V.U Mme Cl. Stiévenart
Av. Armad Huysmans 206, bte 10
B-1050 Bruxelles - Brussel

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Hoofdredacteur

Mr C. Steinkuhler
Rue de la Station 15
B- 1325 Longueville

Rédacteur en chef

Redactiesecretariaat

Mme Cl. Stiévenart
Av. Armand Huysmans 206, bte 10
B- 1050 Bruxelles - Brussel

Secrétaire de Rédaction

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Ce numéro contient les textes d'exposés présentés lors de la réunion organisée par l'Association belge de Radioprotection à Bruxelles, le 8 février 2002

Dit nummer bevat de teksten van de uiteenzettingen gedaan ter gelegenheid van de vergadering van de Belgische Vereniging voor Stralingsbescherming in Brussel, op 8 februari 2002

Overview of the new UNSCEAR publications

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INTRODUCTION TO THE MEETING

J. Maisin
Unité de Radiobiologie et de Radioprotection, UCL RBNT 54/69,
Av. Hippocrate 54, 1200 Bruxelles

Dear Colleagues, dear friends,

In this short introduction, I would like to tell a few words on the history of UNSCEAR and to explain briefly to the participants, who are not members of the Belgian Delegation, how UNSCEAR is working and which topics will be developed in the next UNSCEAR report which must normally be published in the year 2004. This meeting is important to sensitize and to motivate the Belgian Scientists to help the Belgian Delegation in this work.

The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) was established by the General Assembly at its tenth session in 1955.
Its terms of reference are set out in resolution 913. Representatives of 16 countries, including France, USSR, Great Britain and the United States of America originally composed it. A few years later, four additional countries were added including Germany. Later the General Assembly increased the membership of the Committee to a maximum of 21 members and invited China to become a member.

Each member state is represented in the Committee by a representative and by scientific experts. The Belgium delegation is composed actually by myself as representative and A. Debauche, J.M. Van Dam, H. Vanmarcke, P. Smeesters and A. Wambersie as experts.
2 experts of the Netherlands, H.P. Leenhouts and J. Lembrechts, have been invited to join the Belgian delegation.

A Scientific Secretary provides the daily operation of UNSCEAR. A chairman, elected by the Committee for a period of two years, chairs the annual sessions. Since the existence of UNSCEAR, two representatives of Belgium were elected as chairman. Prof. Bacq from Liège, who has been the first chairman of the Committee, and myself, elected for the period 1991-1992.
During its annual session, the Committee is divided in two working groups: the physical and the biological subgroups, which I have the privilege since a few years, to chair.

During its existence, the Committee has assertively attempted in scientific reports to provide the best possible estimates of:

1. doses received by the world's population in the past and expected to be received in the future, from various natural and man-made sources of radiation;
2. risks of induction of various types of harm by radiation, both in the short and the long term, by individuals directly receiving such doses or by their descendants over many generations.

Since its establishment the Committee has published 12 scientific reports. The last of this series was published last year in 2 volumes, including 10 scientific annexes.

The material of these reports and the scientific annexes were developed at the annual session of the Committee, based on working papers prepared by the secretariat of UNSCEAR, that were modified and amended from one session to the next, according to the Committee's requests.

At the previous meeting of the Committee some topics have been listed:

1. the effects of low-level radiation exposure and namely the controversy of the effects of low-level radiation exposure, the mechanisms of cancer induction; the development of potential biological indicators of low-level radiation exposure of non-cancer diseases;
2. radiation consequences of military conflicts;
3. radiation in the environment (high level radioactive waste, ...);
4. lost radiation sources;
5. reporting of other sources of exposure.

Canada has already offered to help on the preparation of such basic papers and is willing to prepare thoughts on the following subjects:

- non-cancerous diseases from exposure to radiation;
- potential biological markers for identifying cancer from low-level radiation exposure;
- effects of environmental radiation exposure resulting from high-level waste.

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UNSCEAR 2000: SOURCES OF IONIZING RADIATION

H. Vanmarcke

SCK•CEN, Departement Stralingsbeschermingsonderzoek,
Boeretang 200, B-2400 Mol, België

ABSTRACT

Volume I of the UNSCEAR 2000 report consists of 5 scientific annexes dealing with radiation sources and levels of exposure:

- Annex A: Dose assessment methodologies (64 pages)
- Annex B: Exposures from natural radiation sources (74 pages)
- Annex C: Exposures to the public from man-made sources of radiation (136 pages)
- Annex D: Medical radiation exposures (204 pages)
- Annex E: Occupational radiation exposures (157 pages)

The report to the General Assembly of the United Nations and the scientific annexes are available from the UNSCEAR website: http://www.unscear.org/reports.htm The annexes contain the expected wealth of data and evaluations. For each annex, there is only time to discuss one or two striking results.

- The use of more realistic values for the atmospheric dispersion model results in lower estimates of the population exposure around nuclear installations and uranium mill tailings.
- The worldwide annual average population exposure to natural sources remains at 2.4 mSv. The population exposure in Belgium is calculated using the UNSCEAR methodologies.
- The radon dose coefficient is maintained at 9 nSv per Bq h m^{-3} (in terms of radon decay products), which is 50% higher than the value given in the new Belgian regulation that is based on ICRP 65.
- The most comprehensive assessment yet is made of the worldwide exposures to fallout from atmospheric nuclear tests.
- The average level of radiation exposure due to the medical applications in developed countries is equivalent to 50% of the global average level of natural exposure. The widespread use of CT in Belgium results in even higher values.
- The collective occupational exposure to natural sources, significantly above background levels, is higher than to man-made sources.

1. DOSE ASSESSMENT METHODOLOGIES

This annex presents and reviews the dose estimation procedures used by the Committee to assess the radiation exposure of human populations. The main features are:

- The use of transfer coefficients or equilibrium modeling. There has been little need for detailed, time-dependent dose modeling because the Committee is in most cases only interested in evaluating the average annual doses.
- Preference for simple empirical methods that are not difficult to understand and relatively easy to apply and adapt by scientists throughout the world to local circumstances.
- The starting point of the calculations is where the fewest steps or assumptions are needed, for example, the concentrations of radionuclides in the human body or in the case of atmospheric nuclear tests the measured deposition densities from fallout radionuclides.

I want to draw your attention to the atmospheric dispersion model used by UNSCEAR to evaluate radiation doses. The average air concentration close to a specific source, such as a stack of a nuclear reactor, is calculated using the long-term sector-averaged Gaussian plume model. In this model, the plume is assumed to spread uniformly across a sector subtended by an angle, usually chosen to be 30°. The variation of air concentration, C_a, with downwind distance beyond 1 km can be approximated by the following simple function, which was also used in previous UNSCEAR assessments:

$$C_a(x) = D_1 \, Q \, x^{-n}$$

where D_1 = the dilution factor at 1 km (s/m³)
Q = the release rate (Bq/s)
x = the distance from the source (km)

When site-specific data are not available, the Committee recommends to use a value of $5\ 10^{-7}$ s/m³ for the dilution factor D_1 and 1.4 for the index parameter n. For noble gases (which do not deposit) and tritium the recommended value for n is 1.2. The value for n is similar to the value of 1.5 used in previous UNSCEAR assessments. The value for the dilution factor is lower by a factor of 6 than the value of $3\ 10^{-6}$ s/m³ suggested in the UNSCEAR 1982 report. The previous value reflected concentrations at a location toward which the wind blows about 50 % of the time, whereas the currently recommended value of $5\ 10^{-7}$ s/m³ assumes a uniform wind rose at the point of release.

The lower dilution factor at 1 km decreases the collective dose estimates from atmospheric releases of nuclear reactors and uranium mill tailings. The latter decreased per unit electrical energy generated, from 150 manSv/(GWyear) in the UNSCEAR 1993 report to 7.5 manSv/(GWyear) in the 2000 report. The two main reasons are:
- a reduction in the dilution factor by a factor of 6 and;
- a reduction in the radon emission rate from abandoned uranium mill tailings by a factor of 3, because of improved decommissioning techniques.

2. EXPOSURES FROM NATURAL RADIATION SOURCES

The exposure of human beings to ionizing radiation from natural sources is a continuing and inescapable feature of life on earth. There are two main contributors to natural radiation exposures: high-energy cosmic ray particles incident on the earth's atmosphere and radioactive nuclides that originated in the earth's crust and are present everywhere in the environment, including the human body itself.

The annual effective doses to the Belgian population are calculated with the methods given in the UNSCEAR 2000 report. *(The world average values of the UNSCEAR 2000 report are given between brackets and in italics.)*

2.1. Cosmic radiation in Belgium

The earth is continually bombarded by high-energy particles that originate in outer space. These cosmic rays interact with the nuclei of atmospheric constituents, producing a cascade of interactions and secondary reaction products that contribute to cosmic ray exposures that decrease in intensity with depth in the atmosphere, from aircraft altitudes to ground level. The cosmic ray interactions also produce a number of radioactive nuclei known as comogenic radionuclides.

The external dose rate outdoors at sea level increases with geomagnetic latitude. The values for the two components of the cosmic radiation field in Belgium *(and worldwide)* are:

- photon and directly ionizing component: 32 nSv/h *(31)*;
- neutron component: 9 nSv/h *(5.5)*.

As Belgium is a low-lying country the altitude correction is small:

- photon and directly ionizing component: 1.02 *(1.25)*;
- neutron component: 1.1 *(2.5)*.

The total effective dose rate outdoors is 32 x 1.02 + 9 x 1.1 = 42.5 nSv/h *(52)*.
Applying the indoor shielding factor of 0.8 and assuming indoor occupancy to be 80 % of time or 7000 h/year the average effective dose is:
$42.5\ (1760 + 7000 \times 0.8)\ 10^{-6} = 0.31$ mSv/year *(0.38)*.

The Committee assessed the internal exposures from the four main cosmogenic radionuclides to be:

- ^{14}C: 0.012 mSv/year;
- ^{22}Na: 0.00015 mSv/year;
- ^{7}Be: 0.00003 mSv/year;
- ^{3}H: 0.00001 mSv/year.

The activity of cosmogenic ^{14}C in the environment, and consequently also in the human body is 230 Bq/kg of carbon.

Including a small contribution from air travel and holidays (for instance winter sports) the average exposure to cosmic radiation in Belgium can be estimated at:
0.31 + 0.012 + air travel and holidays = **0.35 mSv/year** *(0.4)*.

2.2. External terrestrial radiation in Belgium

External exposures arise from terrestrial radionuclides present at trace levels in soil and building materials. Only those radionuclides with half-lives comparable to the age of the earth, and their decay products, exist in significant quantities in these materials. Irradiation is mainly by gamma radiation from radionuclides in the ^{238}U and ^{232}Th series and from ^{40}K.

Hundreds of soil samples from all over Belgium were measured in the eighties by SCK and WIV (Gillard et al., 1988). The average values of the spectrometric analyses of the soil samples, the dose conversion coefficients from the UNSCEAR 2000 report and the resulting absorbed dose rates in air are given in table 1.

