of measurements during the fir. t year following the accident. Thereafter, projections are required to esti. te the further contributions to dose, primarily from caesium-137. T.e projections are bared on experience acquired from past studies of radioactive fall-cut from the atmospheric testing of nuclear weapons,

182. The results of calculations of the first-year committed effective dose equivalents in 34 countries are illuotrated in figure 1. The highest values are for Bulgaria, Austria, Greece and Romania, followed by other countries of northern, eastern and south-eastern Europe. Countries further to the west in Europe and also countries of Asia, North Africa, North and Central America were less affected, which is in accord with the deposition pattern.

183. The dose commitments from the accident are delivered over several years, mostly due to continuing exposures from caesium-137. On average, some 30 per cent of the effective dose equivalent commitments were delivered in the first year following the accident. The dose commitments over all time in wider regions of the world are illustrated in figure 2.

184. The main outcome of the dose assessment is the collective effective dose equivalent commitment. This is estimated to be approximately 600,000 man Sv. Of this amount, 40 per cent will be received in the Soviet Union and 57 per cent in Europe. The remaining 3 per cent will be received by other countries of the northern hemisphere.

185. For comparison with figure 1, the one year effective dose equivalent from natural sources is 2.4 mSv. For comparison with figure 2, it should be noted that most of the dose commitment will be received within 30 years of the accident. The 30-year effective dose equivalent from natural sources is about 70 mSv. In using these comparisons, it should be remembered that the doses are averages over large geographical areas within which there will be local variations, in the doses from Chernobyl and those from natural sources.

FIRST-YEAR EFFECTIVE DOSE EQUIVALENT (mSv)

Countries (top to bottom): BULGARIA, AUSTRIA, GREECE, ROMANIA, FINLAND, YUGOSLAVIA, CZECHOSLOVAKIA, ITALY, SWITZERLAND, POLAND, USSR, HUNGARY, NORWAY, GERMAN DEM. REP., TURKEY, SWEDEN, GERMANY, FED.REP. OF, IRELAND, LUXEMBOURG, ISRAEL, CYPRUS, FRANCE, NETHERLANDS, BELGIUM, DENMARK, UNITED KINGDOM, SYRIA, CHINA, JAPAN, SPAIN, INDIA, PORTUGAL, UNITED STATES, CANADA
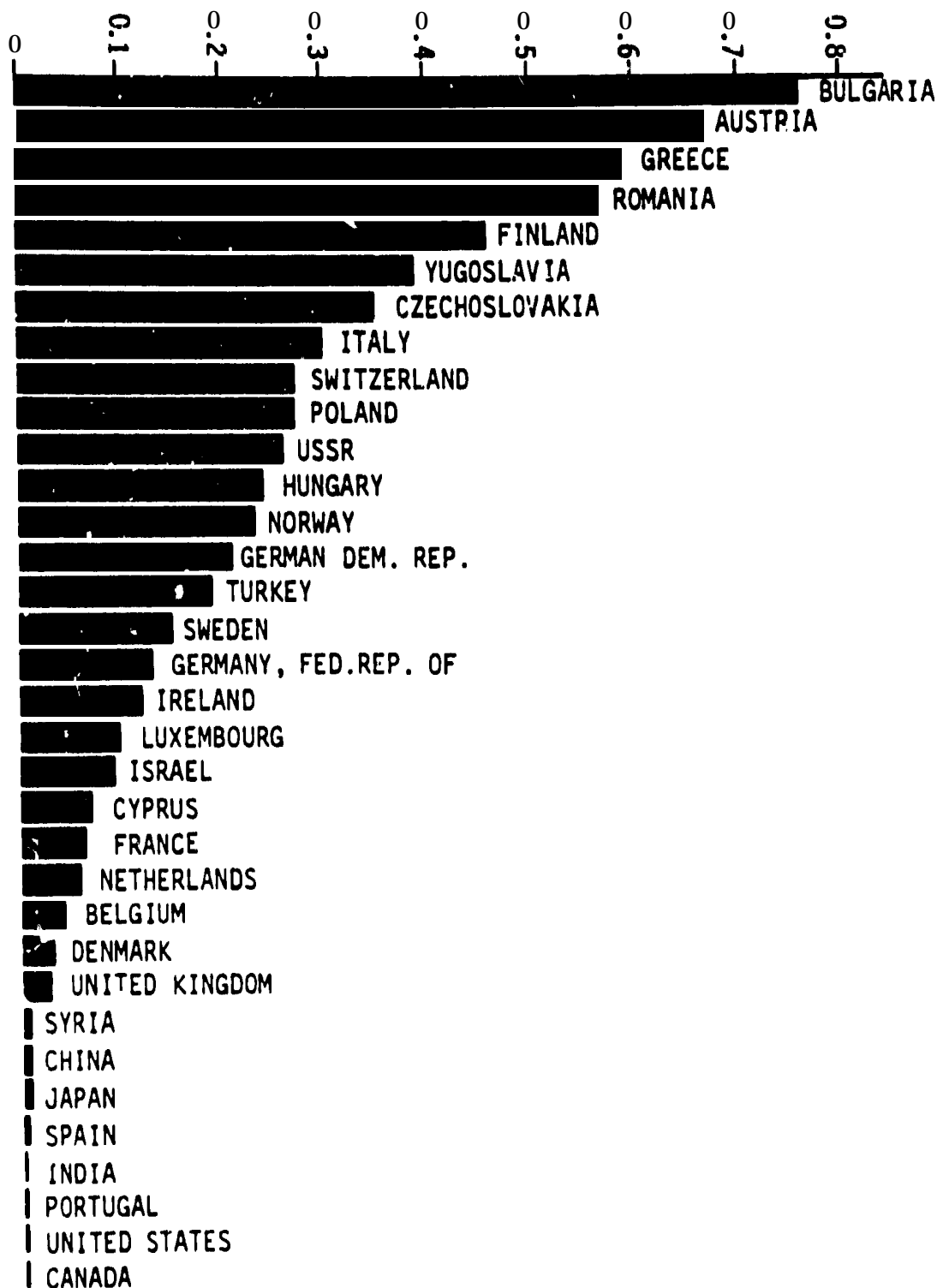
Figure 1. Country average first-year committed effective dose equivalent from the Chernobyl accident

EFFECTIVE DOSE EQUIVALENT COMMITMENT (mSv)

SOUTHEAST EUROPE
NORTH EUROPE
CENTRAL EUROPE
USSR
SOUTHWEST ASIA
WEST EUROPE
NORTH AFRICA
EAST ASIA
GREENLAND
EAST AFRICA
CENTRAL AFRICA
MIDDLE AND SOUTH ASIA
SOUTHWEST EUROPE
WEST AFRICA
NORTH AMERICA
SOUTHEAST ASIA
CARIBBEAN
CENTRAL AMERICA
SOUTH AMERICA

Figure 2. Regional average effective dose equivalent commitment from the Chernobyl accident

