Health Canada Santé Canada

Atomic Energy Control Board
Commission de contrôle de l'énergie atomique

Assessment and Management of Cancer Risks from Radiological and Chemical Hazards

Canada

Assessment and Management of Cancer Risks from Radiological and Chemical Hazards

prepared by a Joint Working Group
assembled from the AECB's Advisory Committees,
AECB Staff, Health Canada Staff,
and Ontario Ministry of Environment and Energy Staff

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Abstract

A Joint Working Group was established in April 1995 by the President of the Atomic Energy Control Board (AECB) and the Assistant Deputy Minister of the Health Protection Branch of Health Canada to examine the similarities, disparities and inconsistencies between the levels of risk considered *acceptable* for regulating ionizing radiation and those considered *acceptable* for regulating chemical and microbiological hazards. During the process of collecting, analyzing and interpreting information, the Joint Working Group realized that its terms of reference as written presented a major difficulty because of the lack of consensus on *acceptable* levels of risk. Consequently it decided that the most reasonable way to proceed was to compare the risk assessment and management processes used to protect the public from radiation, chemicals and microbiological hazards.

This report concentrates on the assessment and management of ionizing radiation and genotoxic chemicals (which both cause cancer by damaging the DNA in cells) and pays less attention to non-genotoxic effects and microbiological hazards. The report also examines public more than occupational exposures and exposures from man-made rather than naturally occurring agents.

Risk assessment methods for ionizing radiation and genotoxic chemicals are well-developed and generally similar in principle. Both depend upon the establishment of dose-response relationships, and prudently assume linearity with no threshold dose. However, there are often differences in the type of data used, the range over which dose-response relationships are characterized, the identification of organ- and species-specific differences to carcinogenic effects, and consideration of the effects of combined exposures.

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. However, the ALARA principle is applied in different ways for radiation and chemicals. While its formal application is more fully developed in radiation protection, it is not applied in a completely systematic manner in either area.

Recognizing that actual levels of exposure are, in general, well below legal limits and guidelines and that observable health effects are largely absent based on current epidemiological methodologies at these exposure levels, the Joint Working Group finds that the risk management strategies for regulated practices for both radiation and genotoxic chemicals provide a high degree of health protection. It is not possible to determine whether environmental exposures to ionizing radiation or genotoxic chemical carcinogens pose the greater risk of cancer at this time.

The consensus of the Joint Working Group is that it does not appear fruitful at this time to consider harmonizing the regulation of ionizing radiation and genotoxic chemicals; however, future opportunities should be considered. In doing so, consideration must be given as to whether public health benefits would be derived from harmonization. Further, discussions should take place in a broader context in which all relevant public health concerns are addressed. For example, in addition to ionizing radiation and genotoxic chemicals, the impact of microbiological agents on public health should be considered.

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Executive Summary

A Joint Working Group composed of representatives from the Atomic Energy Control Board (AECB) advisory bodies and Board staff, Health Canada, and the Ontario Ministry of Environment and Energy, was established in April 1995 to examine the similarities, disparities, and inconsistencies between the levels of risk considered *acceptable* for regulating ionizing radiation, and those considered *acceptable* for regulating chemical and microbiological hazards. The Joint Working Group was formed by agreement of the President of the AECB and the Assistant Deputy Minister of the Health Protection Branch of Health Canada, in part because of a request from the Ontario Minister of Environment and Energy for advice on these issues.

During the process of collecting, analyzing and interpreting information, the Joint Working Group realized that its terms of reference as written presented a major difficulty because of the lack of consensus on *acceptable* levels of risk for regulated radionuclides, chemicals and microbiological hazards. *Acceptable* levels of these hazards vary with the specific application and substance being considered and, in principle, should be as low as reasonably achievable, taking into account not only the hazard but also the social and economic benefits and the available technology (the ALARA Principle). Consequently, the Joint Working Group decided that the most reasonable way to proceed was to compare the risk assessment and risk management processes used in protecting the public from the hazards (see Table 5 of the report for a brief comparison summary).

As the basis for comparison it was decided to focus on cancer from damage to the hereditary material (DNA) present in the cells, which can be caused by either ionizing radiation or genotoxic chemicals. Consequently, the report makes only passing reference to the risks of non-genotoxic effects and of microbiological substances and considers primarily man-made substances, as opposed to naturally occurring ones, given that it is not usually possible to regulate the levels of the latter. The report examines exposures to the general public more than to workers.

Risk is defined in this report as the likelihood of adverse effects, which is the generally accepted scientific definition. Risk thus incorporates two concepts, the adverse effect, or harm itself and the probability that a person will be exposed to the harm in a given period of time. It should be emphasized that the cancer risks of low level environmental exposure to ionizing radiation and carcinogenic chemicals discussed in this report are essentially theoretical calculated values. There is little reliable or reproducible evidence of observable health effects in human populations exposed to the low levels of the cancer-causing agents to which current regulations, guidelines and objectives apply.

Assessing and Managing Health Risks

Organizations in Canada and elsewhere have developed decision-making frameworks for assessing and managing health risks. The framework developed by Health Canada is used as an example in this report, and includes the main elements of risk assessment and management.

Risk Assessment

Risk assessment includes the identification of well defined health hazards and estimation of the associated level of risk. Ionizing radiation and carcinogenic chemicals exist as a result of both natural and man-made processes. There are a limited number of radionuclides, all of which may cause damage by relatively well-understood mechanisms. On the other hand, there are an essentially unlimited number of man-made chemicals, since more are being synthesized all the time, and harm may result from several different mechanisms, which are generally less well understood.

Since public exposures are almost invariably low for both radiological and chemical hazards, risks are rarely detectable by direct observation. Risks at low doses are therefore predicted from effects observed at high doses using the linear, no-threshold hypothesis (LNTH). This assumption has been widely used in cancer risk assessment in the absence of convincing evidence to the contrary.

Given the nature of the available data, and the need to extrapolate from high to low doses or from animals to humans, estimates of human cancer risk can be subject to considerable uncertainty. This is true even when direct epidemiological data are available under actual conditions of human exposure,

because of uncertainties in exposure estimates, errors in disease diagnosis, and the effects of confounding factors. Thus, when performing risk assessments, a range of possible risks is considered, as indicated by a careful analysis of all sources of uncertainty in the data, and conclusions are generally based on appropriately conservative interpretations.

Radionuclides

Exposure to high absorbed doses of ionizing radiation, delivered at high dose rates, can produce a variety of biological effects, in addition to damaging DNA, including localized burns, acute radiation syndrome (in which damage occurs to rapidly dividing cells such as in the bone marrow and the gastro-intestinal system), and death. Exposure to low doses causes damage to DNA which can lead to cancer of various organs, as well as possible genetic disorders in the offspring of exposed individuals.

Radiation risk estimates are largely derived from extrapolation of epidemiological studies of human populations who were exposed to high doses of radiation in the past. The primary source of information on radiological risk comes from studies of Japanese survivors of the 1945 atomic bombings of Hiroshima and Nagasaki. Studies of groups in which past working conditions and past medical practices have led to appreciable levels of exposure also provide useful data. Some attempts have been made to obtain direct estimates of risk from groups which have received low-dose exposures, such as people exposed to radon in homes and nuclear industry workers. Within the limits of sensitivity of these studies, increased cancer risks have not been identified. While evidence is growing that there may be an effective threshold below which there are no adverse effects of low doses of radiation, as discussed in a recent publication of the Advisory Committee on Radiological Protection (ACRP-18, Biological Effects of Low Doses of Radiation at Low Dose Rate), the linear, no-threshold hypothesis continues to be used as a prudent approach to radiation protection.

The risk of cancer from exposure to ionizing radiation depends upon the type of radiation and the sensitivity of the specific organ irradiated. Radiation exposures are therefore usually given in terms of an *effective dose*, which accounts for these variables through the use of weighting factors. One implication of this is that not all cancers are weighted equally: a fatal cancer is weighted more heavily than a curable cancer. The effective dose provides an aggregated risk from radiation, and serves as a broad indicator of the health risk from any type of radiation and any distribution of dose in the body, whether received internally or externally. The impacts on health from all radiation exposures are additive and so can be treated within one constraint or limit. The total hypothetical impact on health from all radiation exposures to a population of interest can be calculated by the "collective dose" to this population. The collective dose is the total radiation dose received by all members of the population, and includes exposure from all potential pathways.

Chemicals

It is useful to distinguish two types of chemical hazards: those which are carcinogenic and those which produce other health effects such as reproductive/ developmental and neurological/behavioural effects. The carcinogenicity of different chemicals which act by a genotoxic mechanism varies, depending upon sources and routes of exposures, potency, and primary target organs.

Genotoxic chemical risk estimates are often based on predictions from high dosage experiments with laboratory animals or on human epidemiology with relatively uncertain exposures. Although considerable data are available on cancer risks in occupationally exposed populations, informative data available for the general population are more limited.

Extrapolations of chemical cancer risk from animal data to human populations are achieved using a number of cautious assumptions. This approach ensures that the actual risk level will probably be lower than the risk criterion used as the basis for risk management, and in many cases may be substantially lower.

In contrast to radiation risk assessment, chemical risk assessment generally considers all forms of cancer, regardless of their lethality, to be equivalent to each other. This is because cancers observed in animal species may not necessarily occur at the same sites in humans, primarily due to inter-species kinetic and metabolic variations.

