ASSESSMENTS BY UNSCEAR OF RADIATION SOURCES AND EFFECTS

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ABSTRACT

Since 1955, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has evaluated the exposures of the world population to the various sources of ionizing radiation and from available radiobiological and epidemiological data has assessed the health effects and risks of radiation. The latest scientific evaluations of the Committee were published in the UNSCEAR 1993 and 1994 Reports. In this paper a summary is presented of the main results of analyses in each of the scientific annexes of these reports.

EXPOSURES FROM NATURAL SOURCES OF RADIATION

The assessment in the UNSCEAR 1993 Report [2] of the annual effective dose from natural sources of ionizing radiation in areas of normal background has not changed from the previous estimate of 2.4 mSv provided in the UNSCEAR 1988 Report [1], although there have been minor adjustments in the various components. One third of the total is due to external exposure to cosmic rays and terrestrial radionuclides and two thirds to internal exposure. The largest component of exposure, half of the total, is from radon and its decay products.

Additional data have been compiled from national surveys of external exposure rates and of radon concentrations. The population-weighted average dose rate in air in areas of normal background is 57 nGy h^{-1} outdoors and 80 nGy h^{-1} indoors. The indoor-to-outdoor ratio is thus 1.5, but the ratio can vary from less than 1 for lightweight houses to around 2 when the construction materials make substantial contributions to exposures. The concentration of radon is typically 10 Bq m^{-3} outdoors and 40 Bq m^{-3} indoors. In tropical areas with houses of lightweight construction and high ventilation, there should be little difference in indoor and outdoor levels. There are, however, too many factors that determine the concentrations, and measurements are necessary in all areas.

The dosimetry of radon is presently under review. UNSCEAR has retained the assumptions used previously, namely equilibrium factors of 0.4 indoors and 0.8 outdoors and a dose conversion factor of 9 nSv h^{-1} per Bq m^{-3} of equilibrium equivalent concentration (EEC) of radon. With these parameters the average annual effective dose from radon and its decay products is estimated to be 0.13 mSv for exposures outdoors and 1.0 mSv for exposures indoors. The average annual effective dose from inhalation of thoron (^{220}Rn) and its decay products is 0.07 mSv.

UNSCEAR has assessed the natural radiation exposures resulting from energy production using coal, oil, peat, natural gas and geothermal energy and the use of phosphate rock in fertilizers and building materials and of mineral sands. The highest exposures result from the use of phosphate by-products in buildings, the domestic use of coal for cooking and heating, and the use of phosphate fertilizers. The overall annual effective dose from all such sources averaged over the world's population is 0.02 mSv.

EXPOSURES FROM MAN-MADE SOURCES OF RADIATION

The assessment of the radiation exposures caused by releases of radionuclides to the environment from man-made practices or events has been updated in the UNSCEAR 1993 Report. Atmospheric testing of nuclear weapons resulted in the largest releases of radionuclides into the environment from man-made sources. Most of the testing occurred in 1952-1958 and 1961-1962. The last atmospheric test was conducted in 1980. From the many measurements that have been made throughout the years, UNSCEAR has evaluated transfer coefficients relating the input of radionuclides into the atmosphere to the resulting dose to humans. The collective effective dose to the world's population from atmospheric nuclear testing is estimated to be 30 million man Sv. Of this total, 86% is due to long-term, low-level exposure from ^{14}C. The contributions to dose in decreasing order of importance are ^{14}C, ^{137}Cs, ^{95}Zr, ^{90}Sr, ^{106}Ru, ^{144}Ce and ^{3}H. Only residual irradiation from ^{14}C, ^{137}Cs, ^{90}Sr and ^{3}H remains to be received by the present and future world population. The collective dose from this practice is equivalent the 2.4 years of exposure of the present world population to natural radiation sources.

There has been an increasing trend in electrical energy generation by nuclear reactors since the practice began in 1956. At present, about 20% of the world's electrical energy is generated by nuclear means. At the end of 1994 there were 432 reactors operating in 29 countries. During routine operation of installations associated with the nuclear fuel cycle (uranium mining and milling, fuel fabrication, reactor operation, reprocessing and waste disposal) radionuclides are released to the environment. The data on radionuclides released are quite extensive and complete, especially for reactor operations.

