Report of the United Nations Scientific Committee on the Effects of Atomic Radiation to the General Assembly

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INTRODUCTION

1. Over the past few years, the United Nations Scientific Committee on the Effects of Atomic Radiation[1] has undertaken a broad review of the sources and effects of ionizing radiation. In the present report,[2] the Committee, drawing on the main conclusions of its scientific assessments, summarizes the developments in radiation science in the years leading up to the new millennium.

2. The present report and its scientific annexes were prepared between the forty-fourth and the forty-ninth sessions of the Committee. The following members of the Committee served as Chairman, Vice-Chairman and Rapporteur, respectively, at the sessions: forty-fourth and forty-fifth sessions: L. Pinillos-Ashton (Peru), A. Kaul (Germany) and G. Bengtsson (Sweden); forty-sixth and forty-seventh sessions: A. Kaul (Germany), L.-E. Holm (Sweden) and J. Lipsztein (Brazil); and forty-eighth and forty-ninth sessions: L.-E. Holm (Sweden), J. Lipsztein (Brazil) and Y. Sasaki (Japan). The names of members of national delegations who attended the forty-fourth to the forty-ninth sessions of the Committee as members of national delegations are listed in Appendix I.

3. The Committee wishes to acknowledge the help and advice of a group of consultants and contributors who helped in the preparation of the scientific annexes (see Appendix II). The sessions of the Committee were attended by representatives of the World Health Organization and the International Atomic Energy Agency. The International Commission on Radiation Units and Measurements and the International Commission on Radiological Protection were also represented. The Committee wishes to acknowledge their contributions to the discussions.

4. In carrying out its work, the Committee applied its scientific judgement to the material it reviewed and took care to assume an independent and neutral position in reaching its conclusions. The results of its work are presented for the general reader in this report to the General Assembly. The supporting scientific annexes are aimed at the general scientific community.

5. The United Nations Scientific Committee on the Effects of Atomic Radiation, a scientific committee of the General Assembly, is the body in the United Nations system with a mandate to assess and report levels and effects of exposure to ionizing radiation. The fact that the Committee holds this specific mandate from such an authoritative body greatly enhances its ability to provide an effective and independent service to the world. The United Nations, through the General Assembly, can take credit for providing that service. The information provided by the Committee assists the General Assembly in making recommendations, in particular those relevant to international collaboration in the health field, to sustainable development and, to some extent, to the maintenance of international peace and security.

6. New challenges as regards global levels of radiation exposure continue to arise and new biological information on the effects of radiation exposure is becoming available. For example, large amounts of radioactive waste have built up as a result of both peaceful uses of nuclear energy and military nuclear operations, and radiation sources used in military and peaceful operations have been abandoned, creating a situation that is prone to illicit trafficking and other criminal activities. Moreover, the potential risks from low-level radiation exposure, that is, exposure to radiation comparable with natural background radiation, are the cause of lively debate and controversy. The Committee is responding to those challenges and will do so further with new initiatives to be included in its future assessments of radiation sources, levels and effects..

7. Governments and organizations throughout the world rely on the Committee's evaluations of the sources and effects of radiation as the scientific basis for estimating radiation risk, establishing radiation protection and safety standards and regulating radiation sources. Within the United Nations system, those estimates are used by the International Atomic Energy Agency in discharging its statutory functions of establishing standards for the radiation protection of health and providing for their application. The Committee is proposing a renewed programme of work to fulfil its obligations to the General Assembly.

I. OVERVIEW

A. THE EFFECTS OF RADIATION EXPOSURE

8. Radiation exposure can damage living cells, causing death in some of them and modifying others. Most organs and tissues of the body are not affected by the loss of even considerable numbers of cells. However, if the number lost is large enough, there will be observable harm to organs that may lead to death. Such harm occurs in individuals who are exposed to radiation in excess of a threshold level. Other radiation damage may also occur in cells that are not killed but modified. Such damage is usually repaired. If the repair is not perfect, the resulting modification will be transmitted to further cells and may eventually lead to cancer. If the cells modified are those transmitting hereditary information to the

descendants of the exposed individual, hereditary disorders may arise.

9. Radiation exposure has been associated with most forms of leukaemia and with cancers of many organs, such as lung, breast and thyroid gland, but not with certain other organs, such as the prostate gland. However, a small addition of radiation exposure (e.g. about the global average level of natural radiation exposure) would produce an exceedingly small increase in the chances of developing an attributable cancer. Moreover, radiation-induced cancer may manifest itself decades after the exposure and does not differ from cancers that arise spontaneously or are attributable to other factors. The major long-term evaluation of populations exposed to radiation is the study of the approximately 86,500 survivors of the atomic bombings of Hiroshima and Nagasaki, Japan. It has revealed an excess of a few hundred cancer deaths in the population studied. Since approximately half of that population is still alive, additional study is necessary in order to obtain the complete cancer experience of the group.

10. Radiation exposure also has the potential to cause hereditary effects in the offspring of persons exposed to radiation. Such effects were once thought to threaten the future of the human race by increasing the rate of natural mutation to an inappropriate degree. However, radiation-induced hereditary effects have yet to be detected in human populations exposed to radiation, although they are known to occur in other species. The Committee is preparing a comprehensive report on hereditary effects of radiation exposures to be submitted to the General Assembly at its fifty-sixth session.

B. LEVELS OF RADIATION EXPOSURE

11. Everyone is exposed to natural radiation. The natural sources of radiation are cosmic rays and naturally occurring radioactive substances existing in the Earth itself and inside the human body. A significant contribution to natural exposure of humans is due to radon gas, which emanates from the soil and may concentrate in dwellings. The level of natural exposure varies around the globe, usually by a factor of about 3. At many locations, however, typical levels of natural radiation exposure exceed the average levels by a factor of 10 and sometimes even by a factor of 100.

12. Human activities involving the use of radiation and radioactive substances cause radiation exposure in addition to the natural exposure. Some of those activities simply enhance the exposure from natural radiation sources. Examples are the mining and use of ores containing naturally radioactive substances and the production of energy by burning coal that contains such substances. Environmental contamination by radioactive residues resulting from nuclear weapons testing continues to be a global source of human radiation exposure. The production of nuclear materials for military purposes has left a legacy of large amounts of radioactive residues in some parts of the world. Nuclear power plants and other nuclear installations release radioactive materials into the environment and produce radioactive waste during operation and on their decommissioning. The use of radioactive materials in industry, agriculture and research is expanding around the globe and people have been harmed by mishandled radiation sources.

13. Such human activities generally give rise to radiation exposures that are only a small fraction of the global average level of natural exposure. However, specific individuals residing near installations releasing radioactive material into the environment may be subject to higher exposures. The exposure of members of the public to regulated releases is restricted by internationally recognized limits, which are set at somewhat less than the global average level of natural exposure. It is to be noted that, should some of the sites with high levels of radioactive residues be inhabited or re-inhabited, the settlers would incur radiation exposures that would be higher than the global average level of natural exposures.

14. The medical use of radiation is the largest and a growing man-made source of radiation exposure. It includes diagnostic radiology, radiotherapy, nuclear medicine and interventional radiology. Large numbers of people (in developing countries in particular) cannot yet take advantage of many of those medical procedures, which are not available worldwide. For the time being, therefore, those people receive less radiation exposure from medical diagnosis and treatment than people living in countries benefiting from advanced medical procedures, a situation that is expected to change in the future and will need to be followed by the Committee.

15. The average levels of radiation exposure due to the medical uses of radiation in developed countries is equivalent to approximately 50% of the global average level of natural exposure. In those countries, computed tomography accounts for only a few per cent of the procedures but for almost half of the exposure involved in medical diagnosis. Severe radiation-related injuries have occurred as a result of poor practice of some interventional techniques (such as radiological procedures to monitor the dilation of coronary arteries) and radiotherapy.

