APPENDIX C

RISK FROM EXPOSURE TO IONIZING RADIATION

Extracted From:
SENES 2003. *Human Exposure to Ionizing Radiation*.
Prepared for OPG in Support of the
Pickering Waste Management Facility
Phase II Environmental Assessment

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APPENDIX C - HUMAN EXPOSURE TO IONIZING RADIATION

The following Appendix is extracted from SENES (2003), a technical appendix, prepared in support of the Environmental Assessment of the expansion of the existing Pickering Waste Management Facility for the dry storage of used fuel.

C1.0 INTRODUCTION

Human populations have always been exposed to ionizing radiation: from cosmic rays; from naturally occurring radionuclides in the air, water, and food; and from gamma radiation from the radionuclides in rocks and soils. The level of exposure to natural radioactivity varies depending mostly on where people live and partly on what they eat or drink.

At least from the middle ages with the mining of radioactive ores in central Europe, people have been exposed to elevated levels of radioactivity arising from man's activities. Since the beginning of the last century, with the discovery of radioactivity, people have increasingly been exposed to additional increments of radiation resulting from human activities of various kinds. Except from medical procedures, these incremental, man-made exposures are typically much smaller than the exposures from natural sources.

Ionizing radiation is ubiquitous in the environment, arising both from natural sources and from various human activities and practices. *Typical* levels of natural and artificially-enhanced (man-made) radiation exposure are shown below in Table C1-1.

Table C1-1
Typical Radiation Dose to the Public[1]

SOURCE OF EXPOSURE	ANNUAL DOSE (μSv)[2]
Natural	
Cosmic	300
Internal (uranium and thorium radionuclides, ^{40}K, ^{14}C)	350
External (terrestrial gamma radiation)	350
Radon	1,000
Natural total	2,000
Man-Made	
Medical	600
Nuclear weapons tests fallout	<10
Nuclear power stations	<1
Miscellaneous	20
Man-Made total (rounded)	620

Note:

1. These "typical" radiation doses are from "Canada: Living with Radiation" (AECB 1995). The actual annual radiation dose to any member of the public will vary greatly by location, housing type, lifestyle, occupation and medical needs. UNSCEAR (2000) indicates a worldwide average annual dose of about 2.4 mSv from natural sources, with a typical range of from 1 to 10 mSv per year.
2. The microsievert [written as μSv] is a measure of effective dose, as defined by ICRP (1996).

Extensive studies of human populations and experimental studies on the effects of ionizing radiation have identified two main categories of radiation effects. At relatively high doses[1], "deterministic" effects occur, principally as a result of extensive cell killing. This can give rise to damage to organs and tissues and, in extreme cases, to death. Deterministic effects, also termed non-stochastic effects, require a threshold dose to be exceeded before they manifest themselves as clinical damage, although sub-cellular damage to cell structures, and even the loss of individual cells will occur at lower doses. Once this threshold is exceeded, the severity of the effect increases with increasing dose; below the threshold, no effect is seen. A deterministic effect usually appears relatively quickly after exposure. Information on deterministic effects of radiation has been reviewed in reports of the scientific committees such as the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) (e.g., UNSCEAR 1988, 1993, 2000).

A different kind of damage can occur at times long after exposure, even at very low doses of ionizing radiation, and without any clinical signs of radiation exposure necessarily appearing earlier, near the time exposure occurred. This arises principally from damage to the nuclear material [deoxyribonucleic acid (DNA)] in the cell, resulting in the development of radiation-induced cancer in those exposed or hereditary disease in their descendants. Although the probability of both cancer and hereditary disease increases with radiation dose, it is generally considered that their severity (if the effects do arise) does not. These are termed "stochastic" effects, and have been the subject of reviews by UNSCEAR (e.g., 1988, 1994) and other scientific bodies such as the committees of the U.S. National Academy of Sciences (e.g., (BEIR V 1990, BEIR VII 1998) and the Advisory Committee on Radiological Protection (ACRP) (1996) (former Advisory Committee to the Canadian Nuclear Safety Commission, CNSC).

For most situations, interest is focused on the second type of damage which is (prudently) assumed to have some finite, dose-related probability of occurring, even at low doses and low dose rates. A recent report by the ACRP defines low dose rates in terms of unavoidable radiation exposure from natural sources (ACRP 1996). The ACRP notes:

> "*The average annual exposures to the whole body are roughly 1 mSv [1,000 μSv] per year in areas of normal background but this is increased to over 4 mSv per year in areas of high exposures due to high concentrations of primordial radionuclides in the soil. Similarly, the average annual effective dose to the lung due to inhalation of radon and its short-lived progeny from natural sources might be taken to be about 1 mSv per year, but this can be increased to 10 mSv per year or even more. On this basis, one might define low dose rates as anything up to say 10 mSv per year or 0.03 mSv per day."*

[1] Above about 0.2 Gy, where Gy (1 Gy = joule/kg) is the abbreviation for gray, a measure of (absorbed) radiation dose.

For the purposes of radiation protection, it is widely assumed that the probability of inducing excess cancers or excess hereditary risk in people exposed to ionizing radiation is directly proportional to the total radiation dose received, even at low doses and low dose rates and that there is no "safe" or threshold dose of radiation below which these biological effects will not be produced. This is commonly referred to as the linear, non-threshold (LNT) model. This model has, for many years, been regarded as a prudent and reasonable hypothesis for radiation protection (ICRP 1991, CRPPH 1998).