There have been generally decreasing trends in normalized releases of radionuclides from nuclear fuel cycle installations as operating practices have improved. This has meant that the trend in collective dose to the world population has been increasing somewhat less than the trend in electrical energy generated. The estimate of collective dose from nuclear power production was 43,000 man Sv during 1990 and 400,000 man Sv for the entire period 1956-1990. Even when the collective dose caused by the Chernobyl accident (600,000 man Sv) is added, the total collective dose has been just 8% of that which the world's population receives in one year from natural radiation sources.

Other man-made sources of radiation exposures caused by releases of radionuclides to the environment that have been assessed in the UNSCEAR 1993 Report include exposures to local populations near the Semipalatinsk, Nevada, Australian and Pacific nuclear test sites, exposures from underground nuclear testing, exposures from nuclear weapons fabrication, exposures from radioisotope production and use and exposures from accidents at the Three Mile Island and Chernobyl reactors, Kyshtym and Windscale plutonium production plants, crashes of airplanes carrying nuclear weapons, satellite re-entries and lost or mishandled radiation sources, as at Goiânia.

MEDICAL RADIATION EXPOSURES

The use of x rays and radiopharmaceuticals for diagnostic examinations and therapeutic treatments is quite common throughout the world. Most of the equipment and the procedures

performed are in industrialized countries, in which 25% of the world's population is located. UNSCEAR has assessed the exposures from medical radiation exposures from information obtained in questionnaires distributed to all countries. Four regions of health care have been designated, based on availability of facilities, and the data have been extrapolated to the world's population. The variations in medical radiation exposures among individuals are great, ranging from no dose to those not examined or treated to high doses to those receiving therapeutic treatments. The largest portion of the total dose from medical radiation sources arises from diagnostic examinations due to their relatively high frequency. At the highest level of health care the annual effective dose averaged over the population from all diagnostic examinations is 1.1 mSv. The comparable value is 0.05 mSv at the lowest level of health care. The population-weighted world average is 0.3 mSv, and the annual collective effective dose is 1.8 million man Sv. The collective effective dose from medical radiation usage has been evaluated to allow comparisons among countries and the evaluation of trends. Much, and optimally most, of the collective dose from medical uses of radiation is offset by direct benefits to the examined or treated patients.

OCCUPATIONAL RADIATION EXPOSURES

Occupational radiation exposures have been assessed from data submitted to UNSCEAR by national authorities in response to questionnaires. The data summarized in the UNSCEAR 1993 Report are quite extensive. Five-year average data for various occupations are reported for the period 1975-1989. The exposures from man-made sources are given the most attention; these data are usually required in countries for regulatory purposes.

The collective effective dose depends on the average individual doses and the number of exposed workers. The highest component of collective dose from man-made sources is from the nuclear fuel cycle (2,500 man Sv). There are 880,000 workers in this industry worldwide. For the 2.2 million medical radiation workers the annual collective dose is 1,000 man Sv. Fewer workers and lower collective doses arise in industrial uses of radiation (510 man Sv) and in defence activities (250 man Sv).

The collective effective dose to workers exposed to natural radiation sources is estimated to be 8,600 man Sv, which is two times higher than that from man-made sources. Some 5.2 million workers have been considered. The individual doses are more uncertain than from man-made sources. The largest component of occupational exposures from natural sources is from underground mining of coal and other minerals. Aircrew and some other occupations form secondary components.

MECHANISMS OF RADIATION ONCOGENESIS

Recent advances in molecular biology have been considered in one of several annexes in the UNSCEAR 1993 Report that deal with biological topics. The principal theories of oncogenesis and the results of experimental cellular and molecular studies are reviewed. The basic processes of induction, promotion and progression are recognized in oncogenesis, but it is not always possible to clearly differentiate these stages. Point mutations, chromosomal translocations and deletions, some of which are common and others specific to different neoplasms, may play roles in initiation and progression. Loss of function of tumour suppressor genes is considered a major

initiating factor in oncogenesis. Evidence of these genes being targets of radiation action comes from studies of germ-line mutations that predispose to cancer. Some of these genes appear to play a central role in control of the cell cycle.