16. Radiation exposure also occurs as a result of occupational activities. It is incurred by workers in industry, medicine and research using radiation or radioactive substances, as well as by passengers and crew during air travel. It is very significant for astronauts.

17. The average level of occupational exposures is generally similar to the global average level of natural radiation exposure. However, a few per cent of workers receive exposures several times higher than the average exposure to natural radiation. The exposure of workers is restricted by internationally recognized limits, which are set at around 10 times the average exposure to natural radiation.

C. THE RADIOLOGICAL CONSEQUENCES OF THE CHERNOBYL ACCIDENT

18. The accident at the Chernobyl nuclear power plant was the most serious accident involving radiation exposure. It caused the deaths, within a few days or weeks, of 30 workers and radiation injuries to over a hundred others. It also brought about the immediate evacuation, in 1986, of about 116,000 people from areas surrounding the reactor and the permanent relocation, after 1986, of about 220,000 people from Belarus, the Russian Federation and Ukraine. It caused serious social and psychological disruption in the lives of those affected and vast economic losses over the entire region. Large areas of the three countries were contaminated, and deposition of released radionuclides was measurable in all countries of the northern hemisphere.

19. There have been about 1,800 cases of thyroid cancer in children who were exposed at the time of the accident, and if the current trend continues, there may be more cases during the next decades. Apart from this increase, there is no evidence of a major public health impact attributable to radiation exposure 14 years after the accident. There is no scientific evidence of increases in overall cancer incidence or mortality or in non-malignant disorders that could be related to radiation exposure. The risk of leukaemia, one of the main concerns owing to its short latency time, does not appear to be elevated, not even among the recovery operation workers. Although those most highly exposed individuals are at an increased risk of radiation-associated effects, the great majority of the population are not likely to experience serious health consequences as a result of radiation from the Chernobyl accident.

II. SOURCES OF RADIATION EXPOSURE

20. Ionizing radiation represents electromagnetic waves and particles that can ionize, that is, remove an electron from an atom or molecule of the medium through which they propagate. Ionizing radiation may be emitted in the process of natural decay of some unstable nuclei or following excitation of atoms and their nuclei in nuclear reactors, cyclotrons, x-ray machines or other instruments. For historical reasons, the photon (electromagnetic) component of ionizing radiation emitted by the excited nucleus is termed gamma rays and that emitted from machines is termed x rays. The charged particles emitted from the nucleus are referred to as alpha particles (helium nuclei) and beta particles (electrons).

21. The process of ionization in living matter necessarily changes atoms and molecules, at least transiently, and may thus damage cells. If cellular damage does occur and is not adequately repaired, it may prevent the cell from surviving or reproducing or performing its normal functions. Alternatively, it may result in a viable but modified cell.

22. The basic quantity used to express the exposure of material such as the human body is the absorbed dose, for which the unit is the gray (Gy). However, the biological effects per unit of absorbed dose varies with the type of radiation and the part of the body exposed. To take account of those variations, a weighted quantity called the effective dose is used, for which the unit is the sievert (Sv). In reporting levels of human exposure, the Committee usually uses the effective dose. In the present report, both the absorbed dose and the effective dose are usually simply called "dose", for which the units provide the necessary differentiation. A radioactive source is described by its activity, which is the number of nuclear disintegrations per unit of time. The unit of activity is the becquerel (Bq). One becquerel is one disintegration per second.

23. To evaluate the effects of exposing a defined population group, the sum of all doses acquired by the members of the group, termed the "collective dose" (in units of man Sv), may be used. The value of the collective dose divided by the number of individuals in the exposed population group is the per caput dose, in Sv. The general procedures used by the Committee to evaluate radiation doses are presented in Annex A of this report, "Dose assessment methodologies".

A. NATURAL RADIATION EXPOSURES

24. All living organisms are continually exposed to ionizing radiation, which has always existed naturally. The sources of that exposure are cosmic rays that come from outer space and from the surface of the Sun, terrestrial radionuclides that occur in the Earth's crust, in building materials and in air, water and foods and in the human body itself. Some of the exposures are fairly constant and uniform for all individuals everywhere, for example, the dose from ingestion of potassium-40 in foods. Other exposures vary widely depending on location. Cosmic rays, for example, are more intense at higher altitudes, and concentrations of uranium and thorium in soils are elevated in localized areas. Exposures can also vary as a result of human activities and practices. In particular, the building materials of houses and the design and ventilation systems strongly influence indoor levels of the radioactive gas radon and its decay products, which contribute significantly to doses through inhalation.

25. The components of the exposures resulting from natural radiation sources have been reassessed in this report based on new information and data from measurements and on further analysis of the processes involved. The results are presented

in Annex B, "Exposures from natural radiation sources". The exposure components have been added to provide an estimate of the global average exposure. The average global exposure does not pertain to any one individual, since there are wide distributions of exposures from each source and the consequent effective doses combine in various ways at each location, depending on the specific concentration of radionuclides in the environment and in the body, the latitude and altitude of the location and many other factors.

26. The annual worldwide per caput effective dose is determined by adding the various components, as summarized in Table 1. The annual global per caput effective dose due to natural radiation sources is 2.4 mSv. However, the range of individual doses is wide. In any large population about 65% would be expected to have annual effective doses between 1 mSv and 3 mSv, about 25% of the population would have annual effective doses less than 1 mSv and 10% would have annual effective doses greater than 3 mSv.

Table 1
Average radiation dose from natural sources

Source	*Worldwide average annual effective dose (mSv)*	*Typical range (mSv)*
External exposure		
Cosmic rays	0.4	0.3-1.0 [a]
Terrestrial gamma rays	0.5	0.3-0.6 [b]
Internal exposure		
Inhalation (mainly radon)	1.2	0.2-10 [c]
Ingestion	0.3	0.2-0.8 [d]
Total	2.4	1-10

a Range from sea level to high ground elevation.
b Depending on radionuclide composition of soil and building materials.
c Depending on indoor accumulation of radon gas.
d Depending on radionuclide composition of foods and drinking water.

B. MAN-MADE ENVIRONMENTAL EXPOSURES

27. Releases of radioactive materials to the environment and exposures of human populations have occurred in several activities, practices and events involving radiation sources. Assessment of the resulting exposures is presented in Annex C of this report, "Exposures to the public from man-made sources of radiation". The main man-made contribution to the exposure of the world's population has come from the testing of nuclear weapons in the atmosphere, from 1945 to 1980. Each nuclear test resulted in unrestrained release into the environment of substantial quantities of radioactive materials, which were widely dispersed in the atmosphere and deposited everywhere on the Earth's surface.

28. The Committee has given special attention to the evaluation of the doses from nuclear explosions in the atmosphere. The worldwide collective effective dose from that practice was evaluated in the UNSCEAR 1982 Report based on numerous measurements of the global deposition of ^{90}Sr and ^{137}Cs and of the occurrence of those and other fallout radionuclides in diet and the human body that were made at the time the testing was taking place.

29. New information has become available on the numbers and yields of nuclear tests. Those data were not fully revealed earlier by the countries that conducted the tests because of military sensitivities. An updated listing of atmospheric nuclear tests conducted at each of the test sites is included in this report (see Annex C). Although the total explosive yields of each test have been divulged, the fission and fusion yields are still mostly suppressed. Some general assumptions have been made to make it possible to specify the fission and fusion yields of each test in order to estimate the amounts of radionuclides produced in the explosions. The estimated total of fission yields of individual tests is in agreement with the global deposition of the main fission radionuclides ^{90}Sr and ^{137}Cs, as determined by worldwide monitoring networks.