44
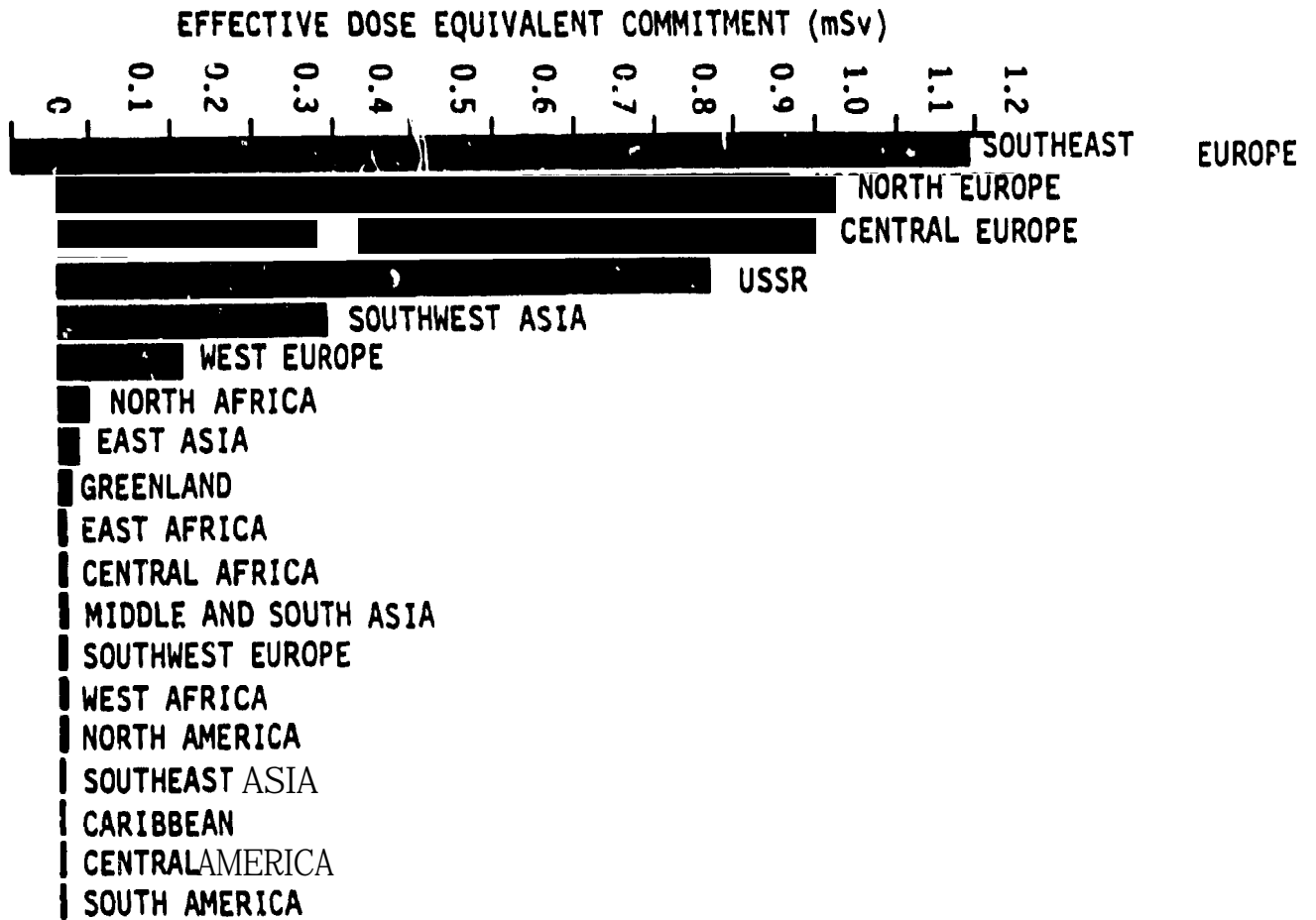
B. Radiation effects

1. Hereditary harm 8/

186. In spite of the considerable progress made during the past few years in understanding the mutation process, there have been no major conceptual changes in the formulation Of risk estimates between the 1986 report and the present one that would Warrant revising of the estimate6 of natural or radiation-induced Mendelian and chromosomal disorders using the doubling dose method. However, an attempt has been made to quantify risks of induction of recessive diseases by this method. New data on the prevalence of congenital anomalies and other disorders of complex aetiology (discussed in 1986) raise a number of questions: Can the doubling dose of 1 Gy be confidently applied to disorder6 of complex aetiology? What is the magnitude of mutational component of these disorders7 Is it meaningful to provide estimates for these disorders in the continuing absence of experimental or human data bearing on the mechanisms of their maintenance in a population and on their possible response to radiation? Until new data become available, the Committee concluded that it was unable to provide meaningful risk estimates for these disorders. Since this situation remains true in 1988, the risk estimates for hereditary effects that the Committee offers at the present time are those shown in table 7. However, an attempt has now been made to quantify risks of induction of recessive disease6 by this method.

187. Using direct methods, the Committee estimated that 10-20 per 10^{-2} Gy per million live born as having genetic diseases caused by induced dominant mutations. The Committee also estimated about 10 extra cases of genetically abnormal children would be expected in the first 10 generations per million live births per 10^{-2} Gy due to recessive mutations. Finally, as to balanced chromosomal rearrangements, the Committee assessed the risk to be between 1 and 15 cases of congenitally malformed children per million live births per 10^{-2} Gy of paternal irradiation (0-5 cases for maternal irradiation). These figures (see table 2) are also thought to remain valid.

188. Although it did not explicitly say so until 1982, the Committee has always realized that simply presenting the number of serious genetic diseases is to ignore the full measure of the harm. In the absence of objective and quantifiable indicators of severity, it is hard to assess the full impact of radiation risks in terms of the individual, familial and social burdens imposed by these diseases. Therefore, starting with the 1982 report, the Committee began systematically to review data bearing on these problems, to gain a better idea of the true detriment associated with hereditary diseases. Although it is confident that an inquiry of this nature will provide a more refined way of assessing the impact of radiation-induced disorders, the Committee feels that its methodology is not yet ready for use.

189. The Committee wishes to stress that there are still no direct data in man on the induction by radiation of hereditary, diseases. Until such data become available there is no alternative but to continue to use data obtained in other mammalian species, suitably corrected to accord with what is known of human genetics, to estimate the risk of hereditary diseases in man.

Table 7. <u>Estimates of risk of severe genetic disease per million
e births in a population exposed to a genetically
significant dose equivalent of 1 Sv per generation of
ow-dose-rate, low-dose irradiation, according to the
doubling dose method</u>

(Based on the 1986 UNSCEAR report and subsequent work)

(The doubling dose equivalent assumed in these calculations is 1 Sv)

Disease classification	Current incidence per million live births	Effect of 1 Sv per generation		
		First generation	Second generation	Equilibrium
Autosomal dominant and X-linked	10 000	1 500	1 300	10 000
Autosomal recessive	2 500	5	5	1 500
Chromosomal				
Due to structural anomalies	400	240	96	400
Due to numerical anomalies	3 400	Probably very small		
Congenital anomalies	60 000)			
Other multifactorial diseases	600 000)	Not estimated		
Early acting dominants)				
Heritable tumours)	Unknown	Not estimated		
Totals of estimated risk		1 700	1 400	12 000

190. All the numerical estimates of genetic risks discussed thus far have been obtained on the basis of genetically significant doses, i.e. on the assumption that the doses are received by individuals before or during the reproductive period. It is obvious that in the exposure of an entire population, the genetically significant doses are markedly less than the total doses received over a lifetime: damage sustained by the germ cells of individuals who are beyond the reproductive period or who are not procreating for any other reason poses no genetic risks. If it is assumed that the mean age at reproduction is 30 years and that the average life expectancy at birth is 75 years, the dose received by age 30 is 40 per cent of the total dose.

191. To derive risk coefficients for genetic diseases in a population, one needs, accordingly, to multiply the genetic risk estimates discussed earlier by 0.40. The calculations shown below make use of the most recent risk estimates presented in table 7 of annex E, "Genetic hazards", and give the risk coefficients per sievert:

(a) Risk coefficient on the basis of **gonadal** dose
in the reproductive segment of the population
(from annex E, table 7); for quantifiable damage
only, over all generations

$12,000/10^6$ or
1.2 per cent

(b) Risk coefficient for the whole population, not
only the reproductive segment, all generations
(0.4 x 1.2 per cent)

0.5 per cent

(c) Risk coefficient for the first two generations,
but otherwise as in (a) above

3, $100/10^6$ or
0.3 per cent

(d) Risk coefficient for the whole population, for
the first two generations (0.4 x 0.3 per cent)

0.1 per cent

2. Radiation carcinoaenesis in man 9/

192. The most recent data in the field of radiation-induced cancer in man have been
examined with the following in mind:

(a) Impressive advances in understanding the molecular mechanisms of cancer
induction:

(b) The analysis made in annex B of the 1986 report, "Dose-response
relationships for radiation-induced cancer":

(c) Extensive additional follow-up data on **major** epidemiological studies such
as those of the survivors of Hiroshima and Nagasaki:

(d) A revised dosimetric **system** for the survivors of Hiroshima and Nagasaki
that allows a better analysis of this important epidemiological series.

193. Several factors influence the probability that an individual exposed to
radiation will develop cancer. Some of these, the host factors, pertain to the
individual, such as his genetic background, age, sex and state of health; others
pertain to the conditions of irradiation, such as the dose delivered, the time
period over which the dose was received and the quality of the radiation: still
others are factors that **may** interact with radiation to affect the susceptibility of
the host, such as his living habits or his exposure to other toxic agents. Thus,
there is no single, simple way to assess the effects, so several approaches have
been taken.

194. One approach is to study the effects of different exposure or host conditions
on biological models of carcinogenesis. This approach allows analysing one or
another aspect of the risk, e.g. its variation with time or with the age of the
exposed individuals. Another approach aims at analysing dose-response and
risk-projection relationships. A **third** approach is the direct regression study of
epidemiological data, especially through modern multiple regression techniques,
which are particularly suited to the complexity of these phenomena.

195. The most informative epidemiologic81 series are those which were carried out in the following groups: (a) people who were chronically exposed to high or intermediate doses of radiation when the dangers of such exposures were as yet unknown) (b) people who were chronically exposed to low doses for occupational, medical or environmental reasons; (c) people who received high doses to some parts of the body over short periods for therapeutic purposes; (d) people who were, and are, exposed to low doses of radiation for medical diagnostic purposes; (e) special cohorts who were irradiated externally as a consequence of the atomic bombings at Hiroshima and Nagasaki or internally as a consequence of fall-out from the testing of nuclear weapons] and finally, (f) isolated individuals who received fairly high doses in accidents of various sorts.

