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I. INTRODUCTION

1. During the last few years the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1/ has undertaken a broad review of the sources and effects of ionizing radiation. Nine scientific annexes on particular subjects were issued in the UNSCEAR 1993 report. Two further annexes have been completed, and these comprise the UNSCEAR 1994 report. This is the twelfth substantive report of the Committee, informing the General Assembly and the scientific and world community of its latest assessments. 2/ The two reports, referred to as the 1993 and 1994 reports, are complementary and provide a coherent summary of the Committee's findings and programme of work.

2. The preparation of the present report and its scientific annexes (see para. 6) 3/ took place from the thirty-eighth to the forty-third sessions of the Committee. Serving as Chairman, Vice-Chairman and Rapporteur, respectively, at the sessions were: thirty-eighth and thirty-ninth sessions: K. Lokan (Australia), J. Maisin (Belgium) and E. Létourneau (Canada); fortieth and forty-first sessions: J. Maisin (Belgium), E. Létourneau (Canada) and L. Pinillos Ashton (Peru); forty-second and forty-third sessions: E. Létourneau (Canada), L. Pinillos Ashton (Peru) and G. Bengtsson (Sweden). The names of members of national delegations who attended the thirty-eighth to the forty-third sessions of the Committee are listed in appendix I.

3. The scientific annexes of the present report were developed at annual sessions of the Committee, based on working papers prepared by the secretariat. The Committee wishes to acknowledge the help and advice of a small group of consultants, appointed by the Secretary-General, who helped in the preparation of the material for the report. Their names are given in appendix II. They were responsible for the preliminary reviews and evaluations of the technical information received by the Committee or available in the open scientific literature, on which rest the final deliberations of the Committee.

4. The sessions of the Committee held during the period under review were attended by representatives of the United Nations Environment Programme (UNEP), the World Health Organization (WHO), the International Atomic Energy Agency (IAEA), the International Commission on Radiological Protection (ICRP), the International Agency for Research on Cancer (IARC) and the International Commission on Radiation Units and Measurements (ICRU). The Committee wishes to acknowledge their contributions to the discussions.

5. In the present report, the Committee summarizes the main conclusions of the two scientific annexes, "Epidemiological studies of radiation carcinogenesis" and "Adaptive responses to radiation in cells and organisms". In addition, the Committee is reviewing the effects of radiation on the natural environment, and although the scientific annex has not yet been completed, a summary of this work in progress is given.

6. Following established practice, only the introductory part of the report is submitted to the General Assembly. The full UNSCEAR 1994 report, including the scientific annexes, will be issued as a United Nations sales publication. This practice is intended to achieve a wider distribution 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 important because they form the basis for the conclusions of the report.

II. EPIDEMIOLOGICAL STUDIES OF RADIATION CARCINOGENESIS

7. The Committee has paid particular attention to the review of results of epidemiological studies of human populations exposed to ionizing radiation, since these form the main basis for quantifying the risks of radiation-induced cancer in man. Several study populations are available, including the survivors of the atomic bombings of Hiroshima and Nagasaki, patients exposed in medical procedures, those exposed occupationally and inhabitants of high natural background or contaminated areas, and these groups are the subject of continuing investigations.

8. Estimates of the risks of cancer caused by radiation exposure were derived in the UNSCEAR 1972, 1977 and 1988 reports and discussed in the UNSCEAR 1993 report. Although all information was considered, the primary estimates of risk were derived from results of the main study population, the survivors of the atomic bombings. An objective of the Committee's present review of this subject is to consider the large number of additional epidemiological studies now contributing quantitative information on the effects in humans of ionizing radiation and to evaluate comparative risk estimates.

9. Studies of disease in human populations must adhere strictly to epidemiological principles in order to achieve valid quantitative results. These include sound case ascertainment, an appropriate comparison group, sufficient follow-up, an accounting for confounding factors and well-characterized dosimetry. Such epidemiological studies are able to provide clear-cut evidence of risks for various sites of cancer, and also to evaluate the factors that modify risks, following high radiation doses. However, at low doses epidemiological studies are not able to detect and quantify statistically significant radiation effects.