30. With improved estimates of the production of each radionuclide in individual tests and using an empirical atmospheric transport model, it is possible to determine the time course of the dispersion and deposition of radionuclides and to estimate the annual doses from various pathways in each hemisphere of the world. In that way it has been calculated that the world average annual effective dose reached a peak of 150 μSv in 1963 and has since decreased to about 5 μSv in 2000, from residual radionuclides in the environment, mainly ^{14}C, ^{90}Sr and ^{137}Cs. The average annual doses are 10% higher in the northern hemisphere, where most of the testing took place, and lower in the southern hemisphere. Although there was considerable concern at the time of testing, the annual doses remained relatively low, reaching at most about 7% of the background level from natural radiation sources.

31. The exposures of local populations surrounding the test sites have also been assessed using available information. The level of detail is still not sufficient to document the exposures with great accuracy. Attention to the local conditions and the possibilities of exposure was not great in the early years of the test programmes. However, dose reconstruction efforts are proceeding to clarify this experience and to document the local and regional exposures and doses that occurred.

32. Underground testing caused exposures beyond the test sites only if radioactive gases leaked or were vented. Most underground tests had much lower yields than atmospheric tests, and it was usually possible to contain the debris. Underground tests were conducted at the rate of 50 or more per year from 1962 to 1990. Although it is the intention of most countries to agree to ban all further tests, both atmospheric and underground, the Comprehensive Nuclear-Test-Ban Treaty (see General Assembly resolution 50/245) has not yet come into force. Further underground testing has occurred. Thus, it cannot yet be stated that the practice has ceased.

33. During the time when nuclear weapon arsenals were being built up, especially in the earlier years (1945-1960), there were releases of radionuclides exposing local populations downwind or downstream of nuclear installations. Since there was little recognition of exposure potentials and monitoring of releases was limited, the assessment must be based on the reconstruction of doses. Results are still being obtained that document the experience. Practices have greatly improved and arsenals are now being reduced. Exposures from the military fuel cycle have thus diminished to very low levels.

34. A continuing practice is the generation of electrical energy by nuclear power reactors. Assuming this practice of generation lasts for 100 years, the maximum collective dose can be estimated from the cumulative doses that occur during the period of the practice. The normalized 100-year truncated figure is 6 man Sv per gigawatt year. Assuming the present annual generation of 250 gigawatt years continues, the truncated collective dose per year of practice is 1,500 man Sv to the world population, giving an estimated maximum per caput dose of less than 0.2 μSv per year.

35. Except in the case of accidents or at sites where wastes have accumulated, causing localized areas to be contaminated to significant levels, there are no other practices that result in important exposures from radionuclides released into the environment. Estimates of releases of isotopes produced and used in industrial and medical applications are being reviewed, but these seem to be associated with rather insignificant levels of exposure. Possible future practices, such as dismantling of weapons, decommissioning of installations and waste management projects, can be reviewed as experience is acquired, but these should all involve little or no release of radionuclides and should cause only negligible doses. For medical practice, the highest individual doses, averaging about 0.5 mSv, may be received by family members who may come into close contact with patients undergoing ^{131}I treatments.

36. When accidents occur, environmental contamination and exposures may become significant. The accident at the Chernobyl nuclear power plant was a notable example. The exposures were highest in the local areas surrounding the reactor, but low-level exposures could be estimated for the European region and for the entire northern hemisphere. In the first year following the accident, the highest regionally averaged annual doses in Europe outside the former Union of Soviet Socialist Republics were less than 50% of the natural background dose. Subsequent exposures decreased rapidly. The higher doses and possible health consequences in the region of the accident are being investigated.

37. There are several industries that process or utilize large volumes of raw materials containing natural radionuclides. Discharges from those industrial plants to air and water and the use of by-products and waste materials may contribute to enhanced exposure of the general public. Estimated maximum exposures arise from phosphoric acid production, mineral sand processing industries and coal-fired power stations. Although annual doses of about 100 μSv could be received by a few local residents, doses of 1-10 μSv would be more common.

C. MEDICAL RADIATION EXPOSURES

38. The use of ionizing radiation for medical diagnosis and therapy is widespread throughout the world. There are significant country-to-country variations in national resources for and practice in medical radiology. In general, medical exposures are confined to an anatomical region of interest and dispensed for specific clinical purposes so as to be of direct benefit to the examined or treated individuals. Diagnostic exposures are characterized by fairly low doses to individual patients (effective doses are typically in the range 0.1-10 mSv) that in principle are just sufficient to provide the required clinical information. The resulting per caput doses to populations are given in Table 2. In contrast, therapeutic exposures involve very much higher doses precisely delivered to the tumour volumes (prescribed doses typically in the range 20-60 Gy) to eradicate disease, principally cancer, or to alleviate symptoms. Relatively small numbers of diagnostic or therapeutic exposures are conducted on volunteers in controlled studies for the purposes of research. Medical radiology is conducted systematically and radiation accidents are fairly infrequent.

39. The Committee has assessed the exposures from medical radiation procedures based on information obtained from questionnaires distributed to all Member States. Four levels of health care have been distinguished based on the number of physicians available to serve the inhabitants of a country. They range from one physician per 1,000 population at the highest level (health-care level I to one physician for

Table 2
Radiation exposures from diagnostic medical x-ray examinations

Health care level	*Population per physician*	*Annual number of examinations per 1,000 population*	*Average annual effective dose to population (mSv)*
I	<1 000	920	1.2
II	1 000-3 000	150	0.14
III	3 000-10 000	20	0.02
IV	>10 000	<20	<0.02
Worldwide average		330	0.4

more than 10,000 population (health-care level IV). The available data have been averaged to obtain representative frequencies of procedures or exposure within countries at each level. These were then extrapolated to the population of all countries within each level and the total population of the world and are presented in Table 2. The detailed results of the Committee's evaluation are presented in Annex D, "Medical radiation exposures".

40. Temporal trends in the estimates of the number of procedures in medical radiology from the various reviews undertaken by the Committee indicate a steady increase. Further increase in the use of medical radiation and resultant doses can be expected following changes in the patterns of health care that are being facilitated by advances in technology and economic developments. For example, increase is likely in the utilization of x rays with, in particular, a growth in importance for computed tomography and interventional procedures. Practice in nuclear medicine will be driven by the use of new and more specific radiopharmaceuticals for diagnosis and therapy, and there will be increased demand for radiotherapy owing to population ageing. In addition, further growth in medical radiology can be expected in developing countries where present facilities and services are often lacking.

41. Accordingly, there is a need for the Committee to undertake further authoritative reviews of global practice, with the systematic compilation of new national survey data, in particular from regions where knowledge is presently sparse, and the exploration of improved modelling in order to provide refined assessments of worldwide exposures. This major task will help monitor and inform on levels and trends in dose from the rapidly evolving and important practice of medical radiology and will also stimulate further assessments and critical review of practices by individual countries.

D. OCCUPATIONAL RADIATION EXPOSURES

42. There are a number of occupations in which workers are exposed to man-made sources of radiation, such as at nuclear installations or medical clinics, and some workers are exposed to enhanced levels of natural radiation. The Committee uses the term occupational exposure to mean exposures at work that are directly due to the work. Occupational radiation exposures have been assessed from data submitted to the Committee by national authorities in response to questionnaires. The data summarized in Annex E, "Occupational radiation exposures", are quite extensive. Five-year average data for various occupations are reported for 1975-1994. The exposures from man-made sources are given the most attention; countries usually record such data for regulatory purposes. Where average exposures over a workforce are needed, the number of workers is taken to be the number of workers monitored.