196. Two methods have been employed in the epidemiological investigation of the groups listed above: (a) cohort studies, in which exposed individuals are analysed retrospectively for their cancer experience and compared with a suitably matched non-exposed control group; and (b) case control studies, in which individuals in an exposed population are matched with individuals of a control population and are followed prospectively. The second method has distinct advantages but of course can be applied only to special experiences.

197. Most of the retrospective studies discussed in the 197'1 report have continued up to the present time, and new results have been reported. In several series, such as that on radiation-induced breast cancer, earlier findings were improved and dose-response patterns were made more precise by combining data from several investigations. In other series, such as that on pelvic irradiation for tumours of the uterine cervix, earlier findings were at least partially called into question. In yet other series, such as those on occupationally exposed groups, the earlier findings have, on closer examination and reinterpretation, been criticiced for different types of investigating and reporting bias. Uncertainties in the dosimetry, tho unsuitability of control groups and potential or actual difficulties in the ascertainment of tumours were some of the problems encountered.

138. All of the most important prospective studies that were in progress in 1977 are still in progress. Three more sets of mortality data, as well as additional incidence data, are now available from the survivors of Hiroshima and Nagasaki, and these have improved the dose-response estimates for some tumour types and have added other malignancies (colon, ovary, multiple myeloma) to the list of those already known to be radiation-induced. Some information has also been added to the studies of people exposed at the Hanfold nuclear facility and to fall-out in the Marshall Islands and of patients exposed for medical conditions ouch as ankylosing spondylitis, mastitis, pneumothorax or thymus-related irradiations. The absolute and relative risks in these cohorts of people continue to increase (save, possibly, in the patients with ankylosing spondylitis and in those who were youngest at the time of the bombings in Hiroshima and Nagasaki). All these studies must obviously continue throughout the lifetimes of the exposed individuals in order to complete the data on dose- and time-response relationships for cancer induction. Moreover, for the relevant information to be genera ized, it is also vital to know to what degree these cohorts are similar to other populations! how, and with what consequences, exposure to non-radiation risks may have changed; and how, for a general population, the risk of a given dose of radiation relates to the beckqround cancer risk. One of the central problems in risk estimation continues to be the shape of the dose-response relationship, an issue extensively treated in the 1986 report.. Although a number of models may be used to analyse the risk, each of them represents no more than an approximation to the true dose-response relation and has potential limitations or pitfalls.

199. The **mortality** experience of Hiroshima and **Nagasaki** survivors has been the single most **important** source of information on the radiation-related risk of cancer induction. A recent re-evaluation of tissue-absorbed doses in these survivors has made clear that their exposure to neutrons was substantially less than had been thought, and the relevant data, particularly those from Hiroshima, are now believed to be much less informative about the effects of neutrons than had once been presumed. The large body of experimental data and the very limited amount of epidemiological evidence on the relative biological effectiveness **(RBE)** of neutrons must therefore be carefully re-examined, with a view to arriving at some estimate of risk for this type of radiation.

ZOO. A new **international** study of patients surviving treatment for carcinoma of the cervix has provided additional data on second cancers at selected sites.

201. Lifetime cancer experience is not yet available for any of the large epidemiological studies. Therefore, to project the overall cancer risk for an exposed population, it is necessary to use models that extrapolate over time data based on only a limited period of the lives of the individuals. Two such projection models have received particular attention: (a) the additive model, which postulates that the annual excess risk arises after a period of latency and then remains constant: and **(b)** the multiplicative model, in which the time distribution of the excess risk follows the same pattern as the time distribution of natural cancers, i.e. the excess (after latency) is given by a constant factor applied to the age dependent incidence of natural cancers in the population. Data are now available that may provide a deeper insight into the applicability of the two models, and recent findings in Japan suggest that the relative risk projection model is the more appropriate, at least for some of the most common cancer types. Firmer conclusions should be possible soon.

202. Cancer is generally understood to develop in a number of stages. That is, for malignancies to be expressed a series of events must occur and the rate at which they occur is thought to be reflected in the way cancers appear in the population over the course of time. Analysis of the various epidemiological series in the light of this notion reveals a number of inconsistencies, so that it is not yet feasible to say which stages in carcinogenesis are affected by radiation or whether more than one stage is affected or whether the multistage model is able to explain the actual process. All of these possibilities may apply to some extent. It **may** even be that events postulated at the cellular or sub-cellular level cannot be easily related to the clinical data on radiation carcinogenesis.

203. A limited number of genes, **known** as oncogenes, have been implicated in the malignant transformation of normal cells. The precise ways in which these oncogenes can be activated by radiation are not known, but so far data have not revealed any modifications that would suggest radiation plays a special role in inducing cancer or that would help to differentiate, at the genetic level, radiation-induced tumours from tumours induced by other carcinogens.

204. The Committee has carried out a detailed review of the information available on time-specific susceptibility to radiation-induced cancer and has considered separately the evidence pertaining to the exposure of children and adult subjects. Data on children show that the thyroid, the bone, the bone marrow and the breast are definitely responsive to the carcinogenic action of radiation. The majority of the children successfully treated by radiation for cancer (i.e. those carrying **localized** primary tumours) who have developed secondary tumours are those whose

primary tumour had a large **heritable** component of cause. **These** children are
obviously more prone to develop **cancer** than a normal child. In general, certain
sites are susceptible, and the genetic evidence Shows **that** this has to do with gene
regions expressed in both **the** tissue involved in the original Primary tumour
(e.g. retinoblastoma) and in the tissue of the second tumour **(e.g.** bon% sarcoma)=
Individuals with the hereditary form of retinoblastoma are **also** known to develop
osteosarcomas away from the irradiated field of in the **absence** of irradiation* The
spontaneous risk of **second** tumours in retinoblastoma patients **is** due to the somatic
development af homosygosity in those children who inherit a Single Copy of the
relevant mutation, but it is not yet known whether this is also the mechanism by
which radiation induces second tumours. There are indications in the case Of
second tumours following retinoblastoma that a multiplicative projection model **may**
apply, as it does to **most** adult tumours.

205. A number of general principles concerning the induction of tumours by
radiation can be derived. **Radiation** is detectably carcinogenic if **the** dose is high
enough, but **no** cancers unique to radiation are induced. Leukaemia (except chronic
lymphatic leukaemia) is the **most** prominently induced cancer but tumours of the
breast, thyroid, lung and bone marrow and at a number of other sites are also
induced. The frequency of induction per Gy varies with the site. **Some** tumours
such as chronic lymphatic leukaemia, squamous cell carcinoma of the cervix and
Hodgkin's disease are not induced by radiation. Induced tumours are expressed **some**
time after exposure, the latency being at least 2-5 years for leukaemia and about
10 years or more for other tumours. Age is the **most** significant host factor but
other factors such as genetics play a role. These features are explained further
in annex F.

206. In general, the results **from** cancer patients are similar to those from other
exposed groups with regard to the post-irradiation pattern of risk. However, in
some instances, the risk in cancer patients appears to be different from that in
the general population. This could be due to differences in susceptibility to
cancer, but it could also be due to differences in exposure to environmental risk
factors, e.g. smoking. Excess cancers occur in both irradiated and non-irradiated
patients, making the estimation of radiogenic risks problematic and suggesting that
inferred results **may** not be generally applicable.

207. The dose-response relationships for various forms of malignancy were discussed
extensively in annex **B** of the 1986 report. The conclusion reached there was that
each type of tumour **may** have a characteristic dose-response pattern and that it is
still difficult to assess satisfactorily the pattern for the majority of the
tumours. However, a general conclusion could be drawn that for low-LET radiations
most dose-response relationships were upward concave reaching a maximum that would
be followed by decline of the response with further increasing of the dose. This
decreasing slope and decline of the curve at high doses **seems** due to killing of the
radiation-initiated cells from which **tumours** eventually arise.

208. The Committee concluded in 1986 that for **some** tumours, i.e. carcinomas of the
female breast and perhaps of the thyroid, a linear relationship at low and
intermediate doses of low-LET radiations **gave** a good fit: for others a linear fit
could not be rejected statistically but other **models,** e.g. linear quadratic and
quadratic, approximated the data equally well. These observations are still
assumed to be basically correct, although evidence presented recently to the
Committee suggests that fractionated doses at very low doses per fraction **may** be
less effective in inducing breast cancer than deduced previously from the linear

relationship **and apparent** lack of dose-fractionation effects. Recent epidemiological studies on patients administered **131-iodine-iodides** for diagnostic purposes **suggest** that **low-LET** radiation at low dose rates is also significantly less effective than intermediate and high doses delivered at high dose rates. This means probably that the dose-response relationship for induction of cancer of the thyroid gland is also non-linear (upward concave), as was suspected in the 1986 report.