A. Effects of external exposures

10. The Committee has examined the epidemiological studies that could be used to derive risk estimates from external, sparsely ionizing (low-linear energy transfer (LET)) radiation exposures at high and low dose rates. The Committee has summarized the main features of these studies, including their strengths and limitations.

11. The primary study for the estimation of risk of cancer induction is the Life Span Study of survivors of the atomic bombings of Hiroshima and Nagasaki. The study, which began in 1950, comprises a large population of all ages and both sexes exposed to a range of doses at high dose rate. Data on cancer mortality and new data on cancer incidence are now available up to 1987. Since most of the original survivors are still living, many more years of follow-up will be necessary to determine the complete lifetime cancer occurrence in this population. Consequently, lifetime risk estimation requires projection beyond the period of observation.

12. Cancers for which statistically significant excess risks have been determined from the Life Span Study mortality data are leukaemia, breast, bladder, colon, liver, lung, oesophagus, ovary, multiple myeloma and stomach. The incidence data are broadly similar, but two of the sites, oesophagus and multiple myeloma, do not show significant risks. The incidence data are probably more definitive than the mortality data. Two additional sites, namely thyroid and skin, have significant excess incident cancers.

13. Studies of other radiation-exposed populations such as cervical cancer patients, ankylosing spondylitics and children treated for tinea capitis serve to clarify and generally support findings from the Life Span Study. Some also provide information on issues that cannot be addressed by the atomic bomb survivor data, such as the effects of low chronic doses, highly fractionated exposures and variability among populations. For some sites of cancer, including breast, leukaemia and thyroid, there are a number of very useful results from studies other than the Life Span Study. In general, there are no great disparities in risk estimates between the Life Span Study and the other studies.

14. Although the Committee has presented risk estimates for specific sites from results of many studies, general estimates of lifetime mortality risks for all cancers must still be derived from the Life Span Study. For this report the Committee has analysed the data from 1950 to 1987 and made projections to the full life-span of the population in several ways. Using the constant relative risk model allowing for sex and age at exposure (a more refined analysis than in the UNSCEAR 1988 report), the estimates of lifetime risk of mortality following an exposure to 1 Sv (weighted dose) is 11 per cent for solid tumours and 1 per cent for leukaemia. Using alternative projection methods allowing for some decline in relative risk with time (as suggested by some epidemiological studies), lifetime risk estimates for solid tumours are 20 per cent to 40 per cent lower. The constant relative risk estimates in the 1988 report were 10 per cent for solid tumours and 1 per cent for leukaemia at 1 Sv.

15. The Committee indicated in the UNSCEAR 1993 report that risk estimates derived at high doses and high dose rates should be divided by a small factor to obtain the risk at low doses (less than 0.2 sievert (Sv)). If a factor of two is used, the risk derived from the UNSCEAR 1988 report would be 5 per cent per Sv and from this report 6 per cent per Sv for a constant relative risk projection. If alternative projection methods are used, however, the risk would be 4 per cent to 6 per cent in the Japanese population (the applicability to other populations involves some additional uncertainty). Consequently, the use of a nominal value of 5 per cent per Sv for mortality owing to leukaemia and solid cancers from irradiation at low doses for a population of all ages (4 per cent per Sv for an adult working population) still seems valid to the Committee.

16. The effects of low-LET radiation delivered at low doses or low dose rates have been examined in studies of occupational, natural background and environmental exposures. Occupational studies offer the most promise of providing results that are statistically significant because they are based on large populations with a range of individual dose estimates and long periods of observation.