Table 1. External exposure rates calculated from the average radionuclide concentrations in soil in Belgium *(and worldwide)*

	Concentration in soil Bq/kg	Dose coefficient nGy/h / (Bq/kg)	Absorbed dose rate nGy/h
^{40}K	380 *(420)*	0.0417	16 *(18)*
^{226}Ra (^{238}U)	26 *(33)*	0.462	12 *(15)*
^{232}Th	27 *(45)*	0.604	16 *(27)*
Total absorbed dose rate outdoors from soil measurements:			44 *(60)*

The three components of the external radiation field make approximately equal contributions to the gamma radiation dose. At the same locations where the soil samples were taken direct measurements of absorbed dose rates in air were carried out. Excluding cosmic ray exposure, an average value of 43 nGy/h *(59)* was found, which is close to the value inferred from the soil concentration results.

Hundreds of absorbed dose rate measurements in air inside dwellings were performed in the same study (Gillard et al., 1988). A somewhat higher average value of 60 nGy/h *(84)* was found, because of the change in source geometry from half-space to a more surrounding configuration indoors.

To estimate annual effective doses, account must be taken of the conversion coefficient from absorbed dose in air to effective dose. Gamma radiation is less absorbed in children and infants resulting in a higher dose conversion coefficient (adults: 0.7, children: 0.8 and infants: 0.9). The annual average effective dose for adults assuming an occupancy factor indoors of 0.8 is:

- Indoors: $60 \times 7000 \times 0.7 \times 10^{-6}$ = 0.30 mSv *(0.41)*
- Outdoors: $43 \times 1760 \times 0.7 \times 10^{-6}$ = 0.05 mSv *(0.07)*

Total = 0.35 mSv *(0.48)*

The values for children and infants are in direct proportion to the increase in the dose conversion coefficient from absorbed dose in air to effective dose:

- Children: 0.40 mSv/year (0.55)
- Infants: 0.45 mSv/year (0.62)

The resulting average effective dose for the whole population from external terrestrial radiation in Belgium is **0.4 mSv/year** *(0.5)*.

2.3. Internal exposures other than radon

Ingestion is the main exposure pathway of the population with significant contributions from ^{40}K and from the ^{238}U and ^{232}Th decay series.

Potassium is more or less uniformly distributed in the body following intake in foods, and its concentration is under homeostatic control:

- Adults: 55 Bq/kg ⇒ 0.165 mSv/year
- Children: 61 Bq/kg ⇒ 0.185 mSv/year

The resulting annual effective dose for the whole population is 0.17 mSv.

There are no control mechanisms to keep the concentration of the uranium- and thorium-series radionuclides in the body at a fixed level, so that the doses are dependent on the intake. The main contributor to this dose is polonium-210. UNSCEAR estimates the effective doses from the ingestion of uranium- and thorium-series radionuclides at:

- Adults: 0.11 mSv/year (^{210}Po contribution = 0.07 mSv/year)
- Children: 0.20 mSv/year (^{210}Po contribution = 0.10 mSv/year)
- Infants: 0.26 mSv/year (^{210}Po contribution = 0.18 mSv/year)

The total effective dose from internal exposures other than radon is assessed at **0.3 mSv/year**.

2.4. Radon (^{222}Rn) and thoron (^{220}Rn) exposure in Belgium

The main contribution to the exposure of the population from natural radiation sources comes from the inhalation of the short-lived radon decay products.

Concentrations of radon in the outdoor environment are affected by the exhalation rates of the soil in the general area and by atmospheric mixing phenomena. Results of radon measurements in thermometer shelters in Belgium gave an average value of 10 Bq/m³ *(10)* (Poffijn, 2001). The radon concentrations indoors are somewhat higher and tend to be log-normally distributed. The average concentration in Belgium is estimated at 48 Bq/m³ *(40)* with a geometric mean of 38 Bq/m³ *(30)* and a geometric standard deviation of 2.0 *(2.3)* (Poffijn et al., 1991). The highest values, up to several thousands of Bq/m³, are found in the Ardennes.

Direct measurements of the concentrations of all short-lived decay products of radon are difficult and limited. They are estimated from considerations of equilibrium (or disequilibrium) between radon and its decay products. An equilibrium factor F is defined that permits the exposure to be estimated from the measurement of the radon gas concentration. The equilibrium factor is the ratio of the Equilibrium Equivalent radon Concentration (C_{EEC}) to the radon concentration (C_{Rn}). The equilibrium equivalent radon concentration is directly proportional to the Potential Alpha Energy Concentration (PAEC) in the following manner:

1 Bq/m³ (EEC) = 5.56 10^{-6} mJ/m³ (PAEC) = 0.27 mWL (Working Level)

$F = C_{EEC}/C_{Rn}$ with $C_{EEC} = 0.105\ C_{218Po} + 0.515\ C_{214Pb} + 0.380\ C_{214Bi}$

where C_{218Po}, C_{214Pb} and C_{214Bi} are the concentrations of the short-lived decay products in air.

The Committee suggests a rounded value for the equilibrium factor of 0.6 for the outdoor environment and 0.4 indoors.

There is no consensus in the scientific community on the value of the dose conversion factor for radon. The epidemiologically based conversion factor of ICRP 65 (1993) is derived from the risk estimate of the superseded BEIR IV report of 1988. The more recent BEIR VI report (1998) suggests an increased risk per unit radon exposure. As the dosimetric evaluation using the ICRP lung model (ICRP 66, 1994) also shows higher values, the UNSCEAR Committee decided to keep its previous value of 3.6 (nSv/h)/(Bq/m³) (= 9 EEC x 0.4 equilibrium factor).
Note that the UNSCEAR dose conversion factor for radon at home is 50 % higher than the value given in the new Belgian regulation that is based on ICRP 65 (ARBIS, 2001):

- *radon at home: 1.1 Sv per J h/m³, which is equivalent to 2.4 (nSv/h)/(Bq/m³);*
- *radon at work: 1.4 Sv per J h/m³, which is equivalent to 3.1 (nSv/h)/(Bq/m³).*

For the representative concentrations of radon, equilibrium and occupancy factors and the dose coefficient in terms of EEC, the following annual effective doses are derived:

- Indoors: $48 \times 0.4 \times 9 \times 7000 \times 10^{-6}$ = 1.2 mSv/year *(1.0)*
- Outdoors: $10 \times 0.6 \times 9 \times 1760 \times 10^{-6}$ = 0.1 mSv/year *(0.1)*

Total = 1.3 mSv/year *(1.1)*

For completeness, the contribution from a minor pathway of exposure to radon can be added, namely dissolution of radon gas in blood with distribution throughout the body. The dose estimate for the representative concentrations of radon in Belgium with the method given in the UNSCEAR report is 0.06 mSv/year *(0.05)*.

The much shorter half-life of thoron (55.6 seconds) compared to radon (3.82 days) limits the thoron exhalation of soil and building materials and thereby the contribution of thoron to the radiation exposure of the population. UNSCEAR estimates the average concentration of thoron outdoors at 10 Bq/m³ and approximately the same indoors. It is not possible to use the concentration of the thoron gas in dose evaluation, since the concentration is strongly dependent on the distance from the source. Starting with the estimated equilibrium equivalent concentrations of thoron indoors of 0.3 Bq/m³ and outdoors of 0.1 Bq/m³ and a dose conversion factor of 40 (nSv/h)/(Bq/m³), the annual effective dose may be derived as follows:

- Indoors: $0.3 \times 40 \times 7000 \times 10^{-6}$ = 0.084 mSv/year
- Outdoors: $0.1 \times 40 \times 1760 \times 10^{-6}$ = 0.007 mSv/year

Total (rounded off) = 0.1 mSv/year (including a minor contribution from thoron gas dissolved in blood)

Note that the UNSCEAR dose conversion factor of 40 (nSv/h)/(Bq/m³) is close to the value in the new Belgian regulation for thoron at work (ARBIS, 2001):
0.5 Sv per J h/m³, which is equivalent to 37.5 (nSv/h)/(Bq/m³).

The average exposure to radon, thoron and their short-lived decay products in Belgium is:
1.3 (radon in air) + 0.06 (radon in blood) + 0.1 (thoron) = **1.45 mSv/year** (rounded off) *(1.2)*.

2.5. Average radiation dose from natural radiation sources in Belgium

Table 2. Average exposure to natural sources in Belgium *(and worldwide)*

Source of exposure	Average annual effective dose mSv	Elevated (*) mSv
Cosmic radiation	0.35 *(0.4)*	2.0
External terrestrial radiation	0.4 *(0.5)*	4.3
Radon and thoron	1.45 *(1.2)*	10
Internal exposures other than radon and thoron	0.3 *(0.3)*	0.6
Total	**2.5 *(2.4)***	

(*) Representative of large regions (UNSCEAR, 1993)

Using the UNSCEAR methodologies, the average annual effective dose to the Belgian population from natural radiation sources is approximately 2.5 mSv/year *(2.4)*. The various components are summarized table 2.

3. EXPOSURES TO THE PUBLIC FROM MAN-MADE SOURCES OF RADIATION

This annex reviews the exposures of human populations resulting from releases to the environment of radioactive materials from man-made sources. I would like to draw your attention to the following two topics: the collective dose from the operation of nuclear fuel cycle installations and the world-wide exposure from the fallout of atmospheric nuclear tests.

3.1. Nuclear fuel cycle

Table 3. Normalized collective effective dose to members of the public from radionuclides released in effluents from the nuclear fuel cycle for the period 1995-1997

Source	Normalized collective effective dose manSv/GWyear
Local and regional component	
Mining	0.19
Milling	0.008
Mine and mill tailings (releases over five years)	0.04
Fuel fabrication	0.003
Reactor operation	
Atmospheric	0.4
Aquatic	0.04
Reprocessing	
Atmospheric	0.04
Aquatic	0.09
Transportation	< 0.1
Total (rounded)	**0.9**
Solid waste disposal and global component	
Mine and mill tailings (releases of radon over 10 000 years)	7.5
Reactor operation	
Low-level waste disposal	0.00005
Intermediate-level waste disposal	0.5
Reprocessing solid waste disposal	0.05
Globally dispersed radionuclides (truncated to 10 000 years)	40
Total (rounded)	**50**

The generation of electrical energy by nuclear power reactors is the most important industrial application of ionizing radiation. In 2000, 57.1 % of the electrical energy in Belgium has been generated by this means. During routine operation of nuclear installations, the releases of radionuclides are low, and exposures must be estimated with environmental transfer models. The collective doses for all fuel cycle operations are summarized in table 3. The estimate for the local and regional collective dose is 0.9 manSv/GWyear. The largest part of this dose is received within a limited number of years after the releases and is mainly due to the normal operation of nuclear reactors and mining operations.

The global dose, which is estimated for 10 000 years, amounts to 50 manSv/GWyear assuming a world population of 10 billion people. The main contribution is from globally dispersed ^{14}C (reactor operation and reprocessing). The collective dose from ^{14}C is delivered over a very long period and

to the entire world population. The individual doses are small compared to the natural background radiation. A continuing practice of 250 GW electrical energy generation each year into the future, as at present, would result in a maximum dose rate of 1 µSv/year. A limited practice of nuclear power generation would result in progressively less annual dose, e.g. a 100 or 200 year practice would cause 0.1 or 0.16 µSv/year respectively.

The release of radon from uranium mill tailings is a source of exposure for the surrounding population. The global dose from these releases over 10 000 years is estimated to be 7.5 manSv/GWyear. As discussed in the annex on dose assessment methodologies, the various revisions in the parameters have led to a considerable reduction from the previously estimated value of 150 manSv/GWyear (UNSCEAR, 2000).