209. Many biological differences among human beings are known to modify their susceptibility to radiation-induced cancer, and the Committee examined these differences, known as host factors. Current information generally suggests sex has little **or** no effect on radiation carcinogenesis, in the sense that the sex ratio for individuals with radiation-induced malignancies (thyroid, breast, lung, **leukaemia)** is similar to that for non-irradiated individuals with the same malignancies. **Data** show further that susceptibility to radiogenic tumours decreases with increasing age, the latency periods being related not so much to age at exposure as to the tissue involved. The mean age and the age distribution of cases in adults exposed to single doses are in general similar to those in the population at large. Data on the effect of genetic constitution suggest that there **may** be a small, but not trivial, fraction of the population who are prone to cancer development **and** could thus be more susceptible to radiation or other carcinogenic agents. To improve the risk estimates, better means of identifying susceptible individuals should be developed.

210. The concluding section of the Committee's study contains an overall analytical summary of radiogenic cancer effects drawn from the **most** comprehensive sources available. From only a few epidemiological studies - primarily the survivors of the atomic bombings and patients exposed during treatment of ankylosing spondylitis or cervical cancer (leukaemia only) - the carcinogenic risk of radiation can be estimated for many different sites. All three studies comprise large numbers of people exposed to **X-** or gamma-radiation for short times and followed for long times; however, each set of data has unique characteristics. The Committee considered the results on tissue-specific **tumours** from these series and compared them with risk estimates produced by various **other** studies. The Committee's evaluation of risk **estimates** is discussed in chapter III, section C.2.

3. Early effects in man of **high** doses of radiation **10/**

211. The Committee has reviewed what is known **about** the effects that occur in man within two to three months from receiving uniformly distributed whole-body doses above **approximately** 1 Gy of X- or gamma-radiation. The data were collated from three main **sources:** accidents, the atomic bombings and radiotherapy treatments. Important information on this subject has recently become available as a consequence of the nuclear accident at the Chernobyl power plant, in the course of which about 100 people were exposed to external and internal irradiation amounting to 1 Gy or more. The Soviet delegation has prepared especially for **UNSCEAR** a detailed report entitled "Acute radiation effects in victims of the Chernobyl nuclear power plant accident", which is presented as an appendix to annex G.

212. **Early** prodromal responses during the first 48 hours after irradiation are mediated through the autonomic nervous **system** and appear as gastrointestinal and neuromuscular signs. The incidence and latency periods for these effects are dose-dependent. For instance, the dose that induces vomiting in 50 per cent of

individuals is approximately 2 Gy, and the wan latency period after this dome is abut 3 hours.

211. Doses higher than 50 Gy generally lead to death within two days from cerebrovascular injury (the so-called neurological syndrome). Uniform, whole-body doses between 10 and 50 Gy cause the gastrointestinal syndrome, which is generally fatal, with most deaths occurring during tho second week after irradiation. In spite of the ● xporienco of those who died after the atomic bombings, there is insufficient information to ● □◆)(■☺◆□ precisely the relationship between the dore and the probability of death duo to this syndrome. The time to death of the gastrointestinal syndrome depends on the renewal time of the intestinal lining and is influenced by secondary factors such as infection, haemorrage, loss of fluid, protein and ● loctrolytos.

214. Uniform, whole-body doses of less than 10 Gy but greater than 1 Gy cause the bona-marrow syndrome, the incidence and severity of which depend on dose. The initial marrow damage after low doses reduces thm number of white cells in the blood, the lymphocytes being the most sensitive indicator8 of injury. Doses of 1-2 Gy reduce the concentration of blood lymphocytes to about 50 per cent of normal within 48 hours of irradiation. Neutrophils show an initial increase over the first few days, then a dose-related fall. Ton dayr after 2-5 Gy, thara is a second abortive rise; however, if the marrow does not recover, a final decline is observed. The loss of neutrophils is associated with the onset of fever and is predictive of survival. The time course of platelet 1088 is broadly similar to that for granulocytes, but without a second abortive rime. Platelet level8 in che blood below 30,000-50,000 per microlitre are associatad with bleeding. People with the bone-marrow syndrome show an increased susceptibility to infection due to injury to the haematopoietic and the immune system.

215. In addition to the ● ymtemic effect8 described, irradiation may also caume damage to many other tissues and organ8 ● xpo8ed raparately. The resulting clinical symptoms vary as to time for appearance and severity. They may or may not be part of the syndromes described, depending upon the tissues irradiated, the does level, the modalities of irradiation and other physical and biological factors.

216. Irradiation of the skin causes lesions that are well known and very dependent on the dome and the area irradiated, in the sense that smaller doses have to take place over larger areas to elicit thr ● ruxe level of damage. Skin lesions include erythema, abnormal hair growth, epilation, desquamation and vascular and dermal injury. The dose in the basal layer of the epidermis determines the amount of cell killing and hence the degree of derquamation.

217. Injury to the mucous membranes in the mouth and throat evokes inflammation and swelling, with ulceration and necrosis after high doses. Mucosal injury is greatest in the cheeks, soft palate and hypoglossa\ region. Acute effects on the eye are also well described and very dependent on the structures irradiated and the doses received.

210. When the thorax is irradiated, pneumonitis is the earliest sign of radiation injury in the lung. It appear8 at 1-3 months for doss6 greater than 8 Gy. The time of onset of pneumonitis is not significantly dose-dependent between 6 and 12 Gy. At Chernobyl there were some patients with early lung reactions, These changes were probably multifactorial in origin.

219. High acute doses of up to 4 Gy induce temporary sterility in some male individuals, but the dose inducing prolonged sterility in all males is at learnt 6 Gy. Although some *of* the differentiating forma $\square$ $\nearrow$ ● parmatogonia respond early and are very radio-sensitive, the sperm count begins to decrease only after six weeks. In women, temporary sterility is induced by high doses up to 4 Gy and prolonged sterility by 4-10 Gy. Oldar women ara more susceptible, probably because the number of ovarian follicles decreases with age.

220. It is of interest to know the dose of radiation that causes, on average, 50 per cent of individual8 to die within 60 dayr ($LD_{50/60}$). The LD_{50} is a concept widely used in experimental *work* but theta is doubt as to its applicability in human radiation biology, except for statistical purposes. The ● pidamiological series available for estimating this dose in man comprise radiotherapy patients, accident cases and the Japanese exposed to atomic bombing8 in the Second World War. The $LD_{50/50}$ reflects marrow failure. The moat recent studies of tha $LD_{50/60}$ from experience in Japan (after revision of tha donor) yield value8 of around 3 Gy. The figure is thought to apply to the very special conditiona prevailing after the bombing for irradiated human beings who have no access, or only minimal access, to medical treatment.

221. Some groups of radiotherapy patiantr hava bran useful for assessment of the $LD_{50/60}$. None of 20 children and adolescents given 3 Gy to the whole body to treat *Ewing's* sarcoma died of marrow failure. The $LD_{50/60}$ for groups of adults irradiated for disseminated cancers was 2.9 Gy in one series and 3.4 Gy in another. All these data indicate that for cancer patients, although they receive supportive treatment, the $LD_{50/60}$ is probably about 3 Gy, while for healthy individuals receiving conventional supportive treatment after irradiation, it may be 4-5 Gy.

222. In the accident at Chernobyl, 43 individuals received doses estimated to have been between 2 and 4 Gy, and 1 of them died. Of 21 people receiving doses between 4.2 Gy and 6.3 Gy, 1 died. Of 20 patients receiving doses between 6 and 16 Gy, 19 died. Because of the complications suffered by many of the patients during the accident, such an thermal and skin injury, it is difficult to derive a value for $LD_{50/60}$ from these data.

223. From its review and discussion of the above data, the Committee concludes that it is impossible to assign a unique value to the LD_{50} in man; it may change substantially depending on aqe, the state of health of the individuals irradiated and on the prophylactic or therapeutic measures adopted before and after irradiation. For the planning of emergency responses, it is important to know which values of the LD_{50} woui? apply in which situation. The Committee underlines, however, the purely statistical nature *of* the LD_{50} *an&* warns that using it to predict the chance of survival *of* a single indi/idual would be totally unwarranted.

224. Neutrons are more efficient in causing acute injury than X- or gamma-radiation, by a factor of 2-3, using single doses. There is little experience in man of the lethal effects of neutrons, except in a few isolated accidents, The neutron component of the doses to the survivors of the atomic bombings is now considered to be much smaller than had previously been estimated so the data collected from this group of people are therefore of little use *in* assessing the effects of neutrons.