43. The estimates of occupational radiation exposure in this report have benefited from a much more extensive and complete database than was previously available to the Committee. The efforts by countries to record and improve dosimetric data were reflected in the responses to the Committee's survey of occupational radiation exposures and have led to improved estimates of occupational doses.

44. The Committee's current estimate of the worldwide collective effective dose to workers from man-made sources for the early 1990s, 2,700 man Sv, is lower by a factor of about 2 than that made by the Committee for the late 1970s. A significant part of the reduction comes in the nuclear power fuel cycle, in particular in uranium mining. However, reductions are seen in all the main categories: industrial uses, medical uses, defence activities and education. This trend is also reflected in the worldwide average annual effective dose, which has fallen from about 1.9 mSv to 0.6 mSv. The average annual doses to workers in the various occupations are given in Table 3.

45. No attempt has been made to deduce any trend in the estimates of dose from occupational exposure to enhanced natural sources of radiation, as the supporting data are somewhat limited. The UNSCEAR 1988 Report made a crude estimate of about 20,000 man Sv from that source, which was subsequently revised downward to 8,600 man Sv in the UNSCEAR 1993 Report. The comparable figure for 1990-1994 is 5,700 man Sv; however, an important new element has been added for this period, namely, occupational exposure to elevated levels of radon and its progeny, bringing the overall estimate of collective dose to 11,700 man Sv. This is still considered to be a crude estimate, and much better data are required. This will be a challenge for the next assessment by the Committee.

Table 3
Occupational radiation exposures

Source / practice	*Number of monitored workers (thousands)*	*Average annual effective dose (mSv)*
Man-made sources		
Nuclear fuel cycle (including uranium mining)	800	1.8
Industrial uses of radiation	700	0.5
Defence activities	420	0.2
Medical uses of radiation	2 320	0.3
Education/veterinary	360	0.1
Total from man-made sources	4 600	0.6
Enhanced natural sources		
Air travel (crew)	250	3.0
Mining (other than coal)	760	2.7
Coal mining	3 910	0.7
Mineral processing	300	1.0
Above ground workplaces (radon)	1 250	4.8
Total from natural sources	6 500	1.8

E. COMPARISON OF EXPOSURES

46. Radiation doses from the various sources of exposure received by the world population are compared in Table 4. Two quantities are appropriate for comparisons. For a source that is constant, or that changes only as the result of natural processes, the annual global per caput effective dose is used. That quantity is also used for a source that delivers all its exposure in a short time. For sources that continue to cause exposure over long periods, it is necessary to indicate the trend over time. The values given in Table 4 are the annual doses averaged over the world population, which are not necessarily the doses that any one individual would experience. Because of considerable variations in exposures, depending on location, personal habits, diet, and so on, doses to individuals differ.

Table 4
Annual per caput effective doses in year 2000 from natural and man-made sources

Source	*Worldwide annual per caput effective dose (mSv)*	*Range or trend in exposure*
Natural background	2.4	Typically ranges from 1-10 mSv, depending on circumstances at particular locations, with sizeable population also at 10-20 mSv.
Diagnostic medical examinations	0.4	Ranges from 0.04-1.0 mSv at lowest and highest levels of health care
Atmospheric nuclear testing	0.005	Has decreased from a maximum of 0.15 mSv in 1963. Higher in northern hemisphere and lower in southern hemisphere
Chernobyl accident	0.002	Has decreased from a maximum of 0.04 mSv in 1986 (average in northern hemisphere). Higher at locations nearer accident site
Nuclear power production (see paragraph 34)	0.0002	Has increased with expansion of programme but decreased with improved practice

47. By far the greatest contribution to exposure comes from natural background radiation. The annual per caput dose is 2.4 mSv and the range in typical circumstances may be between 1 mSv and 10 mSv. There are, however, small groups of persons who may be exposed to much higher levels. In some places, the natural radionuclide content in the soil creates high external exposure levels; these are known as high-background areas. Much more significant and widespread is the variability in the levels of radon concentration in indoor air.

48. The second largest contribution to exposures of individuals worldwide is from medical radiation procedures. There is an increasing trend in such exposures, reflecting the more widespread use and availability of medical radiation services throughout the world.

49. The exposure of the world's population from nuclear test explosions in the atmosphere was considered to be quite dramatic at the time of the most intensive testing (1958-1962), when it was realized how widespread it had been. The practice resulted in the unrestrained release of

large amounts of radioactive materials directly into the atmosphere. Of all man-made practices or events, atmospheric nuclear testing involved the largest releases of radionuclides into the environment. The annual doses reached, on average, 7% of the natural background at their maximum in 1963. Residual levels of longer-lived radionuclides still present in the environment contribute little to the annual exposure of the world population.

III. RADIATION-ASSOCIATED CANCER

50. Radiation effects are caused by the damage inflicted in cells by the radiation interactions. The damage may result in cell death or modifications that can affect the normal functioning of organs and tissues. Most organs and tissues of the body are not affected by the loss of even considerable numbers of cells. However, if the number lost becomes large, there will be observable harm to the organ or tissue and therefore to the individual. Only if the radiation dose is large enough to kill a large number of cells will such harm occur. This type of harm occurs in all individuals who receive an acute dose in excess of the threshold for the effect and is called "deterministic".

51. If the cell is not killed but only modified by the radiation damage, the damage in the viable cell is usually repaired. If the repair is not perfect, the modification will be transmitted to daughter cells and may eventually lead to cancer in the tissue or organ of the exposed individual. If the cells are concerned with transmitting genetic information to the descendants of the exposed individual, hereditary disorders may arise. Such effects in the individuals or in their descendants are called "stochastic", meaning of a random nature.

52. In short, deterministic (acute) effects will occur only if the radiation dose is substantial, such as in accidents. Stochastic effects (cancer and hereditary effects) may be caused by damage in a single cell. As the dose to the tissue increases from a low level, more and more cells are damaged and the probability of stochastic effects occurring increases.

53. Over the 45 years that the Committee has been reviewing information relating to the biological effects of radiation, substantial scientific advances have taken place and an improved understanding has resulted. The present knowledge of radiation effects and the main results of the Committee's assessments are summarized below.

A. RADIOBIOLOGICAL EFFECTS AFTER LOW DOSES OF RADIATION

54. The Committee has reviewed the broad field of experimental studies of radiation effects in cellular systems and in plants and animals. Many of those responses and the factors modifying them form a basis for the knowledge of human radiation effects and can often be evaluated in more detail than studies of humans. Furthermore, fundamental radiobiology nowadays includes the field of molecular radiobiology, which is contributing to an understanding of the mechanisms of radiation response.

55. Damage to deoxyribonucleic acid (DNA) in the nucleus is the main initiating event by which radiation causes long-term harm to organs and tissues of the body. Double-strand breaks in DNA are regarded as the most likely candidate for causing critical damage. Single radiation tracks have the potential to cause double-strand breaks and in the absence of fully efficient repair could result in long-term damage, even at the lowest doses. Damage to other cellular components (epigenetic changes) may influence the functioning of the cell and progression to the malignant state.

56. Numerous genes are involved in cellular response to radiation, including those for DNA damage repair and cell-cycle regulation. Mutation of those genes is reflected in several disorders of humans that confer radiation sensitivity and cancer proneness on the individuals concerned. For example, mutation of one of many so-called checkpoint genes may allow insufficient time to repair damage, because the cell loses its ability to delay progression in the cell cycle following radiation exposure.