3.2. Fallout from nuclear weapons testing

The testing of nuclear weapons in the atmosphere, which took place from 1945 until 1980, involved unrestrained releases of radioactive materials directly to the environment and caused the largest collective dose thus far from man-made sources of radiation. The annual number of atmospheric and underground tests by all countries is summarized in figure 1.

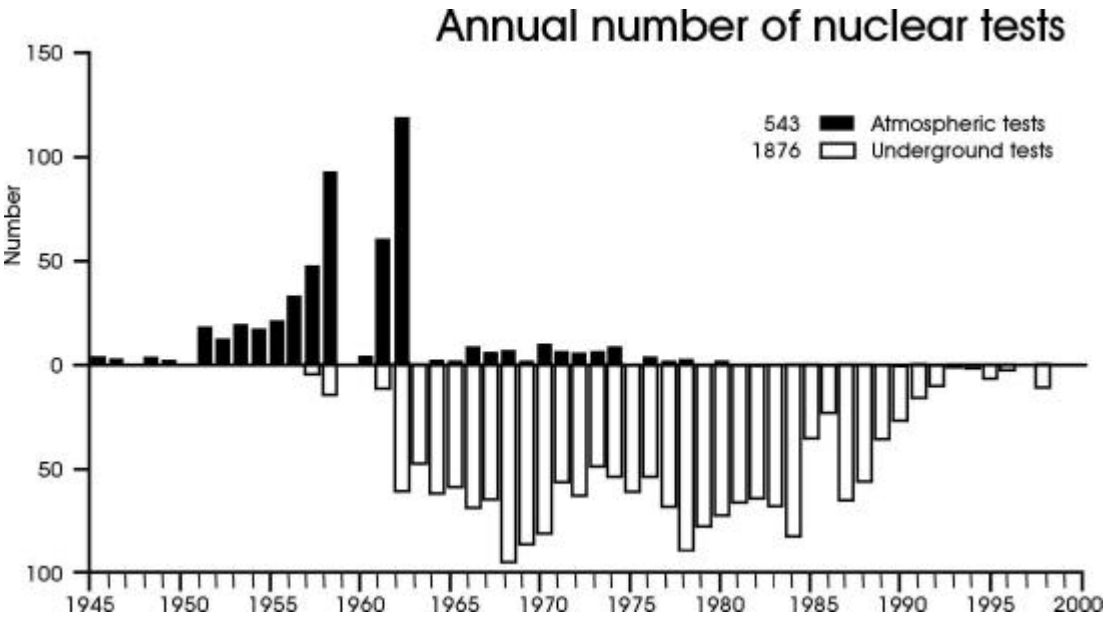

Figure 1. Annual number of tests of nuclear weapons in the atmosphere and underground

Many nuclear weapons were developed and tested during the cold war. A total number of 543 atmospheric tests were conducted by the United States, the Soviet Union and to a lesser extend by France, the United Kingdom and China. The United States detonated 3 nuclear bombs in 1945: a test conducted in the desert of New Mexico followed by combat use destroying the Japanese cities of Hiroshima and Nagasaki.

Underground testing caused exposures beyond the test sites only if radioactive gases leaked or were vented. Following the limited nuclear-test-ban treaty of 1963 between the United States and the former Soviet Union, which banned atmospheric tests, both countries conducted extensive underground test programs until the early 1990s. The underground test programs of France and China continued until 1996. India conducted a single underground test in 1974 and five further tests in

1998. Pakistan reacted some weeks later by conducting six tests. 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 that was formulated in 1996, has not yet come into force. India and Pakistan but also Israel have not yet ratified the treaty, thus it cannot yet be stated that the practice of nuclear weapons testing has ceased.

The annual fission and fusion yields are summarized in figure 2. The total yield was 440 megatons of TNT equivalent (a chemical explosive). The most active years of testing from the standpoint of the total explosive yields were 1962, 1961, 1958 and 1954. The largest test, a 50 Mt hydrogen bomb, conducted by the former Soviet Union in 1961, was reported to have a fission yield of 3 % and a fusion yield of 97 %. The atomic bombs destroying the Japanese cities of Hiroshima and Nagasaki were relatively small nuclear weapons of 15 kt and 21 kt respectively. Most underground tests had a much lower yield than atmospheric tests and it was usually possible to contain the debris.

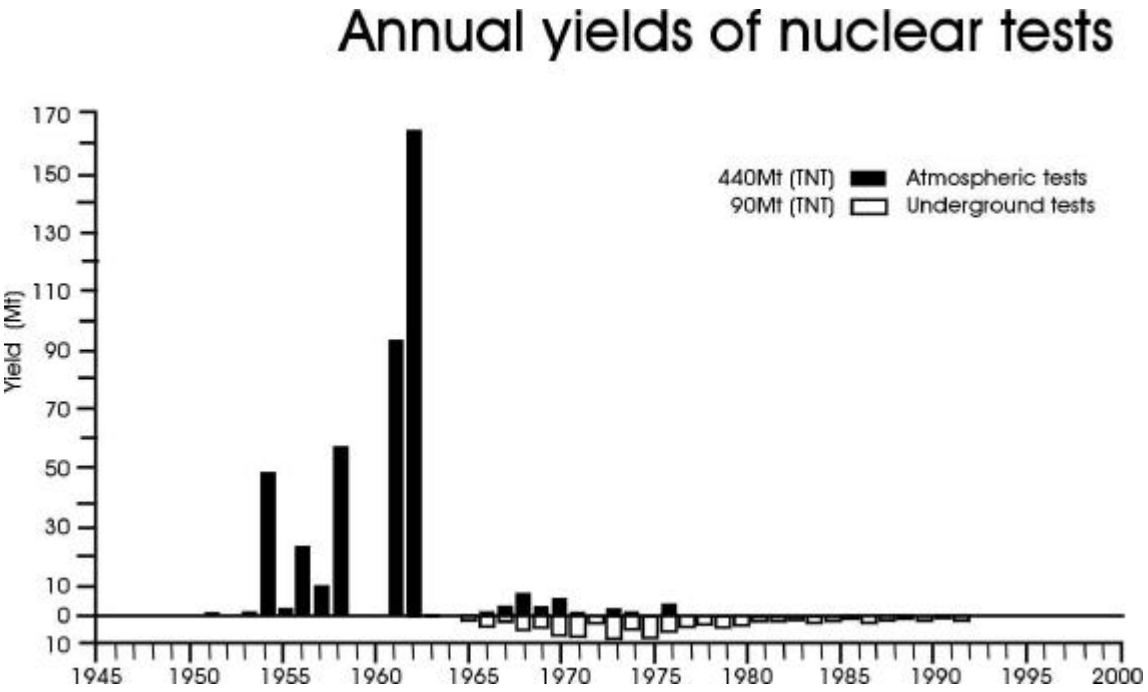

Figure 2. Annual yields of tests of nuclear weapons in the atmosphere and underground

The estimated dose from atmospheric nuclear testing was highest in 1962 and 1963 with a world-wide average exposure of 0.11 mSv/year, which is about 5 % of the background level from natural radiation sources. The doses have since decreased to about 0.005 mSv/year, from residual levels in the environment, mainly of ^{137}Cs, ^{90}Sr and ^{14}C. The cesium-137 and strontium-90 contamination of milk from a farm in Dessel (province of Antwerp) from 1963 through 1990 is illustrated in figures 3 and 4 (Vandecasteele et al., 1997). Both figures show a peak in the 1960s due to the rivalry between the United States and the Soviet Union to detonate the most powerful nuclear weapons. The cesium contamination shows another peak in 1986 from the accident with the Chernobyl nuclear power plant. This peak is absent in the strontium figure because of the small contribution of strontium-90 in the source term of the Chernobyl accident.

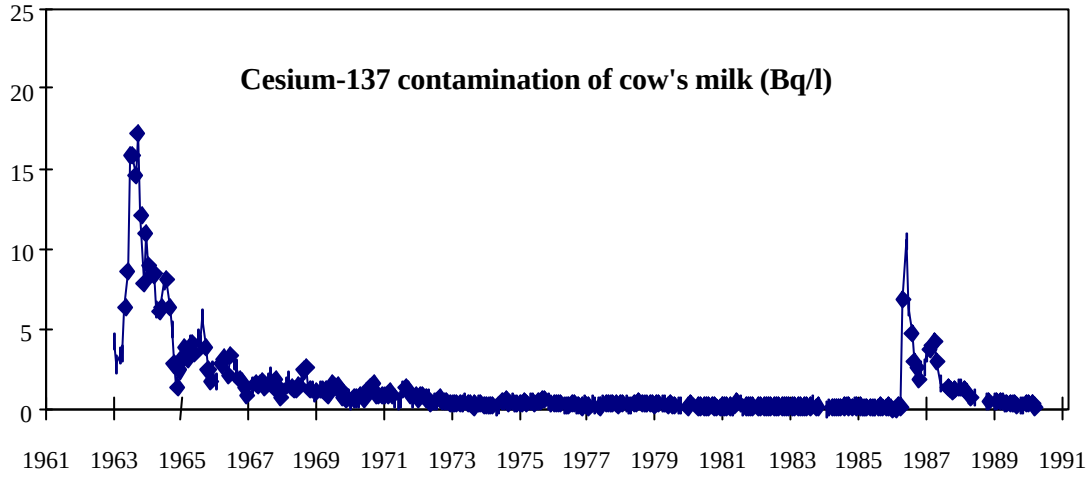

Figure 3. The cesium-137 contamination of milk from a farm in Dessel. For comparison, the potassium-40 content of milk is about 45 Bq/l.

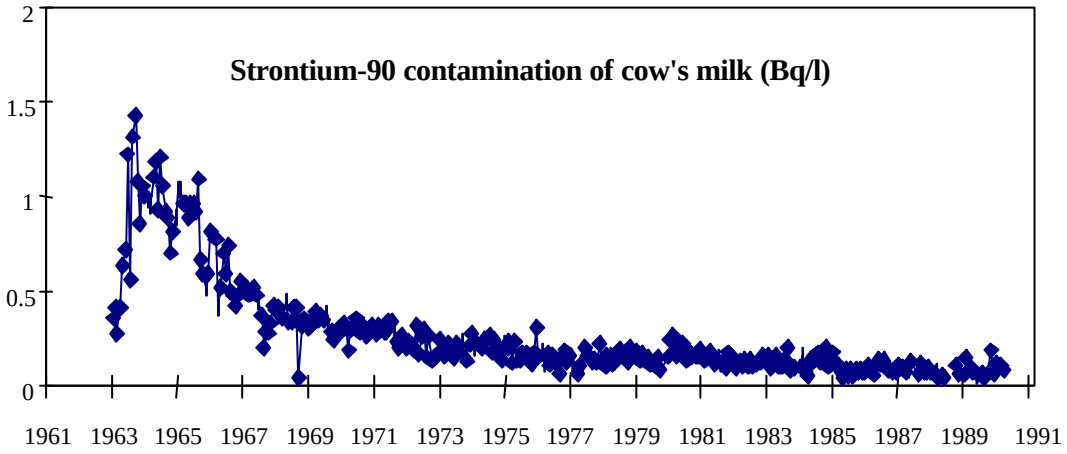

Figure 4. The strontium-90 contamination of milk from a farm in Dessel

The transfer to man of radioactive material dispersed in the environment is illustrated in figure 5. The contamination by cesium-137 in adults in the Mol-Dessel area is shown from 1959 through 1996. The decrease after the limited nuclear-test-ban treaty of 1963 is faster than the physical half-life of cesium-137 (30 years), but slower than the biological half-life of cesium in the human body (about 110 days). The contamination of the food chain decreases because the deposited cesium becomes more and more attached to the soil. The whole body contamination after the Chernobyl accident was 4 times less than in the 1960s.