The ability to measure extremely low levels of radiation implies that it is possible to quantify the radiation effects arising from low doses of radiation. The difficulty is that cancer occurs naturally (in the absence of enhanced radiation exposure) at an appreciable rate, and at low doses it is difficult to determine with reasonable certainty what the level of effect may be, and indeed whether any effect attributable to radiation has occurred. It is important to recognize that a scientific demonstration of significant biological effects has not yet been made for radiation doses less than about 10 mSv to the fetus or below about 50-200 mSv in adults; however, this should not be taken *a priori* as indicating that a threshold exists.

UNSCEAR (2000) suggests that for most types of tumours in experimental animals and in epidemiological studies of humans, a significant risk can only be detected at doses in excess of about 100 mSv of low LET (linear energy transfer) radiation such as gamma or beta radiation. UNSCEAR (2000) also reports site-specific cancer risk estimates, with cancer risks in the range of 4-6% per Sv. For chronic exposure, UNSCEAR (2000) suggests a linear dose response for solid tumours and a nominal risk of about 5% per Sv. For leukemia, UNSCEAR (2000) adopts a linear-quadratic dose response and a risk of about 1% per Sv for an acute radiation dose. However, in view of the linear-quadratic dose response relation, the risk from an acute dose of radiation of 0.1 Sv is about 20 fold smaller than the risk of leukemia from an acute dose of 1 Sv. Overall, the radiation risk estimates developed in UNSCEAR (2000) are comparable to those previously reported by UNSCEAR (1994).

The purpose of this Appendix is to summarize the potential health effects that may be associated with human exposure to ionizing radiation and the various factors that affect how the potential health affects are estimated.

C2.0 STUDIES OF PEOPLE LIVING NEAR CANADIAN NUCLEAR GENERATING STATIONS

Over about the past 10 years, a variety of studies have been carried out in Canada to provide information about the possible impact of the Nuclear Generating Stations (NGS) on the health of local residents. These will be discussed chronologically.

In 1991, the AECB (Atomic Energy Control Board, predecessor to the CNSC) published a research report, prepared for the AECB by epidemiologists at Health Canada, concerning the possible relation between releases of tritium from the Pickering NGS and birth defects and infant mortality in the local community (Johnson and Rouleau 1991). This project was carried out in response to the concerns of a local citizen who claimed, in 1988, to have found such an association. Many potential radiation effects were reviewed, and for only one, Down syndrome, was there evidence of a statistical increase. However, because no correlation with emissions from the station could be established, the authors concluded that the result could have occurred by chance. In fact, an elevated rate of Down syndrome was also identified in another Ontario community, far from any nuclear facility.

The authors' direct conclusions were as follows:

> "*Overall, this analysis does not support a hypothesis of increased rates of stillbirths, neonatal mortality or infant mortality in the vicinity of Pickering Nuclear Generating Station. Since the plant's start up in 1971, the rates of these conditions were neither high overall, nor were the patterns of yearly rates unexpected among any of the communities in the vicinity of the plant. Furthermore, the analysis does not support a hypothesis of increased birth prevalence of birth defects in the vicinity of Pickering Nuclear Generating Station for 21 of the 22 diagnostic categories into which the birth defects were divided.*"
>
> "*The birth prevalence of Down syndrome was elevated in both Pickering and Ajax; however interpretation of this elevated risk must be very cautious. There was no consistent pattern between tritium release levels and Down syndrome birth prevalence...*" (Johnson and Rouleau 1991)

The authors stressed that the multiple testing of data that was carried out readily turns up associations by chance.

In 1991, the AECB also published the final report of a project, carried out for them by researchers at the Ontario Cancer Treatment and Research Foundation, to determine whether childhood leukemia rates in populations local to Ontario nuclear facilities, including Pickering, differed from those in Ontario as a whole (Clarke *et al.* 1991). Mortality ratios were examined for the periods "before" and "after" the start of the Pickering NGS. The number of leukemia deaths in the "before" period was slightly higher than expected in the nearby community. In addition, there was no difference between the mortality ratios in the "before" and "after" period. Overall, the authors found no evidence of a statistically-significant increase for any of the populations studied.

The 1991 study of childhood leukemia (Clarke *et al.* 1991) was subsequently published in a refereed journal by McLaughlin *et al* (1993). This paper discusses the incidence and mortality

from leukemia in children up to 15 years of age born to mothers living within 25 km of nuclear facilities in Ontario, including the nuclear generating stations at Pickering and Bruce. The stated objective of the study was to investigate whether the frequency of leukemia in these children differed from the provincial average. This study was an ecological comparison of leukemia rates for various geographic regions and therefore, it was not possible to investigate specific risk factors. The statistical power of the study was also limited due to the rarity of childhood leukemia and the small observed and expected numbers of leukemia. With these limitations, the authors found that the childhood leukemia risks (i.e., observed/expected) ranged from less than expected to more than expected but none of the differences were statistically significant. Overall, the authors found no indication of a birth cohort effect as there was no consistent pattern in mortality ratios between children born in the vicinity of a nuclear facility as compared to mortality ratios based on residence at death.

