UNSCEAR: Present and Future Activities

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ABSTRACT

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) was established in 1955. The Committee reviews the exposure of the world population to all sources of radiation under normal circumstances as well as after accidents, and is also the prime international scientific body reviewing and assessing the health risks of exposure to ionizing radiation. Its estimates have been and are still being used by major international bodies including the ICRP, IAEA, and WHO. UNSCEAR's publications form the scientific basis on which international and national agencies develop appropriate radiation protection standards for workers, patients and the general public.

UNSCEAR reports yearly to the General Assembly and submits regularly comprehensive reports with detailed scientific annexes on the sources and biological effects of ionizing radiation. In 1995, UNSCEAR began a new program of review of the sources, exposure and biological effects of ionizing radiation. A comprehensive report with scientific annexes will be published in the year 2000. The report will include exposures from natural and man-made radiation sources, occupational and medical radiation exposures, and dose assessment methodologies. The biological effects will cover epidemiological evaluation of radiation-induced cancer, DNA repair and mutagenesis, hereditary effects, combined effects of radiation and other agents, as well as models, mechanisms and uncertainties of biological effects at low radiation doses. An assessment of exposures and effects of the Chernobyl accident is also made. A brief overview is presented here of the range of subjects considered by the Committee.

INTRODUCTION

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) was established in 1955 to advise the General Assembly of the sources and biological effects of ionizing radiation. Its main objective is to assess radiation exposures and the possible consequences for human health. The Committee's publications form the scientific basis on which international and national agencies develop appropriate radiation protection standards for workers, patients and the general public (1-4). During the last few years, UNSCEAR has undertaken a broad review of the sources and effects of ionizing radiation, and will this year publish the results of these evaluations.

RADIATION SOURCES AND EXPOSURES

Radiation exposures arise from both natural and man-made sources. The natural background radiation normally contributes the greatest exposure to individuals, and forms the baseline to which the man-made exposures are added. These include practices or events that have resulted in releases of radionuclides to the environment, atmospheric nuclear weapons testing, the operation of nuclear fuel cycle installations, and accidents. Many individuals are exposed from time to time to medical radiation examinations or treatments. Certain individuals are also exposed to radiation in their work.

Natural sources

The world average annual effective dose in areas of normal background is about 2.2 mSv (range 1 - 10 mSv), of which 40% is external exposure and 60% internal exposure (Table 1). About half of this exposure comes from radon and its decay products. The dose from external irradiation is about 0.9 mSv/year, and comes approximately equally from cosmic radiation and terrestrial sources. Ingestion of natural radionuclides in foods and drinking water accounts for about 0.3 mSv/year. Variations about the mean values by factors of 5-10 are not unusual for many components of exposure. The greatest variation occurs for indoor radon concentrations, which span more than four orders of magnitude.

Table 1. **Worldwide average annual effective doses from natural radiation sources.**

Source	Annual effective dose (mSv/year)	Typical range (mSv/year)
External exposure		
Cosmic rays	0.4	0.3-1.0
Terrestrial gamma rays	0.5	0.3-0.6
Internal exposure		
Inhalation (mainly radon)	1.0	0.2-10
Ingestion	0.3	0.2-0.8
Total	**2.2**	**1-10**

Man-made radiation

The testing of nuclear weapons in the atmosphere has caused the greatest man-made release of radioactive materials to the environment. Most of the testing occurred in 1952- 1962. In recent years, additional information on the atmospheric tests has been published. The maximum annual dose occurred in 1963 (world average, 0.15 mSv). No atmospheric test has occurred since 1980 and the annual doses have decreased to small fractions of the natural background. The present world average dose from atmospheric testing is about 0.005 mSv/year. Some individuals living locally have received higher exposures from atmospheric nuclear tests, e.g. residents of the Marshall Islands exposed in 1954 or around the Semipalatinsk test site in Kazakstan. These exposures are of concern to the individuals involved, but they have contributed little to the world average doses. Notable exposure of local residents have occurred near installations producing nuclear weapons, particularly from the Hanford plant in the United States and from the Chelyabinsk plant in the former USSR.