57. Cells have a number of biochemical pathways capable of recognizing and dealing with specific forms of damage. This subject is reviewed in Annex F, "DNA repair and mutagenesis". One gene that plays a key role is the tumour suppressor *TP53*, which is lost or mutated in more than half of all human tumours. The p53 protein produced by the gene controls both arrest of the cell cycle and one pathway of apoptosis (the programmed cell death that is instrumental in preventing some damaged cells from progressing to the transformed, malignant growth stage). Some such biochemical pathways are also implicated in stress response or adaptation processes that act to limit the extent or outcome of damage. Even with such protective processes induced and acting, it is clear that misrepaired radiation damage gives the potential for progression to cancer induction or hereditary disease.

58. Proto-oncogenes (genes that may be activated inappropriately and then participate in tumorigenesis) and tumour-suppressor genes control a complex array of biochemical pathways involved in cellular signalling and interaction, growth, mitogenesis, apoptosis, genomic stability and differentiation. Mutation of those genes can compromise those controls and contribute to the multi-stage development of cancer.

59. Proto-oncogene activation by chromosomal translocation is often associated with early stages in the development of leukaemias and lymphomas, although gene loss also occurs. For many solid tumours there is a requirement for a loss-of-function mutation of tumour-suppressor genes that control cellular proliferation in specific tissues. The subsequent onset of genomic instability through further mutations in clones of cells may be a critical event in the transformation from benign to malignant state. Loss of apoptotic control is also believed to be important throughout tumorigenesis.

60. The multi-stage nature of tumorigenesis is considered in Annex G, "Biological effects at low radiation doses". Much knowledge about the process remains to be learned. Although the concept of sequential, interacting gene mutations as the driving force for tumorigenesis is more firmly established, there is a lack of understanding of the complex interplay between those events and the consequences for cellular behaviour and tissue homeostasis; uncertainty also exists about the contribution made to malignant development of non-mutational (epigenetic) cellular events such as gene silencing and cellular communication changes.

61. Direct evidence on the nature of radiation-associated initiating events in human tumours is sparse, and rapid progress in the area should not be anticipated. By contrast, good progress is being made in resolving early events in radiation-associated tumours in mouse models. Those molecular observations strengthen the view expressed in the UNSCEAR 1993 Report that radiation-induced tumorigenesis will tend to proceed via gene-specific losses; a contribution from early arising epigenetic events should not, however, be discounted.

62. Much information points to the crucial importance of DNA repair and other damage-response functions in tumorigenesis. DNA damage-response functions influence the appearance of initial events in the multi-stage process and reduce the probability that a benign tumour will spontaneously acquire the secondary mutations necessary for full malignant development. Thus, mutations of DNA damage-response genes in tumours play an important role in the spontaneous development of genomic instability.

63. The repair of sometimes complex DNA double-strand lesions is largely error-prone and is an important determinant of dose, dose rate and radiation quality effects in cells. Uncertainties continue to surround the significance to tumorigenesis of adaptive responses to DNA damage; the mechanistic basis of such responses has yet to be well characterized, although associations with the induction of biochemical stress responses seems likely. Recent scientific advances highlight the differences in complexity and reparability between spontaneously arising and radiation-induced DNA lesions. Those data argue against basing judgements concerning low-dose response on comparisons of overall lesion abundance rather than their nature.

64. The research findings on the adaptive responses to radiation in cells and organisms were reviewed in the UNSCEAR 1994 Report, and the typical expression of an adaptive response is described there. The phenomenon has been interpreted as being the result of an initial small (priming) dose activating a repair mechanism that reduces the response to a subsequent larger (challenge) dose. Apparently, the range of priming doses is limited, the time for presenting the challenge dose is critical and the challenge dose needs to be of a reasonable magnitude. The response varies greatly between individual donors of lymphocytes. Nevertheless, the adaptive response has been seen in many systems, including human lymphocytes, a variety of mouse cells and with some chemical agents such as hydrogen peroxide and bleomycin as well as with radiation. However, so far there appears to be no generally reproducible reduction in tumour induction following low-dose irradiation.

65. The basic premises of radiation response are that any radiation interaction with DNA results in damage that if not repaired or if incorrectly repaired may represent an initiating event in the tumorigenesis pathway. The mutation of genes commonly results in modulation of their expression, with loss of gene products (proteins) or alteration in their properties or amounts. The biochemical balance of the cell may then be disrupted, compromising the control of cell signalling or the proliferation and differentiation schedules. In that way, mutated cells, instead of being checked or killed, may be allowed to proceed to clonal growth. Some non-mutational (epigenetic) events or damage may be involved or contribute to those changes. In some cases the genome may be destabilized, allowing further mutations to accumulate, which may promote the progression of tumorigenesis.

66. The judgement as to whether there might be a threshold level of exposure below which biological response does not occur can be guided by mechanistic considerations. Specifically, there is a need to know whether at very low doses the repair processes are more efficient and perhaps enhanced by the adaptive response, preventing any damage to the cellular components. Such a threshold could occur only if repair processes were totally effective in that dose range or if a single track were unable to produce an effect. The absence of consistent indications of significant departures from linearity of tumorigenic response at low doses in cellular endpoints (chromosome aberrations, gene mutation, cell transformation), the activity of well characterized error-prone DNA repair pathways and the evidence on the nature of spontaneous DNA damage in mammalian cells argue against adaptive or other processes that might provide for a dose threshold for radiation effects. The cellular processes such as apoptosis and cellular differentiation that can protect against later phases of tumorigenesis are judged to be efficient but can be bypassed; there is no reason to believe that those defences act differently on spontaneous and radiation-induced tumours or have specific dose dependencies.

67. It may therefore be concluded that, as far as is known, even at low doses radiation may act as a mutational initiator of tumorigenesis and that anti-tumorigenic defences are unlikely to show low-dose dependency. In general, tumorigenic response does not therefore appear to be a complex

function of increasing dose. The simplest representation is a linear relationship, which is consistent with most of the available mechanistic and quantitative data. There may be differences in response for different types of tumour and statistical variations in each data set are inevitable. A departure from linearity is noted for leukaemia data, for which a linear-quadratic function is used. Skin cancer and some cancers induced by alpha emitters may have virtual thresholds. Because of the multi-step nature of the tumorigenesis process, linear or linear-quadratic functions are used for representational purposes only in evaluating possible radiation risks. The actual response may involve multiple and competing processes that cannot yet be separately distinguished.

B. COMBINED EFFECTS

68. Combined exposures to radiation and other physical, chemical or biological agents in the environment are a characteristic of life. The characteristics and effects of combined exposures are reviewed in Annex H, "Combined effects of radiation and other agents". Although both synergistic and antagonistic combined effects are common at high exposures, there is no firm evidence for large deviations from additivity at controlled occupational or environmental exposures. This holds for mechanistic considerations, animal studies and epidemiology-based assessments. Therefore, in spite of the potential importance of combined effects, results from assessments of the effects of single agents on human health are generally deemed applicable to exposure situations involving multiple agents.

69. Deviation from additivity depends on the specificity of the agents for the different steps in the sequence leading to clinical effect. Such effects are, however, only to be expected in cases where both agents are responsible for a large fraction of the total transitions through the sequence. For agents acting independently and through different mechanisms and pathways, simple additivity is predicted.

70. Because exposure to both cigarette smoke and radon is so prevalent, that combined effect is of special importance. Cigarette smoke is a complex mixture of chemical and physical agents and there is still no clear picture of the interaction mechanisms. Epidemiological data clearly indicate that the interaction at intermediate to high exposure levels leads to more-than-additive effects on lung cancer. For example, enhanced radiation risks (more than additive but less than multiplicative) to smokers are evident in the radon miner studies.