In an affidavit prepared on behalf of the Inverhuron and district ratepayers association in 1999, Dr. Hoel (Hoel 1999) commented on the ecological studies of leukemia incidence and mortality discussed above (Clarke *et al.* 1991, McLaughlin *et al.* 1993). Dr. Hoel challenged the starting hypothesis of these papers, namely to investigate whether there is a difference in the frequencies of leukemia between birth cohorts near nuclear sites and the provincial average, suggesting that the objective of the studies should have been to look for an excess (i.e., one tail test) rather than a difference (i.e., a two tail test). Moreover, Hoel suggests the use of a 90% confidence interval rather than the 95% confidence interval used in Clarke *et al.* (1991) and McLaughlin *et al.* (1993). The reality is that the studies by McLaughlin et al. were clearly stated to be exploratory in nature. In such situations, the two-tailed test is appropriate. The use of a 95% confidence limit for such tests is conventional. In any event, as clearly stated in the paper by McLaughlin *et al.* (1993) and Clarke et al. (1991), childhood leukemia is rare, and the numbers of expected and observed cases is small, and the power[2] is necessarily low, and the confidence limits of the relative risk (i.e., ratios of observed/expected) all include 1.

A 1999 paper by Laurier and Bard reviews epidemiological studies of leukemia in people under 25 years of age who live near nuclear sites (Laurier and Bard 1999). With respect to descriptive studies such as the ecological studies reported by Clarke *et al.* (1991, and McLaughlin *et al.* 1993), Laurier and Bard, although acknowledging the limitations of such studies, conclude that these studies " … *do not support the hypothesis that the frequency of leukemia generally increases among young people living near nuclear sites*". These authors also note that to date (i.e., 1999) analytical studies looking for causes of leukemia clusters "*have not yet provided a definitive explanation for the clusters observed*" and suggest the implementation of a system of surveillance of leukemia incidence cases around nuclear sites. The current status of a proposed Canadian cancer surveillance system is described below.

[2] In lay terms, power refers to the probability of missing an effect (excess risk) if one is present.

In 1992, the AECB published a study with the objective of assessing whether radiation exposure of fathers affected the risk of leukemia in offspring (McLaughlin *et al.* 1992). The study, which included workers at both Bruce and Pickering NGS, did not identify any statistically significant association between childhood leukemia and the fathers' radiation exposure (at any level of dose). Moreover, the study found there was "no apparent gradient of effect with increasing dose (ibid p. 27). Thus, while acknowledging the limitations of the (statistical) power of the study, the authors found no evidence for such a birth risk (McLaughlin *et al.* 1992).

In November 1996, the Durham Region Health Department published a survey, "Radiation and Health in Durham Region". The study compared health indicators, which can be associated with radiation for Durham Region versus Ontario. This was done for municipalities within Durham Region (grouped into Ajax-Pickering, Oshawa-Whitby, Clarington and North Durham) as well. The sources of their data are of high quality: the Ontario Cancer Registry for assessing deaths from cancer and the Canadian Congenital Anomalies Surveillance system for birth defects. The Durham Region Health Department document shows no evidence that emissions from CANDU stations at PNGS or Darlington Nuclear Generating Station (DNGS) have any adverse health impacts on nearby residents. The overall conclusion from the study was as follows:

> "*The surveillance framework developed by the Health Department to categorize health indicators according to their level of association with radiation is a useful tool for assessing whether populations close to the Pickering and Darlington NGSs have higher than expected rates of certain diseases and conditions. Results show no consistent pattern among significant and possible radiological health indicators to suggest that ionizing radiation is affecting the health of Durham Region residents. Inconsistent radiological health indicators show areas of concern with multiple myeloma and prostate cancer but these do not suggest a radiological effect because the patterns are not consistent with known latency periods. Other indicators in this category were at or lower than provincial levels. Theoretical radiological health indicators are difficult to interpret because studies of humans have failed to find elevated rates of these health outcomes in those exposed to ionizing radiation. Only one area of concern was identified -- higher than expected rates of Down syndrome in Ajax-Pickering over the entire 1978-91 time period. The rate of Down syndrome has declined since peaking in 1984-86.*
>
> *Overall, the pattern of results do not suggest adverse health effects in Ajax-Pickering or Clarington from the nuclear generating stations.*" (Durham Health Department 1996).

Finally, a study was initiated by the AECB ("Cancer Incidence Surveillance in Regions Proximal to Canadian Nuclear Facilities Research Project" in the 99/00 AECB Work Programme Summary) and Health Canada, to develop a methodology for monitoring cancer incidence near

nuclear facilities. The purpose of the study is for the development, implementation and testing of a pilot surveillance system for cancer around the Pickering NGS (PNGS). The primary objective of the system would be to assess whether the cancer incidence rates in the population are potentially related to exposures from PNGS. Ultimately, the objective of such a cohort surveillance system would be to assess the potential use of a (large) cohort living reasonably close to a source of environmental contamination to assess the potential cancer risk arising from a source such as Pickering or Bruce NGS. Recently, the CNSC has indicated that "to date, the feasibility study has been prepared and a protocol for the pilot study was partially complete" (Laurier and Bard 1999). The CNSC also states that work on the surveillance system is on hold until the CNSC completes a review of its regulatory mandate (Laurier and Bard 1999).

Studies in other jurisdictions on whether cancer rates are elevated in areas around nuclear facilities also provide useful information on this issue.

UNSCEAR periodically prepares definitive reviews of levels of radiation in the world and current knowledge of the effects of radiation. In its 1994 Report to the United Nations General Assembly, UNSCEAR reviewed the current information on cancer in populations living near nuclear facilities. It considered the results from studies in the United States, the UK, France, Germany and Canada. The overall conclusion was that there was no general increase in cancer deaths in the vicinity of these nuclear facilities (UNSCEAR 1994).