Also shown in figure 5 is the aerosol activity in Mol from 1957 through 1996. The average value during the Chernobyl accident was 4 times higher than at the height of the atmospheric testing in the 1960s but the increase lasted only one month so that less cesium came available for transfer to man.

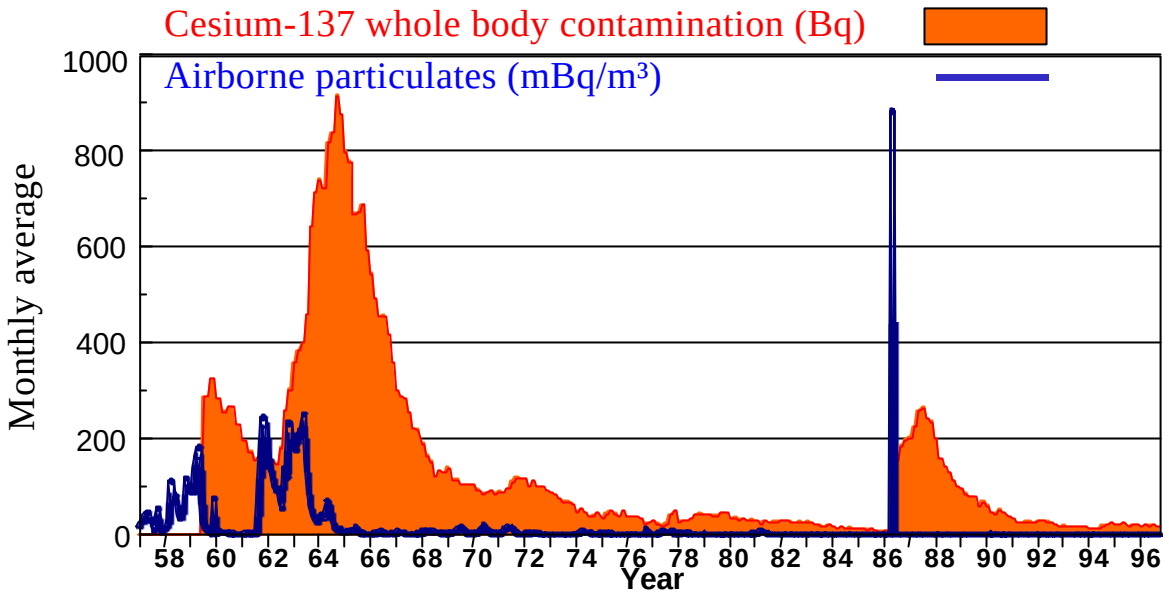

Figure 5. The colored area is the cesium-137 whole body contamination in the Mol-Dessel region. The results are normalized for a body weight of 70 kg (Genicot et al., 2001).
The line represents the monthly average activity of airborne particulates in Mol in mBq/m³

4. MEDICAL RADIATION EXPOSURES

Over the last 100 years, ionizing radiation has been increasingly applied in medicine and is now firmly established as an essential tool for diagnosis and therapy. The overwhelming benefits accruing to patients from properly conducted procedures have fostered the widespread practice of medical radiology, with the result that medical radiation exposures have become an important component of the total radiation exposure of populations. In Belgium, like in most developed countries with an advanced health care system, medical exposures are now the most important single source of ionizing radiation. Recent Flemish data collected for the yearly report on the environment and nature in Flanders (Vanmarcke et al., 2001 (MIRA report)) will be given and compared to the world average values of the UNSCEAR 2000 report *(between brackets and in italics)*.

The utilization of x-rays for diagnosis is the most widespread medical application. According to social security data (RIZIV) the average Fleming undergoes 1.2 examinations a year (excluding dental x-rays). Differences in the patterns of practice from 1990 through 1999 are shown in figure 6. Most notably, increases in the relative number of examinations are apparent from CT (computed tomography) and mammography, while the number of examinations of chest and extremities (limbs and joints) remained constant at a high level.

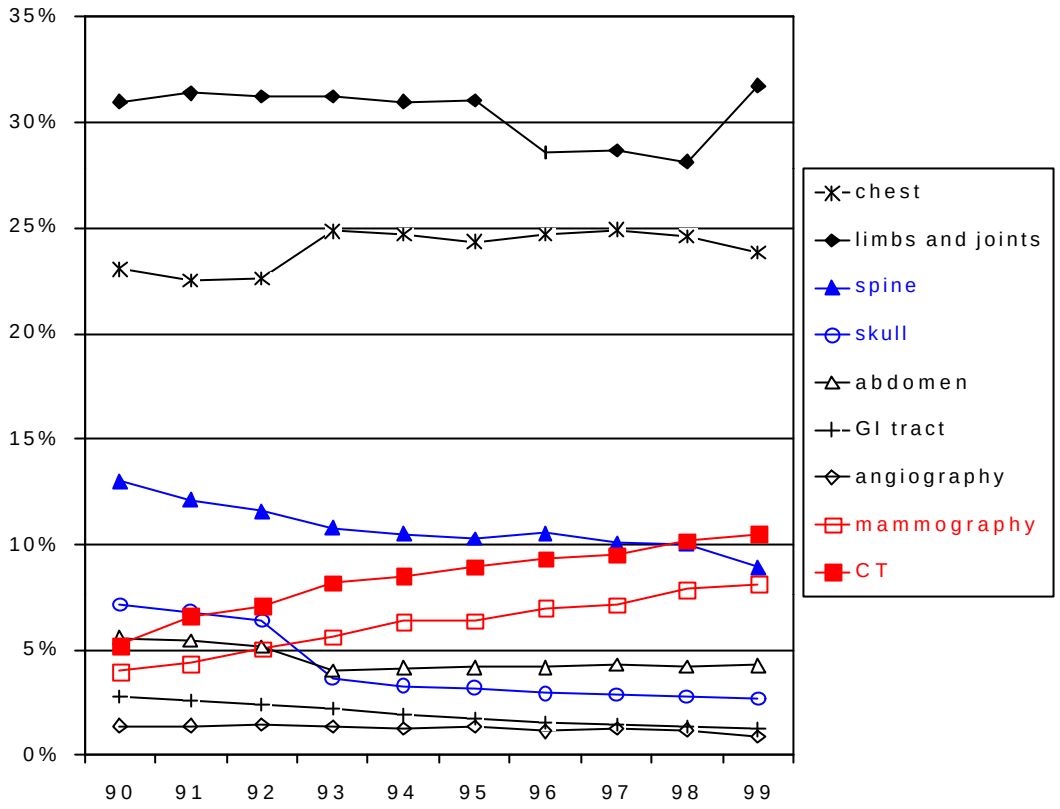

Figure 6. Trends in diagnostic radiology practice in Flanders

The average effective dose per type of examination is compared in table 4 from three different sources. The values of the UNSCEAR 1993 report have been adapted in the UNSCEAR 2000 report to the continuing developments in medical imaging. The results of a recent study in 20 Flemish hospitals for 5 important types of examinations, including CT, are in line with the values of the UNSCEAR 2000 report (Mol, 2001). Relatively high levels of patient doses are received with CT, GI tract, angiography and spine, while the doses from chest examinations and extremities are low.

Multiplying the RIZIV-data on the number of examinations with the effective dose per examination gives the dose distribution shown in figure 7. The dosimetric data from the UNSCEAR 2000 report was used when no local data was available (Mol, 2001). The population exposure is dominated by CT, which provides 54 % of the annual effective dose. With 123 CT-scans per year per 1000 population and an average dose of 7.7 mSv per examination, the average contribution from CT amounts to 0.95 mSv/year.

Table 4. Comparison of patient doses from diagnostic x-ray examinations (in mSv effective dose per examination)

Type of examination	UNSCEAR 1993	UNSCEAR 2000	Mol 2001
Chest	0.14	0.14	0.15
Limbs and joints	0.06	0.06	-
Spine	1.7	1.8	1.7
Pelvis and hips	1.2	0.83	-
Head	0.16	0.07	-
Abdomen	1.1	0.53	0.92
GI tract	5.7	5.0	-
Cholesystography	1.5	2.3	-
Urography	3.1	3.7	7.9
Angiography	6.8	12	-
PTCA	-	22	-
Mammography	1	0.51	-
CT	4.1	8.8	7.7

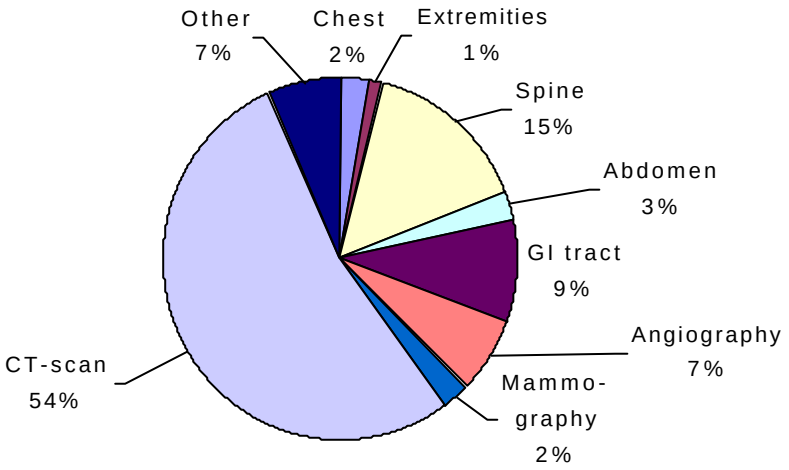

Figure 7. Dose distribution from diagnostic x-ray examinations in Flanders in 1999

Excluding dental x-rays, the Flemish population undergoes on average 1200 diagnostic x-ray examinations per 1000 population per year *(920)* resulting in an average effective dose of 1.78 mSv/year. *(The UNSCEAR estimate for countries with an advanced health care system is 1.2 mSv/year.)* The high value for Flanders comes from a higher number of examinations and in that a larger share of CT.

The number of diagnostic administrations of radiopharmaceuticals to patients, broadly referred to as nuclear medicine, in Flanders was 42 *(19)* per 1000 population per year in 1999. UNSCEAR estimates the mean effective dose per nuclear medicine procedure in countries with an advanced health

care system at 4.3 mSv. Multiplying the two numbers results in an average dose of 0.18 mSv/year *(0.08)*.

Adding the contributions from radiology and nuclear medicine leads up to an average medical exposure in Flanders of **1.95 mSv/year** (rounded off) *(1.3)*. The medical practice in Brussels and in the Walloon provinces is quite similar so that the Flemish results can be extrapolated to the whole of Belgium.

5. OCCUPATIONAL RADIATION EXPOSURES

There is a wide variety of situations in which people at work are exposed to man-made sources of radiation, such as nuclear installations or medical clinics, and some workers are exposed to enhanced levels of natural radiation. For this annex, I want to call your attention to the data on exposures of workers in nuclear power plants and to give an overview of all occupational exposures from man-made and natural sources of ionizing radiation.