The action of specific chemicals, hormones and growth factors on cell surface receptors alter proliferative responses of cells and lead to neoplastic progression. In some cases, the enzyme protein kinase C is thought to mediate promotional processes. Mutagenesis and repair of DNA damage are being studied in *in vivo* and *in vitro* systems. Although the complexities are great, the application of modern methods of cell and molecular biology in studies of radiation oncogenesis offer promising prospects of better understanding. Some aspects of research needs and future perspectives are briefly considered in this annex.

INFLUENCE OF DOSE AND DOSE RATE ON STOCHASTIC EFFECTS OF RADIATION

It is generally recognized that the effectiveness of radiation exposures becomes more than proportionally less at low doses and low dose rates. This is reflected in a quadratic term usually needed in describing the radiation response relationship. The factor of reduction may vary with the specific neoplasm and the physical and biological conditions of the exposure. In this annex of the UNSCEAR 1993 Report, the biological models of dose response in cells and organisms are reviewed and the experimental data available from studies of animals and cells in culture are analysed to derive the range of dose and dose-rate effectiveness factors.

The human epidemiological data on dose response are limited. Data from the survivors of the atomic bombings in Japan indicate that a reduction factor of about 2 would be appropriate for leukaemia but not much in excess of 1 for solid tumours. The results of studies of radiation workers are consistent with low values of the reduction factor. Information on thyroid cancer induction by acute external irradiation compared with low dose-rate exposure from intakes of ^{131}I are consistent with a reduction factor of about 3, although there are questions about the heterogeneity of the dose and uncertainties in the dose estimates and the effect that age makes to the overall reduction in risk. For female breast cancer, the information is conflicting, and a range of reduction factors from 1 to 3 can be derived.

For application of reduction factors the Committee considered that dose rates less than 0.1 mGy min^{-1} (averaged over about an hour) or acute doses less than 200 mGy may be regarded as low. The Committee concluded that reduction factors for low-LET exposures should be considered to be similar to those derived from the atomic bomb survivor data. Insufficient data are available to make recommendations for specific tissues. For high-LET radiation, there is little evidence of a consistent dose-rate or dose fractionation effect at low to intermediate doses. For hereditary disease, the adoption of a reduction factor of 3 is supported by experimental data in male mice; one study indicated the factor may be higher for female mice.

HEREDITARY EFFECTS OF RADIATION

It has not been possible to directly confirm radiation-induced mutations in human populations, so genetic risk estimates have had to rely on general knowledge of human genetics and extrapolation of results from animal experiments.

The understanding of human genetics at the molecular level is increasing rapidly. More precise analysis of the type of genetic damage caused by various agents, including radiation, is possible with new laboratory techniques. More recognition is also being obtained of non-traditional types of inheritance, such as mosaicism, genomic imprinting, uniparental disomy, gene amplification and cytoplasmic inheritance. The complexities involved may seem to make genetic risk estimation even more difficult and uncertain.

The specification of the genetic component of diseases and especially of the many so-called multifactorial diseases, which may occur throughout life and with varying severity, is a difficult problem. If some of the non-traditional mechanisms are involved, there could be trans-generational effects with manifestation of effects only after the F_1 or F_2 generations. There are few data to quantify these risks.

The Committee has concluded that there is no basis to alter present genetic risk estimates. Both the direct and indirect (doubling dose) methods of analysing animal data should be used, with due recognition of limitations of both methods. Radiation effects on multifactorial disease, gene regulation and non-traditional forms of inheritance are not well understood and may require different methods of estimation. The study of children of the atomic bomb survivors may be useful in setting outside limits on genetic risk estimates. These data indicate that hereditary effects from moderate acute exposure of a large human population are minimal. Further results are needed of both human and animal data analysed at the molecular level.

RADIATION EFFECTS ON THE DEVELOPING HUMAN BRAIN

The developing human brain is especially sensitive to ionizing radiation. This sensitivity reflects the structural complexity of the brain, its long developmental (and hence sensitive) period, the vulnerability of the undifferentiated neural cells, the need for cell migration to functional positions and the inability of the brain to replace most lost neurons.