17. The most comprehensive occupational study to date involves nuclear workers in the United Kingdom of Great Britain and Northern Ireland. This study reports a significant excess risk for leukaemia and a positive, but non-significant excess for all cancers as a group. A smaller study carried out in the United States found non-significant deficits of cases among exposed workers. In a combined analysis of these two studies, the results of which were non-significant, there was excess incidence of leukaemia and all cancers, which were about half the estimates for the atomic bomb survivors. Initial findings in studies of workers in the atomic energy programme of the former Soviet Union with exposures of the order of several sievert accumulated over several years show clear excesses of cancer in the highest dose groups broadly consistent with the levels of risk seen in the survivors of the atomic bombings.

18. Comparisons of cancer incidence in areas of high and low natural radiation background have been undertaken in China, France, Japan, Sweden, the United Kingdom and the United States of America. None, including the largest, that in China, has produced statistically significant associations.

19. Populations exposed to environmental releases of radionuclides have provided little information on risk. However, one circumstance of special interest concerns releases of fission products into the Techa River in the former Soviet Union during 1948-1951. In the 28,000 people studied there was some evidence of an excess of leukaemia not inconsistent with results derivable from the study of the survivors of the atomic bombings.

B. Effects of internal exposures

20. Of the radionuclides emitting low-LET radiation that may enter the body, iodine-131 is the most important, since it is used to diagnose thyroid conditions and to treat hyperthyroidism and thyroid carcinoma. Environmental exposures to iodine-131 from fallout and from accidents at nuclear installations have also occurred. Iodine-131 appears to be less effective than external radiation in causing thyroid cancer, perhaps by a factor of three to five. More studies are needed to clarify the possibly greater risks in children than in adults, as indicated by external radiation exposure. The Committee is aware of reports of thyroid cancer incidence in locally exposed individuals following the Chernobyl accident and intends to examine this issue in a future report.

21. More densely ionizing (high-LET) radiation exposures result from alpha-particle-emitting radionuclides, such as radon and its decay products and radium and thorium used in medical and industrial applications. High-LET radiation is more effective in causing damage in tissue than low-LET radiation. Alpha-radiation is not very penetrating, however, so exposures occur only when the radionuclides in air, food or water are taken into the body. The Committee has examined the few epidemiological studies that can provide risk estimates.

22. Radon is an important source of exposure of the public in houses and other buildings. The risk of lung cancer caused from exposure to radon is derived from studies of miners of uranium and other minerals. There is no consistent evidence that radon causes cancer in tissues other than the lung. The excess incidence of lung cancer from radon is concentrated in the period 5 to 14 years after exposure and decreases with time. The risks for low and protracted exposures are likely to be more appropriate for applying to exposure levels experienced by the public. The numerous studies of residential radon exposure have so far contributed little to radon risk estimation, mainly because of their low statistical power. Important issues that must be addressed include the impact of confounding factors such as smoking and arsenic-containing dusts in mines.

23. Estimates of carcinogenic risk in bone and liver have been derived from exposures to alpha-emitting radionuclides: radium-224 in the case of bone and Thorotrast, a thorium-based x-ray contrast agent, in the case of liver.

24. Long-lived radium-226 and radium-228 at high levels have caused bone sarcomas and carcinomas of the paranasal sinuses in radium dial painters, and the risk extends over the long periods in which these radionuclides are lodged in bone. Precise risk estimates have not been derived. No excess cancers were identified in workers exposed to small amounts of plutonium or to uranium dusts.

Workers exposed in Russia to a combination of external radiation and plutonium did have excess lung cancers at the higher exposure levels.

C. Other relevant studies

25. In the last decade there were many studies of the incidence of leukaemia near nuclear installations in the United Kingdom following the identification of several leukaemia clusters. One report suggested paternal exposure as a cause. However, in the light of more recent reports it is unlikely that any of these clusters or excesses are the result of either environmental radiation or paternal exposure. A possible explanation is that the excesses result from the spread of infection that occurs when populations from urban and rural areas mix. No such pattern of clusters was found in subsequent studies around nuclear installations in Canada, France, Germany and the United States.