225. As is well known in the field of radio-biology, dose protraction and fractionation cause lore effect than the same total dose given singly. The early effects of high doses in man are no exception to this general rule. Thus, prodromal responses are somewhat alleviated by dose protraction or fractionation. Similarly, low-dose-rate or multi-fractionated irradiation markedly reduces injury to the intestine and the bone marrow in all species including man. Various quantitative formulas have been proposed to estimate the changes in dose or effect brought about by protracted irradiation] however, because the data base for many tissues is sparse, these formulas are only very rough guidelines for prediction. There is, moreover, one exception – the testis – to the general rule on protraction and fractionation; the progression of cells into sensitive phases makes this organ more sensitive to fractionated doses than to single doses.

226. In general, large amounts of internal emitters are required to produce early effects in man. Bone marrow depression is observed after single large intakes of iodine-131 and caesium-137. Gold radio-colloids have produced mild radiation sickness and haamotological complications, as have phosphorus-32 and sulphur-35. Severe acute intestinal injury in man from internal emitters has not been reported, and luny injury has been rare. Treatments for internal contamination with radio-nuclides ars based on local removal, reduced retention, enhanced excretion and diminished tranalocation.

227. A small fraction of the population may be particularly sensitive to early radiation injury by virtue of inherited genetic disorders, such as ataxia telangiectasia. Persons with this disease are more radio-sensitivr than normal. Many other genetic disorders predispose to increased chromosomol r cellular injury, but quantitative estimates of this increase are not available.

228. It is difficult to form a prognosis in irradiated patients solely from an estimate of the dose. There are many confounding factors, including intercurrent disease, dose protraction and radiation quality, The type and duration of prodromal symptoms, including erythema, may assist in the prognosis. Haematoloqical signs, particularly the lymphocyte count, are good prognostic indicators. The lowest blood counts and their time of occurrence for the various blood cell types are also important, as is the duration of marrow aplasia after high doses. The appearance and persistence of immature cells in the blood is usually a favourable *sign* of marrow recovery. A valid prognosis must be founded on a wide range of different types of data and constantly updated.

229. The information provided by the Soviet Union and contained in the appendix to annex G on the victims of the Chernobyl accident is exhaustive and valuable. While the nature of the lesions observed is not unexpected, the degree of precision achieved in the analysis of their time of onset and their magnitude and duration adds considerably to our understanding of the biological effects of high doses of radiation in man. Further analysis of these findings is definitely warranted, particularly with respect to the following points: the precise assessments of the doses received by the victims; the correlation of the various symptoms and signs with the causal agents (the pattern of exposure was complex and involved internal and external irradiation, as well as thermal injury). These new studies will substantially enhance the present knowledge and will eventually allow the data collected at Chernobyl to be consolidated with other findings discussed in annex G. The Committee is indebted to all those who contributed to the appendix for their willingness to share this experience and wishes to commend them for the professional skill and the human compassion shown on such a tragic occasion.

4. Effects of pre-natal irradiation

230. In its latest study of the biological effects of pre-natal irradiation, contained in the 1986 report, the Committee reviewed the most recent information on developmental events, particularly in the brain of mammalian embryos and fetuses; the irradiation of experimental animals before birth; and children exposed to radiation pre-natally by the atomic bombings at Hiroshima and Nagasaki. Its review centred as much as possible on human experience and included effecto that had not previously been considered before in this light, such as the carcinogenic effects of irradiation in utero.

231. The 1986 data showed that mental retardation is the most likely type of developmental abnormality to appear in the human species. In essence, analysis as a function of time showed that the probability of radiation-related mental retardation is essentially zero with exposure before 8 weeks from conception, is maximum with irradiation between 8 and 15 weeks, and decreases between 16 and 25 weeks. After 25 weeks and for doses below 1 Gy, no case of severe mental retardation had been reported, On the assumption that the induction of the effect is linear with dose (as the data seemed to indicate), the probability of induction per unit absorbed dose was estimated at 0.4 per Gy at the time of the peak sensitivity and at 0.1 per Gy between 16 and 25 weeks from conception.

232. Using all the data available, the Committee attempted to derive quantitative risk estimates for the radiation effects for which there is positive evidence or, at least, reasonable presumption of induction. In addition to men' al retardation, these effects include mortality and the induction of malformations, leukaemia and other malignancies. Under a number of qualifying assumptions, the Committee estimated that a dose to the conceptus of 0.01 Gy delivered over the whole pregnancy would add a probability of adverse health effects in the live born of less than 0.002. The normal risk of a non-irradiated live born carrying the same conditions is about 0.06. Information becoming available suggests that the risk estimates in the last two paragraphs may need substantial revision downward (particularly in the low-dose ranges). The Committee intends to review this in the near future.

C. Derivation of risk coefficients

233. In the situations described in the annexes, people are exposed to a range of types of radiation, and the resulting doses in their bodies ar often non-uniform. In order to add the doses from groups of sources, e.g. natural sources, it is necessary to use a quantity that takes account of these different kinds of radiation and dose distributions in the body. The quantity used by the Committee is the effective dose equivalent. This quantity is obtained b weighting the absorbed dose in a tissue of the body, first by a factor to take account of the effectiveness of the type of radiation and then by a factor to take account of the different biological sensitivities of the tissues. The sum of these weighted absorbed doses is the effective dose equivalent.

234. The values of the two sets of weighting factors are those recommended by ICRP. From time to time, the Committee has considered other systems of weighting, but has so far decided that the effective dose equivalent remains adequate for its purposes. The use of the effective dose equivalent is limit-d to assessments of long-term effects such as carcinogenesis. For assessing the early effects of high doses, the absorbed dose is an appropriate quantity.

235. When it uses the term "risk" (in a quantitative sense) the Committee means the probability of a harmful event, e.g. a radiation-induced death, and often expresses this probability in per cent, The number of projected events in a population is expressed either a8 cases per thousand or cases prr million. The term "risk coefficient" is used in a general way to indicate the risk per unit dose (risk per gray in the case of absu ed dose or risk per sievert in the case □✗ ● ffective dose equivalent). Since the relationship between dorm and risk is not always proportional, it is sometimes necessary also to specify the dose Or dose range for which the coefficient is valid.

236. In addition to estimating risk, the Committee has also estimated the projected number of years of life lost in an exposed population due to radiation-induced mortality. This quantity and also the projected number of cases or deaths in an exposed population are sometimes called measure8 of collective detriment.

1. Hereditary harm

237. Genetic risk coefficient8 may be defined to apply either to the gonad dose equivalent or the effective dose equivalent. It is al80 necessary to decide whether they should apply to genetically significant dose8 (i.e. doses to reproductive individuals) or average doses to the population at large. Opting for the latter might seem absurd from the scientific point of view, but sometimes only average doses or total collective doses are knownr moreover, risk coefficients for cancer often apply to average doses.

238. In the 1986 report and in annex E of the present report, "Genetic hazards", the Committee has reviewed the present body of knowledge of the hereditary effects of ionizing radiation. These reviews are summarised in chapter II, section D.1. There are several tustomary ways of presenting the scientific information. One is to make the assessment for an equilibrium situation, wherein a stable population hao been exposed over many generations, with each reproductive individual, male or female, receiving a unit gonad dose, and to estimate the fraction of tl.j offspring who would then be expected to be affected bv hereditary harm. Another way is to assess the affected numb\..r of offspring to a parent generation where the parent generation, males or females or both, have received a given collective dose.

239. In both cases, the nformation can be translated into a risk coefficient that expresses either the probability of a reproductive individual giving birth to a child affected by hereditary harm or the expected number of affected children, per unit individual or collective gonad dose to reproductive individuals. The risk coefficient may also be extended to include harm in all future generations.

240. Such risk coefficients can be applied directly to estimates of the genetically significant dose, such as those which have been made for various medical diagnostic X-ray procedures. However, they cannot be applied to effective dose equivalents unless there is uniform whole-body exposure. In other cases, the applicable genetic risk coefficient could range from zero (if the gonads are not exposed) to four times the risk coefficient that is applicable to the gonad dose (in the case that only the gonads are exposed), the organ weighting factor for the gonads being 1/4.

241. If the effective do86 equivalent is assessed not for reproductive individuals but for average individuals in the population at large, then the relevant risk coefficient is Only F/L of the genetic risk coefficient that would apply to reproductive individuals, F being the main reproductive age and L the life expectancy at birth. If F is about 30 years and L about 75 years, the gonatic risk coefficient for the average individual become8 40 per cent of the coefficient for reproductive individuals.

242. Table 8 summarise6 the Committee'8 present estimates of genetic risk coefficients. Extensive information about the nature of the genetic risk was presented in the 1986 report.

243. A comparison wih previous estimates (see table 1) shows that the present estimates are lower than those made in 1977. The 1977 estimates were used when ICRP defined the effective dose equivalent, The risk coefficient8 refer Only to the expected number of cases of quantifiable, severe, hereditary disease. What this means in terms of dstrimont is a question the Committee will continue to study.