At present, 17% of the world's electrical energy is generated by nuclear fission. Over the 44-year period that nuclear power has been produced, there have been decreasing trends in normalized releases of radionuclides from such installations, mainly due to improved operating practices and to newer plants coming into operation. The doses to individuals living near the installations are low, and estimated to be of the order of 0.001 mSv/year. Some released radionuclides have long half-lives (e.g. ^{14}C) or are released over long periods (radon from mill tailings). Such long-term exposures involve dose rates to individuals that are far below the natural radiation background level. Accidents at nuclear installations could result in greater exposures to affected populations. The releases from the accident at the Three Mile Island reactor in the United States in 1979 were small due to the containment building. The releases from the Chernobyl accident were extensive, and the dispersed radionuclides could be measured over a large part of the Northern Hemisphere. In the first year after the Chernobyl accident, doses of 0.3-0.8 mSv were estimated in countries of Eastern and Central Europe.

Medical radiation

The use of x-rays and radiopharmaceuticals for diagnostic and therapeutic purposes is common and over 90% of the total dose come from x-ray examinations. Most of the equipment and the procedures performed are found in industrialized countries, having 25% of the world's population. In industrialized countries, the average annual effective dose is 1.1 mSv from all diagnostic examinations, and the world average is 0.3 mSv. At the highest level of health care (defined as less than 1000 people per physician), the average annual effective dose is 1 mSv/year and 0.04 mSv/year at the lowest level (more than 10 000 people per physician). Much, and optimally most, of the doses from medical uses of radiation are offset by direct benefits to the exposed patients.

Occupational radiation

The average annual doses to workers have been declining over the past 20 years. At present the effective dose to workers exposed to man-made sources is 1.1 mSv/year (Table 2). The doses to workers exposed to natural sources of radiation have been less thoroughly studied and are estimated at 1.7 mSv/year. Further attention should be given to evaluation of occupational exposures arising from natural sources. The individual doses from natural sources are more uncertain than the doses from man-made sources. The largest component of occupational exposures from natural sources comes from underground mining of coal and other minerals.

Table 2. Number of monitored workers and occupational radiation exposures in relation to type of occupation.

Source	Number of monitored workers (10^3)	Average effective dose (mSv/year)
Man-made sources		
Nuclear fuel cycle	900	2.9
Industrial uses of radiation	600	0.9
Defense activities	400	0.7
Medical uses of radiation	2 200	0.5
Total	**4 100**	**1.1**
Natural sources:		
Mining (other than coal)	700	6
Aircrew	250	3
Coal mining	3 900	0.9
Other	~300	<1
Total	**5 200**	**1.7**

The annual effective doses received by the world population at present are presented in Table 3. These are the annual doses averaged over the world population and not necessarily the doses that any one individual would experience. Doses to individuals differ because of considerable variations in exposures, depending on location, personal habits etc.

Table 3. World average effective doses at present from natural and man-made radiation sources.

Source	World average effective dose (mSv/year)	*Comments*
Natural background	2.2	Typical range 1-10 mSv/year.
Diagnostic medical examinations	0.3	Range 0.04-1.0 mSv/year at lowest and highest levels of health care.
Atmospheric nuclear testing	0.005	Maximum of 0.15 mSv in 1963.
Chernobyl accident	0.002	Maximum of 0.04 mSv in 1986.
Nuclear power production	0.001	Increasing with expansion of program.

BIOLOGICAL EFFECTS OF RADIATION

Over the 45 years that UNSCEAR has been in existence, an improved understanding of the biological effects of radiation has been achieved. Information needed to evaluate the risks of biological effects following radiation exposure comes direct studies of the effects of radiation on humans (epidemiology), studies of animals and plants (experimental radiobiology), and fundamental studies of cells and their components (cellular and molecular biology). The key to understanding the health effects of radiation is the interaction between these sources of information. In its present report, the Committee's main emphasis is on carcinogenesis and hereditary effects.. The Committee has also given special attention to the consequences of the Chernobyl accident that occurred in 1986.

CARCINOGENESIS

Cancers induced by radiation are similar to those that are caused by many other agents. The effects can therefore only be demonstrated as a statistical excess of cancers in the exposed population. UNSCEAR has reviewed epidemiological studies of cancer mortality and incidence up to the 1990s among the A-bomb survivors in Japan, patients exposed to radiation for diagnostic or therapeutic purposes, workers exposed to radiation and individuals exposed to environmental radiation.