It should be noted that there is always a fluctuation in rates for particular cancers in particular periods, and in particular areas. This is a statistical limitation of epidemiological surveys. Some fluctuations are high; some are low. UNSCEAR (1994) provides examples. In the UK, lymphoid leukemia among persons under 25 years old was found to be increased around nuclear fuel reprocessing facilities but not around nuclear power plants. Mortality from Hodgkin's disease in ages 0-24 years was also increased, but mortality from lymphoid leukemia in ages 25-64 was significantly reduced. In France, communities near nuclear installations experienced slightly lower rates of leukemia in comparison with both national rates and the rates observed in control communities.

A United States study (as summarized in UNSCEAR 1994) was the most extensive. Over 900,000 cancer deaths in 113 counties in the United States containing or adjacent to 62 nuclear facilities were compared to 1,800,000 cancer deaths in control counties with similar population and socio-economic characteristics. UNSCEAR (1994) notes:

> "*Overall, and for specific groups of nuclear installations, there was no evidence that mortality for any cancer, including childhood leukemia, was higher in counties with nuclear reactors than in control counties.*"

C3.0 SCIENTIFIC BASIS FOR ESTIMATING RISKS FROM EXPOSURE TO IONIZING RADIATION

Judgements about the potential health effects arising from exposure to ionizing radiation and the probability that a given radiation dose will cause an effect are provided by international and national committees established for that purpose.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)
This Committee was established by the United Nations General Assembly in 1955 to evaluate the levels of ionizing radiation and radioactivity in the environment and any health effects that may possibly arise. UNSCEAR currently consists of 70-100 scientists (physicists, biologists, physicians and others) from more than 20 countries. The committee reviews scientific literature and, at about five year intervals, produces voluminous reviews of recent scientific information on the levels and current state of knowledge about the biological effects of ionizing radiation. Since 1955, UNSCEAR has issued 13 major reports to the United Nations General Assembly, each containing detailed annexes on discrete topics, on ionizing radiation sources and effects. The most recent UNSCEAR document was published in 2000 (UNSCEAR 2000). Initially, the driving force for formation of UNSCEAR was concern about fallout from the testing of nuclear weapons in the atmosphere and the possible induction of genetic effects in exposed persons. UNSCEAR's scientific reviews have led to a diminution of concern about the role of induced genetic effects. Attention has now shifted to the possibility of developing radiation-related cancers many years after populations are exposed to enhanced levels of radiation.

Over time, UNSCEAR's activities have expanded to assess a wide variety of sources and effects related to ionizing radiation. For example, there is a growing realization of the extent to which people are exposed to natural sources of ionizing radiation, to artificial radionuclides used in medicine, agriculture, industry and from the large growth of a civilian nuclear power production program, to elevated radiation levels from air travel, from exposures of patients from nuclear medicine, from interventional radiology, from new diagnostic and treatment procedures, and from accidents. UNSCEAR is now the major world authority which reviews information on the exposures and effects of ionizing radiation.

Most national authorities, including those in Canada, cite UNSCEAR as the definitive authority on radiation effects, as it furnishes a balanced view of levels of exposure and of health effects from those exposures. The UNSCEAR documents also play an important role in guiding research directions in the field of radiological protection. Perhaps most importantly, the UNSCEAR assessments are employed as guidance by the following commission, which issues the recommendations for radiation protection which are the basis for formulating national regulations in most developed countries.

International Commission on Radiological Protection (ICRP)
The ICRP was formed (under a different name) in 1928 to deal with protection against undue exposure to medical x-rays and to radium. With the advent of nuclear weapons, nuclear reactors and high-energy accelerators, this committee was reorganized and given its present name in 1950.

The ICRP reviews the scientific literature on biological effects of radiation and issues reports with recommendations on various aspects of the protection of humans against all sources of ionizing radiation. The ICRP provides a leadership role internationally with respect to developing both the philosophical structure and the scientific basis for radiation protection. The recommendations of the ICRP are followed in many countries, including Canada.

Biological Effects of Ionizing Radiation (BEIR) Committees
A third important source of information on potential health effects of radiation are the reports of the BEIR committees. These committees operate under the auspices of the United States National Academy of Sciences (NAS) and are funded by the United States Environmental Protection Agency, United States Department of Defense, United States Department of Energy, and the Nuclear Regulatory Commission. Like UNSCEAR, the BEIR reports are concerned only with the assessment of effects and do not make any recommendations on radiation protection as, for example, is done by the ICRP.

Advisory Committee on Radiological Protection (ACRP)
Many industrialized countries have national committees on radiation protection, for example, the National Radiological Protection Board (NRPB) in the UK and the National Council on Radiation Protection and Measurements (NCRP) in the United States.

In Canada, this role was filled by the ACRP, which reported directly to the president of the Canadian regulatory agency, the CNSC. The ACRP reviewed published literature on health effects of radiation, co-sponsored public scientific symposia on relevant topics, and made recommendations on dose limits in Canada. As noted previously, the recommendations of the national committees in Canada and in other countries closely follow the recommendations of the ICRP. In October 2001, the CNSC disbanded their advisory committees. To date, no replacement for the committees has been proposed.