Risk assessment for combined exposures to chemicals is still at an early stage of development for a number of reasons: most cancer risk assessments determined from animal studies involve exposure to individual carcinogens, often via only one exposure pathway; chemicals may interact with many different sites in the body and in many ways; and the effects of combined exposures are not always additive, with synergistic or antagonistic effects often occurring. Such effects are largely unknown and are often difficult to characterize. Although aggregation of risks associated with different chemicals is difficult, in some cases the combined risk of several substances within the same chemical class can be established.

Microbiological Hazards

Microbiological agents in food and drinking water, including bacteria, protozoa, viruses, and fungi, do not typically cause cancer, although they can cause a variety of other illnesses. Such agents may produce toxins which can cause health effects ranging from short-term, mild symptoms to long-term or life-threatening illnesses. Alternatively, they may produce an infection which can have pathological consequences. Both qualitative and quantitative methods are used for risk assessment, depending on the availability of data.

Risk Management

Risk management is the process in which the results of risk assessment along with other considerations are used to select one or more strategies for controlling a risk. Risk management methodologies for ionizing radiation and genotoxic chemicals

are generally well developed. Guidelines used for risk management are set on the basis of the estimated magnitude of risk, as well as consideration of benefits, and technological, economic, and social factors.

The federal government has jurisdiction over activities that are considered to be of national interest, or of inter-provincial or international concern, and in setting minimum standards for protecting the health and the environment of all Canadians. Provincial and territorial governments are responsible for the health and safety of their citizens, and have jurisdiction over industries within their borders, and in setting and enforcing provincial standards for health. In general, these standards cannot be less stringent than federal standards or requirements. Nuclear industries are regulated federally. Chemical industries are regulated primarily by the provinces.

Approaches to risk management by the responsible federal and provincial authorities generally take the form of source control, point-of-use control, or educational strategies. Management options include legally enforceable limits, regulations, and standards, as well as recommended operating targets, guidelines, or goals for source and point-of-use controls.

Radionuclides

Radiation risk-control strategies developed under the assumption that a balance of the risks and benefits of radiation and radiation-producing technologies was necessary. In Canada, as in most countries of the world, the recommendations of the International Commission on Radiological Protection (ICRP) form the basis for risk management involving regulated practices. All such practices must produce a net benefit to society, be optimized with respect to benefits versus costs, and include a system of dose limitation for individuals. While legal dose limits have been established for occupational and public radiation exposures arising from regulated practices, they do not apply to exposures from natural sources, or to exposures of patients from medical applications.

Laws governing the use of radioactive materials, radiation emitting devices and ionizing radiation exposures exist in Canada at both the federal and provincial levels, and are generally applied at the source. The principal legal instruments at the federal level are the *Atomic Energy Control Act*[1] and *Regulations*, and the *Radiation Emitting Devices Act* and *Regulations*. The *Atomic Energy Control Act* regulates, among other things, the use of radioactive and fissionable materials or processes that could be used in a nuclear chain reaction. This *Act* is administered by the AECB, which has the lead role in the regulation of nuclear facilities and the use of nuclear materials. The *Radiation Emitting Devices Act*, administered by Health Canada, pertains to specific classes of radiation emitting devices used both occupationally (e.g. X-ray equipment, lasers, ultrasound therapy devices) and residentially (e.g. microwave ovens, television receivers). Natural radiation is not covered under either *Act*.

The dose limits recommended by the ICRP for occupational and public exposures are generally adopted by regulators, including the AECB, for legal purposes and must not be exceeded under normal circumstances. The public dose limits apply to the sum of all exposures from all regulated practices and do not represent a threshold between *safe* and *unsafe*, but are based on a consideration of estimated health risk in comparison with the risks generally accepted in society. These public dose limits are believed by the ICRP to be sufficiently restrictive to protect the survival of other species. The AECB sets a legal annual limit to the radiation risk of individuals in the population group at greatest risk (i.e. the critical group) for licensed activities (expressed as an annual exposure) and then requires licensees to apply an ALARA process to derive a level of exposure (typically a few percent of the legal limit) that should not be exceeded under normal conditions. Thus, the latter exposure defines the *acceptable* level of risk for radiation.

At present, the current legal limits in Canada are 50 millisieverts per year (mSv/year) for occupational exposures, and 5 mSv/year for public exposures. The ICRP has recommended dose limits of 20 mSv/year averaged over five years for occupational exposures, and 1 mSv/year for public exposures, from all licensed practices. The AECB is in the process of adopting the latest ICRP recommendations on dose limits. The new public dose limit is about half the average exposure to radiation from natural sources in Canada and less than its variation across Canada.

As a condition of licensing, the AECB requires nuclear facilities to ensure that, under normal operating conditions, maximum doses to members of the public by all pathways are kept to a few percent of the legal annual limit. Actual maximum annual doses are less than these operating targets and annual doses to individual members of the population at large are smaller still.

Chemicals

Chemical risk management practices developed from the early assumption that the public could be completely protected from all risk, and thus that no level of risk was acceptable. While the ultimate goal of risk management for chemical hazards, though limited by practical constraints, is to reduce risk to levels that are as low as possible, in recent years consideration has been given to balancing risks and benefits. For chemicals, the ALARA principle is applied in setting the guidelines or legal limits for the risk, which thus constitutes the *acceptable* risk. As a consequence of the adherence to ALARA, the *acceptable* risk varies from application to application. Currently, the presence of natural sources of carcinogens is being given increasing attention in risk management strategies, although for many chemicals significant natural sources are absent.

In Canada, the management of risks from chemicals is primarily a provincial responsibility, although the federal government exercises regulatory control under some pieces of legislation, including the *Canadian Environmental Protection Act*, the *Food and Drugs Act*, and the *Pest Control Products*

1. A new act to replace the *Atomic Energy Control Act* has been passed by Parliament. The new *Nuclear Safety and Control Act*, which recognizes the many changes since 1946 when the existing Act was passed, had not yet come into force at the time of publication of this report.

Act. Both source controls and point-of-use controls are applied. Regulatory limits for industrial, commercial, institutional and other releases of chemical contaminants to air, water, and land are established under provincial authority. Exposure guidelines for chemical carcinogens in drinking water, food and air are established for individual pollutants through federal-provincial-territorial discussion. Although management approaches and requirements are generally similar across the country, the details vary amongst provinces and territories, depending on the regulations which apply. Ontario has been used as an example in this report, but is not intended to be representative of other provinces or territories.

Risk management decisions are made following consultation with affected parties, and involve judicious balancing of the estimated risks against the associated costs, feasibility of controls, and benefits to society. For example, management strategies pertaining to Priority Substances which are found to be toxic under the *Canadian Environmental Protection Act* take into account risks, benefits, and costs, including the cost effectiveness of available control technologies.

Using applicable legislation, public exposure limits are established for individual carcinogens and are dependent on the above factors, and background levels of the chemical of concern. Limits apply to individual chemicals, often via only one route of exposure. Given the growing number of potential chemical carcinogens, and the complexities associated with identifying the effects of combined exposures, it is not possible to calculate the total risk associated with all individual limits.

In general, regulations governing chemical releases into the environment are based on both ecological and human-health effects. For carcinogens, release limits are established on a case-specific basis, with some consideration given to the presence of natural sources, the available scientific information, and the implications of proposed controls. In Ontario, for example, short-term (30 minutes) atmospheric emissions of carcinogens must not exceed Point of Impact (POI) standards. These POI standards are set at a factor of 15 times the annual ambient air quality criteria. Such ambient air quality criteria are case specific, but generally based on a lifetime risk of cancer of one in a million to ten in a million for specific chemicals, in the absence of significant technical and economic limitations.

Microbiological Hazards

Risk management practices for microbiological hazards are usually not directed towards achieving a defined level of risk, but rather towards reducing the risk to the extent possible and then minimizing its reoccurrence. This is because microbiological hazards, unlike radiological or chemical hazards, are highly sensitive to environmental conditions such as temperature changes. Point-of-consumption approaches are used in the management of microbiological hazards, and are carried out at both federal and provincial levels. Risk management strategies to control microbiological hazards are usually developed on an *ad hoc* basis rather than on the basis of quantitative risk assessments. Microbiological risks are managed by various means, including surveillance of human infections and disease, and monitoring of microbiological pathogens in the environment.

Drinking Water

The *Guidelines for Canadian Drinking Water Quality* are described in the report as an example of how radiological, chemical, and microbiological risk assessment and management practices are combined within a flexible risk control strategy. The *Guidelines* have been established through the Federal-Provincial-Territorial Committee on Environmental and Occupational Health, and are intended to facilitate consistency in drinking water quality across the country. The *Guidelines* have been designed to accommodate the diverse needs of the various jurisdictions involved. Although not mandatory, the *Guidelines* may be used by the provinces and territories as a basis for setting maximum permissible levels for radionuclides, chemicals, and microbiological hazards. Since water quality is essentially a provincial responsibility in Canada, the provinces may adopt the *Guidelines* in whole or in part, or may establish their own criteria.

Guidelines for radionuclides in drinking water are based on a reference dose (0.1 mSv per year) which applies to the total dose from all radionuclides in the water supply, whatever their source. This is in conformance with international radiation protection methodologies recommended by the World Health Organization. Actual concentrations of radionuclides, particularly in surface drinking waters, are usually orders of magnitude (e.g. 100 fold) lower than the guideline value.

For individual carcinogenic chemicals in drinking water, guideline values are set on the basis of achievability at a reasonable cost, and the reliability of detection. It is not instructive to compare lifetime cancer risks associated with the guideline values for chemicals in drinking water to those for radionuclides since the former may represent only a small proportion of the number that may be present while the latter are for all radionuclides combined. For reasons noted previously, it is not possible to evaluate or regulate the total risk from all chemical carcinogens combined.