Table 8. Revised genetic risk coefficients

(Per cent per Sv) a/

| | For gonad dose equivalent | | For effective dose equivalent | |
	Reproductive population	Total populstion	Reproductive population	Total population
First two generations	0.3	0.1	o-1.2	o-o.5
All generation6	1.2	0.5	0-5.0	0-2.0

a/ Ris vs from diseases of complex aetiology were not estimated.

2. Cancer

244. Cancer risk coefficients may be expressed either as (a) the site-specific individual probability of future radiation-induced cancer (death) per unit dose or (b) the collective detriment. The latter may be presented either as the expected number of cancer deaths (or cases) in the expooed population or as the number of person years lost because of cancer deaths per unit collective dose.

245. The new assessments in annex F, "Radiation carcinogenesis in man", relate to the cancer risk at doses of 1 Gy at high dose rate of low-LET, radiation. It has to be stressed, however, that statistically significant excess cancer mortality in Hiroshima and Nagasaki has been observed foi the first time fcr some cancers and at several specific sites at doses between 0.2 and 0.5 Gy. Not only have the risks from nine types of cancer been assessed with reasonable confidence, but also the total risk from all other types of cancer has been independently assessed. The risk estimates include a projection into the future of observations on the exposed popu 1 at. ions at Hiroshima and Nagasaki. The new estimates have taken into account the revised dosimetry, All of this has had the combined effect of making the risk estimates at these doses and dose rates higher than before.

(a) Site-specific individual risk

246. Table 9 shows the results of the Hiroshima-Nagasaki study with regard to the individual probability of death from site-specific radiation-induced cancer. Two sets of numbers are given: one is derived from projections based on the additive (absolute) risk model, the other from projections based on the multiplicative (relative) risk model.

247. The total cancer mortality risk coefficient for the average individual (averaged also *over* both sexes) is 4.5 per cent per gray on the additive risk model And 7.1 per cent par gray on the multiplicative risk model. These numbers may be compared with the 1977 estimate for high doses, which war about 2.5 per cent par sievert on the basis of the Additive modal (see table 3). Further summary values *of* risk coefficients for populations *of* other ages and other circumstances are given in table 10.

248. The problems in deriving risk coefficients that are also applicable at low doses are the same as before. Such risk coefficients can only be inferred from the observed values at moderate, to high doses. In 1977, when the total cancer risk coefficient at high domes was estimated to be about 2.5 per cent per sievert, the Committee pointed out some *of* the uncertainties; the fact that this estimate was an overestimate in the sense that the risk per unit dose at low doses was believed to be lower than the estimates for high doses.

249. In the present report, the prob ems in deriving risk coefficients at low doses and for low dose rates remain. The Committee agreed that there was A need *for a* reduction factor to modify the risks shown in table 9 for low doses and low dose rates. The Committee considered that such a factor certainly varies very widely with individual tumour type And with doss rate range. However, an appropriate range to be applied to total risk *for* low dose and low dose rate ahould lie between 2 and 10. The Committee intends to study this matter in detail in the near future.

Table 9 . Per caput lifetime excess cancer deaths probability
a
high dose rate of low-LET radiation

(Per cent)

(Based on the population of Japan using an average age
risk coefficient)

	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.97	0.93
All cancers except leukaemia	6.1	3.6
Bladder	0.39	0.23
Breast a/	0.6	0.43
Colon	0.79	0.29
Lung	...	
Multiple myeloma	0.22	0.59 0.09
Ovary a/	0.31	0.26
Oesophagus	0.34	0.16
stomach	1.3	0.86
Remainder	1.1	1.0
Total	7.1	4.5

a/   Value has to be divided by 2 to calculate the total
and other organ risks.

Table 10. Estimates of projected lifetime risks for 1,000 persons
(500 males and 500 females) exposed to 1 Gy of high
dose rate low-LET radiation

(Based upon the population of Japan)

	Risk projection model	Excess fatal cases	Years of life lost
Total population	Additive	30-50	700-1 200
	Multiplicative	70-100	950-1 400
Working population (aged 18-65 years)	Additive	40	880
	Multiplicative	80	970
Adult population (over 25 years)	Additive	30	510
	Multiplicative	60	650

250. The Committee has not presented risk estimates for high-LET radiation in general in the present report (except for the exposure to radon of uranium minors). For low doses of external high-LET radiation it would be necessary to multiply the risks for low-LET radiation by an appropriate quality factor. No dose or dose rate reduction factor is considered necessary for high-LET external radiation at low doses.

(b) Collective detriment

251. The product of risk coefficients appropriate for individual risk and the relevant collective dose will give the expected number of cancer death6 in the ● xpo6ed population, provided that the collective dose is at least of the order of 100 man Sv. If the collectire dove is only a few man Sv, the most likely outcome is zero deaths.

252. The Committee ha6 also assessed the person years lost per unit collective dose because of radiation-induced cancer mortality. The results at high dose6 and high dose rates of low-LET radiation are summarised in table 10. The total loss amounts to about 1 person year per man Gy, with both projection models.

D. Comparison of exposures

1. Previous UNSCEAR comparisons

253. The way in which to present radiation exposures from various sources has always been a problem for the Committee. In its 1958 report, the Committee assessed the per caput mean marrow dose and the genetically significant dose to the world population from various sources and practices. At that time, the Committee even calculated the expected number of cases of leukaemia and hereditary harm from natural background radiation and nuclear explosions.

254. In its 1962 report, the Committee assessed the per caput dose6 from natural irradiation of the gonads, the bone surface layers and red bone marrow. It also calculated the dose commitments to the world population for the same organs. The genetically significant dose was assessed for medical and occupational exposures. However, in that report the Committee felt that it had less confidence in the risk coefficients used in the 1958 report and that it was not able to assess any detriments. It stated, instead, that the estimated doses and dose commitments could be used for comparative risk assessment6 and gave this comparative risk in relation to natural background radiation, which was assigned the value of unity. This comparison was made for medical exposures and nuclear explosions with reference to leukaemia, bone tumours and hereditary effects. On the same basis, the Committee said, the detriment. of various sources could be expressed in terms of exposure to natural background radiation that would give the same per caput dose or dose commitment.

255. In the 1964, 1969 and 1972 reports, the Committee continued to express the risk from nuclear explosions in terms of the equivalent period of oxposure to natural background radiation Until 1972 the Committee had calculated per caput. doses or dose commitments for the whole world population. For A population of a given number, this implies an assessment of the collective dose from each source. In the 1977 report, the Committee for the first time explicitly presented collective dose & sessmants for various sources and practices. At the same time,

however, it also drew comparisons on the basis *of* equivalent periods of natural background exposure. In the 1982 report, the Committee included more information on the ways in which individual exposures vary and assessed collective dose commitments. In the summary and conclusions, the collective dose equivalents were translated into equivalent periods of natural background radiation,

256. From this short review it can be seen that comparison with the natural background dose rate has always played an important role in the Committee'6 presentation of its assessments. When, in 1958, the Committee estimated the number *of* affected persons, it drew a comparison with the natural occurrence *of* cancer and hereditary disease. Since then, per caput and collective doses have been compared with the corresponding doses caused by natural radiation.

2. Purpose of comparisons

257. Comparisons usually have a purpose and may be presented in different ways depending on that purpose. Comparisons with doses or detriment6 caused by natural sources of radiation may help to clarify the relative radiological importance of man-made radiation sources, but they say little about justifiability or acceptability *of* these other sources. Information on where doses are low or high in relation to the natural background may help in determining whether there is a potential for meaningful epidemiological studies. Comparing the radiation dose6 or risks of alternative procedures for achieving one and the same objective, e.g. medical diagnostic information, may disclose what might be preferable from the radiation protection point of view, but it will not reveal other risks or disadvantages. Since the Committee has no use *of* its own for comparisons, it wishes to present its data in such a form that they can be used for a number *of* different purposes.

3. Comparison of collective doses

258. If risk coefficients are known and if proportionality between dose and response can be assumed, radiation detriments, such as the expected number of cancer deaths, can be calculated from information on collective dose commitments. For relative comparisons, however, it suffices to compare collective doses or per caput doses (which amount6 to the same thing, from the various sources, thereby eliminating the uncertainty in the risk coefficients, "" such compar ieons, the annual collective dose from natural sources of radiation may be taken a6 the reference; the contribution from other sources may be expressed in terms of the equivalent periods *of* natural background radiation, as has been the Committee's practice since 1962.