C4.0 STOCHASTIC EFFECTS

The main potential effects of concern arising from exposure to ionizing radiation at low dose and low dose rates are increased risks of cancer in exposed persons and of hereditary (genetic) effects in their offspring. The risks of cancer or hereditary disease arise principally from damage to the nuclear material (DNA) of a cell. Both cancer and hereditary effects, referred to as stochastic

effects, have been studied by UNSCEAR and other committees including the U.S. BEIR Committees and the ACRP.

General Considerations

Quantitative information on radiation-induced cancer is available from epidemiological studies of a number of human populations. These include the survivors of the atomic bombings in Japan and other groups that have been exposed to external radiation or incorporated radionuclides, either at work, in the environment or for medical reasons. Such studies provide direct, quantitative information on the risk of cancer at intermediate to high doses. Recent reviews include those of UNSCEAR (e.g., 1988, 1993, 1994, 2000), and those of the U.S. BEIR Committees (e.g., 1990 and 1992) and the ACRP (1996).

However, for the majority of situations in which people are exposed to ionizing radiation, the principal concern is with exposure at low doses and/or low dose rates. Estimation of the expected incidence of cancer or hereditary disease following radiation exposure in such circumstances is presently based upon the hypothesis that their frequency increases not just monotonically but linearly with radiation dose. Typically, this LNT dose-response model, which assumes that the risk of cancer increases with increasing exposure and that there is no dose below which there is absolutely no risk, has been adopted by national and international bodies for assessing the risks resulting from exposures to low doses of ionizing radiation.

Over a more extended dose range, a linear-quadratic dose-response, again without a threshold and, if necessary, with a cell-killing function at the highest doses, has frequently been applied, and sometimes offers a demonstrably better fit to the data (e.g., radiogenic leukemia). The experimental and epidemiological data on which the LNT model is based have come largely from studies following exposures at moderate to high doses and/or dose rates. Actually, a degree of non-linearity is implicitly recognized by the so-called LNT model. Based on the dose response observed with experimentally-irradiated laboratory animals (NCRP 1980), most national and international organizations have applied a reduction factor (value >1) by which the risks calculated from exposures to low- LET[4] radiation at high doses and high dose rates are divided for application to low doses and low dose rates. This reduction factor has been termed a "dose and dose rate effectiveness factor (DDREF)". The basis for the application of such a reduction factor is described in (UNSCEAR 1993). For high-LET radiation, however, no reduction factor has generally been applied on the basis that the dose response for high LET-induced cancer and hereditary disease is essentially linear up to doses at which cell killing becomes a factor in the dose response.

[4] Abbreviation for low linear energy transfer (LET) radiation such as gamma or beta radiation. High LET radiation is densely- ionizing radiation which deposits a large dose to cells that receive a dose. High LET radiation includes alpha particles and neutrons.

Epidemiological studies give information on dose-response relationships for tumour induction and provide the basis for quantitative risk estimates for human populations. The data available have been the subject of substantive reviews by various committees.

Overall, available data on tumour induction, whether from experimental or epidemiological studies, do not generally provide direct information on the shape of the dose-response relationship at low doses. Because of difficulties in detecting significant increases in risk at low doses, it is unlikely that this position will change in the foreseeable future.

No evidence of any statistically significant increase in genetic or partially genetic defects has ever been observed in any group of irradiated humans that has been studied, including the children of the atomic bomb survivors (ICRP 1991, UNSCEAR 2000).

Through the development and application of modern molecular methods, the understanding of the mechanisms of tumorigenesis has, in recent years, increased substantially. In the future, it is expected that this will lead to biologically-based models for estimating effects at low doses. Coupled with this has been an equivalent increase in knowledge of radiation action in cellular DNA, of control of the reproductive cell cycle, of the mechanisms of DNA repair, genomic maintenance and mutagenesis and of non-mutational mechanisms of stable cellular changes. All this information has potential relevance to assessing the shape of the dose response for both radiation-induced cancer and hereditary disease at low doses and dose rates and in the future will likely be the basis for estimating the effects of radiation exposures at levels below those for which direct information is available.

Cancer Risk

For estimating the potential effects of low doses of radiation, a risk projection model is necessary since it is difficult to observe effects in populations exposed to low doses. The ACRP (1996) provided a summary of the lowest doses at which a statistically significant increase in cancer in various epidemiology studies has been observed[5] (Table C4-1).

[5] This should not be interpreted as equivalent to a threshold.

Table C4-1
Lowest Doses for Detection of Significantly Elevated Effects

Childhood leukaemia and other cancers after X-irradiation of the fetus.	10-20 mSv
Thyroid cancers after X-irradiation of the thyroid gland in children.	60 mSv
Leukemia and other cancers after irradiation of the whole population of the Japanese Atomic bomb survivors.	200 mSv
Bone cancer in adults after ingestion of radium-226.	16,000 mSv (0.8 Gy) to 200,000 mSv (10 Gy)

Other scientists suggest somewhat lower detection "thresholds" of about 50 mSv or so for acute exposure (Pierce *et al.* 1996) and about 10 mGy for in-utero exposure. (Doll 1998)

The Nuclear Energy Agency's expert group, while noting the limitations of epidemiological study at low doses and low dose rates, has indicated that the lack of epidemiological evidence for effects at low doses and dose rates does not prove that such effects do not exist (CRPPH 1998).

In Canada, the ACRP [1401] has considered the LNT relationship as very useful for regulatory purposes, even though it may not always be the best model of the relationship between risk and dose.