For microbiological hazards in drinking water, risk management strategies do not attempt to achieve a defined level of risk but mainly to treat drinking water so as to reduce microbial organism concentrations to very low levels and to prevent re-contamination by appropriate technical means.

Conclusions

Risk assessment methods for ionizing radiation and genotoxic chemicals are well-developed and generally similar in principle. Both depend upon the establishment of dose-response relationships, and prudently assume linearity with no threshold dose. This enables the estimation of risk well below the observable range for health protection purposes. However, there are often differences in the type of data used, the range over which dose-response relationships are characterized, the identification of organ-and species-specific differences to carcinogenic effects, and consideration of the effects of combined exposures.

Radiation risk estimates are based mainly on epidemiological data while genotoxic chemical risk estimates are based mainly on toxicological data derived from laboratory

experiments. As a result, organ-specific susceptibilities have been established for radiation but not for genotoxic chemical exposures. Where past working conditions and medical practices have led to appreciable levels of human exposure, this experience has been useful in identifying carcinogenic agents and in establishing dose- response relationships, particularly for radiation. 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.

Because humans may be exposed to more than one carcinogenic agent (e.g., a genotoxic chemical and radiation) simultaneously, there is a need to consider risk assessment methods for joint exposures and mixtures. The simple approach is to assume that risks are additive but in some cases interactions, especially synergistic effects, may exist and need to be taken into account when assessing risks.

Risk estimates can be subject to considerable uncertainty, particularly when extrapolation is necessary beyond the conditions under which the original data were collected. Such uncertainties are believed to be smaller for ionizing radiation than for genotoxic chemical hazards. This is mainly because of the type of data generally used, and a greater understanding of the mechanisms of radiation carcinogenesis compared to those for chemical carcinogenesis. The Joint Working Group recognizes that it is important to characterize uncertainties in all risk estimates and urges that this be done to the extent possible.

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. However, the ALARA principle is applied in different ways for radiation and chemicals. While its formal application is more fully developed in radiation protection, it is not applied in a completely systematic manner in either area.

There is a lack of consensus regarding levels of risk acceptability for ionizing radiation or genotoxic chemical hazards. Rather, the *acceptable* levels of risk associated with established guidelines vary up to a million-fold. These guidelines take into account to varying degrees the specific application and agent or process being regulated, the economic and social costs and benefits and technology factors.

Recognizing that actual levels of exposure are, in general, well below legal limits and operational target levels and that there is an absence of observable health effects by current epidemiological methodologies at these exposure levels, 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. It is not possible to determine whether environmental exposures to ionizing radiation or genotoxic chemicals pose the greater risk of cancer at this time.

The consensus of the Joint Working Group is that it does not appear fruitful at this time to consider harmonizing the regulation of ionizing radiation and genotoxic chemicals. Future opportunities for harmonization should, however, be considered. In doing so, consideration must be given as to whether public health benefits would be derived from harmonization. Further, discussions should take place in a broader context in which all relevant public health concerns are ddressed. For example, in addition to ionizing radiation and genotoxic chemicals, the impact of microbiological agents on public health should be considered.

1. Introduction

In 1994 the Ontario Advisory Committee on Environmental Standards [ACES, 1994] recommended an interim guideline for tritium in drinking water of 100 Bq/L (becquerel per litre) based on risk considerations similar to those for individual chemicals. At about the same time, the Ontario Minister of Environment and Energy (OMEE) issued a document establishing an interim objective for tritium in drinking water of 7,000 Bq/L based on internationally-recommended radiological protection approaches. The different approaches used within these two documents prompted the OMEE Minister to request guidance from Health Canada regarding the apparent differences between acceptable risk levels used in regulating radionuclides and chemicals.

In January of 1995, Joint Working Group 6 (JWG-6) of the Advisory Committees of the Atomic Energy Control Board of Canada (AECB) was formed at the request of the President of the AECB. The purpose of the Joint Working Group was to conduct a close examination of the disparities and inconsistencies in the levels of risk considered *acceptable* for regulating radioactive materials and those in use for regulating industrial chemicals and pesticides. The Joint Working Group was to be composed of representatives from the Board's Advisory Committees on Nuclear Safety (ACNS) and Radiological Protection (ACRP) and the Group of Medical Advisors (GMA). These advisory bodies are composed of individuals selected by the AECB for their scientific expertise.

In response to the OMEE request for advice, and given the establishment of JWG-6, the Assistant Deputy Minister of the Health Protection Branch of Health Canada proposed to the President of the AECB that the Joint Working Group be expanded to include Health Canada representatives. This proposal was accepted, and the first meeting of the Joint AECB Advisory Committees/Health Canada Working Group was held on April 27, 1995. The membership list is provided in Appendix A. After some discussion, the Terms of Reference were established as follows: "To examine the similarities, disparities, and inconsistencies between the levels of risk considered *acceptable* for regulating ionizing radiation and those considered *acceptable* for regulating chemical and microbiological hazards". Further details are provided in Appendix B.

During the process of collecting, analyzing and interpreting information, the Joint Working Group realized that its terms of reference as written presented a major difficulty because of the lack of consensus on *acceptable* levels of risk for regulated radionuclides, chemicals and microbiological hazards. *Acceptable* levels vary by up to a million-fold, as will be described later in this report, depending upon the specific application and substance being considered. In principle, *acceptable* levels should be as low as reasonably achievable, taking into account not only the hazards but also the social and economic benefits and the available technology (the ALARA Principle). Consequently, the Joint Working Group decided that the most reasonable way to proceed was to compare the risk assessment and risk management principles and practices used in protecting the public from the hazards associated with regulated radionuclides, chemicals and microbiological agents.

As the basis for comparison, it was decided to focus on cancer resulting from damage to the hereditary material present in the cells, which may be caused by either ionizing radiation or genotoxic chemicals. This material, called deoxyribonucleic acid (DNA), contains coded instructions for all life processes in living cells. It should be emphasized that the cancer risks of low level exposure to radiation and carcinogenic chemicals discussed in this report are essentially theoretical calculated values. Except for tobacco smoke and possibly radon gas, there is no reliable or reproducible evidence of observable health effects in human populations exposed to the low levels of cancer-causing agents to which current regulations, guidelines and objectives apply. For health protection purposes, it is assumed that the dose-response relationship for ionizing radiation and genotoxic chemicals is linear with no threshold dose below which deleterious effects would be absent; this implies that there is a probability of an adverse health effect at any level of exposure, no matter how low.

Potential effects on humans from exposure to extremely high levels of ionizing radiation or genotoxic chemicals were not considered in detail. Some comparison is made with risk assessment and management practices associated with naturally occurring radionuclides and chemicals, non-carcinogenic chemicals, microbiological hazards, and occupational exposures. Risks associated with communicable diseases were

considered to be beyond the Terms of Reference of the Joint Working Group. Although the Joint Working Group recognizes that differences in risk management approaches exist among provinces and territories, Ontario has been chosen as the working example in this report.

Copies of earlier drafts of this report were sent for comment to selected scientists in Canada, the United States, and the United Kingdom, and to members of the AECB Advisory Committees and Group of Medical Advisors and the Federal-Provincial-Territorial Committee on Environmental and Occupational Health (see the *Acknowledgements* section of this report for more information).

2. Frameworks for Health Risk Assessment and Management

Protection of public health from environmental risks is complex and involves many considerations. Decision-making frameworks have been developed by several organizations in Canada and elsewhere, to provide structured approaches to health risk assessment and management [Krewski and Birkwood 1987a, 1987b].

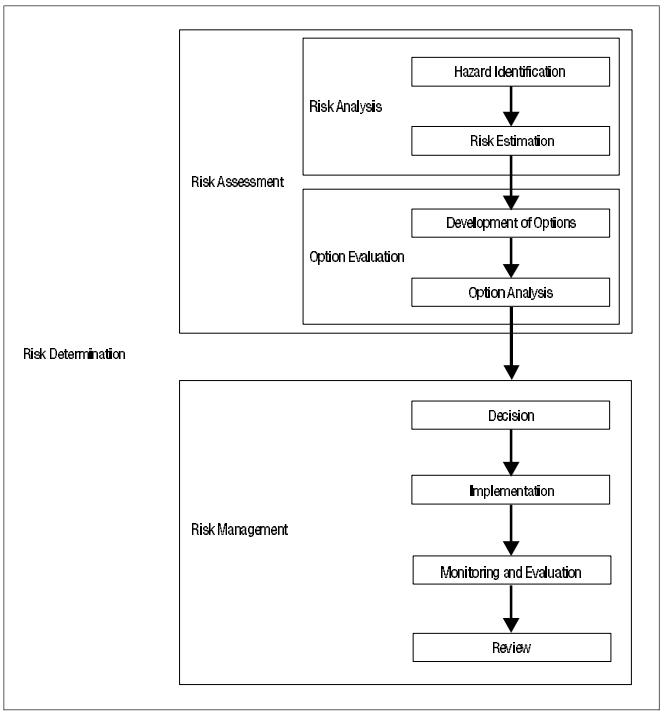
Besides providing an analytical and structured guideline for decision-making, these frameworks also provide the flexibility to address specific health hazards as required. While frameworks are generally consistent in principle, differences may exist in terminology, scope of considerations, level of detail, and the role of factors such as risk communication, and the involvement of stakeholders in the overall process [Krewski and Birkwood 1987].

While there is no *Canadian* framework for risk assessment and risk management, two relevant consensus standards have been developed by the Canadian Standards Association. One of these provides general requirements and guidelines for selecting and implementing risk analysis techniques primarily for technological hazards [CSA 1991]. The other is intended to assist decision makers in managing all risks, including those pertaining to health and environmental hazards [CSA 1997].