26. Initial excesses in leukaemia were observed following a single nuclear test explosion in the United States and following that explosions carried out by the United Kingdom, but the observation seems to be the result of chance in the first case and an unusually low incidence in controls for the British participants in the tests and unusual latencies in the cohort of New Zealand participants in the second case. No clear effect is evident.

27. People with certain recessive hereditary diseases, such as ataxia-telangiectasia and retinoblastoma, are known to be sensitive to radiation exposure and are more likely to develop second cancers if treated with radiation. There are indications that those who do not have the disease but are genetic carriers may also be more sensitive than normal individuals to cancer induction, possibly by radiation exposure, but studies so far are not definitive.

III. ADAPTIVE RESPONSES TO RADIATION IN CELLS AND ORGANISMS

28. The scientific community has been aware for many years of the possibility that low doses of radiation may result in changes in cells and organisms, which reflects an ability to adapt to the effects of radiation.

29. It has been suggested in recent years that conventional estimates of the risks of stochastic effects of low doses of ionizing radiation may have been overstated because no allowance was made for the process referred to as adaptation. This is the name given to the possibility that a small dose of radiation may condition cells in such a way as to stimulate cellular repair processes and thus reduce either the natural incidence of malignant conditions or the likelihood of excess malignancy being produced by radiation.

30. There is substantial evidence that the number of radiation-induced chromosomal aberrations and mutations can be reduced by a small prior conditioning dose in proliferating mammalian cells in vitro and in vivo. It seems likely that this effect is linked to an increased capacity for deoxyribonucleic acid (DNA) repair. While it has been observed under specified and clearly defined conditions, it has not been seen with all cell systems.

31. There is increasing evidence that cellular repair mechanisms are stimulated after radiation-induced damage. It has to be resolved whether these are related to increased DNA repair. Whatever the mechanisms, they seem able to act not only on the lesions induced by ionizing radiation but also on at least a portion of the lesions induced by some other toxic agents. There appears to be similar overlap in regard to the type of DNA damage that induces adaptive response.

32. It remains doubtful whether the immune system plays any role in these processes. In annex E of the UNSCEAR 1993 report, entitled "Mechanisms of radiation oncogenesis", the Committee concluded that the immune system may not have a significant influence on radiation carcinogenesis after low doses. In annex B to the report, entitled "Adaptive responses to radiation in cells and organisms", that conclusion is not altered, although some transient effects on the immune system have been identified.

33. Extensive data from animal experiments and limited human data provide no evidence to support the view that the adaptive response in cells decreases the incidence of late effects such as cancer induction in humans after low doses. However, further experimental studies should be conducted.

34. As to the biological plausibility of a radiation-induced adaptive response, it is recognized that the effectiveness of DNA repair in mammalian cells is not absolute. The mechanisms of adaptation are likely to coexist with the mechanisms induced by low doses that may result in malignant transformations. An important question, therefore, is to judge the balance between stimulated cellular repair and residual damage. The Committee hopes that more data will become available and stresses that at this stage it would be premature to draw conclusions for radiological protection purposes.

IV. EFFECTS OF RADIATION ON THE NATURAL ENVIRONMENT

35. All living organisms are exposed to radiation from natural sources (cosmic rays and the natural radionuclides present in all components of the terrestrial and aquatic environments) and from local, regional and global contamination arising from human activities.

36. The Committee has not previously attempted to review the effects of radiation on plants and animals in the environment. There is, however, a substantial body of information that can form the basis for such a review. The accumulation of radionuclides in plants and animals in the environment has been considered, particularly from the viewpoint of their transfer through food chains leading to man but also in terms of basic physiology. These data may be developed to provide estimates of the possible concomitant radiation exposure.

37. Previous reports of the Committee have presented summaries of the extensive laboratory studies of the effects of radiation on a variety of animals. In addition, data on radiation effects have been obtained from use of large, sealed sources of gamma rays in the environment and from investigations of the effects, actual or potential, in contaminated areas. Together, these data may be used to assess the relative radiosensitivities of a wide range of organisms and the effects of radiation exposure on those individual attributes (mortality, fertility, fecundity, etc.) that are essential for the maintenance of healthy natural populations.