The ICRP (1991), in its most recent recommendations for radiation protection purposes adopted the LNT model for projecting risks at low doses. In developing its risk coefficients, the ICRP considered the available epidemiology data, as summarized for example in the UNSCEAR (1988, 2000) and BEIR V (1990) reports, and applied a dose rate reduction effectiveness factor (DRREF) of 2 by which risk estimates derived for acutely-delivered low-LET radiation (such as to the survivors of the atomic bombings at Hiroshima and Nagasaki) were divided. On this basis, the ICRP (1991) adopted the risk (probability) coefficients for stochastic radiation effects, for workers and for the whole population including children, shown in Table C4-2.

Table C4-2
Nominal Risk (Probability) Coefficients for Stochastic Effects

EXPOSED POPULATION	ADULT WORKERS	WHOLE POPULATION
	Risk (% per Sv)	
Fatal cancer	4.0	5.0*
Non-fatal cancer	0.8	1.0
Severe hereditary Effects	0.8	1.3
Total	5.6	7.3**

* = Reduced to 4.4 in ICRP draft recommendations (2005).
** = Reduced to 6.5 in ICRP draft recommendations (2005).

UNSCEAR (1994) based on its review of the available data, concluded that the use of a nominal value of 5% per Sv for mortality (due to excess fatal leukemias and solid cancers) from irradiation at low doses for a population of all ages (4% per Sv for an adult working population) remained valid. Risk estimates developed in the most recent UNSCEAR report (UNSCEAR 2000) are comparable to the earlier estimates.

To provide a context for the applicability of the ICRP risk coefficients, consider that in Canada, about 28% of all deaths in 1991 and 1992 were due to cancer (Doll 1998, Statistics Canada 1995). Radiation exposures from natural sources are about 2 mSv per year in Canada. Assuming an average life expectancy of about 70 years and a theoretical probability of 5×10^{-5} fatal cancers per mSv (i.e., 5% per Sv) for the public, then the theoretical probability that radiation from all natural sources would induce a fatal cancer at some point during an average lifetime would be about 1.0%. Compared to the natural mortality rate from cancer, this theoretically suggests that about 4% of the normal probability of death from cancer in Canada may be due to natural background radiation. Studies of populations which live in areas where the natural background is several-fold higher than average have failed to identify any increased mortality from cancer (UNSCEAR 1994).

Hereditary Risk

The types of permanent genetic changes which can be induced in DNA by ionizing radiation include deletions of part of the DNA, translocations of part of this material from one chromosome to another or to another part of the same chromosome, and inversions of portions of the hereditary code. These same types of damage also occur in the absence of ionizing radiation, which means that no exclusive "molecular signature" of ionizing radiation-induced cancers has been identified yet. Point mutations in the form of DNA base changes can also occur but are relatively less common after exposure to ionizing radiation than in the case of radiomimetic chemicals (UNSCEAR 1993). Deletions, translocations and inversions all result from incorrect repair of initial DNA damage. That is to say, any permanent genetic change in the hereditary material of the germ cell (sperm or ovum) which does not lead to death of the developing embryo is potentially sufficient to produce an inherited disorder in the live-born offspring.

Hereditary effects vary widely in their severity. To date, radiation has not been identified as a cause of hereditary effects in humans; however, effects have been observed in experiments in plant and animals and suggest that genetic effects may occur in humans. The consequences of hereditary effects may range from the undetectably trivial, through gross malformations or loss of function, to premature death (ICRP 1991).

In 2001, UNSCEAR produced a scientific Annex addressing the hereditary effects of radiation (UNSCEAR 2001). UNSCEAR (2001) observed that, to-date, there are no observed hereditary effects on human populations exposed to radiation. However, since hereditary effects have been

observed in experimental studies in plants and animals, UNSCEAR developed risk estimates based on spontaneous mutations in humans and radiation induced effects in mice. Overall, UNSCEAR (2001) used a doubling dose[6] of 1 Sv for risk estimation; essentially the same value used in earlier UNSCEAR reports.

Overall, hereditary risks are assumed to follow a linear, non-threshold dose-response relationship. The ICRP's nominal probability coefficients for hereditary effects are included in Table C4-2.

C5.0 RISKS FROM PRENATAL EXPOSURE

Both developmental effects in infants and late effects such as childhood cancer may be caused by the irradiation of the embryo and fetus. Information on the effects of prenatal exposure can be found in various reports including UNSCEAR 1993, ICRP 1991, and ICRP 1986 which form the basis for the following discussion.

Developmental Effects

Experimental studies with laboratory animals have shown that mammalian embryos are sensitive to ionizing radiation, especially during organogenesis. In humans, the irradiation of pregnant women can result, after high doses, in malformations in offspring. The most frequent site of malformations is the central nervous system resulting in diminution of intelligence, mental retardation in infants and microcephaly following doses in the order of 0.3 -2.5 Gy (UNSCEAR 1993).

Cancer

Cancer can also be induced by prenatal exposure. The induction of childhood cancers, leukemia, and solid cancers as the result of exposure to X-rays has been the subject of much interest since the publication of the Oxford Survey in 1958 which studied the risk of childhood cancer associated with prenatal exposure (Stewart *et al*. 1958).