The framework developed by the Health Protection Branch of Health Canada is used as an example in this report to illustrate the general process of risk assessment and risk management. This framework was developed as a guideline to assist the Branch in protecting health and safety with respect to foodborne, drug-related, and environmental risks, as well as controlling disease and injury (see Figure 1).

Within this framework, risk is defined as a product of the hazard to health from exposure to an agent, and the probability of its occurrence [HPB 1990]. Risk may be defined differently by other agencies. Risk assessment consists of four steps: hazard identification, risk estimation, development of options, and option analysis. Risk management also consists of four steps: decision, implementation, monitoring and evaluation, and review. Communication with stakeholders may occur at any step of the risk assessment/risk management process.

Figure 1. Risk Assessment/Risk Management Framework



Source: Health Risk Determination:
The Challenge of Health Protection [HPB 1993]

Hazard identification involves recognition that a particular agent (e.g. a specific chemical) can cause a specific adverse health outcome. Hazards may be identified through various means, including epidemiological investigations, toxicological studies, and, in the specific case of chemicals, structure/activity analysis.

Epidemiological studies provide information about health hazards in humans and, in the case of radiation risk assessment, are the primary source of data. However, they can be difficult to conduct due to high costs and the complexity of the human

environment, possible insensitivities to small effects, and long-term study periods, possibly of many years [Health and Welfare Canada 1991]. Consequently, they are often supplemented with other types of investigations, as discussed below.

Toxicological experiments are typically performed in laboratories on non-human models, and are widely used to identify possible human health hazards, especially for chemicals. Extrapolations are used to relate the results of tests involving high doses of substances in different species to relatively low doses of substances in humans. Highly sensitive tests are available to examine a variety of deleterious effects, including tests of acute and chronic toxicity in animals, metabolism of chemicals, reproductive and developmental effects, and long-term and carcinogenic effects.

Biological markers are useful in the study of chemical hazards. These are biochemical changes that indicate that an exposure has occurred, but that are not necessarily linked to a clinically harmful effect. They may be studied to evaluate exposure, health effects or susceptibility, to assess intra- and inter-subject variability, to clarify mechanisms, or to identify dose-response relationships. Their ultimate usefulness is the extent to which they can predict disease occurrence.

Structure/activity relationship studies use the chemical structure of a compound to predict toxic or carcinogenic effects. Predictions are often based on the known behaviour of similar compounds considering specific properties and attributes. However, while such classification rules are useful, they are not perfect predictors of health effects.

Risk estimation involves determination of the likelihood that a particular adverse health outcome will occur. *Hazard identification* and *risk estimation* comprise the process of *risk analysis*. Risk estimates may be obtained through quantitative analysis of toxicological or epidemiological data. Given that these scientific data are often incomplete or not available however, such estimations must often be supplemented with more qualitative approximations and consideration of uncertainties.

Option evaluation involves the *development* and *analysis of options* to control risk. Such options may be regulatory or non-regulatory in nature, depending on such factors as the mandate of the organization, the program objectives and policies, the current regulatory environment and the availa- bility of non-regulatory alternatives. Options are evaluated in light of several factors including: the nature of the health hazard involved and the likelihood of its occurrence; uncertainties in risk estimation; public perception of risk; the health benefits and technical feasibility of the option; economic and environmental impacts; social, political and cultural concerns; and the viewpoint involved (e.g. individual or societal).

Risk management begins once a *decision* is made (i.e. an option is selected) and the necessary resources committed. *Implementation* is accompanied by communication with affected parties in order to improve their understanding of the changes which are to take place.

Monitoring and *evaluation* of the impacts of the decision are conducted in order to determine its effectiveness. Techniques include environmental sampling, post-market surveillance, prospective epidemiology, evaluation of new health risk information, and formal and informal information gathering from the public.

The next step in the process involves *review* of new information, which may lead to a reconsideration and revision of any previous step in the process.

3. Risk Assessment

Risk assessment for ionizing radiation has developed within a markedly different framework than has been the case for chemicals. Radiation risk assessment has been largely derived from long-term follow-up studies of humans exposed to relatively well-known high doses of radiation, while chemical carcinogen risk assessments are more often based on projections from high dosage experiments with laboratory animals or on human epidemiology with relatively uncertain exposures. While various effects have been observed at high exposures, the primary effect predicted at lower doses is cancer. Cancers caused by radiological and chemical agents have been observed in virtually every organ of the body, depending upon the agent in question, the species, and the conditions of exposure [NCRP 1989]. The appearance of the induced cancer is generally preceded by a long latency period, which varies by the type of malignancy and age at time of exposure. In general, the same types of cancers are observed in exposed individuals as those observed in unexposed populations (though there are exceptions such as mesothelioma, which is associated with exposure to asbestos).

Whereas the number of radionuclides is relatively well known, the number of chemicals suspected of being carcinogenic continues to increase each year, although not all are detected in the environment. ICRP Publication 38 [ICRP 1983] lists just over 800 radionuclides. About 50 of these are of potential concern because of their abundance in emissions or wastes or their toxicity. There are a variety of estimates of the number of chemicals which can cause cancer. At present, some 69 agents or industrial processes have been shown to cause cancer in humans [IARC 1995]. Chronic long term studies in rats and mice have shown that more than half the 1300 chemicals tested in the Carcinogenic Potency Database are capable of causing cancer at high exposure levels [Gold et al 1997]. Ames *et al* [1990, 1990a] have estimated that about half of all chemicals tested, whether synthetic or from natural sources, will cause cancer when fed in high doses to animals over long periods of time.

Since environmental exposures are low for both radiological and chemical hazards, risk levels are rarely detectable from direct observational studies of human populations. To assess the risk at lower doses, an appropriate dose-response model must be selected in order to extrapolate from observed high-dose effects to estimated low-dose effects. Similar models for quantitative risk assessment are used for both radiation and chemicals. Although dose-response curves may be non linear at high doses, it is generally assumed that the dose-response curve for ionizing radiation and genotoxic chemical carcinogens is linear at low doses. Since threshold doses are assumed not to exist, some risk is presumed to exist even at the lowest dose levels, although unobserved and unobservable [NCRP 1989].

A desirable feature for any model is the ability to provide the best estimate of the risk and an indication of its uncertainty. With the limited information provided by epidemiological and toxicological studies, it is possible to postulate different models that fit the data equally well, but which provide estimates of risk at low doses that differ by several orders of magnitude [Food and Drug Administration 1971]. Biologically-based models could provide a more realistic basis for risk estimates by incorporating toxic mechanisms of action [Goddard and Krewski 1995]. This would assist in evaluating model-based predictions of risk and in extrapolating beyond the conditions under which the original data were obtained.

The multistage model, the most widely used model for cancer risk estimation, is based on the number of stages in the carcinogenesis process [Armitage and Doll 1961]. For practical applications, Crump and Howe [1984] proposed the modified, *linearized multistage model*. Another class of biologically-based models of carcinogenesis [Moolgavkar and Luebeck 1990] is based on the assumption that initiated cells are formed following the occurrence of a single mutation in a normal stem cell and that initiated cells can sustain a second mutation and progress to a cancerous cell. The initiated cell population can also be promoted by clonal expansion, increasing the pool of cells available for progression to malignancy. An important advantage of this type of model is that its parameters are interpretable in biological terms and can, in some cases, be obtained experimentally.

Physiologically based pharmacokinetic (PBPK) models are an important tool in mechanistic modelling. Broadly, the focus of PBPK models is to predict the dose of reactive metabolites which reach target tissues. The use of an appropriate

measure of tissue dose rather than an external measure of exposure can lead to more accurate estimates of low-dose cancer risks [Krewski *et al* 1994].

Due to the lack of directly observable effects at low doses, estimates of cancer risk are subject to uncertainty. Some uncertainty is due to inherent variability, such as measurement error in dose and exposure estimates. Actual effects may also depend on physiological parameters such as body weight, respiratory rate and cardiac output, which vary among individuals. Sometimes, only incomplete or subjective information is available for risk estimation. For example, exposure estimates in epidemiological studies on chemicals may be uncertain since historical information on individual exposures may be poorly documented. Additional sources of uncertainty include determination of health outcomes, extrapolation from animals to humans, and extrapolation between routes of exposure. Risk estimation may also be highly sensitive to the choice of dose-response model. These uncertainties are believed to be smaller for the estimates of radiation-induced cancer risk, which are primarily based on human population studies, than for risk of cancer induced by chemicals, which are often based on studies of animals.

3.1 Ionizing Radiation

When ionizing radiation passes through matter, including tissue, it deposits some of its energy in the traversed material as a result of electrical interactions. The resulting ionization of body tissue causes chemical changes in the irradiated cells that can potentially lead to biological damage. The fundamental dosimetric measure of this energy transfer is the *absorbed dose*, which is defined as the amount of energy deposited by the radiation in the tissues and organs of the body. The unit of absorbed dose is the gray (Gy); one Gy is an absorbed dose of one joule of energy per kilogram of material irradiated. The absorbed dose is independent of the type and energy of the radiation; however, the extent of radiation damage varies with the type and energy of the radiation. Absorbed dose is therefore multiplied by a radiation weighting factor to give an equivalent dose to the exposed organ, which is measured in sieverts (Sv). Weighting factors of 1 to 20 have been assigned to the different types of radiation [ICRP 1991].