The main effects of radiation and the sensitive periods have been derived from survivors of the atomic bombings in Japan exposed *in utero*. Thirty cases of severe mental retardation have been observed in 1,541 survivors. Most cases occurred in those exposed during the period 8-15 weeks following conception. A secondary period of reduced sensitivity occurred 16-25 weeks following conception.

The results indicate that damage caused by exposure to 1 Gy within the most vulnerable period (8-15 weeks following conception) increases the frequency of mental retardation to about 40% (background frequency: 0.8%) and lowers IQ by 25-30 points. The latter result is consistent with the observed increase in mental retardation. Exposure in the critical period also causes a decrement in average school performance and increases the risk of unprovoked seizures. There are no clear indications of thresholds for effects in those exposed in the most critical period. For the period 16-25 weeks, no cases of severe mental retardation were observed at exposure of less than 0.5 Gy. It is reasonable to assume the risks would be smaller for chronic exposures, but the data are too limited to provide quantitative estimates.

LATE DETERMINISTIC EFFECTS IN CHILDREN

Deterministic effects of ionizing radiation are the result of exposures that cause sufficient cell damage or killing to impair function in the irradiated tissue or organ. All tissues and organs may be affected, but tissues vary in their sensitivity to radiation. The ovary, testis, bone marrow, lymphatic tissue and lens of the eye belong to the most radiosensitive tissues.

Deterministic effects in children, with tissues actively growing, are often more severe than in adults. Examples of deterministic damage following radiation exposure in childhood include effects on growth and development, hormonal deficiencies, organ dysfunctions and effects on intellectual and cognitive functions. The review of deterministic effects of radiation in children stems mainly from the study of late clinical effects in children given radiotherapy treatments. As survival rates increase, some of the effects are becoming more apparent. The study groups are small, however, and the follow-up times are limited. The treatment modalities have usually included surgery and chemotherapy in addition to radiotherapy; it is thus not always possible to single out the effects of radiation alone.

The effects in tissues reviewed in this annex include those in the brain, endocrine system, gonads, skeleton, eye, cardiovascular system, lung, breast, liver and gastrointestinal tract, kidney and bone marrow. One objective was to determine the critical dose levels for the appearance of clinical deterministic effects. In general, younger children are more sensitive than older children. Owing to the paucity of data, however, it is not possible to quantify the effects by age in most situations.

EPIDEMIOLOGICAL STUDIES

The Committee previously reviewed the results of epidemiological studies in the UNSCEAR 1988 Report. The main basis for risk estimates in that report was the results of the Life Span Study of survivors of the atomic bombings of Hiroshima and Nagasaki. In the first of two annexes in the UNSCEAR 1994 Report [3], "Epidemiological studies of radiation carcinogenesis", the Committee presents a review of the wide range of epidemiological studies and provides comparative listings of risk estimates that can be derived from these results. The Committee feels that such broader analyses are necessary to establish more reliably the risk estimates. Results from a single study, although ostensibly providing statistically significant results for a particular site or type of cancer, may not represent the general case for one reason or another.

The epidemiological studies considered in the annex, in addition to the Life Span Study of survivors of the atomic bombings, include medically irradiated patients, occupationally exposed workers, individuals exposed to radionuclides released to the environment on various occasions and those exposed to elevated levels of natural background radiation. The characteristics of these studies, along with their strengths and weaknesses, are tabulated in the report.

The Life Span Study of survivors of the atomic bombings continues to be a primary source of epidemiological data on radiation effects. The large cohort includes individuals of both sexes and all ages with good dosimetric data covering a wide range of doses. Cancer incidence data for 1958-1987 are available for the first time, and the cancer mortality data have been extended for

another two years, available now for the period 1950-1987, for use in analysis of risk estimates in this annex.

The Life Span Study incidence and mortality data are broadly similar, with both sets of data demonstrating statistically significant effects 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, and kidney or renal pelvis. An association with radiation exposure is noted for several types of leukaemia but not for lymphoma or multiple myeloma. Earlier data had indicated a possible association between radiation and multiple myeloma, but the new, extended analyses no longer indicate a statistically significant relationship.