The Life Span Study (LSS) cohort of the A-bomb survivors continues to be a primary source of epidemiological data on radiation effects, including individuals of both sexes and all ages with good dosimetric data over a wide range of doses. Among the about 86 500 individuals in the LSS cohort, there were about 7 800 deaths from malignant diseases between 1950 and1990. Of these, about 5% can be attributed to radiation exposure. The cancer incidence and cancer mortality data are broadly similar, both demonstrating statistically significant effects for all solid tumors as a group, as well as for cancers of the stomach, colon, liver, lung, breast, ovary, and bladder. The incidence data also show excess radiation risks for thyroid cancer and non-melanoma skin cancers. Statistically significant excess risks have not been observed in either the incidence or the mortality data for cancers of the rectum, gallbladder, pancreas, larynx, uterine cervix, uterine corpus, prostate, kidney or renal pelvis. An association with radiation exposure is noted for several types of leukemia but not for lymphoma or multiple myeloma.

The numbers of solid tumors associated with radiation exposure are not sufficient to permit detailed analysis of the dose-response relationship for specific sites or types of cancer. For all solid tumors together, the slope of the dose-response curve is linear up to about 2-3 Sv. The dose-response curve for leukemia is best described by a linear-quadratic function. Statistically significant risks for solid tumors in the LSS cohort are presently seen only above 100 mSv. An inherent limitation of epidemiological studies is to quantify results at doses because of the low statistical power of the available results.

The epidemiological results may be used to infer risk estimates for radiation-induced cancer. The estimates of lifetime risk following exposure of 1 Sv, using sex- and age-at-exposure-specific relative risks estimated from the LSS mortality data and using the demographic structure for Japan, are 0.9% for leukemia and 11.2% for solid tumors. In the UNSCEAR 1994 Report (3), the estimates were 1.1% for leukemia and 10.9% for solid tumors. Alternative assumptions can be used to project risk beyond the present observation period. The relative risk can be assumed to either remain constant to the end of life or decrease to lower values at times greater than 40 years after exposure, as has been the case in some epidemiological studies. The estimate using the assumptions decreasing risk beyond 40 years after exposure is 20%-30% less than the total risk estimate quoted above, i.e. 9-10% instead of 12% for an exposure of 1 Sv.

The application of a dose-and-dose-rate-effectiveness factor of less than 3 was recommended in the UNSCEAR 1993 Report (2). If a factor of 2 is applied, as was done by ICRP (5), the risk estimate derived from the UNSCEAR 1988 Report (1) would be 5% per Sv and from the UNSCEAR 1994 Report (3) 6% per Sv for a constant relative risk projection. The alternative projection methods would yield values between 4% and 6% in the Japanese population (the applicability to other populations involves additional uncertainty). The use of a nominal value of 5% per Sv for mortality from cancer after exposure to low doses for a population of all ages, as recommended by ICRP, thus still seems valid to the Committee based on these latest analyses.

Studies of other exposed populations, e.g. cervical cancer patients, ankylosing spondylitics, and children treated for benign conditions, provide risk estimates that generally support those derived from the LSS data. Other epidemiological studies provide additional information on issues that cannot be addressed by the A-bomb survivor data, such as the effects of low chronic exposures, alpha exposures to the lung from radon, highly fractionated exposures, and variability among populations. Large studies of occupationally exposed persons are also contributing tentative risk estimates. In general, there are no great discrepancies in risk estimates between the LSS and the other studies. Valuable information on the effects of low-LET exposure of humans is available from many other epidemiological studies. Studies of persons with partial-body exposures from medical examinations or treatments provide valuable information on risks for specific cancers. In addition to individuals exposed to low-LET radiation, various groups with exposure to high-LET radiation have been studied. These exposures have occurred in occupational settings (e.g. radon in mines or plutonium in nuclear facilities), from medical interventions (e.g. injections with ^{224}Ra or Thorotrast) and environmentally (e.g. radon in homes).

Thyroid cancer is of particular importance in children, and the risk of a cancer being induced by radiation in children under 15 years is about 10 times that in adults. Children aged 0-5 years appear to be five times more sensitive than the 10-14-year-old group. In view of this sensitivity, it is not surprising that thyroid cancer has appeared in children in Belarus, the Russian Federation, and Ukraine following the Chernobyl accident in 1986.