The harm induced by radiation exposure has also been found to depend on the specific organ or tissue irradiated. The risk of induced cancer or hereditary (genetic) disorders varies between organs for the same equivalent dose. To account for the various susceptibilities of the organs and tissues, a set of tissue weighting factors has been developed [ICRP 1991]. The effective dose, in sieverts, is obtained by multiplying the equivalent dose in each organ by the corresponding tissue weighting factor, and summing the result for each organ to give a total effective dose to the body. Tissue weighting factors range from about 0.01 for skin and bone surfaces to 0.2 for the gonads. The sum of all tissue weighting factors is one; therefore, a uniform dose over the whole body will give an effective dose numerically equal to the equivalent dose. For low levels of radiation, the harm resulting from a given effective dose will be approximately the same regardless of the type of radiation or the tissues irradiated.

Radionuclides taken into the body by inhalation, ingestion, or absorption through the skin may remain in certain tissues and organs for extended periods of time; in some cases, the resulting dose to the internal organs may extend over several days or years. The *committed dose* is the total effective dose received from a radioactive substance in the body during the remainder of an individual's life, taken to be 50 years for an adult, and 70 years for a child. The committed dose is implicitly included in any calculations of effective dose. The total *effective dose* therefore serves as a broad indicator of the risk to human health from any type of radiation and any distribution of dose in the body, whether received internally or externally. The impact on health from all combined exposures can therefore be treated within one constraint or limit. This unifying approach to radiation risk assessment is possible because it is generally agreed that, regardless of the radiation source or the tissue irradiated, the types of health effects are similar. This makes for significant differences from current practice with carcinogenic chemicals.

The *committed dose* depends on the chemical form of each radioactive isotope, any selective uptake into target organs or tissues, internal metabolism and rate of elimination from the body, and age of the individual at the time of intake. Tables of committed tissue equivalent dose and committed dose per unit intake are published in ICRP Publications 30, 61, 67-69, and 72 [ICRP 1979-1988, 1991a, 1993a-1996]. Dose coefficients are derived primarily from human data, supplemented by studies in laboratory animals, and are calculated using standard biokinetic methods and human reference models.

Another useful concept is that of the *collective dose*, which is a measure of the total radiation dose to a group of people or a whole population. The collective dose is obtained by summing the doses received by all individuals in a population from all exposure pathways. The unit of measure is person-Sv.

Effective dose is frequently abbreviated to dose, and collective effective dose to collective dose. This terminology is adopted in subsequent usage in this document.

3.1.1 Biological Effects of Ionizing Radiation

Exposure to ionizing radiation can cause two kinds of health effects. High absorbed doses of radiation delivered at high dose rates, for example 5,000 mSv in a few minutes, can produce a variety of effects, including death within a few weeks or months after exposure. These effects result from the inability of the body to cope with the damage associated with the death of a significant number of cells in certain tissues or organs. The severity of these early effects, such as radiation burns from localized exposure, or acute radiation syndrome (ARS) due to whole body exposure, increases with dose above a clinical threshold. ARS represents the clinical expression of damage to many important organs but particularly those in which cells are subject to continual and rapid replacement such as the bone marrow and gastro-intestinal system. The threshold for

observable early effects such as nausea or temporary blood cell changes is about 250-500 mSv in a short period of time [ICRP 1991]. The average doses received by members of the public in Canada from natural sources are typically about 2 mSv per year, and from routine exposures from regulated practices are about 0.0001 mSv per year (derived from Table 4, footnote b, assuming a Canadian population of 30 million). These doses are far below the threshold doses cited above.

Low doses of radiation [less than 200 mSv; UNSCEAR, 1993] may result in effects that are manifested later in life. The effects of primary concern associated with low doses of ionizing radiation are an increased incidence of cancer in exposed persons, and potential genetic disorders in their offspring. The probability of occurrence of late effects is assumed to be proportional to dose, and it is generally assumed that there is no threshold below which they do not occur.

Late effects arise as a result of damage to the DNA. Usually, cellular damage is repaired through a natural process; however, if it is not adequately repaired, it may result in a viable but modified cell. The reproduction of a modified somatic cell may result, after a prolonged and variable latency period, in the appearance of a cancer. The risk of cancer is the principal concern in radiation protection. Specific cancers observed in exposed populations include leukemia and cancers of the thyroid, lung, breast, and bone. Damage occurring in a cell whose function is to transmit genetic information may result in effects which are expressed in the offspring of the exposed individual. Although hereditary effects due to radiation have been observed in experimental animals, there is no direct evidence of their occurrence in humans.

Unlike responses to chemical exposures, there are no known immunological hypersensitivity responses to radiation exposure. Differences in individual radiation responsiveness which have been presumed to be due to differences in the efficacy of DNA repair are not believed to be immunologically related.

3.1.2 Quantitative Risk Assessment for Ionizing Radiation

Estimates of radiological cancer risk are based on epidemiological studies of human populations exposed to high doses of radiation. The main source of information on the risk of radiation-induced cancer following whole-body exposure to external radiation comes from the follow-up studies on the Japanese survivors of the 1945 atomic bombings of Hiroshima and Nagasaki. Other studied populations include miners exposed to high concentrations of radon and its decay products in air, early radium dial painters who inadvertently ingested appreciable amounts of radium, and patients treated with high doses of medical X-rays, or given radium-224, radium-226 or Thorotrast (thorium oxide). Additional information has been derived from extensive experiments on animals and other organisms. Since no significant excess of hereditary diseases has been observed even in the children of the Japanese bomb survivors, estimates of this probability are derived from studies on experimental animals.

Information of this nature is reviewed periodically by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), which publishes a series of authoritative reports to the United Nations General Assembly at about five-year intervals; the last UNSCEAR report was published in 1994. The International Commission on Radiological Protection (ICRP) reviews the body of scientific literature on the biological effects of radiation, and issues reports with recommendations on various aspects of radiological protection. The United States Committees on the Biological Effects of Ionizing Radiation (BEIR) also analyze the available data and issue reports. Like UNSCEAR reports, the BEIR reports are concerned only with the assessment of effects, and do not make any recommendations on radiation protection. Risk estimates provided by these committees are in good general agreement [BEIR-VI 1998, BEIR-V 1990, ICRP 1991, UNSCEAR 1993, NCRP 1993].

Problems associated with the use of data on excess cancers in Japanese bomb survivors to predict consequences at lower levels of dose and dose rate have been extensively discussed in BEIR, UNSCEAR and ICRP reports. One problem is how to extrapolate the data on increased numbers of cancer experienced by the bomb survivors in the first 40 years following exposure to predict the increase that will occur over the total lifespan of the population. Various models relating the increase of cancer with age after exposure have been used to obtain lifetime risk estimates.

A second problem is how to apply the lifetime risk of various cancers occurring in a Japanese population to other populations. Because of different cancer incidence patterns in Japan and other countries, extrapolation to other populations is difficult. The 1991 ICRP estimates were obtained by averaging results from two different extrapolation models, and applying them to the population of five countries.

A third problem is the extrapolation of data from populations exposed to various whole body doses of external radiation at high dose rates, to predicted effects of radiation at low dose rates. Based on theoretical considerations, experimental animal studies, and some limited human data, ICRP Publication 60 [1991] has adopted the convention of dividing the cancer risks observed at high doses and high dose rates of X- and gamma-rays by a dose and dose rate effectiveness factor of two in order to obtain cancer risk estimates for low doses of ionizing radiation at low dose rates. In other words, a low radiation dose delivered at low dose rates is about half as effective at producing long-term effects as the same dose delivered at a high dose rate. UNSCEAR [1993] currently defines low doses as less than 200 mSv and low dose rates as less than 0.1 mSv per minute or 6 mSv per hour; it should be noted that these doses and dose rates are very high compared to typical public doses.

Finally, there is the question of the applicability of the data to individuals exposed to radiation doses orders of magnitude less than the atomic bomb survivors. The international radiation protection community has conservatively assumed that any increase in radiation exposure will result in a proportional increase in cancer risk and the risk of genetic disorders (although there is some evidence to the contrary). This

assu mption, referred to as the linear no-threshold model (i.e. linear dose response down to zero dose), has been examined in ACRP-18 [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 [BEIR-IV 1988]. There is also some evidence of a reduction of cancer rates in humans on exposure to very low doses of radioactivity, resulting from the stimulation of repair mechanisms. However, the available data are not sufficient at present to take this into account in radiological protection [ACRP-18 1996].

Despite these problems and the uncertainties involved, an estimate of the probability of radiation-induced cancers is needed for use in radiation protection. Based on extrapolations from high dose epidemiological studies, ICRP Publication 60 [1991] recommends lifetime fatal cancer risk estimates of 0.04 per Sv for the adult population, and 0.05 per Sv for the entire population including all age groups, following a protracted whole body exposure of low dose and low dose rate radiation. The ICRP risk estimates represent a convergence of international scientific opinion, and may overestimate risk at low doses. Although the linear no-threshold model cannot be used to predict the outcome of actual exposures to an individual or a population, it is an important radiation protection tool, and can be used in comparing risk management and regulatory options.

In addition to fatal cancer, risk coefficients have been estimated for total harm produced by all late effects, including fatal cancers, non-fatal cancers weighted for severity and ease of curing, the years of life lost or seriously impaired, and risk of serious genetic disorders developing in subsequent generations. For example, a radiation-induced leukemia (which is potentially fatal) is weighted more heavily than a radiation-induced skin cancer, which is readily curable. Incorporating these factors, the ICRP has recommended a risk coefficient for total harm of 0.056 per Sv for an adult population, and 0.073 per Sv for the general public [ICRP 1991]. Risk estimates for genetic disorders are inferred from mouse data exposed over a wide range of doses and dose rates, due to a lack of direct evidence of these effects in human populations. Teratogenic effects (i.e. abnormalities in the developing embryo) have also been considered, but are believed to be zero below the dose limits recommended by the ICRP [1991] for public exposure.