5.1. Reactor operation

The types of reactor used for electrical energy generation are characterized by their coolant system and moderator: light-water-moderated and -cooled pressurized or boiling water reactors (PWRs, BWRs), heavy-water-moderated and -cooled reactors (HWRs) and gas-cooled, graphite-moderated reactors (GCRs) in which the gas coolant, either carbon dioxide or helium, flows through a solid graphite moderator. These are all thermal reactors in which the moderator material is used to slow down fast fission neutrons to thermal energies. The collective doses of the main reactor types are summarized in figure 8. The data have been averaged over five-year periods and expressed per unit electrical energy generated. The collective doses have decreased by a factor of 3 over a period of 15 years.

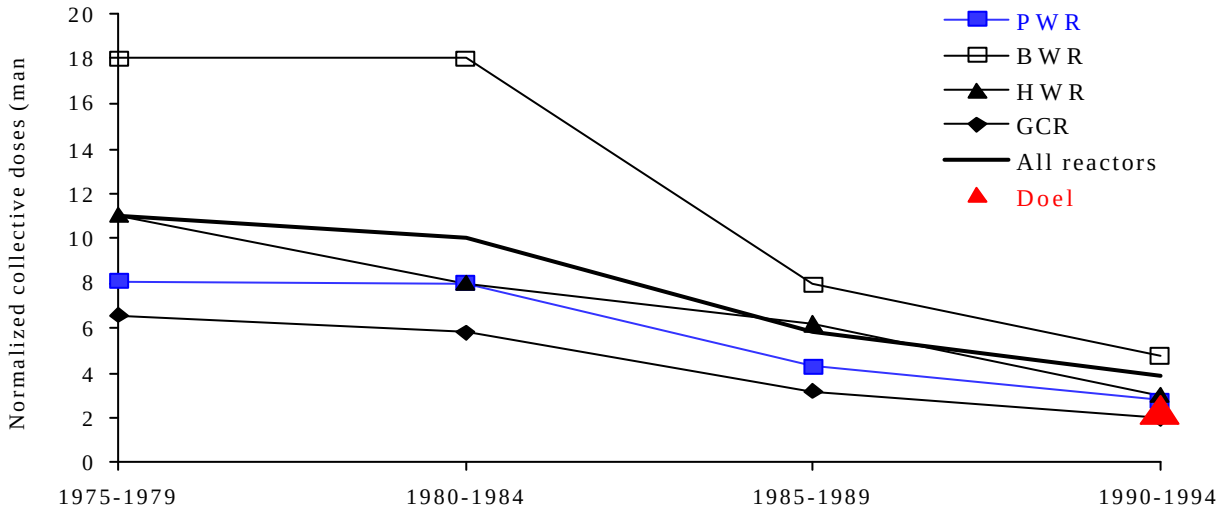

Figure 8. Trends in occupational radiation exposures in nuclear power plants. The collective dose at Doel is given for 1990 - 1994

Table 5. Worldwide occupational exposures for 1990 - 1994

Practice	Number of monitored workers thousands	Average collective effective dose manSv/year	Average effective dose mSv/year
Man-made			
Nuclear fuel cycle			
Mining	69	310	4.5
Milling	6	20	3.3
Enrichment	13	1	0.12
Fuel fabrication	21	22	1.03
Reactor operation	530	900	1.4
Reprocessing	45	67	1.5
Research	120	90	0.78
Total	*800*	*1 400*	*1.75*
Medical uses of radiation			
Diagnostic radiology	950	470	0.50
Dental practice	265	16	0.06
Nuclear medicine	115	90	0.79
Radiotherapy	120	65	0.55
Total	*2 320*	*760*	*0.33*
Industrial uses of radiation			
Radiography	106	170	1.58
Radioisotope production	24	47	1.93
Other	570	140	0.25
Total	*700*	*360*	*0.51*
Defense activities			
Weapons	380	75	0.19
Nuclear ships and support	40	25	0.82
Total	*420*	*100*	*0.24*
Miscellaneous uses			
Education	310	33	0.11
Veterinary medicine	45	8	0.18
Total	*360*	*40*	*0.11*
Total (man-made)	**4 600**	**2 700**	**0.6**
Natural radiation			
Coal mining	3 910	2 600	0.7
Other mining	760	2 000	2.7
Mineral processing	300	300	1.0
Radon in workplaces	1 250	6 000	4.8
Aircrew	250	800	3.0
Total (natural)	**6 500**	**11 700**	**1.8**
Total (man-made + natural)	**11 100**	**14 000**	**1.3**

The nuclear reactors of Doel and Tihange are pressurized water reactors (PWRs). The collective dose of the workers at the 4 reactors of Doel for 1990 - 1994 was 2.4 manSv/GWyear, comparable to the worldwide average for PWR reactors over the same period of 2.8 manSv/GWyear. Since then the doses at Doel have decreased by another factor of 4 to 0.6 manSv/GWyear in 2000.

5.2. Worldwide overview of occupational exposures

Occupational radiation exposures have been evaluated for six broad categories of work: the nuclear fuel cycle, medical uses of radiation, industrial uses, defense activities, education and veterinary uses, and occupations where enhanced exposures to natural sources of radiation may occur. The contribution of each category for 1990 - 1994 is summarized in table 5. The collective dose is estimated to be about 14 000 manSv/year: 2 700 manSv/year from man-made sources and 11 700 manSv/year from natural sources. The largest component of this, 6 000 manSv/year, comes from the exposure of workers to radon and its progeny significantly above background levels. *(As might be expected from the radon levels in residential buildings, the highest radon concentrations in above-ground workplaces in Belgium are found in the Ardennes.)* Of the remainder, the largest components are 2 600 manSv/year for coal mining and 2 000 manSv/year for other mining operations (excluding uranium mining, which is dealt with in the nuclear fuel cycle). There are contributions of 800 manSv/year to aircrew from exposure to cosmic radiation and 300 manSv/year to those involved in the minerals processing industries. The estimated collective dose from natural sources is, however, associated with much greater uncertainty than that from man-made sources of radiation.

Of the collective dose from exposure to man-made sources of radiation (2 700 manSv/year), about 50% arises from operations in the nuclear fuel cycle (1 400 manSv/year), about 30% from medical uses (760 manSv/year), about 14% from industrial uses of radiation (360 manSv/year), about 4% from defense activities (100 manSv/year), and about 2% from educational and veterinary activities (40 manSv/year).

6. SOURCES AND TRENDS OF RADIATION EXPOSURE IN BELGIUM

The radiation exposure of the Belgian population from natural and man-made sources is compared in table 6 to the average exposure for countries with an advanced health care system. The average annual dose in Belgium is 4.5 mSv. The greatest contribution comes from diagnostic medical examinations, which is estimated on the basis of social security data to be 1.95 mSv in Flanders. The second largest contribution is from radon and thoron exposure. The annual dose, calculated with the UNSCEAR dose conversion factor, is 1.45 mSv. Note that the UNSCEAR dose conversion factor for radon is 50% higher than the ICRP 65 conversion convention that was adopted in the new Belgian regulation (ARBIS, 2001). Much more significant than the average values is the variability in the levels of radon concentration in indoor air and in the diagnostic exposures to patients. For instance, the dose limit for occupationally exposed workers of 20 mSv/year is equivalent to two or three CT-scans.

The average effective dose in Belgium has almost doubled over the last 100 years from 2.3 mSv/year in 1899 to 4.5 mSv/year in 1999. Of this increase about 0.2 mSv/year comes from natural sources and 2 mSv/year from human activities involving the use of radiation and radioactive substances, mainly in medicine:

- An increase of the radon exposure from about 1.3 mSv/year in 1899 to 1.45 mSv/year in 1999. The causes are the reduced ventilation of residential buildings and the application of building materials with enhanced radium levels, such as phosphogypsum and fly ashes.
- A small increase of the cosmic radiation of about 0.05 mSv/year from air travel and holidays (for instance winter sports).
- The medical use of radiation is the largest and a growing man-made source of radiation exposure. The contribution has increased from nothing in 1899, shortly after the discovery of x-rays by Röntgen, to 1.95 mSv/year in 1999.
- A small contribution from all other man-made sources of 0.05 mSv/year.

Table 6. Average exposure from radiation sources in Belgium and worldwide. The medical exposure is for developed countries with an advanced health care system

Source	Average annual effective dose	
	Belgium mSv/year	Worldwide mSv/year
Natural radiation		
Cosmic radiation	0.35	0.4
External terrestrial radiation	0.4	0.5
Radon and thoron	1.45	1.2
Internal exposures other than radon	0.3	0.3
Total	2.5	2.4
Man-made		
Diagnostic medical examinations	1.95	1.3
Other man-made exposures	0.05	0.05
Total	2.0	1.35
Total	4.5	3.75

I would like to conclude this overview on sources of ionizing radiation with the trends in life-time exposure in Belgium. At the end of the nineteenth century the average life expectancy in Belgium for man and women was only 48 and 51 years, respectively. This increased in 1999 to 74 and 80 years. During the same period the annual doses doubled from 2.3 mSv to 4.5 mSv, resulting in a tripling of the average life-time exposure:

- for man from 110 mSv in 1899 to 338 mSv in 1999 and;
- for women from 117 mSv in 1899 to 360 mSv in 1999.

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UNSCEAR 2000: Effects of ionizing radiation (biological annexes)

Dr. P. Smeesters
Radiation Protection Adviser

Federal Agency for Nuclear Control
Ravenstein street 36, B-1000 Brussels

Abstract

This article aims to lay out an overall and comprehensive view of UNSCEAR 2000 Report's Volume II. Three annexes of this volume are dealing mainly with biological mechanisms and effects: Annex F concerning "DNA repair and mutagenesis", Annex G about " Biological effects at low radiation doses" and Annex H dealing with "Combined effects of radiation and other agents" .
Annex I represents an important state of the art as regards "Epidemiological evaluation of radiation-induced cancer". The choice of the projection models for the evaluation of the lifetime risks is a particularly important issue that will receive special attention. Annex J deals with "Exposures and effects of the Chernobyl accident", as they were evaluated at that moment. This annex has given rise to a controversy into which we will try to give some insight.
Beside the presentation of the main conclusions and issues raised, the article will try to give some insight into the way UNSCEAR is working and into the way UNSCEAR's work can be used.

Introduction

The biological annexes (Volume II) of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 2000 Report aim to be a state of the art regarding a series of scientific issues related to the health effects of human exposure to ionizing radiation. These annexes contain a huge amount of data. In this article, I try to lay out an overall, short but comprehensive, view of this UNSCEAR 2000 Report's Volume II.
Three annexes of this volume are dealing mainly with biological mechanisms and effects: Annex F about "DNA repair and mutagenesis", Annex G about " Biological effects at low radiation doses" and Annex H dealing with "Combined effects of radiation and other agents".
The radiation-induction of cancer at low doses and low dose rates was *the* major concern in Annex F (DNA repair and mutagenesis) and G (Biological effects at low radiation doses). So, the data concerning the non-cancer effects of irradiation in utero have not even been presented. These two annexes will therefore be discussed here, together, under the title "Radiation-induction of cancer at low doses".
Annex I deals with "Epidemiological evaluation of radiation-induced cancer". The choice of the projection models for the evaluation of the lifetime risks is a particularly important issue and will receive special attention. Annex J gives the Committee's evaluation of the "Exposures and effects of the Chernobyl accident". This annex has given rise to a controversy into which I will try to give some insight.