Notwithstanding that no risks either in childhood or as adults have been observed in the survivors of the atomic bombings exposed *in utero*, ICRP (1991) assumes that the risk of childhood cancers induced from *in utero* exposure are similar to the risks from irradiation as a young child. Doll and Wakeford (1997) suggest that a risk of about 6% per Gy has been demonstrated for exposures above 10 mGy. This is detectable in main because of the very low background rate of cancers in young children. Overall, the value of the absolute risk coefficient for prenatally exposed children is very similar to that for a whole population of all ages, i.e. 5% per Sv (Table C4-2).

6 The doubling dose is the amount of radiation estimated to produce the same number of radiation-induced mutations as occur spontaneously.

C6.0 NON-STOCHASTIC RISKS

Very large radiation doses can injure a large number of cells and result in substantial cell death. If a sufficient number of cells are affected and some threshold dose is exceeded, the function of the tissue or organ may be impaired. An example of this is the routine use of high doses of radiation in medical radiotherapy to kill malignant tissues. Below the threshold dose, no clinically-detectable effect is observed. Above the threshold dose, the greater the number of cells damaged, the greater the effect becomes. Since an effect will be evident above the threshold dose, non-stochastic effects are also referred to as deterministic effects. Various organs including the blood forming organs, reproductive tissues, the skin, and the gastrointestinal tract are considered to be radiosensitive in this respect. (Also currently being studied is the cardiovascular risk resulting from, mainly, high dose medical exposures. This status of information on this subject will be reported on in the next report of the UNSCEAR Committee which is expected in about 2005).

Although there is variation in the sensitivity among individuals for deterministic effects, the threshold levels of dose above which deterministic effects arise are quite well known. For example, the threshold for depression of red blood cell production (i.e., resulting from the exposure of the bone marrow) is about 0.5 Gy of acute exposure and the dose for reddening of the skin (erythema) is between 3 - 5 Gy (UNSCEAR 1993).

C7.0 REGULATORY ISSUES

This section provides a brief overview of several issues related to the regulation of ionizing radiation.

Linear Non-Threshold (LNT) Model

Although either the linear non-threshold model or the linear-quadratic (without threshold) models have been widely used for assessing biological effects at low doses of low-LET radiation, there has been extensive debate as to what the shape of the dose-response relationship is at doses below those at which radiation-related effects can be directly determined. The effects of radiation exposure at low doses and dose rates has been examined in (ACRP 1996). Some evidence from both human and animal studies suggests that in certain cases, notably for the induction of bone cancer by radium-226, a practical threshold dose exists below which the chance of producing a bone cancer within the normal lifespan is virtually zero (ACRP 1996). There is also some evidence of a reduction of cancer rates on exposure to very low doses of radiation, resulting from the stimulation of repair mechanisms (UNSCEAR 1994); overall, however, UNSCEAR concluded

> *"Extensive data from animal experiments and limited human data provide no evidence to support the view that the adaptive response in cells decreases the incidence of late effects such as cancer induction in humans at low doses*" (UNSCEAR 1994).

In reviewing the available data on the health effects of exposure to low levels of ionizing radiation, BEIR VII (1998) has recommended a critical evaluation of all data

> *"that might affect the shape of the dose-response curve at low doses, in particular, evidence of thresholds or lack thereof in dose-response relationships and the influence of adaptive responses and radiation hormesis".*

Addressing the latter topic, however, the Organization of Economic Corporation and Development (OECD) *Nuclear Energy Agency* recently concluded (CRPPH 1998), in its summary of the current status of knowledge in radiation protection research, that

> *"No positive biological effects have been observed in humans exposed to acute doses of ionizing radiation".*

A recent report of the United States NCRP concludes that existing epidemiological data on the effects of low-level irradiation are inconclusive and suggests that

> *"for radiation protection purposes, pending further clarification of dose-response" [that] "no alternative dose-response model seems more plausible than the LNT model"* (U.S. NCRP 2001).

Overall, the advice of the ACRP (1996) which suggests the continued use of LNT for regulatory purposes seems prudent with the caveat that, at low doses and dose rates, there is a possibility of no (excess) risk.

Age and Sex Dependence of Risk Factors

The ICRP (1991) derived nominal values of risk to be used for radiation exposures of workers or members of the public. The risk factors apply to low dose, low-dose rate exposures such as are relevant to this assessment. As previously stated, for members of the public, the risk factor for cancer is 0.06 Sv^{-1}, comprising 0.05 Sv^{-1} for fatal cancers and 0.01 Sv^{-1} for non-fatal cancers (Table C4-2). These values are averages over both sexes and all ages (from birth to 90 y) of the whole population.

The age at exposure is of interest because of the risk projection model used by the ICRP. Age-dependent risk factors have become available in recent years, although there is still considerable

uncertainty in the factors. The risk factor (for fatal cancer) for exposure of children and adolescents to external gamma radiation is larger than the risk factor for the whole population, by about a factor of 2.9 for ages from birth to 9 y and by a factor of 1.7 for ages 10 – 19 y (Almen and Mattsson 1996). Since the risk of non-fatal cancers is considered by the ICRP to be proportional to the risk of fatal cancers, it may be assumed that these age variations also apply to the total cancer risk.

The ICRP (1991) has stated that, although there are differences between the sexes, they are not so large as to necessitate the use of different risk coefficients. The ICRP does however provide age dependent dose conversion factors for internally-deposited radioactivity (ICRP 1996).

Risks from Chemicals and Ionizing Radiation

The World Health Organization (WHO 1993) recommends a limit for tritium in drinking water of 7,800 Bq/L as being acceptable for continuous, life-long consumption (at 2 L/day), based on a dose equivalent to 0.1 mSv per year.