An important consequence of the assumption of a linear no-threshold relationship between dose and risk, is that the collective dose becomes an indicator of communal risk (aggregated risk to the whole community). If a very large number of individuals were exposed to low doses of radiation from various sources, below any limit set for individuals, the total dose to the population as a whole could be appreciable. Under the linear no-threshold model, the likelihood of adverse health effects due to radiation are assumed to increase linearly with dose, and the potential societal harm would be determined by the total population dose. It should be noted however that the AECB Advisory Committee on Radiological Protection has recently recommended that, in calculating collective or total population doses, those from individual doses of less than 10 microsieverts per year (roughly 5 percent of the radiation dose which everyone receives on average each year from natural sources) should be categorized separately from and not added to those of higher individual doses. This recommendation reflects the lower level of concern about the health risks of such doses which are considered negligible even if the linear no-threshold hypothesis is assumed to be correct [ACRP-18 1996].

Many reports have been published that present data on workers who have been occupationally exposed to well quantified low-dose radiation, the most important of which are the detailed observations on radiation workers in the nuclear industry by IARC [1994], Kato and Cardis [1994], and Kendall *et al* [1992]. The IARC study was the largest investigation of cancer risks associated with occupational radiation exposures, and included over 90,000 nuclear workers in Canada, the U.S., and the United Kingdom. This study failed to establish a clear cancer risk at low levels of dose, and is therefore of limited value in making inferences about risks associated with environmental radiation exposures. [Cardis *et al* 1995].

Some attempts have been made to obtain direct estimates of risk from populations who have received low dose exposures, such as those resulting from residential exposure to radon [Létourneau *et al* 1994, Alavanja *et al* 1994, Pershagen *et al* 1994, Lubin *et al* 1994, ACRP-18 1996)]. No definitive results have been obtained. Although it is difficult to clearly establish excess lung cancer risks based on studies of the general population (Lubin and Buice 1997), the BEIR-VI (1998) report concluded that, based on the radiobiological considerations and studies of miners exposed to high doses of radon, about 10-15% of lung cancer in the general population may be due to residential radon exposures.

3.2 Chemical Hazards

The hazards associated with chemicals are generally categorized into two types: cancer and non-cancer (the latter including reproductive/developmental, and neurological/ behavioural effects). Although the focus of this report is on carcinogens, mention is made of non-carcinogenic effects for comparative purposes.

3.2.1 Biological Effects of Carcinogenic Chemicals

Information on the effects of exposure to chemical agents is obtained primarily from toxicological studies of animal species and occasionally from epidemiological studies on human populations. Both the route of exposure and enzyme activation are thought to be major determinants of the site of carcinogenesis. In carcinogenic risk assessment, all forms of cancer are generally given more or less the same weight. This is because cancers observed in animal species may not necessarily occur at the same sites or be of the same types as in humans.

Genotoxic chemicals are defined as those which can damage the genetic material (DNA) present in all living cells, and which thus possess the ability to induce cancer, heritable

disorders and abnormalities in the developing embryo. The carcinogenicity of different genotoxic chemicals varies depending upon the level and route of exposure, potency, and target organs. Chemicals may be directly genotoxic in their parental form (e.g. ethylene oxide, alkylating agents) or may become genotoxic following biotransformation to a reactive metabolite (e.g. polycyclic aromatic hydrocarbons). The cancer-causing effects of certain chemicals on humans and laboratory animals are similar to those of ionizing radiation.

Non-genotoxic (or epigenetic) carcinogens cause cancer without directly interacting with DNA. For example, cytotoxic (toxic to living cells) chemicals may lead to compensatory cell regeneration and an increase in the rate of cell proliferation. Cell proliferation can lead to increased opportunities for endogenous (originating or produced within the body) DNA damage during cell division, or proliferation of premalignant cells that have already sustained one or more mutations. Non-genotoxic chemical carcinogens may also influence the hormonal status of the exposed individual, or disrupt cell-to-cell communication. Whereas genotoxic chemicals may damage DNA at even the lowest levels of exposure, non-genotoxic agents are believed to have a critical threshold level that must be exceeded for harmful effects to occur [Scientific and Organizing Committee 1991; International Expert Panel on Carcinogen Risk Assessment, 1996].

3.2.2 Quantitative Risk Assessment for Chemicals

Data regarding human cancer incidence following chemical exposure are occasionally obtained through epidemiological studies of occupational exposure, such as workers involved in beta-naphthylamine distillation or in asbestos mining and processing. These data, together with estimates of exposure levels, are important in assessing the risk of carcinogenic chemicals in occupational settings. Similar studies and data are not typically available for the general population.

Most estimates of cancer risk arising from low-dose chemical exposure are necessarily derived, in the absence of direct human data, from toxicological studies on laboratory animals. These studies are usually carried out with long-term, chronic exposures of the laboratory animals (e.g. rats and mice) to two or three dose levels of the chemical in question [NRC 1993]. The highest dose level is usually the maximum dose which does not cause other serious health problems for the animals in question, and is referred to as the maximum tolerated dose. The frequency of cancers induced by these different levels of exposure is normally fitted to a linear no-threshold dose-response curve, as for ionizing radiation. A detailed discussion of this assumption can be found in a number of publications [e.g. Zeise *et al* 1987; Bailer *et al* 1988; McClellan, 1994 and 1995].

Extrapolations of cancer risk from animal data to human populations are achieved using a number of cautious assumptions. They are generally based on the upper 95% confidence limit of the linear fit to the experimental data, rather than on the basis of the best linear fit. Data on rates of metabolism and excretion of chemicals in different species, including humans, are used in these extrapolations where they are adequate. This method of dealing with uncertainty ensures that the actual risk level will probably be lower than the risk criterion used as the basis for risk management, and in many cases may be substantially lower.

Although it is possible to calculate numerical estimates of cancer risk due to human exposure to carcinogenic chemicals found in small concentrations in the environment, such estimates must be interpreted and used with care. This is primarily because of the considerable uncertainties associated with low-dose extrapolation of data for chemicals. To characterize cancer risk for *Priority Substances* under the *Canadian Environmental Protection Act (CEPA)*, quantitative estimates of the carcinogenic potency are compared to the estimated exposure of the general population in Canada [Meek *et al* 1994b]. Potency is expressed as the concentration or dose which induces a 5% increase in tumour incidence or mortality. The resulting index is then classified according to high, medium, or low priority for further action relative to the other non-threshold substances.

The results of comparing exposures with quantitative estimates of carcinogenic potency for fourteen compounds (or groups thereof) considered to be carcinogenic on the first *Priority Substances List* under *CEPA* provide information on the magnitude of risks associated with selected chemical carcinogens in the general environment in Canada. For seven of these fourteen compounds, taking into account exposure of Canadians from all media, exposure potency indices were high (corresponding to lifetime risks of more than one in one hundred thousand). For three of the fourteen compounds, values were moderate (corresponding to lifetime risks of one in ten million to one in one hundred thousand). For four of the fourteen compounds values were low (corresponding to lifetime risks of less than one in ten million) (see Table 1).

Risk assessment for combined exposures to chemicals is still at an early stage of development. Most cancer risk assessments have been determined from bioassays of animals exposed to individual carcinogens, often via only one exposure pathway. Chemicals, however, may interact in many ways and affect absorption, distribution, bio-transformation and excretion, as well as molecular and cellular changes, with possible toxic outcomes. The effects of combined exposures may be simply additive, or more than additive, or alternatively, a reduction or an antagonistic effect may be seen [NCRP 1989]. As a result, chemical risk assessment approaches currently lack a single unified approach similar to that used for radiation risk assessment.

Recently, there has been development of a toxic equivalency factor (TEF) approach for some families of chemicals where the mechanisms of carcinogenesis are considered to be similar, such as dioxins and dibenzofurans, co-planar polychlorinated biphenyls (PCBs), and polycyclic aromatic hydrocarbons (PAHs). TEFs are designed to permit the conversion of concentrations of different members of a family of chemicals to an equivalent concentration in terms of potential toxicity so that the total toxicity of a mixture containing these chemicals can be assessed and compared to mixtures of different composition [Krewski *et al* 1989; US EPA 1993; Safe 1990]. International agreement has been reached on a TEF scheme for dioxins and dibenzofurans which has been used in the development of environmental standards in Canada [NATO/CCMS

Table 1. Exposure Potency Indices for Carcinogenic Compounds on the First Priority Substances List under CEPA

High Exposure Potency Index ($> 10^{-5}$ Lifetime Risk)	Moderate Exposure Potency Index ($> 10^{-7}$ to $< 10^{-5}$ Lifetime Risk)	Low Exposure Potency Index ($< 10^{-7}$ Lifetime Risk)
Arsenic & Its Compounds Benzene Cadmium-Inorganic Compounds (Inhalation) Chromium VI (Inhalation) Hexachlorobenzene Oxidic, Sulphidic & Soluble Nickel 5 PAHs	1,2 -Dichloroethane Dichloromethane (pbpk Modified) Trichloroethylene	Refractory Ceramic Fibre Benzidine BCME & CMME 3,3'-Dichlorobenzidine

PBPK: Physiologically based pharmacokinetic (models)
BCME: Bis(chloromethyl)ether
CMME: Chloromethyl methyl ether
PAHs: Polycyclic aromatic hydrocarbons

1989]. relative carcinogenic potencies have been developed for subsets of PAHs present in the general environment [Meek *et al* 1994a].