259. When collective doses from different sources are compared, it is important that the comparison be on a relevant basis. This is simple for sources and practices aimed at achieving one and the same objective, such as energy production or medical diagnostic information. In other cases, one must be careful to find a common basis for comparison. For example, it is of doubtful relevance to compare collective doses to arbitrarily selected populations an' time periods. However, although comparisons of collective doses from entirely different practices will often not be very meaningful, they may sometimes help in setting priorities for dealing with concern6 of radiological consequences.

4. Comparison of individual doses

260. The radiation doses an individual receives from various man-made sources are normally compared with the dose he receives from natural sources Of radiation. An extra dose that is small in relation to the background dose will not significantly affect an individual, i.e. it will not change his total exposure situation noticeably. While the individual might still wish to avoid such a small extra dose, he would know that it does not in itself present any substantial risk. This does not mean that the dose is acceptable just because it is small: rather, acceptability would depend on the total harm the source is likely to cause and on society's appraisal of that harm,

261. Comparing per caput doses in the case of an uneven dose distribution within a population may be misleading, since no individual may actually receive the per caput dose but instead will receive either higher or lower doses. In that case, comparing typical doses as well as extreme doses may be more appropriate.

5. Summary of dose comparisons

262. Table 11 summarizes the various estimates of radiation doses. Au in previous reports, the equivalent period of exposure to natural background radiation is given along with the collective dose commitments. In comparing these estimates with those in previous reports, it should be remembered that the estimate of the annual dose from natural background radiation has increased, from less than 100 mrad (corresponding to about 1 mSv) in the 1977 report to 2.4 mSv in the present report. This increase came about for two reasons! (a) instead of giving a number of organ doses, the effective dose equivalent is now given and (b) the large contribution from radon daughter products has been recognised.

263. Table 11 is of necessity a considerable condensation of the available information. It is worth noting that about half of the natural background radiation is contributed by lung irradiation by radon daughters. Occupational exposures are experienced by those who work in the medical field as well as those who work in the nuclear power industry and in industrial radiography. Exposures from nuclear power production are due to radio-nuclides released from uranium mining and waste disposal activities, as well as from the operation of reactors to produce electric energy. About one third of the current exposures from nuclear power is attributable to radon emissions from mine tailings and another third to carbon-14 discharges from reactor operation, primarily heavy water reactors,

264. Of the collective effective dose equivalent commitment (other than from carbon-14) from all atmospheric test explosions, 1.5 million man Sv have been contributed by short-lived radio-nuclides and 3.5 million man Sv represent contributions to present individual life-time doses primarily from strontium-90 and caesium-,137. Because the Chernobyl accident led to doses mainly in Europe, the collective effective dose equivalent commitment rather than the global per caput dose is presentad.

Table 11. Summary of estimates of effective dose equivalent

Source or practice	Present annual individual doses (mSv)		Collective dose commitments	
	Per caput (world population)	Typical (exposed individuals)	Million man Sv	Equivalent years *of* background
ANNUAL			**Per year of practice**	
Natural background	2.4	1.0-5.0	11	1
Medical exposures (diagnostic)	0.4-1.0	0.1-10.0	2-5	0 .i-0.5
Occupational exposure	0.002	0.5-5.0	3.01	0.001
Nuclear power production	0.0002	0.001-0.1	0.001 (0.03) a/	0.0001 (0.004) a/
SINGLE			**Per total practice**	
All teet explosions toqether	0.01	0.01	ᴄ (26) a/	0.5 (2.4) a/
Nuclear accidents			0.6	

a/ The additional long-term collective dose commitments from radon and carbon-14 for nuclear power production and carbon-14 for test explosions are given in parentheses,

6. Direct comparison of detriments

265. In the present report, the Committee has reviewed the existing knowledge on radiation risks and has ventured to indicate the magnitude of the risk factors for low doses as well as for high doses. The Committee has also assessed the collective doses from various sources and practice@. It is tempting to combine the estimates and calculate the expected number of cases of cancer and hereditary disease.

266. Many estimates of this type, with different degrees of reliability, depending on the risk coefficients assumed, and with widely different purposes on the part of those who made them, have been reported. The results have been very scattered, depending on the general assumptions. The Committee hesitates, for a number of reasons, to add its own detriment assessments to those already provided for the various sources of radiation.

267. First, the Committee needs to bear in mind the terms of reference under which it operates: its purpose is to evaluate doses, not to make value judgements or engage in setting standards. As is made clear by the discussion in, chapter III, section D.4, even those assessments of risk that purport to be scientific involve

arrumptionr *and* decisions that are not, strictly speaking, scientific. Indeed, the physical quantities used by the Committee reflect such arrumptionr. For example, the affective dose equivalent, by definition, includes weighting factor8 that depend on subjective judgements a8 to what constitutes radiation-induced harm. For each further step in processing the basic! information, non-scientific judgements are likely to be needed or implied.

268. Next, the way *in* which the basic scientific fact8 are presented influence8 the impression they give. For example, thousands of cancer death8 from a single accident would undoubtedly be a high number of deaths. However, since such deaths could be expected to occur over a long period of time, tha annual incidence will be low. This moans a vary small increase of tha normal incidence of cancer, an increare that is not expected to be noticeable in health statistics. This shows that it is possible, by selecting the form of presentation, to convey different impressions.

269, Lastly, there is the graat uncertainty of such estimates. It was stressed i n chapter III, section C, that the risk coefficients for cancer at low doses can only be inferred from observation8 at high doses and that the risk coefficients for hereditary effect8 are not even deduced from observations in man, Even though the Committee believes that it8 estimates ara the best that can be given At the current state of knowledge, it must qualify them by drawing attantion to the underlying assumptions and uncertainties. Unfortunately, *any* estimate of a finite number of cancer death8 is soon taken out of context and the qualifications forgotten.

270. For these reasons, the Committee prefer8 to follow its previous practice of comparing collective dose commitment8 from the main radiation sources rather than estimated detriments.

Notes

1/ The United Nation8 Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session, *in* 1955. Its terms of reference are ret out *in* reeolution 913 (X) of 3 December 1955. It was originally composed of tha following Mamber States: Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, Union of Soviet Socialist Republics, United Kingdom of Great Britain and Northern Ireland and United State8 of America. The membership of the Committee was subsequently enlarged by the Ganoral Aseembly in its resolution 3154 C (XXVIII) of 14 December 1973 to include the Federal Republic of Germany, Indonesia, Peru, Poland and the Sudan. By reeolution 41/62 B of 3 December 1986, the General Assembly increased the membership of the Committea to a maximum of 21 and invited China to become a member.

2/ For the previous substantive reports of UNSCEAR to the General Assembly, see Official Records o f the General Assembly, Thirteenth Session, Supplement No. 17 (A/3838); ibid., Seventeenth Session, Supplement No. 16 (A/5216); ibid., Nineteenth Session, Supplement No. 14 (A/5814); ibid., Twenty-first Session, Supplement No. 14 (A/6314 and Corr.1); ibid.. Twenty-fourth Session, Supplement No. 13 (A/7613 and Corr.1); ibid., Twenty-seventh Session, Supplement No.2.5 (A/8725 and Corr.1); ibid., Thirty-second Session, Supplement No. 40 (A/32/40); ibid., Thirty-seventh Session, Supplement No. 45 (A/37/45); and ibid., Forty-first Session, Supplement No. 16 (A/41/16). These documents are referred to as tho 1958, 1962, 1964, 1966, 1369, 1972, 1977, 1982 and 1986 reports, respectively. The 1972

report with scientific annexes was published as: <u>Ionising Radiation: Levels and Effects, Volume I: Levels</u> and <u>VolumeEffects</u> (United Nations publication, Sale8 No. E.72.1X.17 and 18). The 1977 report with rcientific annrxer was published as: <u>Sources and Effects of Ionising Radiation</u> (United Nation8 publication, Sale8 No, E.77.IX.1). The 1962 report with rcientific annexer was published as: <u>Ionising Radiation: Sources and Biological Effects</u> (United Nation8 publication, Sales No. E.82.1X.8). Tha 1986 report with rcientific annexer war published as: <u>Genetic and Somatic Effects of Ionising Radiation</u> (United Nations publication- Sale8 No. E.86.IX.9).

3/   This subject is raviawed extensively in annax A, "Exposures from natural sources of radiation",

4/   This subject is raviewad extensively *in* annex B, "Exposures from nuclear power production".

5/   This subject is reviewed extensively in annex C, "Exposures from medical uses of radiation",

6/   This subject is reviewed in annex B, "Exposures f tom nuclear power production", and in annex C, "Exposures from medical uses of radiation".

7/   This subject is reviewed extenrively in annex D, "Exposures from the Chernobyl accident".

8/   This subject is reviewed extenrively in annex E, "Genetic hasards".

9/   This subject is reviewed extensively in annex F, "Radiation carcinogenesis in man".

10/   This subject is reviewed extensively in annex G, "Early effect8 in man *of* high doses of radiation".