Radiation-induction of cancer at low doses

o *What is a low dose?*

The definition the Committee gives of "low doses" is directly related to the low dose *concern*, namely: how to form a reasoned judgment about cancer induction at exposures *below which* no confident direct information is available?
Criteria for choosing a numerical value can be derived from three possible approaches:

- epidemiological: in most studies, the effects of exposure to ionizing radiation under 100 mGy are either not statistically significant or subject to discussions; this value of 100 mGy could be taken as the low dose boundary, although there is strong evidence that childhood cancers appear after irradiation *in utero* with doses as low as 10-20 mGy; radiobiological : the limit (with some exceptions) for detection of effects in experimental systems is ~ 20 mGy (chromosome aberrations);
- microdosimetric: single track damage (< 2 % «targets » receiving more than 1 track) correponds with ~1 mGy .

On the basis of the epidemiological approach, the low doses were taken to be the doses ***under 100 mGy.***

o *Mechanistic considerations about tumorigenesis*

The development of tumours is currently described with a ***multistage model***. The first step is a damage to DNA, which the cell's repair mechanisms fail to correct. This results in an *initiating mutation*. In most tumours, *single* target stem-like cells are implicated.
The second stage, ie the *promotional growth*, shows clonal developments of pre-neoplastic lesions, in which cellular environment plays an important role.
The third stage is the *conversion* into a malignant phenotype: this conversion is driven by *further mutations*. The fourth stage is the *progression*, characterized by the spread of the tumour (also driven by further mutations).

Many genes – not only the well-known DNA-repair genes- are involved in the response to DNA-damage. DNA-repair is a part of a ***complex response system*** (damage-signalling genes; adaptive or stress-response; genes blocking chekpoints; immune genes) and the puzzle is not yet assembled.

There are several pathways for DNA-repair, some of which being *error-prone* (non-homologous end joining producing deletions and re-arrangements); others are rather error-free (homologous recombination using the template of parental copy or sister-chromatid after duplication), but not always: in heterozygotes, the *bad* allele can be copied! This produces a "loss of heterozygoty".
These "protective" genes are affected in a series of ***human genetic disorders.***
The so-called *high penetrance* disorders are characterized by a strong expression: the (rare) affected individuals show *radiosensitivity* after acute exposure (radiotherapy, chromosome-damage tests) and *cancer-proneness* (in general and after irradiation).
The effects of the (frequent) *low penetrance* disorders (with generally subtle mutations or polymorphisms) are still poorly known: they could have the same

potential risks, at some degree. Research with rodent models is currently ongoing and its results could lead to an ethical challenge in the future.

While non-mutational effects (epigenetic effects, like the bystander effects) can play a role in it, ***mutations are*** currently considered as ***the driving force*** in the tumorigenesis. Mutations of proto-oncogenes (gain-of-function genes) are frequent in leukaemias and lymphomas, while mutations of tumour-suppressor genes (loss-of-function genes or cellular « gatekeepers »; p.ex. p53) are required in many solid tumours. These genes control a complex array of cellular responses. An important point is the onset, after the initiating mutation, of ***genetic instability*** that causes *further* mutations. This means that additional external attacks are not needed anymore and that the defences of the cells can be bypassed.

o *Radiation-induction of cancer : current evidence*

The current evidence concerning radiation-induction of cancer can be summarized on the following way:

- The main mechanism is the ***initiation*** of mutations in critical target cells (mainly: gross deletions affecting tumour-suppressor genes). This increases the *general pool* of tumour-initiated cells later subjected to age and environment. Such a view supports the use of *relative* risk projections.
- The principal damages are ***complex DNA double-strand lesions*** (« multiply damaged sites »). The lesions are different from the spontaneous lesions, but are no fingerprints. The radiation-induction of complex DNA double-strand lesions is *possible with single tracks* (i.e.at very low doses) .
- There is ***no expectation of wholly error-free repair*** of these lesions even at low doses and dose rates. Note that error-free repair is not increased by adaptive response.
- ***Cell defenses*** (apoptosis, telomere erosion, cellular communication, immuno-surveillance, …) ***can be bypassed*** by specific mutations.

o *Radiation-induction of cancer : overall judgment*

On the basis of the current evidence, the UNSCEAR expressed the following overall judgment concerning the risk of radiation-induction of cancer at low doses and low dose rates:

–there is ***no threshold dose;***
–the cancer risk is rising as a function of dose;
–the **L and LQ** dose-effect relation is **« the most scientifically defensible approximation »**

Combined effects of radiation and other agents (stochastic endpoints)

During the early phases of tumorigenesis, combined effects are only observed with *high* concentrations of chemicals during irradiation or repair. This is not relevant for radiation protection.
During the organ phase of cancer development (long duration), there are many interaction opportunities (genotoxic agents or not). Supra-additivity has been observed with ***smoking*** (lung cancer), ***UV*** (skin cancer), ***asbestos***, ***diet***, ***arsenic***, ***hormones*** like diethylstilbestrol (breast cancers), …..In the radon miner studies, the radiation risk was enhanced in smokers: the effect was more than additive but less than multiplicative.
There is "no firm evidence for large deviations from additivity at chronic low doses", with the exception of radiation and smoking.
Nevertheless, the lack of data does not imply the absence of combined effects at low doses.

Epidemiological evaluation of radiation-induced cancer

o *Lifetime cancer risk estimate*

In the UNSCEAR's document, the risk is expressed as the "risk of exposure-induced *death*" (REID): this measure of the risk includes cases who would have died of cancer in the absence of exposure but who died *earlier* as a result of the exposure. The values given are *a global estimate* (all ages, both sexes, all cancers) after irradiation of the *Japanese population* at *high dose/high dose rate* and are based principally on the *Life Span Study* (LSS) with use of *projection models* (unavoidable because most survivors in the LSS are still alive). Leukaemia cases are included.

Under the above-mentioned conditions, the UNSCEAR 2000 Report gives two evaluations of the lifetime radiation-induced cancer risk, according to the projection model that is used:

- with the *age-at-exposure model* (time-constant relative risk) : *12.1 % Sv^{-1}*
- with the *attained age model* (age at death by cancer): *8.3 % Sv^{-1}*

It concludes with a global estimate of **about 12% Sv^{-1}** , with an *uncertainty factor of 2 (higher or lower).*
This estimation is roughly the same as the UNSCEAR 1994 value (between 8.6 and 12% Sv^{-1} using relative risk models in which the relative risk either remains constant or decreases in varying ways with time since exposure).

o *The role of the age at exposure*

On the basis of the attained age model, the importance of the age at exposure is considerably reduced. Yet the UNSCEAR Report considers that the lifetime cancer risk might be multiplied by two for those exposed as children. The choice of the projection model has been discussed again, in the presence of the rapporteur for this UNSCEAR's annexe, during a scientific seminar organised on 9 November 2000 by the European Commission, in cooperation with the Group of experts referred to in Article 31 of the Euratom Treaty [1] . During this seminar, the attention has been drawn

[1] Low Dose Ionizing Radiation and Cancer Risk- Proceedings of a scientific seminar held in Luxembourg on 9 November 2000. Radiation Protection 125, European Commission.

to the following point: although neither the age-at-exposure model nor the attained age model describes all the observations, the fact that, in the LSS, the Excess Absolute Risk (EAR) is still, *at a given attained age, higher* for exposure at younger ages than for exposure at older ages is not very consistent with the attained age model. In the conclusions of this seminar, it is stated that the ***age-at-exposure model*** continues to provide a reasonable fit to the observations, and therefore ***should be retained at present.*** A change in our current estimation that the risk is higher for those exposed as children is therefore not justified.

o *Other causes of variations in the lifetime cancer risk estimates:*

Besides the age-at-exposure effect, there are various other factors that influence the lifetime cancer risk estimates after exposure to ionizing radiation:

- The risk for ***women*** is ***30% higher*** than for men (all solid cancers).
- The cancer risk estimates are multiplied by a factor of two when considering cancer ***induction*** (the above mentioned values of REID are based, by definition, on cancer *mortality*).
- Potential changes of the estimation of neutron doses in the LSS may cause only a small decrease *(max 10 %)* of the slope of the dose-response curve.
- Under ***chronic*** exposure conditions, the above-mentioned REID should be reduced by a factor 2, with an uncertainty factor of 2 (higher or lower).
- Last but not least, due to different *baseline* incidences for *specific* cancer types, the choice of an absolute or relative model of ***transfer of risk between populations*** has a profound influence on the cancer risk estimation: so, the estimation of the lifetime risk for breast cancer may vary from 1.3 to about 6 % Sv^{-1} between Japan and the USA .

o *Dose response relationship*In the LSS, a ***linear*** dose-response is observed for solid cancers taken as a whole, while a reduction factor of 2 is observed at low doses for leukaemias (LQ relationship). On this basis, ***the DDREF*** (Dose and Dose Rate Effectiveness Factor) ***can be taken as 1*** (no reduction of the risk at low dose) ***for the low doses delivered at high dose rate.*** Although this is not relevant for most situations in the radiation protection field, it must be remembered that the doses in the medical field are often delivered at high dose rate.

Exposures and effects of the Chernobyl accident

o *Exposures of individuals*

Estimations of the exposure of various categories of individuals as a result of the Chernobyl accident are found in the UNSCEAR 2000 Report. Here is a summary of these estimations:

- as regards the 600 emergency workers:
  - gamma doses from 2 to 16 Gy
  - beta doses up to 400-500 Gy

    - remember that there were 134 acute radiation diseases; 30 persons died
- as regards the 600 000 ***liquidators*****:**
    - there is an important uncertainty in the dose estimations
    - a reasonable ***average*** effective dose in the first year could be **100 mSv**
- as regards the 116 000 evacuated persons:
    - average effective dose: 30 mSv
    - ***average thyroid dose: 0.47 Gy***
- as regards the residents of the contaminated areas:
    - average effective dose: 10 mSv/y, with a range of one order of magnitude higher or lower
    - ***average thyroid dose: 0.2 Gy, with a range of two orders of magnitude higher or lower***
    - ***the dose to the thyroid in children is 10 times higher than in adults***

Globally, the lifetime doses are estimated to be 2 to 5 times the first year dose.
Individual dose reconstruction, necessary for conducting epidemiological studies, constitutes a challenge for the future.

o *Thyroid cancers*

The most striking, unexpected and least questionable effect of the Chernobyl accident was found to be a significant increase of thyroid cancer in children, in the areas most exposed to the initial radioactive clouds. About ***1800*** cases of thyroid cancers had been observed at the time of the UNSCEAR 200 Report in those individuals exposed in childhood. This number is « considerably greater than expected, ...even after taking confounding influences into consideration». Young children seem particularly vulnerable and were affected by thyroid cancers of an ***aggressive or invasive nature*** and with a ***short latency period.*** The age distribution analysis of the thyroid cancers suggests that the relative risk for the ***children who were the youngest at the time of the exposure*** is much higher than for older children and is especially much more pronounced than was predicted on the basis of previous observations.
While the incidence currently seems to decrease for the 5-9 years old at exposure cohort,the increase continues for those exposed before the age of 5.

o *Other health effects*

An increased incidence is also observed for other cancers, but the interpretation is difficult because this increase already existed before the accident and is not limited to the areas affected by the accident. No significant increase of leukaemias has been observed, but ***very sound epidemiological studies are necessary*** in order to ensure correct diagnosis and to reveal small increases, all the more so since the study is complicated by the socio-political context.
Many other health effects have been observed in the various categories of exposed persons, but the UNSCEAR Report presents them all as non radiation-related (or as psychosomatic). Nevertheless observations from the LSS (Hiroshima-Nagasaki) ask for more cautiousness, as the frequency of several non-cancer diseases increases in function of the dose. While, on this basis, some experts were reluctant to adopt a too definite conclusion about the cause of the various pathologies observed in the areas around Chernobyl, there has been a great pressure from some delegations to rule out any allusion to a possible responsability of the radiation exposures.