3.2.3 Other Toxic Effects

Any chemical which is not carcinogenic can be toxic when the concentrations are sufficiently high. This statement also applies to essential chemical nutrients such as vitamin D and minerals [ICME 1996]. However, these harmful effects are generally not observed below a given threshold dose. Exposures to chemicals can lead to serious non-malignant effects which are generally classified in the following broad categories: organ-specific, neurological/behavioural, reproductive/developmental, and immunological. Such effects vary depending upon the dosage, route of exposure (ingestion, inhalation or dermal absorption), frequency and duration of exposure, physiological state, and sex and age of the exposed population. Toxic effects resulting from chemical exposure may be brief or prolonged, reversible or irreversible, immediate or delayed. The nature, severity, incidence and prevalence of these effects in populations exposed to chemical substances generally increase with dose, above some threshold exposure level.

Toxic effects can occur following either acute exposure to high concentrations or chronic exposure to generally lower concentrations. Some examples include mucus hypersecretion or obstructive lung disease from chronic exposure to inorganic dusts, as well as neurobehavioural dysfunction from chronic heavy metal exposure. For example, neurological effects in the native population at Grassy Narrows, Ontario were associated with high levels of organic mercury in the Native population from their fish-based diet; however, due to numerous confounders no proven diagnosis of methylmercury poisoning could be made [Wheatley 1979].

The tolerable levels for non-malignant effects from chemical exposure are generally derived from effect levels observed in laboratory animal studies or epidemiological investigations, divided by an uncertainty factor. Uncertainty or safety factors are applied on a case-by-case basis and depend principally on the quality of the database. They may vary slightly in different regulatory agencies or programs.

Some chemicals can cause individual hypersensitivities; these effects are not dose-related but are triggered by the immune system. Immediate hypersensitivity reactions occur within 12 hours of exposure. Anaphylaxis, which causes death if not treated, and urticaria, are well known examples. Delayed hypersensitivity reactions occur at least 24-48 hours following exposure. One example of this type of reaction is hypersensitivity pneumonitis caused by exposure to chemicals such as beryllium. Approaches to risk assessment for individual hypersensitivities are not well developed; risks are managed on an individual basis by attending physicians.

3.3 Microbiological Hazards

Like radiation and chemical contaminants, microbiological agents in food and drinking water can present a health risk. These agents include bacteria, protozoa, viruses, and fungi. While most of these are harmless and often beneficial, many are capable of causing illness and death in humans. Microbiological hazards are major causes of human disease in situations where control measures are inadequate. In spite of this, risk assessment methodologies have only recently begun to evolve for foodborne microbiological and other biological hazards. Microbiological agents are usually measured as the number of organisms present in a given sample volume.

Consideration of microbiological risk assessment for food and water is relevant to the present report since measures to control microbiological hazards may, in turn, increase long-term chemical risk. For example:

- the chlorination of municipal drinking water supplies to control microbiological hazards produces waterborne carcinogens, notably chloromethanes (e.g. chloroform and carbon tetrachloride); and

- the addition of sodium nitrite to meats to control growth of *Clostridium botulinum*, which causes foodborne botulism, leads to the subsequent production of carcinogenic nitrosamines.

An appropriate balance between the need to control disease-causing bacteria and the need to control the presence of disinfecting carcinogenic chemicals in food and drinking water is essential to ensure optimal public health protection.

In addition to illnesses that occur shortly after exposure or infection, microbial risk assessment must take into consideration pathogens that can precipitate serious chronic diseases such as Guillain-Barré Syndrome, reactive arthritides, and congenital toxoplasmosis. These are documented, often life-long consequences in otherwise healthy individuals, and must be assessed in context with the acute effects of microbial hazards.

Adverse health effects may result from either *intoxication* or *infection*. Intoxication is brought about by the production of toxins which can cause symptoms ranging from mild, temporary effects to severe intoxications that can cause long-term or life-threatening consequences. Infection is caused by exposure to live bacterial cells, viruses or parasites capable of infecting the host and producing a pathological response.

In rare cases, microorganisms are directly associated with carcinogenesis. It has recently been recognized that gastritis, gastric and intestinal ulcers, gastric carcinoma, and primary gastric B-cell lymphoma are associated with gastro-intestinal infections with the bacterium *Helicobacter pylori* [Blaser *et al* 1995]. It has been suggested that elimination of *H. pylori* infection may prevent most gastric carcinomas and primary gastric lymphomas [Graham 1994]. Proper diet, including fresh fruits and vegetables, combined with a reduction in the infection rate with *H. pylori* may eventually lead to a reduction in gastric cancer in the general population. Other examples of microbiological hazards which may induce cancer include aflatoxin, produced by a mould on peanuts and certain other foodstuffs, and hepatitis B virus, prevalent in some parts of the world.

Risk assessment associated with microbiological pathogens presents unique challenges. Risk assessment methods for foodborne bacteria are complicated by factors resulting from procedures used to grow, process, store and prepare food for consumption. These can vary greatly depending on cultural and geographical differences.

In many cases sufficient data are not available to support a quantitative assessment of risk associated with pathogenic bacteria in foods. By default, the qualitative approach to characterizing risk may be the only available alternative. This depends on experience with a specific food, a knowledge of the ecology of pathogens, epidemiological data, and expert judgement regarding hazards associated with the manner in which food is produced, processed, stored, and prepared for consumption.

4. Risk Management

Risk management is the process in which the results of risk assessment along with other considerations are used to select and implement one or more strategies for controlling a risk. Standards used for risk management are set on the basis of not only the magnitude of risk, but also technical, economic, and socio-political factors. In Canada, there is no consensus on *acceptable* levels of risk for radiological, chemical, and microbiological hazards. The *acceptability* of risk is often determined by judgements on such factors as the weight of scientific evidence, the nature, extent and severity of the hazard based on risk assessment evaluations, the degree of public concern, the benefits associated with the substance, product, or process, the cost and feasibility of reducing exposures, and regulatory agency policies defining limits on *acceptable* risks. In addition, consultation with stakeholders is necessary to determine *de facto* the *acceptable* residual risk after implementing risk reduction controls or measures. Judgements of *acceptable* or tolerable levels of risk are often difficult as a result of polarized positions which exist within society. In Canada, the weight given to various factors in risk management decision-making depends on the context in which risk management decisions are to be taken, including consideration of the legislation which applies.

If a cancer risk is judged to be *significant* or *unacceptable*, then it is generally expected that some action will be taken to reduce or eliminate the risk. In contrast, a *de minimis* or *essentially negligible* risk is one that is so small that no action needs to be taken. If a risk is judged to be *insignificant* or *acceptable*, however, this does not necessarily mean that it is *de minimis* or *negligible* [Sadowitz and Graham 1994].

The federal government has jurisdiction over activities that are considered to be of national interest, or of inter-provincial or international concern, and in setting minimum standards for protecting the health of Canadians and the environment. Provincial and territorial governments are responsible for the health and safety of their citizens, and have jurisdiction over industries within their borders, and in setting and enforcing provincial standards for health. In general, these standards cannot be less stringent than federal standards.

Approaches to risk management generally take the form of source control, point-of-use control, or educational strategies, and are the responsibility of both the federal and provincial governments. The application of legislative, technical and procedural controls may be different for source control and point-of-use control. Management tools include legally enforceable limits, regulations, and standards, as well as non- enforceable operating targets, guidelines, or goals for source and point-of-use control. Source control strategies limit human health risk by imposing regulations and operating criteria on the industry or process in question. Criteria governing the release of chemical contaminants into the environment are primarily set by provincial authorities, although the federal government has some jurisdiction under CEPA and its regulations. The regulation of radioactive emissions from nuclear facilities is entirely a federal responsibility.

Whereas source control strategies are source-specific, point-of-use control strategies are concerned with the levels of contaminants in air, food, and drinking water from all pollutant sources. Maximum allowable levels for contaminants are set through cooperation between the federal and provincial governments, and apply in addition to, but independently of, controls at the source. Such standards are not permits to contaminate up to the maximum values, but rather limits below which actual levels should be kept. This approach to risk management will be discussed later in this report in the context of the *Guidelines for Canadian Drinking Water Quality* [Health Canada 1996].

Education strategies can be used to inform the public when situations of potentially higher risk may exist. Such information may include air pollution advisories or suggestions on limiting consumption of particular types of foods, for example, sport fish [OMEE 1995a]. In general, source control and point-of-use control are the most important strategies in radiological and chemical risk management.

An overview of the responsibilities of the AECB and Health Canada in risk management is provided in Appendix C.

4.1 Ionizing Radiation

4.1.1 Philosophy

Although the usefulness of radiation in medicine and science was recognized soon after the discovery of X-rays in

1895, accumulating reports of harmful effects created a need for basic safety rules. Thus, from the very beginning of the use of these sources, radiation risk-reduction strategies were developed in parallel under the assumption that a balance of the risks and benefits of radiation and radiation-producing technologies was necessary. These strategies have evolved over the last century in light of an increasing knowledge of dose-response characteristics, risks and benefits resulting from radiation practices, and an environment that includes unavoidable natural background radiation. Under normal situations, radiation protection practices are concerned primarily with control at the source.

In Canada, as in most countries of the world, the system of radiological protection is based on the recommendations of the International Commission on Radiological Protection (ICRP). This body was first established in 1928 to focus on safety aspects of medical radiology. Its scope was expanded in 1950 with the widespread use of radiation outside the sphere of medicine. Members of the ICRP and its committees are chosen on the basis of their recognized expertise in the fields of medical radiology, radiation protection, physics, health physics, biology, genetics, biochemistry and biophysics. The risk management philosophy recommended by the ICRP focuses on controlling hazards from nuclear facilities at their source.