Of the some 86,300 individuals in the Life Span Study cohort there were 6,900 deaths from solid tumours during 1950-1987. From comparisons with the control group, approximately 300 of these cancer deaths can be attributed to radiation exposure. The data for leukaemia incidence in this same period indicate that 75 cases can be attributed to radiation exposure.

The numbers of solid tumours associated with radiation exposure are not sufficient to permit detailed analysis of the dose response for specific sites or types of cancer. For all solid tumours together the slope of the dose-response curve is linear up to about 2 Sv. The dose-response curve for leukaemia is non-linear and is best described by a linear-quadratic function. Statistically significant risks for solid tumours in the Life Span Study are presently seen only above 0.2 Sv. The relative risks in the lower dose categories (0.01-0.05, 0.06-0.09, 0.10-0.19 Sv) all have positive nominal risk estimates, but they are not statistically different from unity. The slope of the dose response for doses lower than 0.5 Sv, while lower than the slope for all doses up to 4 Sv, does not differ significantly from it. An inherent limitation of epidemiological studies is to quantify results at doses less than 0.2 Sv because of the low statistical power of the available results.

Because of concerns about the role of cell-killing and the impact of errors in individual dose estimates on the slope of the dose-response curves at high doses, and because the Life Span Study risk estimates are primarily used to evaluate the effects at low doses, the analysis by UNSCEAR in this annex of lifetime risks have been limited to use of the data on individuals with shielded kerma of less than 4 Gy. The models for lifetime risk estimation allowed for differences in age at exposure and sex of the exposed individuals. Alternative assumptions were used for projections of risk beyond the present observational period. The relative risk was either assumed to remain constant to the end of life or to decrease to lower values at times greater than 40 years after exposure, as has been indicated to be the case in some epidemiological studies. The relative risks of leukaemia or lung cancer, for example, seem to decline after more than 20 years after exposure, although cancers of the GI tract can continue to occur for years longer.

The estimates of lifetime risk following exposure of 1 Sv, computed using sex- and age-at-exposure-specific relative risks estimated from the Life Span Study mortality data for 1950-1987 and using the demographic structure for Japan and the Japanese background cancer mortality rates for 1985, are 1.1% for leukaemia and 10.9% for solid tumours. These results may be compared with comparable values derived in the UNSCEAR 1988 Report of 1.0% for leukaemia and 9.7%

for solid tumours. The estimates for the alternative assumptions of risk beyond 40 years after exposure are 20%-30% less than the total risk estimate quoted above of 12% for an exposure of 1 Sv.

The estimates of risk are presented without adjustment for decreased effectiveness of radiation at low doses and low dose rates. The application of a small (<3) dose and dose-rate effectiveness factor was recommended in the UNSCEAR 1993 Report. If a factor of 2 is applied (as was used by ICRP in their 1990 recommendations), the risk estimate derived from the UNSCEAR 1988 Report would be 5% per Sv and from the 1994 Report 6% per Sv for a constant relative risk projection. The alternative projection methods would yield values from 4%-6% in the Japanese population (the applicability to other populations involves some additional uncertainty). Consequently, the use of a nominal value of 5% per Sv for mortality due to leukaemia and solid tumours from irradiation at low doses for a population of all ages (4% per Sv for an adult working population), as recommended by ICRP, still seems valid to the Committee based on these latest analyses.

Studies of other radiation-exposed populations, such as cervical cancer patients, ankylosing spondylitics and children treated for tinea capitis, provide risk estimates that generally support those derived from the data of the survivors of the atomic bombings. The other epidemiological studies provide additional 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 leukaemia, breast and thyroid, and for bone and liver cancer from exposure to radium and thorium radionuclides, there are a number of very useful results from studies other than the Life Span Study. Large studies of occupationally exposed persons are also contributing tentative risk estimates. In general, there are no great discrepancies in risk estimates between the Life Span Study and the other studies.

Two studies provide some indications of risk at doses less than 0.2 Sv in sensitive subgroups of the population. These studies include cancers among those prenatally exposed to x rays with doses of about 0.01 Gy and thyroid cancer in children with doses of roughly 0.1 Gy. The risk estimates are in both cases, however, not yet well established.