71. With the exception of radiation and smoking, there is little indication from epidemiological data for a need to adjust for strong antagonistic or synergistic combined effects. The lack of pertinent data on combined effects does not imply per se that interactions between radiation and other agents do not occur and have no influence on the radiation risk at low doses. Indeed, substances with tumour promoter and/or inhibitor activities are found in the daily diet and cancer risk therefore depends on lifestyle, in particular eating habits. Not only can those agents modify the natural or spontaneous cancer incidence, but they may also modify the carcinogenic potential of radiation. Such modifications would influence the outcome in particular when radiation risks were projected relative to the spontaneous cancer incidence.

72. In general, it can be concluded that genotoxic agents with similar biological and mechanistic behaviour and acting at the same time will interact in a concentration-additive manner (isoadditive). This means that concurrent exposures to ionizing radiation and other DNA-damaging agents with no specific affinity to those DNA sequences which are critically involved in carcinogenesis will generally result in effects not far from isoadditive.

C. CANCER EPIDEMIOLOGY

73. Radiation-associated cancer in humans is studied in population groups that have been exposed to radiation doses such that cancer cases in excess of the normal background incidence may be identified. Estimates of risk may be derived from populations for whom individual doses can be reasonably estimated. Those populations include survivors of the atomic bombings, medically irradiated patients, those occupationally exposed, individuals exposed to radionuclides released into the environment, and people exposed to elevated levels of natural background radiation. Since the Committee's assessment of the risks of radiation-induced cancer in the UNSCEAR 1994 Report, additional important information has become available from epidemiological studies. Those data are summarized in Annex I, "Epidemiological evaluation of radiation-induced cancer".

74. It is now known that radiation can cause cancer in almost any tissue or organ in the body, although some sites are much more prone than others (see paragraph 77). A clearer understanding of physiological modifying factors, such as sex and age, has developed over the last few years. Although differences in the absolute risk of tumour induction with sex are not large and vary with site, for most solid cancers the absolute risk is higher in women than in men. People who were young at the time of radiation exposure have higher relative and absolute risks than older people, but again this varies by site.

75. Further follow-up of radiation-exposed cohorts has demonstrated that excess cancers continue to occur at long times after radiation exposure and, therefore, large uncertainties can arise in the projection of lifetime risks. Data for the Japanese atomic bomb survivors are consistent with a linear or linear-quadratic dose response over a wide range of doses, but quantifying risks at low doses is less certain because of the limitations of statistical precision, potential residual biases or other methodological problems and the possibility of chance findings due to multiple statistical testing. Longer follow-up of cohorts with a wide range of doses, such as the atomic bomb survivors, will provide more essential informa-

tion at low doses, but epidemiology alone will not be able to resolve the issue of whether there are low-dose thresholds. It should be noted, however, that the inability to detect increased risks at very low doses does not mean that those increases in risk do not exist.

76. The studies of the Japanese survivors are particularly important because the cohort includes a large exposed population of both sexes, a wide distribution of doses and the full range of ages. The results of that research provide the primary basis for estimating the risk of radiation-induced cancer. Among the 86,572 individuals in the Life Span Study cohort of survivors of the atomic bombings, there were 7,578 deaths from solid tumours during 1950-1990. Of those cancer deaths, 334 can be attributed to radiation exposure. During the same period, 87 of 249 leukaemia deaths can be attributed to radiation exposure. In 1991, at the time of the latest evaluation, some 48,000 persons (56%) were still living. It is projected that 44% of the population will still be living in 2000.

77. The Life Span Study cancer incidence and mortality data are broadly similar, demonstrating statistically significant effects of radiation for all solid tumours as a group, as well as for cancers of the stomach, colon, liver, lung, breast, ovary and bladder. The incidence data also provide evidence of excess radiation risks for thyroid cancer and non-melanoma skin cancers. Statistically significant risks were not seen in either the incidence or the mortality data for cancers of the rectum, gall bladder, pancreas, larynx, uterine cervix, uterine corpus, prostate gland and kidney or renal pelvis. An association with radiation exposure is noted for most types of leukaemia, but not for lymphoma or multiple myeloma.

78. The numbers of solid tumours associated with radiation exposure are not sufficient to permit detailed analysis of the dose response for many specific sites or types of cancer. For all solid tumours combined, the slope of the dose-response curve is linear up to about 3 Sv, but the dose-response curve for leukaemia is best described by a linear-quadratic function. Statistically significant risks for cancer in the Life Span Study are seen at organ doses above about 100 mSv.

79. Studies of populations exposed to medical, occupational or environmental radiation provide information on issues that cannot be addressed by the atomic bomb survivor data, such as the effects of chronic low doses, alpha doses to the lung from radon, highly fractionated doses and variability among populations. For some cancer sites, including leukaemia, breast, thyroid gland, bone and liver, very useful results come from investigations other than the Life Span Study. Risk estimates derived from those studies generally agree well with those from the Life Span Study.

80. Large studies of occupationally exposed persons are also contributing valuable data on low-dose effects. A combined analysis of data for a large number of nuclear workers indicates that the risk of leukaemia increases with increasing dose. However, the statistical precision of such studies is still low in comparison with the results at high-dose rate from the atomic bomb survivors. As a result, it is difficult to arrive at a definitive conclusion on the effects of dose rate on cancer risks, in particular since those effects may differ among cancer types. However, the conclusions reached in the UNSCEAR 1993 Report, based on both epidemiological and experimental evidence that suggested a reduction factor of less than 3 when extrapolating to low doses or low-dose rates, still appear to be reasonable in general.

81. Information on the effects of internal doses, from both low- and high- linear energy transfer (LET) radiation, has increased since the time of the UNSCEAR 1994 Report. In particular, an elevated risk of thyroid cancer in parts of Belarus, the Russian Federation and Ukraine contaminated as a result of the Chernobyl accident shows a link with radioactive iodine exposure during childhood. However, risk estimation associated with those findings is complicated by difficulties in dose estimation and in quantifying the effect of screening for the disease. Other studies in the former Soviet Union have provided further information on internal doses, for example, an increased risk of lung cancer among workers at the Mayak plant. Leukaemia was elevated in the population living near the Techa River. However, the different sources of radiation exposure (both external and internal) and, in the case of the Techa River studies, the potential effects of migration, affect the quantification of risks. Results from several case-control studies of lung cancer and indoor radon have been published in recent years that, in combination, are consistent with extrapolations from data on radon-exposed miners, although the statistical uncertainties in those findings are still large.

82. Particular attention has been paid in Annex I to risks for specific cancer sites. Again, the new information that has become available in recent years has helped in the examination of some risks. However, for some cancer sites there remain problems in characterizing risks, owing to the low statistical precision associated with moderate or small excess numbers of cases. This can limit, for example, the ability to estimate trends in risk in relation to factors such as age at exposure, time since exposure and gender. An exception is breast cancer, where a comparison of data on the Japanese atomic bomb survivors and women with medical exposures in North America points to an absolute transfer of risks between populations. There are some cancer sites for which there is little evidence for an association with radiation (e.g. non-Hodgkin's lymphoma, Hodgkin's disease and multiple myeloma). While the evaluations for the lymphomas are affected in part by the small numbers of cases in several studies, they should be contrasted with the evaluations for leukaemia (excluding chronic lymphocytic leukaemia), which, while also a rare disease, has clearly been related to radiation in many populations.

83. Lifetime risk estimates are sensitive to variations in background tumour rates and the variability can lead to differences that are comparable to differences associated with the transport method across populations or the method of risk projection. The variability in such projections

highlights the difficulty of choosing a single value to represent the lifetime risk of radiation-induced cancer. Furthermore, uncertainties in estimates of risk for specific types of cancer are generally greater than for all cancers combined.