APPENDIX I

Members of national delegations attending the thirty-first to thirty-seventh sessions of the Committee

ARGENTINA

D. Beninson (Representative), D. Cancio, A, J. Gonsales, E. Palacios

AUSTRALIA

K. H. Lokan (Representative)

BELGIUM

M. Errera (Representative), J. Maisin (Representative), J. Aten, P. Lohman, F. H. Sobels, A. D. Tates

BRAZIL

E. Penna Franca (Representative), L. R. Caldas (Representative)

CANADA

E. G. Letourneau (Representative), A. M. Marko (Represesntative), W. R. Bush, G. C. Butler, B. C. Lentle, D. K. Myers

CZECHOSLOVAKIA

M. Klímek (Representative)

CHINA

Wei Lüxin (Representative), Li Deping, Wu Dechang

EGYPT

S. El-Din Hashish (Representative), H. Roushdy (Representative), M. El-Kharadly

FRANCE

H. Jammet (Representative), A. Bouville, R. Coulon, M. Bertin, B. Dutrillaux, J. Lafuma, G. Lemaire, R. Masse, P. Pellerin, M. R. Tubiana, G. Uzzan

GERMANY, FEDERAL REPUBLIC OF

A. Kaul (Representative), U. Ehling, W. Jacobi, H. Kriegel, F. E. Stieve, C. Streffer

INDIA

N. K. Notani (Representative), K. Sundaram (Representative)

INDONESIA

S. Wiryosimin (Representative), A. Baiquni (Representative), O. Iskandar (Representative), M. Ridwan (Representative), C. J. Sugiarto

JAPAN

T. Kumatori (Representative), H. Matsudaira (Representative), T. Terasima (Representative), A. Kasai, A. Yamato

MEXICO

E. Araico (Representative), J. R. Ortiz Magaña (Representative)

PERU

L. V. Pinillos Ashton (Representative), M. Zaharia (Representative)

POLAND

2. Jaworowski (Representative), J. Liniecki (Representative), Z. Stot

SUDAN

A. Hidayatalla (Representative), A. A. Yousif

SWEDEN

B. Lindell (Representative), G. Bengtsson, K. Edvarson, L.-E. Holm, K. G. Lüning, S. Mattsson, J. O. Snihs, J. Valentin, G. Walinder

UNION OF SOVIET SOCIALIST REPUBLICS

L. A. Ilyin (Representative), A. Guskova (Representative), K. M. Barkchudarov, V. Denim, E. Golubkin, D. F. Khokhlova, A. A. Moiseev, Yu. I. Moskalev, V. Pavlinov, O. Pavlovsky, O. Piatak, V. V. Redkin, V. A. Shevchsnko

UNITED KINGDOM OF GREAT BRITAIN AND NORTHERN IRELAND

J. Dunrtrr (Representative), R , H. Clarke, S. C . Darby, J. Denekamp, J. H. Edwards
K. E. Halnan, P. S. Harper, A. Searle

UNITED STATES OF AMERICA

F. A. Mottler (Representative), R. D. Moseley (Representative), R. E. Anderson,
L. R. Anspaugh, R . Baker, C . Edington, J. H. Harley, R. C. Ricks, H. H. Rossi,
W. L. Russell, P. B. Selby, W. K. Sinclair, J. W. Thiessen, E. W. Webster,
H. O. Wyckoff

APPENDIX II

<u>**List of scientific staff and consultants who have co-operated with the Committee in the preparation of the report**</u>

L . R. Anspaugh

B. G. Bennett

A . Bouville

R. H. Clarke

F . Fagnani

L. Frittelli

A, Hagen

J. Hendry

B. Lindell

F. A, Mettler

M. Morrey

0. Pavlovrky

W. J. Schull

G. Silini

F. D. Sowby

K. Sankaranarayanan

G. A, M. Webb

K. Weiss

List of reports received by the Committee

1. Listed below are reports received by the Committee from Governments between 19 April 1986 and 17 Juno 1988.

2. Reports received by the Committee before 19 April 1986 were listed in earlier reports of the Committee to the General Assembly.

Document	Country	Title
A/AC.82/G/L.1732	United Kingdom of Great Britain an'. Northern Ireland	Environmental radioactivity surveillance programme1 results for thr UK for 1984, 21 April 1986
1733	Japan	Radioactivity Survey Data in Japan, number 72, March 1985, 16 July 1906
1734	Japan	Radioactivity Survey Data in Japan, number 73, June 1985, 16 July 1986
1735	United States of America	Environmental Measurements Laboratory! A compendium of the EML's research projects related t o the Chernobyl nuclea: accident, 10 December 1966
1736	United States of America	Environmental Measurements Laboratory : The high altitude sampling programme: radioactivity in the stratosphere, 10 April 1987
1737	Japan	Radioactivity Survey Data in Japan, number 74, September 1985, 10 April 1387
1738	Japan	Radioactivity Survey Data in Japan, number 75, December 1985, 10 April 1987
1739	Union of Soviet Socialist Republics	Assessment of population doses from X-ray examination in the USSR (1970-1980), 13 April 1987

Document	Country	Title
1740	Union of Soviet Socialist Republic6	Genetic effects of radio-nuclide decay, 13 April 1907
1741	Union of Soviet. Socialirt Republica	Acute radiation effects *in* man, 13 April 1987
1742	Union of Soviet Socialist Republics	Production and release of aarbon-14 in nuclear power stations with RBMK reactors, 13 April 1987
1743	Union of Soviet Socialist Republic6	Body burden of fall-out caesium-137 in thr inhabitant6 of Moscow 1980-1983, 13 April 1987
1744	Union of Soviet Socialist Republic6	Radiation doses to the inhabitants of the far north, 13 April 1987
1745	Union of Soviet Social ist Republica	Occupational exposure of radiographic workers, 13 April 1907
1746	Japan	Radioactivity Survey Data in Japan, numbrr 76, March 1986, 2 July 1987
1747	Japan	Radioactivity Survey Data in Japan, number 77, Juno 1986, 2 July 1987
1748	Japan	Radioactivity Survey Data in Japan, number 78, October 1987, 18 December 1987
1749	Japan	Radioactivity Survey Data in Japan, number 79, October 1987, 18 December 1987
1750	Union of Soviet Socialist Republic6	Proposals for rotting possible intake limit6 for transuranium radio-nuclider absorbed from the gastro-intestinal tract, 31 May 1988
1751	Union of Soviet. Socialist. Republics	The evaluation of non-stochastic effects in man from low doses of internal irradiation, 31 May 1988

Document	country	Title
1752	Union of Soviet Socialist Republics	Tritium production in LWGR power plants and its release into the environment, 31 May 1988
1753	Union of Sovirt Socialist Republics	Medical treatment in the case of uranium intoxication, 31 May 1988
1754	Union of Soviet Socialist Republics	Dynamics of ● ffoctivr dose equivalent from intake of strontium-90 and caesium-137, 31 May 1988
1755	Union of Soviet Socialist Republics	Specific activities of natural radio-nuclides in building materials used in the Soviet Union, 31 May 1988

كيفية الحصول على منشورات الأمم المتحدة

يمكن الحصول على منشورات الأمم المتحدة من المكتبات ودور التوزيع في جميع أنحاء العالم ٠ استعلم عنها من المكتبة التي تتعامل معها أو اكتب إلى ٠ الأمم المتحدة ٠ قسم البيع في نيويورك أو في جنيف ٠

如何购取联合国出版物

联合国出版物在全世界各地的书店和经售处均有发售。 请向书店询问或写信到纽约或日内瓦的联合国销售组。

HOW TO OBTAIN UNITED NATIONS PUBLICATIONS

United Nations publications mtty be obtained from book ores and distributors throughout the world Consult your bookstore or write to United Nations, Sales Section, New York or Geneva

COMMENT SE PROCURER LES PUBLICATIONS DES NATIONS UNIES

Les publications der Nations Unies sont en vente dans les librairies et les agences dépositaires du monde entier Informez-vous auprès de votre libraire ou adressez vous à Nations Unies, Section des ventes, New York ou Genève

КАК ПОЛУЧИТЬ ИЗДАНИЯ ОРГАНИЗАЦИИ ОБЪЕДИНЕННЫХ НАЦИЙ

Издания Организации Объединенных Наций можно купить в книжных магазинах и агентствах во всех районах мира Наводите справьк об изданиях tt вашем книжном магазине или пишите по адресу: Организация Объединенных Наций, Секция по продаже изданий, Нью-Йорк или Женева.

COMO CONSEGUIR PUBLICACIONES DE LAS NACIONES UNIDAS

Las publicaciones Jr las Naciones Unidas están en venta cn librerías y casas distribuidoras en todas partes del mundo. Consulte a su librero o diríjase a Naciones Unidas, Sección de Ventas, Nueva York o Ginebra