UNSCEAR has further considered the reported incidences of leukaemia near nuclear installations in the United Kingdom, now thought unlikely to be related to environmental radiation or paternal exposure, and the evidence of cancer occurrence among participants of atmospheric nuclear tests. All such issues and results of epidemiological studies are reviewed in some detail in this annex.

ADAPTIVE RESPONSES

In a second annex in the UNSCEAR 1994 Report, "Adaptive responses to radiation in cells and organisms", the Committee considers the more recent research findings that are being reported on this interesting aspect of radiation response. The main impetus for the studies of adaptive responses has been the observation that human lymphocytes exposed to a conditioning dose of some 10 mGy while maintained with growth stimulants in culture media suffer about 50% fewer chromosome aberrations when subsequently irradiated with x rays to a dose of 1.5 Gy than when

exposed without the conditioning dose. It seems likely that the effect is linked to an increased capacity for DNA repair.

The adaptive response has been observed, as well, with proliferating bone marrow cells, spermatocytes and fibroblasts, but not with pre-implantation embryo cells. In some cases, *in vivo* exposure of an experimental animal (mouse or rabbit) has been able to provide the conditioning features which result in an adaptive response during subsequent *in vitro* exposure of lymphocytes.

Investigators have determined that the adaptive response for lymphocytes *in vitro* requires a conditioning dose of at least 5 mGy, delivered at a dose rate greater than 200 mGy min^{-1}, and no more than 200 mGy. The adaptive response occurs between 4 and 6 hours after conditioning and lasts for 3 cell cycles. The composition of the culture medium is quite important; it requires a narrow range of pH to be maintained and growth stimulating factors to be present. The degree of response depends on the stage of the cell cycle. Cells in active phases just prior to cell division are more likely to show the response than cells in other stages. Lymphocytes from different donors show variable response. Thus, the adaptive response is by no means a generally occurring phenomena; it requires rather carefully controlled experimental conditions to elicit the behaviour.

The possible mechanisms of adaptive responses are being investigated in current research. Cell cycle delay has been noted at relatively higher conditioning doses (>200 mGy). The delay allows cell damage to be repaired before the cell proceeds through the cell cycle. Since the adaptive response occurs at lower doses when this type of cell cycle delay is not apparent, this cannot be a central mechanism in the response at lower doses.

The evidence becoming available indicates that following radiation damage to cells a number of changes occur. Among these changes are the activation of genes that code for the synthesis of enzymes involved in the control of the cell cycle, proliferation of cells and repair of damage. Some of the enzymes seem to be similar to those induced by damage caused by other toxic agents. The adaptive response may therefore be part of a common mechanism involving cellular response to damage.

In addition to gene activation, enzyme production and at higher doses cell cycle delay, other cellular mechanisms involved may be detoxification of reactive radicals and activation of membrane-bound receptors stimulating cell proliferation. The immune response of the organism may operate at least transiently following radiation exposures in accelerating programmed cell death of damaged cells. All of these process are subjects of continuing investigation.

It is generally more difficult to demonstrate adaptive responses in the whole organism. Some earlier experiments reported seemingly stimulatory effects following low-level exposures. More recent experiments with rodents and beagle dogs exposed at various ages to low dose rates of low-LET radiation have been unable to demonstrate statistically significant differences in life-span of irradiated and control groups after accumulated doses of up to about one gray nor has reduced tumour induction been an outcome of low-dose exposures in these experiments. An important point to note is that along with adaptive response in cells, selective damage leading to malignant cellular transformations may also occur in parallel. This may explain the observed responses in these animal experiments.

The Committee thus judges that adaptive responses manifested by improved repair of cellular damage that take place under specific conditions in experimental cellular studies probably do not completely eliminate residual damage in cells that may ultimately result in malignant transformation. It will be important to continue these studies in order to understand more fully the molecular processes that occur following radiation exposure of cells and how these changes might be manifested in overall response of the organism. The Committee states that "at this stage it would be premature to draw conclusions for radiological protection purposes".

UNSCEAR has now begun a new programme of review of the sources and effects of ionizing radiation. The next scientific report of the Committee will be published probably in 1998.

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