84. Based on the available epidemiological data, the Committee has derived risk estimates for radiation-induced cancer. For a population of all ages and both genders with an acute dose of 1 Sv (low-LET), it is suggested that lifetime risk estimates for solid cancer mortality might be taken as 9% for men and 13% for women. The uncertainties in the estimates may be a factor of about 2, higher or lower. The estimates could be reduced by 50% for chronic exposures, as discussed in the UNSCEAR 1993 Report, again with an uncertainty factor of 2, higher or lower. Solid cancer incidence risks can be taken as being roughly twice those for mortality. Lifetime solid cancer risks estimates for those exposed as children might be twice the estimates for a population exposed at all ages. However, continued follow-up in studies of such groups will be important in determining lifetime risks. The experience of the Japanese atomic bomb survivors provides compelling evidence for linearity in estimating excess risks of solid cancers; therefore, as a first approximation, linear extrapolation of the estimates at 1 Sv could be used for estimating solid cancer risks at lower doses.

85. The estimates of lifetime risks for leukaemia are less variable. The lifetime risk of death from leukaemia may be taken as 1%, for either gender, following an acute dose of 1 Sv. The uncertainty in the estimate may be about a factor of 2, higher or lower. In view of non-linearity in the dose response, decreasing the dose tenfold, from 1 Sv to 0.1 Sv, will result in a 20-fold decrease in the lifetime risk if the dose is acute. The risks of solid cancer and leukaemia are broadly similar to those estimated in the UNSCEAR 1994 Report.

86. One radiation-associated cancer of particular importance in children is cancer of the thyroid gland. There is strong evidence that the risk of thyroid cancer decreases with increasing age at exposure, so that the risk in children under 15 years of age is substantially larger than in adults. Among children, those aged 0-5 years are five times more sensitive than those aged 10-14 years. In view of that sensitivity, it is not surprising that large increases in thyroid cancer incidence have been observed in children in Belarus, the Russian Federation and Ukraine following the Chernobyl accident in 1986. The incidence rate of thyroid cancer in children from regions of those countries was ten times higher in 1991-1994 than in the preceding five years. About 1,800 cases of childhood thyroid cancer had occurred as at 1998. The topic is reviewed extensively in Annex J of this report, “Exposures and effects of the Chernobyl accident”.

87. Cancer may be induced by prenatal exposure. In humans, the induction of childhood cancers, leukaemia and solid cancers as a result of exposure to x rays was first reported in 1958, when the Oxford Survey established an increased incidence of childhood tumours in the first 15 years of life for those exposed to x rays *in utero* compared with those who were not exposed. The attribution of that increase to radiation exposure has been criticized by some on the grounds that the exposed women may have had medical or other conditions that were responsible for the increased cancer rates. Support for the causal role of radiation is found in some other studies, and the risk, if genuine, was estimated to be about 5 % per Sv. No such effects were observed in survivors of the atomic bombings irradiated *in utero*.

88. Risks of induced cancer expressed in adulthood among those exposed *in utero* are more difficult to evaluate. Nevertheless, the fact that relative risks increase with decreasing age at exposure among the survivors of the atomic bombings causes concern about a potentially greater sensitivity to cancer induction for those exposed *in utero* than for those exposed at young ages. The atomic bomb survivors exposed *in utero* are now 55 years old. Thus it is especially important to evaluate their cancer risk experience later in life.

III. THE CHERNOBYL ACCIDENT

89. The Committee has given special attention to the accident at the Chernobyl nuclear reactor that occurred on 26 April 1986. It was the most serious accident ever to occur in the nuclear power industry. The reactor was destroyed in the accident, considerable amounts of radioactive materials were released to the environment and many workers were exposed to high doses of radiation that had serious, even fatal, health consequences (see below). Among the residents of Belarus, the Russian Federation and Ukraine, well over a thousand cases of thyroid cancer (about 1,800) have been reported in children. Notwithstanding problems associated with screening, those cancers were most likely caused by radiation exposures received at the time of the accident. Many other health problems have been noted in the populations that are less likely to be related to radiation exposures. From a scientific point of view, there is a need to evaluate and understand the technical causes and effects of the accident. From a human point of view, there is also an obligation to provide an objective analysis of the health consequences of the accident for the people involved. The Committee has prepared a further assessment of the accident with both objectives in mind.

90. Soon after the accident, the deposition of dispersed radionuclides and the exposures that resulted were measured and evaluated throughout the region affected. The Committee made use of those data to evaluate the average individual and

population doses for the various regions and countries and for the northern hemisphere as a whole. The results were presented in the UNSCEAR 1988 Report, Annex D, "Exposures from the Chernobyl accident". The experience gained in treating the immediate radiation injuries of workers and firefighters involved in controlling the accident were also reviewed in the Appendix to Annex G, "Early effects in man of high doses of radiation", of the same report.

91. Evaluating the exposures received by the people who were evacuated or who still reside in the areas most affected by the accident has required much time and effort. The initial measurements must be supplemented by information on such things as the location and diet of the people in each settlement. The accumulation of data on late health effects has also required further time. Only now, some 15 years after the accident, can an initial assessment of the local exposures and effects of the accident be made. The detailed results of the Committee's assessment are presented in Annex J of this report, "Exposures and effects of the Chernobyl accident".

A. RELEASE OF RADIONUCLIDES

92. The accident at the Chernobyl reactor happened during an experimental test of the electrical control system as the reactor was being shut down for routine maintenance. The operators, in violation of safety regulations, had switched off important control systems and allowed the reactor to reach unstable, low-power conditions. A sudden power surge caused a steam explosion that ruptured the reactor vessel, allowing further violent fuel-steam interactions that destroyed the reactor core and severely damaged the reactor building.

93. It is noteworthy that an earlier accident in 1979 at the Three Mile Island reactor in the United States of America also resulted in serious damage to the reactor core but without a steam explosion. In that case, however, the containment building surrounding the reactor prevented the release of all but trace amounts of radioactive gases. The Chernobyl reactor lacked the containment feature. Following the explosions, an intense graphite fire burned for 10 days. Under those conditions, large releases of radioactive materials took place.

94. The radioactive gases and particles released in the accident were initially carried by the wind in westerly and northerly directions. On subsequent days, the winds came from all directions. The deposition of radionuclides was governed primarily by precipitation occurring during the passage of the radioactive cloud, leading to a complex and variable exposure pattern throughout the affected region.

B. EXPOSURE OF INDIVIDUALS

95. The radionuclides released from the reactor that caused exposure of individuals were mainly iodine-131, caesium-134 and caesium-137. Iodine-131 has a short radioactive half-life (eight days), but it can be transferred to humans relatively rapidly from the air and through milk and leafy vegetables. Iodine becomes localized in the thyroid gland. For reasons related to the intake of those foods by infants and children, as well as the size of their thyroid glands and their metabolism, the radiation doses are usually higher for them than for adults.

96. The isotopes of caesium have relatively longer half-lives (caesium-134 has a half-life of 2 years while that of caesium-137 is 30 years). These radionuclides cause longer-term exposures through the ingestion pathway and through external exposure from their deposition on the ground. Many other radionuclides were associated with the accident, which have also been considered in the exposure assessments.

97. Average doses to those persons most affected by the accident were about 100 mSv for 240,000 recovery operation workers, 30 mSv for 116,000 evacuated persons and 10 mSv during the first decade after the accident to those who continued to reside in contaminated areas. Maximum values of the dose may be an order of magnitude higher. Outside Belarus, the Russian Federation and Ukraine, other European countries were affected by the accident. Doses there were at most 1 mSv in the first year after the accident with progressively decreasing doses in subsequent years. The dose over a lifetime was estimated to be 2-5 times the first-year dose. These doses are comparable to an annual dose from natural background radiation and are, therefore, of little radiological significance.

98. The exposures were much higher for those involved in mitigating the effects of the accident and those who resided nearby. Those exposures are reviewed in great detail in the assessment of the Committee.