Over the years there has been a steady increase in the number of cancers known to be induced by radiation and also for which risk estimates have been made. Most tissues or organs in the body can be the site of an induced cancer, although some sites are more likely than others. In a few tissues, no radiation-induced cancers have been identified. It is also known that there are physiological modifying factors such as sex and age. Differences in the absolute risk of tumor induction with sex are not large and vary with the site, but for most solid cancers absolute risk is higher in females than in males. People young at the time of exposure usually have higher relative and absolute risks than older people.

The epidemiological evidence on radiation-induced cancer has been derived from exposures that were in the high-dose region and at doses down to about 100 mSv. Below this dose, the epidemiological evidence of the effects is weakened by the statistical limitations. For all cancers, the data on the survivors of the atomic bombings appear to be linear and demonstrate effects down to about 100-200 mSv. Some worker studies with average doses of the order of 30 mSv indicate a possible risk for leukemia. The association of leukemia and cancer in children after prenatal x-ray exposure has been noted for minimum doses of about 10 mGy. Despite these findings, there is still controversy at doses below 100 mSv. Mechanistic considerations of radiation response must therefore be relied on for risk evaluation at lower doses.

Cancer induction following radiation exposure has also been extensively studied in animals. Dose-response relationships have been determined in relation to the effect of radiations of different quality (RBE) and dose rate, and age, strain and species. The data are not always directly relevant, but some general observations may be made. For example, protraction of continuous or fractionated radiation generally reduces the carcinogenic effect. Younger animals are more sensitive to cancer induction. Differences between strain and species suggest considerable genetic control. The development of qualitative generalizations gives some hope that quantitative inferences may ultimately be possible. Some experimental studies show linear dose response and indications of effects down to 10 mGy or less.

The question of whether there might be a threshold level of exposure below which biological response does not occur has been addressed from mechanistic considerations. 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 carcinogenic response at low doses in cellular endpoints (chromosome aberrations, gene mutation, cell transformation) and the presence of spontaneous double-strand breaks in mammalian cells argue against adaptive or other processes that might provide error-free repair at some level of radiation exposure. It should be noted that all man-made radiation exposures occur above the natural radiation background, which limits the need to consider extrapolations to exceedingly low levels.

It may thus be concluded that as far as is known, even at low doses radiation may act as an initiator of neoplasia. The dose response does not appear to be a complex function of increasing dose. The simplest representation is a linear relationship, and this is consistent with most of the available quantitative data. There may be differences in response for different types of tumors, and statistical variations in each dataset are inevitable. A departure from linearity is noted for leukemia data, for which a linear-quadratic function is used. Because of the multi-step nature of the carcinogenesis 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.

HEREDITARY EFFECTS

UNSCEAR has given particular and continued attention to assessing the broad features of heritable damage after radiation exposure. It has not been possible to directly confirm radiation-induced mutations in human populations. Estimation of genetic risk therefore has to rely on knowledge of human genetics and extrapolation of results from animal experiments. There is a considerable background of spontaneous mutations that give rise to congenital defects and hereditary disease in humans, and experimental studies have demonstrated radiation-induced genetic effects in plants and animals. The basic effects of dominant and recessive mutations in the transmission of heritable disease have long been known. The understanding of human genetics at the molecular level is increasing rapidly, and more precise analysis of the type of genetic damage caused by radiation and other agents is possible with new techniques. There is a broad range of so-called multifactorial diseases, such as coronary heart disease, diabetes, and essential hypertension, which may occur throughout life and with varying severity and which may be affected to varying degrees by genetic and environmental influences. The specification of the genetic component of diseases, and especially of the many multifactorial diseases, is a difficult problem.