In 1993, the Ontario Ministry of the Environment (MOE) issued a document establishing an interim objective for tritium in drinking water of 7,000 Bq/L based on internationally recommended radiological protection approaches (MOE 1993). Subsequently, in 2000, the MOE confirmed the 7,000 Bq/L limit for tritium (MOE 2000). In 1994, the Ontario Advisory Committee on Environmental Standards (ACES 1994) recommended an interim guideline for tritium in drinking water of 100 Bq/L based on risk considerations (i.e. 10^{-6} lifetime theoretical risk) similar to those assumed for individual chemicals. The different approaches used within these two documents prompted the Ontario Environment Minister to request guidance from Health Canada regarding the apparent differences between acceptable risk levels used in regulating radionuclides and chemicals.

A joint AECB Advisory Committee/Health Canada Working Group was established to address this issue. The Joint Committee focused on the potential for concerns from either ionizing radiation or genotoxic chemicals. The Joint Committee examined the risk assessment and decision making frameworks used for ionizing radiation and genotoxic chemicals. The committee concluded that:

- "*Risk assessment methods for ionizing radiation and genotoxic chemicals are well developed and generally similar in principle.*
- *Radiation risk estimates are based mainly on epidemiological data while genotoxic chemical risk estimates are based mainly on toxicological data derived from laboratory experiments.*
- *In radiation risk assessment, the combined risks for exposures to different radionuclides by different pathways are routinely calculated. This is generally not*

done for genotoxic chemicals, given their varying nature, their large and increasing number, and the synergistic and antagonistic effects which can exist among them.

- *Risk management strategies for both ionizing radiation and genotoxic chemicals are also well-developed and are similar in that they both set legal limits to exposures, endorse the ALARA principle, and employ approaches such as source controls, point-of-use controls, and education.*
- *The Joint Working Group finds that the risk management strategies for regulated practices for both ionizing radiation and genotoxic chemicals provide a high degree of health protection." (Health Canada 1998).*

Maximum Acceptable Concentrations (MACs) of Radionuclides in Water

Guidelines for radionuclides in drinking water conform to international radiation protection methodologies, including specific recommendations formulated by WHO (1993). As a result of the method of dose limitation recommended by WHO (0.1 mSv/y from drinking water), the levels of risk associated with the guideline dose, although low, are somewhat higher than the basic criteria for most individual chemical carcinogens in water. However, for radionuclides, the guideline dose applies to the total dose received from all radionuclides in the water supply, whereas chemicals are regulated singly, often in single media.

Maximum acceptable concentrations (MAC) for radionuclides in drinking water in Canada are based on a committed effective dose of 0.1 mSv per year from consumption of drinking water, consumed at the rate of two litres per day, or one tenth of the ICRP's recommendation of 1 mSv per year on total public exposure from regulated sources (Health Canada 1996). The guideline reference dose is based on the total radioactivity in a water sample, what the radionuclides are, whether the radionuclides appear singly or in combination, and includes the dose due to natural as well as to anthropogenic radionuclides. Individual MACs therefore apply only in the event that a single radionuclide is found in the water supply. If multiple radionuclides are detected, the dose received from all radionuclides should not exceed the guideline dose of 0.1 mSv per year. The guideline reference dose corresponds to a lifetime risk of fatal and weighted non-fatal cancer of about four in ten thousand ($4x10^{-4}$).

As noted previously, the MOE has established regulatory criterion of 7,000 Bq/L for tritium in drinking water (MOE 2000). Ontario nuclear generating stations, the only significant industrial source of tritium in Ontario, have agreed to keep average annual concentrations of tritium in drinking water at nearby municipal pumping stations to less than 100 Bq/L (i.e., < 1/70 of the MAC). Actual monitoring data for tritium at water supply plants near Bruce NGS show annual average levels below 10 Bq/L.

For someone who drinks 2 L of water per day 365 days per year, this value (i.e., 7,000 Bq/L) corresponds to an annual dose of 0.1 mSv, 1/10 of the annual dose limit for a member of the public. For someone who drinks water containing tritium at 100 Bq/L for their entire life, the theoretical lifetime risk of a (fatal) cancer is about five in a million (5×10^{-6}). Drinking water at 10 Bq/L results in a lifetime risk one tenth of this. In any event, the concentrations in water required to reach the MACs for radionuclides are orders of magnitude greater than concentrations currently observed.

C8.0 SUMMARY

People have always been exposed to ionizing radiation: from cosmic rays; from naturally occurring radionuclides in the air, water, and food; and from gamma radiation from the radionuclides in rocks and soils. The level of exposure to this background radiation varies widely depending mostly on where people live and partly on what they eat or drink. Typical annual background radiation doses to members of the public in Canada are about 2,000 μSv per year. This can be compared to the dose limit for nuclear facilities of 1,000 μSv per year above background for a member of the public.

For the purposes of radiation protection, it is widely assumed that the probability of inducing excess cancers in people exposed to ionizing radiation is directly proportional to the total radiation dose received, even at low doses and low dose rates, and that there is no threshold dose of radiation below which these biological effects will not be produced. This is commonly referred to as the linear, non-threshold (LNT) model. This model has, for many years, been regarded as a prudent and reasonable hypothesis for radiation protection. The nominal risk factor used to assess the lifetime risk of a fatal cancer arising from radiation exposure to the general public is 0.05 per Sv [5×10^{-8} per μSv].

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