Some considerations about UNSCEAR's work

An assertion commonly heard during UNSCEAR's meetings is that this committee must be considered as "purely scientific". This is also the way the Committee is commonly perceived outside.

Let us remember that, if scientific experts are supposed to be « competent, specialized, recognized » - which is certainly the case with UNSCEAR's participants -, they have also to be « neutral and objective ». These last conditions are difficult to meet in general and UNSCEAR is not an exception.

Whether we want it or not, everyone of us, including experts, has a mandate, the nature of which influence the primary goals which are aimed at. This can obviously affect the expected degree of neutrality.

Moreover ethical issues are often deeply imbricated *within* the area of experts' evaluations, particularly as regards the management of uncertainty or the transparency or ambiguity of the messages. These ethical problems can be sources of varying opinions, including from "objective" experts.

Finally, as regards "objectivity", reflection about science shows clearly that the role of the *subject* who observes (as an isolated individual or, more often, as a representative of a current of thoughts) is essential, whether this is in the process of selecting observations, or in the formulation of hypotheses or in the theoretical construction. When appealing to the *consensus* of the scientists, as a guarantee for objectivity, one forgets that the scientific experts, coming from the same melting pot, from the same "clubs", often share the ***same*** interpretative language, the same views and the same paradigm.

This means that, in spite of the great interest of UNSCEAR's work, these reports have to be read with an ounce of critical mind.

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THE UNSCEAR 2001 REPORT ON HEREDITARY EFFECTS OF RADIATION

K. Sankaranarayanan
Department of Radiation Genetics and Chemical Mutagenesis
Leiden University Medical Centre, Sylvius Laboratories, Wassenaarseweg 72
2333 AL Leiden, The Netherlands

Abstract

Starting in the late 1950s, UNSCEAR has published a total of 10 reports on hereditary effects of ionizing radiation of which the one published last year (UNSCEAR 2001) is the latest. It reviews the advances in the science behind genetic risk estimation in eight chapters and presents estimates of risk in Chapter VIII. The report contains an extended summary and a glossary of technical terms. Several important concepts have been introduced in this report and the risk estimates presented in Chapter VIII build on these concepts. It is worth noting that for the first time in its history, UNSCEAR has been able to: (i) provide risk estimates for all classes of genetic diseases; (ii) incorporate advances in molecular biology in risk estimation; and (iii) reconcile the results from the largest of human studies ever conducted (namely, those carried out on the children of A-bomb survivors in Japan) with its own risk estimates in showing that genetic risks at low doses of chronic low LET radiation are indeed small compared to the baseline risks of such diseases in the population.
For a population exposed to low dose chronic, low LET irradiation, the estimated risks to the first generation progeny are the following (all estimates per million progeny per Gy): autosomal dominant and X-linked diseases, ~ 750 to 1,500 cases (compared to 16,500 cases per million of naturally-occurring ones); autosomal recessive diseases, essentially zero cases (compared to 7,500 per million naturally-occurring ones); chronic diseases, ~ 250 to1,200 cases (compared to 650,000 per million naturally-occurring-ones) and multi-system developmental abnormalities, ~ 2,000 cases. The total risk per Gy for the first generation progeny, is only about 0.41 to 0.64% of the baseline risk of 738,000 per million.

1. INTRODUCTION

The goal of genetic risk estimation is to estimate the added risk of “inducible genetic diseases” in human populations exposed to ionizing radiation, over and above that which occurs naturally as a result of spontaneous mutations. Efforts at genetic risk estimation over the past four decades have all been driven by mouse data on radiation-induced mutations, human data on baseline frequencies of genetic diseases, population genetic theory and models, and a number of plausible assumptions. They are therefore essentially of the nature of predictions.

These predictions, however, have to be viewed against the background of human studies especially the one carried out on A-bomb survivors in Japan which showed no statistically significant adverse genetic effects of parental radiation exposure. Since the Japanese data on genetic effects could not be used for expressing risks quantitatively in terms of genetic diseases, they remained on the sidelines of mainstream efforts at risk estimation. Nonetheless, the notion remains in the public mind familiar with the Japanese studies, that the estimates published by

scientific committees such as UNSCEAR and the BEIR Committee of the US National Academy of Sciences are probably overestimates of genetic risks of radiation exposure.

The key words in genetic risk estimation are "inducible genetic diseases". The aim of this paper is to briefly review recent progress in UNSCEAR's efforts in this regard, highlight those aspects that have emerged from the incorporation of advances in human molecular biology into the conceptual framework of genetic risk estimation and show how the new estimates of genetic risks are consistent with the findings in the Japanese studies.

2. GENETIC DISEASES

Genetic diseases are those that arise as a result of mutations in germ cells and are transmitted to the progeny. Diseases caused by mutations in single genes are called Mendelian diseases and are further subdivided into autosomal dominant, autosomal recessive and X-linked recessive depending on the chromosomal location and the transmission patterns of the mutant genes. The important point with respect to Mendelian diseases is that the relationship between mutation and disease is straightforward and the pattern of transmission is simple and predictable.

Multifactorial diseases are those due to the joint action of multiple genetic and environmental factors. Examples include the common congenital abnormalities (e.g., neural tube defects, congenital heart defects, cleft lip with or without cleft palate etc) which are present at birth and chronic diseases of adults such as diabetes, essential hypertension, coronary heart disease etc. These diseases do not show simple patterns of inheritance i.e., the relationship between mutation and disease is complex. However, these diseases do "run" in families.

3. THE DOUBLING DOSE METHOD OF RISK ESTIMATION

The method that has been used for risk estimation over the past decades is what is referred to as the doubling dose method. It is based on the equilibrium theory which population geneticists use to explain the dynamics of mutant genes in populations. The basic concept is that the stability of mutant gene frequencies (and thus of disease frequencies) in a population is the result of the existence of a balance or equilibrium between the rates at which spontaneous mutations enter the gene pool in every generation and the rates at which they are eliminated by natural selection i.e., through failure of survival or reproduction. Under conditions of irradiation, this balance is disturbed by the influx of induced mutations, but the population will eventually reach a new equilibrium between mutation and selection. The amount of increase in mutant and thus of disease frequency, the time it takes to reach the new equilibrium and the rate of approach to it are all dependent on induced mutation rates, the intensity of selection, the type of genetic disease and whether radiation exposure occurs in one generation only or generation after generation.

With the DD method, the risk is estimated as a product of three quantities:

$$\text{Risk per unit dose} = P \times [1/DD] \times MC \qquad (1)$$

Where P is the baseline frequency of the disease class under study,
DD is the doubling dose and MC is the "mutation component"
Advances have been made with respect to each of these quantities.

3.1. Baseline frequencies of genetic diseases

Recent estimates (Table 1) suggest that about 2.4% of all live born children suffer from one or another Mendelian disease (1.5%, autosomal dominants; 0.75%, autosomal recessives and 0.15%, X-linked). Additionally, about 0.4% of live births are affected by diseases due to numerical or structural abnormalities of chromosomes (chromosomal diseases), 6% by congenital abnormalities and over 65% of the population will develop one or another chronic disease in adult life. Although not shown in Table 1, the frequency of 2.4% for Mendelian diseases is about twice that which has been used until the 1993 UNSCEAR report (UNSCEAR 1993).

Table 1. Baseline frequencies of genetic diseases

Disease class		Frequency/million[a]
Mendelian diseases[b]		24,000
Autosomal dominant	15,000	
X-linked	1,500	
Autosomal recessive	7,500	
Chromosomal diseases		4,000
Multifactorial diseases		710,000
Chronic diseases	650,000	
Congenital abnormalities	60,000	
Total		738,000

a/ For Mendelian and chromosomal diseases and for congenital abnormalities, the frequencies are per million live births and for chronic multifactorial diseases, per million of the population

b/ Based on Sankaranarayanan (1998)

3.2. The Doubling dose

The second quantity in the risk equation (1) is the doubling dose (DD) which is defined as the amount of radiation required to produce as many mutations as those that occur spontaneously (i.e., in the absence of radiation) in a generation. It is estimated by dividing the average spontaneous mutation rate of a set of defined genes by the average rate of induced mutations in the same set of genes. The reciprocal of the DD (i.e., 1/DD), is the relative mutation risk (RMR) per unit dose and Gy is the unit of radiation dose. Since RMR is a fraction, one can readily note that, a small DD implies high RMR and a large DD implies low RMR. The DD so far used in risk estimation is 1 Gy and was based on mouse data on spontaneous and induced recessive mutations in 7 genes which have been extensively studied.

The use of the DD estimate based entirely on mouse data for risk estimation in humans entails the assumption that the average spontaneous rates and the average rates of induced mutations in mice and humans are similar. There are now reasons to believe that:

(i) the assumption of similarity of induced mutation rates in both species, while unavoidable, is reasonable;

(ii) the assumption of similarity of spontaneous mutation rates in the two species is flawed and avoidable and

(iii) additional uncertainties have now arisen in the calculation of spontaneous mutation rates in mice. UNSCEAR adopted the view that the prudent way forward is to use human spontaneous rates and mouse induced rates for DD calculations as was first done in the 1972 BEIR report (NAS 1972).

The average spontaneous mutation rate of human genes that can now be estimated is $(2.95 \pm 0.64).10^{-6}$/gene and is based on some 135 genes resulting in 26 autosomal dominant disease phenotypes. A similar analysis of mouse data on induced mutations, pertaining now to 32 genes permit an average estimate of $(3.6 \pm 0.10).10^{-6}$/locus/Gy for chronic low LET radiation conditions. The DD therefore becomes (0.82 ± 0.29 Gy) not very different from the 1 Gy thus far used. UNSCEAR has suggested the continued use of the 1 Gy estimate for the DD to avoid the impression of undue precision, emphasizing, however, that an important conceptual change has been made (i.e., the use of human data on spontaneous mutation rates and mouse data on induced mutation rates for DD calculations) and that the present estimate is based on more data than has been the case so far.

3.3. Mutation component

The third quantity in the risk equation is what is referred to as the mutation component (MC). It provides a measure of how the disease frequencies will increase when the mutation rate is increased, as for example with radiation. The reason for having this quantity in the risk equation is that the relationship between mutation and disease varies between different classes of genetic diseases. It is simple for autosomal dominant and X-linked diseases, slightly complex for autosomal recessive diseases and very complex for multifactorial diseases.