Specific values of parameters needed in the risk estimation are derived from both human and mouse data. The doubling dose reflects the sensitivity of genes to induced mutations. It was previously estimated to be 1.0 Gy based on spontaneous and induced mutation rates in mice. UNSCEAR has now estimated the doubling dose to be 1.5 Gy based on spontaneous rates in humans and induced rates in mice. Another variable that has been reevaluated by the Committee is the mutational component, which reflects the degree to which changes in the mutation rate modify the occurrence of the diseases. Values range from 0.3 for autosomal dominant and X-linked diseases to 0.01-0.02 for multifactorial diseases and 0 for first-generation autosomal recessive diseases. Risk estimates are derived from the background incidence of genetic diseases in human populations. Estimates of risk have now been more completely specified for the various types of genetic diseases. The estimates are lower than before for the Mendelian diseases. Abnormalities in growth and development can be expected to be the main effects of damage to the genome as a whole. Such effects, however, are not as easy to delineate as the more specific effects that are related to mutations in single genes. Although chronic, multifactorial diseases are common in the population, any change in incidence caused by a dose of 10 mGy is relatively small.

Advances in molecular genetics are contributing to a better understanding of the structural and functional changes in genes that underlie genetic diseases. Present risk estimates are consistent with available empirical data on humans, specifically the survivors of the atomic bombings, and should thus provide reasonably accurate expectations for genetic effects of radiation exposures. Hereditary effects are now known to be less important than the induced cancers, and the total heritable effect is estimated to be less than 1% per Sv, something which may be compared with 5% per Sv for all cancer.

THE CHERNOBYL ACCIDENT

The Chernobyl accident that occurred in 1986 resulted in widespread radioactive contamination in areas of Belarus, the Russian Federation, and Ukraine inhabited by several million people. The radionuclides released from the reactor that caused exposure of individuals were mainly ^{131}I, ^{134}Cs, and ^{137}Cs. Outside Belarus, the Russian Federation, and Ukraine, the countries most affected by the accident were in Eastern, Central, and Northern Europe, where exposures were at most 1 mSv in the first year after the accident and 2-5 times the first-year dose over a lifetime.

Among the residents of the local regions of Belarus, the Russian Federation, and Ukraine well over 1000 thyroid cancers have been reported in children. These cancers were most likely caused by radiation exposures received at the time of the accident. The high incidence and the short induction period have not been experienced in other exposed populations, and other factors are likely to influence the risk. If the current trend continues, more thyroid cancers can be expected to occur, especially in those exposed at young ages. The most recent findings indicate that the thyroid cancer risk for those older than 10 years of age at the time of the accident appears to be leveling off, while the increase continues for those younger than 4-5 years at the time of exposure.

Apart from the increase in thyroid cancer after childhood exposure, there has been no evidence of a major public health impact 14 years after the Chernobyl accident. No increases in overall cancer incidence or mortality have been observed that could be attributed to ionizing radiation. Risk of leukemia, which is the first malignancy to appear after radiation exposure due to its short latency time, is not elevated – not even among the recovery workers. Neither is there any scientific proof of other non-malignant disorders that are related to ionizing radiation. In addition to radiation exposure, the accident caused long-term changes in the lives of the people living in the contaminated districts, due to e.g. resettlement, changes in food supplies, and restrictions on the activities of individuals and families. These changes were later on accompanied by other economic, social, and political changes that took place when the former Soviet Union disintegrated.

For the last decade, attention has been devoted to etiological research on the association between exposure to fallout from radionuclides from the Chernobyl accident and late effects, particularly thyroid cancer, after childhood exposure. As long as individual dosimetry is not performed, it is unclear whether the effects might be radiation-related, and it is also impossible to make reliable quantitative estimates. The reconstruction of individual doses is a key element for future research on radiation-associated cancers related to the Chernobyl accident.

CONCLUSIONS

In 1995, UNSCEAR began a new program of reviewing the sources, exposure and biological effects of ionizing radiation. A comprehensive report will be published later this year. Considerable data are today available that allow detailed, quantitative analysis of the levels of exposure from the various sources and the health effects of radiation exposure. However, there are still many questions about the response at the lowest levels of exposure that must be answered in order to evaluate the risks of radiation exposures such as occur in normal circumstances at home and in the workplace. The Committee intends to pursue these challenges and to address widespread concerns in its continued review and evaluation of the sources and effects of ionizing radiation. The assessments of UNSCEAR will be useful to evaluate and compare radiation exposures from the various sources, and will provide better understanding of the mechanisms of cellular damage and the biological effects in organisms, something which will provide a sounder basis for establishing the risks of radiation exposure.

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