C. HEALTH EFFECTS

99. The Chernobyl accident caused many severe radiation effects almost immediately. Of 600 workers present on the site during the early morning of 26 April 1986, 134 received high doses (0.7-13.4 Gy) and suffered from radiation sickness. Of these, 28 died in the first three months and another 2 soon afterwards. In addition, during 1986 and 1987, about 200,000 recovery operation workers received doses of between 0.01 Gy and 0.5 Gy. That cohort is at potential risk of late consequences such as cancer and other diseases and their health will be followed closely.

100. The Chernobyl accident also resulted in widespread radioactive contamination in areas of Belarus, the Russian Federation and Ukraine inhabited by several million people. In addition to causing radiation exposure, the accident caused long-term changes in the lives of the people living in the contaminated districts, since the measures intended to limit radiation doses included resettlement, changes in food supplies and restrictions on the activities of individuals and families. Later on, those changes were accompanied by the major economic, social, and political changes that took place when the former Soviet Union broke up.

101. For the last 14 years, attention has been focused on investigating the association between exposure caused by radionuclides released in the Chernobyl accident and late effects, in particular thyroid cancer in children. A majority of the studies completed to date are of the descriptive type, in which average population exposures are correlated with the average rates of cancer incidence over specific periods of time. As long as individual dosimetry is not available, it is difficult to determine whether the effects are radiation-related and it is also impossible to make reliable quantitative estimates of risk. The reconstruction of individual doses is a key element for future research on radiation-associated cancers related to the Chernobyl accident.

102. The number of thyroid cancers (about 1,800) in individuals exposed in childhood, in particular in the severely contaminated areas of the three affected countries, is considerably greater than expected based on previous knowledge. The high incidence and the short induction period are unusual. Other factors may be influencing the risk. If the current trend continues, additional thyroid cancers can be expected to occur, especially in those who were exposed at young ages.

103. Apart from the increase in thyroid cancer after childhood exposure, no increases in overall cancer incidence or mortality have been observed that could be attributed to ionizing radiation. The risk of leukaemia, one of the main concerns (leukaemia is the first cancer to appear after radiation exposure owing to its short latency time of 2-10 years), does not appear to be elevated, even among the recovery operation workers. Neither is there any proof of other non-malignant disorders that are related to ionizing radiation. However, there were widespread psychological reactions to the accident, which were due to fear of the radiation, not to the actual radiation doses.

104. There is a tendency to attribute increases in the rates of all cancers over time to the Chernobyl accident, but it should be noted that increases were also observed before the accident in the affected areas. Moreover, a general increase in mortality has been reported in recent years in most areas of the former Soviet Union, and this must be taken into account when interpreting the results of Chernobyl-related studies.

105. The present understanding of the late effects of protracted exposure to ionizing radiation is limited, since the dose-response assessments rely heavily on studies of exposure to high doses and animal experiments; extrapolations are needed, which always involves uncertainty. The Chernobyl accident might shed light on the late effects of protracted exposure, but given the low doses received by the majority of exposed individuals, any increase in cancer incidence or mortality will be difficult to detect in epidemiological studies. One future challenge will be to develop individual dose estimates including estimates of uncertainty, and to determine the effects of doses accumulated over a long period of time.

Notes

1 The United Nations 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 set out in resolution 913 (X) of 3 December 1955. The Committee was originally composed of the following Member 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 States of America. The membership of the Committee was subsequently enlarged by the Assembly in its resolution 3154 C (XXVIII) of 14 December 1973 to include the Federal Republic of Germany, Indonesia, Peru, Poland and the Sudan. By its resolution 41/62 B of 3 December 1986, the General Assembly increased the membership of the Committee to a maximum of 21 members and invited China to become a member.

2 For the previous substantive reports of the United Nations Scientific Committee on the Effects of Atomic Radiation to the General Assembly, see *Official Records of 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. 25* (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); ibid., *Forty-first Session, Supplement No. 16* (A/41/16); ibid., *Forty-third Session, Supplement No. 45* (A/43/45), ibid., *Forty-eighth Session, Supplement No. 46* (A/48/46); ibid., *Forty-ninth Session, Supplement No. 46* (A/49/46); ibid. *Fifty-first Session, Supplement No. 46* (A/51/46). These documents are referred to as the 1958, 1962, 1964, 1966, 1969, 1972, 1977, 1982, 1986, 1988, 1993, 1994 and 1996 reports, respectively. The 1972 report, with scientific annexes, was published as *Ionizing Radiation: Levels and Effects, Volume I: Levels* and *Volume II: Effects* (United Nations publication, Sales Nos. E.72.IX.17 and 18). The 1977 report, with scientific annexes, was published as *Sources and Effects of Ionizing Radiation* (United Nations publication, Sales No. E.77.IX.1). The 1982 report, with scientific annexes, was published as *Ionizing Radiation: Sources and Biological Effects* (United Nations publication, Sales No. E.82.IX.8). The 1986 report, with scientific annexes, was published as *Genetic and Somatic Effects of Ionizing Radiation* (United Nations publication, Sales No. E.86.IX.9). The 1988 report, with scientific annexes, was published as *Sources, Effects and Risks of Ionizing Radiation* (United Nations publication, Sales No. E.88.IX.7). The 1993, 1994 and 1996 reports, with scientific annexes, were published as *Sources and Effects of Ionizing Radiation* (United Nations publication, Sales Nos. E.94.IX.2, No. E.94.IX.11 and E.96.IX.3, respectively).

Appendix I

Members of national delegations attending the forty-fourth to forty-ninth sessions

Argentina	D. Beninson (Representative), E. D'Amato, D. Cancio
Australia	P. A. Burns (Representative), K. H. Lokan (Representative), J. Loy, D. I. Macnab
Belgium	J. R. Maisin (Representative), A. Debauche, R. Kirchmann, H. P. Leenhouts, J. Lembrechts, K. Sankaranarayanan, P. Smeesters, J. van Dam, H. Vanmarcke, A. Wambersie
Brazil	J. L. Lipsztein (Representative), D. Melo, A. T. Ramalho, E. R. Rochedo
Canada	R. M. Chatterjee (Representative), D. B. Chambers, R. J. Cornett, N. E. Gentner (Representative), R. V. Osborne (Representative), S. Vlahovich (Representative)
China	Z. Pan (Representative), N. Gu, F. He, Q. He, J. Ma, B. Mao, K. Li, P. Liu, Y. Song, Z. Tao, K. Wei, B. Xiu, H. Yang, L. Zhang, Y. Zhao, J. Zhou, B. Zhu
Egypt	A. M. El-Naggar (Representative), F. Hammad (Representative), M. A. Gomaa
France	J. F. Lacronique (Representative), A. Aurengo, M. Bourguignon, A. Flüry-Hérard, J. Lallemand, C. Luccioni, R. Masse (Representative), J. Piéchowski, A. Rannou
Germany	W. Burkart (Representative), U. Ehling, W. Jacobi, T. Jung, A. Kaul (Representative), A. Kellerer, J. Kiefer, G. Kirchner, W. Köhnlein, C. Reiners, F.E. Stieve, C. Streffer
India	K. B. Sainis (Representative), P. C. Kesavan (Representative)
Indonesia	K. Wiharto (Representative), T. Suprihadi, S. Zahir
Japan	Y. Sasaki (Representative), T. Asano, H. Iizuka, T. Isoyama, S. Kumazawa, S. Mizushita, K. Morita, Y. Muramatsu, N. Nakagawa, J. Onodera, K. Sato, T. Sato, Y. Taguchi, K. Tatsumi
Mexico	J. R. Ortiz-Magaña (Representative), E. Araico (Representative)
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Appendix II

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