Until recently, mathematical procedures were available for estimating MC for Mendelian diseases which constitute a small part (2.4%) of the total genetic disease burden and not for multifactorial diseases which affect over 70% of people. During the last few years, it had become possible to develop the MC concept fully for both Mendelian and multifactorial diseases with all the necessary algebraic formulations. This effort was carried out within the framework of an ICRP Task group, the report of which was published in 2000 (ICRP 2000).

For example, for the first generation following radiation exposure, the MC can now be estimated to be of the order of about 0.3 for autosomal dominant and X-linked diseases, close to zero for autosomal recessive diseases and about 0.01 to 0.02 for chronic multifactorial diseases. For congenital abnormalities, the other sub-group of multifactorials, MC cannot be reliably estimated. But this does not pose any serious problems as discussed later.

4. ADVANCES IN HUMAN MOLECULAR BIOLOGY AND THEIR IMPACT ON RISK ESTIMATION

The concept of Potential Recoverability Correction Factor (PRCF). One important question in risk estimation concerns the appropriateness of the estimated induced rate of mutations in 7 mouse genes for estimating the risk of inducible genetic diseases in humans. It should be realized that the risk equation: Risk per unit dose = P x 1/DD x MC, is a predictive one based

on population genetic theory. In making this prediction, it is assumed that the genes which underlie the diseases included under P will all respond to radiation-induced mutations (which are assumed to be deletions predominantly), that such deletions will be compatible with live births and hence recoverable in the progeny of irradiated individuals. But no one has seen a single radiation-induced genetic disease. Why is this so? It is now clear that this is so because the assumptions used are incorrect.

Advances in molecular studies of spontaneous disease-causing mutations in humans and of radiation-induced mutations in experimental systems have now highlighted a number of differences between them, both in terms of their nature and the mechanisms by which they arise. For example, spontaneous mutations include point mutations, small and large intragenic DNA deletions, some large multi-gene deletions etc. Most radiation-induced mutations, however, are DNA deletions, often including more than one gene. At the functional level, spontaneous mutations include those which cause loss of function as well as gain of function. Radiation-induced mutations, because they are often multi-gene deletions, are mostly loss of function mutations.

Spontaneous mutations arise by a number of different mechanisms that are dependent on DNA sequence organization of the genes and their genomic context. In contrast, radiation-induced deletions originate through random deposition of energy in the cell i.e., one can assume that the initial probability of inducing a deletion may not be different between genomic regions. However, their recoverability in live births seems more dependent on whether the loss of the gene or genomic region is compatible with viability in heterozygotes.

It is now clear that the success in experimental radiation mutagenesis studies is mainly due to the fortunate choice of genes that are non-essential for survival of the animal or the cell and also happen to be located in non-essential regions of the genome. Most human disease-causing genes are *not* of this type. There are a number of structural and functional constraints associated with the recoverability of induced disease-causing mutations in humans which explains why no one has seen a radiation-induced germ cell mutation, let alone an induced genetic disease in humans!

These findings considered together lend strong support to the view that only in a small proportion of human genes of interest from the disease point of view, induced mutations may be potentially recoverable in live births. Since there is no alternative to the use of mouse radiation data for risk estimation, there is a need to bridge the gap between the rates of induced mutations in mice and those of induced mutations that are potentially recoverable in humans and result in disease.

The approach that has been pursued is to introduce a disease-class specific correction factor called *P*otential *R*ecoverability *C*orrection *F*actor (PRCF) in the risk equation such that the risk becomes a product of four factors instead of the original three:

$$\text{Risk per unit dose} = P \times [1/DD] \times MC \times PRCF \qquad (2)$$

In order to estimate PRCF, certain criteria for potential recoverability of induced mutations in human live births were defined on the basis of molecularly-analysed mutations in experimental systems. These criteria were then applied, on a gene-by-gene basis, to human genes of interest from the disease point of view taking into account gene structure, function, mechanisms, the

known spectrum of naturally-occurring mutations, the genomic context of the gene of interest etc. The question asked was: if a deletion is induced in this gene/genomic region, is it potentially recoverable in a live birth?

The analysis has shown that among the human autosomal and X-linked genes studied, only 15 to 30% may be responsive to induced mutations that are potentially recoverable in live births. These fractions of 0.15 to 0.30 are the PRCFs for autosomal dominant and X-linked diseases.

For chronic multifactorial diseases, the PRCFs are expected to be much less since one has to calculate the probability of recovering induced mutations simultaneously in a minimum of two genes to call the disease multifactorial. A rough approximation is that the PRCFs for these diseases is the n^{th} power of those for a Mendelian disease where n = the number of genes assumed to underlie a given multifactorial disease. With just 2 loci, the figures become $(0.15)^2$ to $(0.30)^2$ or 0.02 to 0.09. One does not need PRCFs for autosomal recessive diseases and for congenital abnormalities, it is not possible to estimate them reliably. The important general point is that the concept of PRCF is one of the outcomes of the integration of molecular biology into the conceptual framework of genetic risk estimation.

Phenotypes of radiation-induced genetic damage in humans. As discussed earlier, in genetic risk estimation, the radiation risks are expressed as increases in the frequencies of genetic diseases. It should be realized, however, that

(i) radiation produces genetic damage by random deposition of energy;
(ii) most radiation-induced mutations studied in experimental systems are multi-gene deletions;
(iii) for their recoverability in live births, the deletions must be compatible with survival.

Obviously, radiation does not "know" that the risk estimators, are interested in specific societally relevant diseases and does not "bother" to respect the classification schemes for these diseases which we use for our convenience of study. It will produce a deletion somewhere in the genome and whether it will be recoverable in a live birth or not and what the clinical phenotype will be, will depend on what gene functions have been lost because of the deletion.

Some insights into the potential phenotypes of radiation-induced genetic damage in humans come from studies of the so called microdeletions in humans. These are deletions of multiple, functionally unrelated yet physically contiguous genes that are compatible with survival in the individuals receiving them. Many examples of such naturally-occurring microdeletions have been and continue to be reported in the human genetics literature. They show that their distribution in different chromosomes is non-random. This is not unexpected in the light of differences in gene density in different chromosomes and chromosomal regions. However, the important point is that despite their occurrence in different chromosomes, they share some common features: mental retardation, growth retardation, specific patterns of dysmorphic features, serious malformations etc. This is because of the fact that genes involved in developmental processes are enormous in number and are distributed in nearly all the chromosomes.

It has therefore been suggested that the main adverse genetic effects of radiation will be manifest as multi-system developmental abnormalities which we call congenital abnormalities. Unlike naturally-occurring congenital abnormalities most of which are interpreted as being of

multifactorial origin, the induced developmental defects, by and large, are predicted to show autosomal dominant patterns of inheritance, since they arise as a consequence of induced multi-gene deletions. Such predictions have been fulfilled. There are some radiation data on congenital malformations ascertained *in utero*, growth retardation, dominant skeletal defects and dominant cataracts in the progeny of irradiated mice. These data provide a basis for making a provisional estimate of risk of adverse developmental effects. The estimate is $\sim 20 \times 10^{-4}$/Gy for low LET chronic irradiation of both sexes. Note that one does not need to use the DD method here.

5. THE UNSCEAR 2001 ESTIMATES OF GENETIC RISKS

Using estimates of disease-class specific parameter values that were presented earlier, risk estimates have been made for all classes of genetic diseases. For simplicity, the estimates for the first generation progeny of an irradiated population are presented here (Table 2).

Table 2 UNSCEAR (2001) estimates of genetic risks from continuing exposure to low-LET, low-dose or chronic irradiation.
(Assumed doubling dose: 1 Gy)

Disease class	Baseline frequency per million live births	Risk per Gy per million first generation progeny
Mendelian		
Autosomal dominant	16,500	~750 to 1,500
Autosomal recessive	7,500	0
Chromosomal	4,000	[a]
Multifactorial		
Chronic diseases	650,000	~250 to 1,200
Congenital abnormalities	60,000	~2,000[b]
Total	738,000	~3,000 to 4,700
Total risk per Gy expressed as per cent of baseline		~0.41 to 0.64

[a] Assumed to be subsumed in part under the risk of autosomal dominant and X-linked diseases and in part under congenital abnormalities
[b] Estimate obtained using mouse data on developmental abnormalities and not with the doubling dose method

As can be noted, the risk of autosomal dominant and X-linked diseases is of the order of 750 to 1,500 cases per million progeny per Gy of chronic low LET radiation (compared to 16,500 cases per million of naturally-occurring ones). The risk of autosomal recessive diseases is essentially zero (compared to 7,500 per million naturally-occurring ones). The risk of chronic diseases is of the order of 250 to1,200 cases per million per Gy (compared to 650,000 per million naturally-occurring-ones). The risk of multisystem developmental abnormalities may be of the order of about 2,000 cases per million per Gy. Note that the total risk per Gy, is only about 0.41 to 0.64% of the baseline risk of 738,000 per million live births, a very small proportion indeed!

6. RECONCILIATION OF THE PRESENT RISK ESTIMATES WITH THE FINDINGS FROM THE JAPANESE STUDIES

As mentioned earlier, the genetic studies carried out on A-bomb survivors in Japan did not show any measurable adverse effects of parental radiation exposure. Neel and colleagues who carried out this monumental work have published a number of DD estimates over the years and the most recent ones are 3.4 to 4.5 Sv (Neel et al. 1990). Comparisons are often made between these DDs and the one used by scientific committees such as UNSCEAR and the BEIR Committee, which all happen to be 1 Gy. Since a high DD translates into a low relative mutation risk per unit dose, these findings were interpreted to make the erroneous point that these committees have overestimated the risks.

UNSCEAR has pointed out that such comparisons are inappropriate because the DDs in the Japanese studies are *retrospectively* estimated from empirical data showing no significant differences in endpoints of damage which are totally different. On the other hand, the DD used by UNSCEAR and the BEIR Committee is calculated from different sets of data on mutations in defined genes and is *prospectively used as one of the quantities in the risk equation to predict risk*. However, despite these differences, the main message from the Japanese studies and from the present UNSCEAR estimates is basically the same, namely that at low doses, the genetic risks are small, compared to the baseline risks of naturally occurring genetic diseases.

7. PERSONAL REFLECTIONS

Soon after his discovery of the mutagenic effects of ionizing radiation in Drosophila in the late 1920s (Muller 1927), for which he received the Nobel Prize in Medicine in 1946, Muller started to alert the medical profession to the genetic consequences of carelessly and avoidably exposing the human gonads to radiation. Muller was not only one of the greatest intellects of the 20th century, but also a great humanist; he was genuinely concerned about human welfare. The field of radiation genetics prospered. From about the mid-1950s onwards, the genetic effects of radiation became an integral component in radiological protection recommendations by ICRP and other organizations.

Starting in the early 1990s, advances in molecular biology began to be incorporated into the conceptual framework of risk estimation. Now, at the end of 20th century, the advances in our science suggest that genetic risks of radiation exposure at low doses are probably not as high as Muller feared they might be and we are in a position to scientifically reconcile the findings of the Japanese studies with the estimates that can be made now. This is an important achievement of 20th century science in our field.

As one who has been involved in genetic risk estimation for the past more than three decades, I am convinced that further progress and refinements in this field in the coming years will be intimately linked to and spearheaded by advances in human molecular biology engendered by the human genome projects, past and on-going. The enterprise of genetic risk estimation in the 21st century calls for active participation of practicing human and molecular biologists and informatics specialists besides those coming from the traditional fields of radiation biology, epidemiology and population genetics.

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