38. The Committee is in the process of reviewing these data and intends to provide a scientific assessment of the impact of increased radiation exposure on the natural environment in a future report.

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, and its terms of reference were 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 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 resolution 41/62 B of 3 December 1986, the 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 UNSCEAR 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) and ibid., Forty-eighth Session, Supplement No. 46 (A/48/46). These documents are referred to as the

1958, 1962, 1964, 1966, 1969, 1972, 1977, 1982, 1986, 1988 and 1993 reports, respectively. The 1972 report, with scientific annexes, was entitled 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 entitled 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 entitled Genetic and Somatic Effects of Ionizing Radiation (United Nations publication, Sales No. E.86.IX.9). The 1988 report, with scientific annexes, was entitled Sources, Effects and Risks of Ionizing Radiation (United Nations publication, Sales No. E.88.IX.7). The 1993 report, with annexes, was entitled Sources and Effects of Ionizing Radiation (United Nations publication, Sales No. E.94.IX.2).

3/ To be issued as a sales publication.

APPENDIX I

Members of national delegations attending the thirty-eighth
to forty-third sessions

ARGENTINA	D. Beninson (Representative), E. d'Amato, C. Arias, D. Cancio, A. Curti, E. Palacios
AUSTRALIA	K. H. Lokan (Representative)
BELGIUM	J. Maisin (Representative), P. Govaerts, R. Kirchmann, H. P. Leenhouts, P. H. M. Lohman, K. Sankaranarayanan, D. Smeesters, A. Wambersie
BRAZIL	J. Landmann-Lipsztein (Representative), E. Penna Franca (Representative)
CANADA	E. G. Létourneau (Representative), A. Arsenault, D. R. Champ, R. M. Chatterjee, P. J. Duport, V. Elaguppilai, N. E. Gentner, B. C. Lentle, D. K. Myers, R. V. Osborne
CHINA	Wei Kedao (Representative), Li Deping (Representative), Liu Hongxiang (Representative), Wei Lüxin (Representative), Leng Ruiping, Pan Ziqiang, Tao Zufan, Wu Dechang
EGYPT	F. H. Hammad (Representative), M. F. Ahmed (Representative), F. Mohamed (Representative), H. M. Roushdy (Representative), S. E. Hashish
FRANCE	P. Pellerin (Representative), E. Cardis, R. Coulon, H. Dutrillaux, A. Flury-Hérard, H. Jammet, J. Lafuma, G. Lemaire, R. Masse
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INDONESIA	S. Soekarno (Representative), S. Wiryosimin (Representative), S. Zahir (Representative), K. Wiharto
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PERU	L. V. Pinillos Ashton (Representative)
POLAND	Z. Jaworowski (Representative), J. Jankowski, J. Liniecki, M. Waligórski, O. Rosiek, S. Sterlinski, I. Szumiel

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SUDAN	O. I. Elamin (Representative), A. Hidayatalla (Representative)
SWEDEN	G. Bengtsson (Representative), L. E. Holm, J. O. Snihs, L. Sjöberg, J. Valentin
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UNITED STATES OF AMERICA	F. A. Mettler (Representative), L. R. Anspaugh, J. D. Boice, C. W. Edington, J. H. Harley, N. H. Harley, C. Meinhold, P. B. Selby, W. K. Sinclair, E. W. Webster, H. O. Wyckoff

Notes

a/ At the thirty-eighth and thirty-ninth sessions: Federal Republic of Germany.

b/ At the thirty-eighth, thirty-ninth and fortieth sessions: Union of Soviet Socialist Republics.

c/ At the thirty-eighth, thirty-ninth, fortieth and forty-first sessions: Czechoslovakia.

APPENDIX II

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