Initially, ICRP recommendations were based on the prevention of observable harmful effects, such as skin reddening, among medical radiologists. Tolerance doses were recommended based on the concept of a threshold value for these effects. Late effects were not immediately recognized because of the long latency period between radiation exposure and expression of a cancer.

A major change in radiation protection philosophy occurred in ICRP Publication 2 [ICRP 1960], in which genetic damage was assumed to be the main effect to be prevented. The assumption of a zero threshold for genetic and carcinogenic effects resulted in the basic precept that there should be no man-made exposure without the expectation of benefit. By 1977, continuing observations of radiation effects in the Japanese atomic bomb survivors, including the absence of observable genetic effects, led the ICRP to update its radiation safety recommendations. Publication 26 [ICRP 1977] recognized cancer as the main effect to be avoided. It also recognized that the various tissues and organs of the body have different susceptibilities to radiation-induced cancer. This led to the concept of effective dose, and the recommendation of a maximum effective dose expressed as an annual dose limit that included the sum of external radiation dose and the dose from internally deposited radionuclides [Cember 1996]. Criteria for maximum effective dose were based on quantitative risk estimates and comparisons with non-radiological risks considered acceptable by society.

New dose limits for occupational and public exposure were recommended in ICRP Publication 60 [1991], based on continued study of the Japanese bomb survivors. Publication 60 sets forth a comprehensive framework for radiation protection, with the goals of preventing early effects, and controlling the risk of radiation-induced cancers and serious genetic disorders to levels deemed to be acceptable to society. The three basic principles of radiation protection recommended by the ICRP [1977 and 1991] may be summarized as follows:

Justification: No practice involving exposures to radiation should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes.

Optimization: In relation to any particular source within a practice, the magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures should all be kept as low as reasonably achievable, economic and social factors being taken into account (ALARA principle).

Dose limitation: The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits. ICRP dose limits are set such that continued exposure at a dose just above the limit would be unacceptable on any reasonable basis.

The system of dose limitation applies to all exposures arising from all regulated practices. Recommended dose limits do not apply to radiation exposures received by patients in the course of medical diagnosis or treatment, by persons carrying out lifesaving procedures in an emergency, or by the public from natural sources.

Prior to its 1990 recommendations, the ICRP set dose limits for public exposure arising from regulated radiation practices based on an acceptability of fatal risk that was 10-50 times lower than that for occupational risks, depending on whether public exposures were considered for a single year or for a complete lifetime. The ICRP indicated that a risk in the range of one in a million (10^{-6}) to one in one hundred thousand (10^{-5}) per year would likely be acceptable to any individual member of the public [ICRP 1977]. Occupational limits were set such that the corresponding radiation risk would be no greater than the risk of accidental death in other industries not associated with radiation, or no greater than one in one thousand (10^{-3}) per year.

This approach to dose limitation was modified in 1990 to incorporate not only fatal risk, but also non-fatal conditions. In addition, the ICRP felt that it was difficult to assess the acceptability of risk for public exposures in the same manner as for occupational exposures. Therefore, in choosing the new dose limits for the public, the ICRP has taken into account both the concept of *acceptable* risk, and the variations in the existing level of dose from natural sources. The fact that a man-made radiation practice causes doses which are small in comparison to background does not necessarily imply that the practice is justified, but it does imply that the total radiological risk to the exposed individual is not significantly changed [ICRP 1991]. The annual dose from natural sources, including radon exposure, is about 2 mSv.

On the basis of these judgements, the ICRP recommends limits of 20 mSv per year averaged over 5 years for occupational exposure, and 1 mSv per year for public exposure arising from all regulated radiation practices. The public dose limit is

roughly half the average exposure to radiation from natural sources and considerably smaller than the normal variation in exposures to radiation from natural sources.

At an exposure of 1 mSv, the total risk of excess, radiation-induced fatal cancers, weighted non-fatal cancers and hereditary disorders summed over all future generations would be about seven per hundred thousand. The ICRP has indicated that continued exposures at or near the recommended limit for many years is not acceptable.

The use of dose limits is aimed at ensuring that no individual is exposed to radiation risks that are judged to be unacceptable in any normal circumstance. In Canada, design, manufacturing and operating practices have kept the actual maximum exposures of the public well below the legal dose limits set by the AECB. These practices have been interpreted as fulfilling the intent of the ALARA principle [AECB C-129 1994], even though rigorous ALARA processes such as those recommended by the AECB Advisory Committees [AC-2 1991] were not applied.

Consideration of relative costs and health benefits using the ALARA principle has been recommended by the ICRP, the U.S. EPA Science Advisory Board [US EPA SAB 1992], the Joint Committee on Health and Safety of the Royal Society of Canada and the Canadian Academy of Engineering [JCHS 1993], the AECB Advisory Committees [AC-2, 1991], and other organizations including the American Medical Association**.** The AECB Advisory Committees also believe that all risks arising from a nuclear facility should be included in the ALARA analysis; the AECB, however, may not have the legal authority to control non-radiological risk. In spite of any differences of approach and the difficulties that will be associated with its application, the Advisory Committees believe that the ALARA principle offers the best approach for establishing acceptable risk by balancing risks, costs and benefits.

Finally, the Advisory Committees have recommended that individual exposures of 0.01 mSv per year or less to members of the public from regulated practices could be regarded as a *de minimis* level, as it carries negligible risk to human health [AC-1 1990, ACNS-20 1995]. Exposures at this level would be regarded as safe, requiring no further mitigative action. Radiation protection groups in the U.S. and other countries have recommended similar values.

4.1.2 Regulatory Control

Laws governing the use of radioactive materials, radiation emitting devices and ionizing radiation exposures exist in Canada at both the federal and provincial levels, and are generally applied at the source. The principal legal instruments at the federal level are the *Atomic Energy Control Act*[1] and *Regulations*, and the *Radiation Emitting Devices Act* and *Regulations*. The *Atomic Energy Control Act* regulates, among other things, the use of radioactive materials and fissile material or processes which could be used in a nuclear chain reaction. This *Act* is administered by the AECB, which has the lead role in the regulation of nuclear facilities and the use of nuclear materials. Discussion of the *Act* will focus on the nuclear industry. The *Radiation Emitting Devices Act*, administered by Health Canada, pertains to specific classes of radiation emitting devices used both occupationally (e.g. X-ray equipment, lasers, ultrasound therapy devices) and residentially (e.g. microwave ovens, television receivers). Background radiation from natural sources is not covered under either *Act*.

Federal Legislation

The Atomic Energy Control Act

Nuclear facilities regulated by the AECB under the *Atomic Energy Control Act* include power and research reactors, uranium mines, mills and refineries, nuclear fuel fabrication plants, high energy particle accelerators, heavy water plants, and radioactive waste management facilities. The AECB is also responsible for the regulation of radioisotopes and the transport of radioactive materials (together with Transport Canada). The three major stages of licensing for all nuclear facilities are site acceptance, construction approval, and issuance of an operating licence. The applicant is required at each stage to show that its facility can be built and operated without undue risk to workers, the public, and the environment. The AECB monitors the facility and carries out inspections throughout the facility's lifespan to ensure that licensing criteria are met. In addition, the International Atomic Energy Agency inspects the nuclear generating stations of all member states, including Canada, to ensure compliance with the Nuclear Non-Proliferation Treaty. In the specific case of Ontario Hydro, the utility also participates in the Peer Evaluation Program adopted from the Institute of Nuclear Power Operators in the U.S. At the end of the useful life of a facility, the AECB must approve all plans for decommissioning.

Limits on both occupational and public exposures are stated in the *AECB Regulations* and are written into licences issued by the AECB. The current occupational dose limit for radiation workers is 50 mSv per year but the AECB is in the process of adopting the 1990 ICRP recommendation of 20 mSv per year [AECB 1991]. All radiation workers in Canada are required to wear dosimeters to monitor both annual and cumulative radiation exposures. These are currently recorded in the National Dose Registry maintained by Health Canada. According to the Registry, the average occupational exposures at nuclear generating stations in Ontario, for example, were about 0.6 mSv in 1994 [Ontario Hydro Nuclear 1995a, Myers 1996]. This average occupational exposure of 0.6 mSv per year is approximately 30 percent of the average annual exposure of all Canadians to radiation from natural sources (about 2 mSv per year).This occupational exposure corresponds to a total theoretical risk of about 0.12 fatal cancers per year [ICRP 1991a] for the total of all Ontario Hydro radiation workers, with the fatal cancers possibly developing some 20 or so years after the radiation exposure, in contrast to fatal accidents which result in an immediate loss of life.

1. A new act to replace the *Atomic Energy Control Act* has been passed by Parliament. The new *Nuclear Safety and Control Act*, which recognizes the many changes since 1946 when the existing Act was passed, had not yet come into force at the time of publication of this report.

The National Dose Registry, together with the accompanying mortality database and cancer incidence files maintained by Statistics Canada, provide a wealth of useful information. Epidemiological studies on correlations between recorded radiation doses in the National Dose Registry and causes of death and cancer incidence are currently in progress. Similar registries do not exist for workers exposed to carcinogenic chemicals.

Within the operating licence, the AECB sets annual maximum release limits for radioactive emissions from the facility in order to limit exposures to the general population. Operation at these limits for a full year would result in a maximum estimated dose to individuals in the population group at greatest risk, the critical group, equal to the AECB legal dose limit for public exposure. The nature of the critical group depends on the facility. For example, the critical group might be assumed to reside at the site boundary, and to derive all of their food and water from local sources. Release limits for individual radionuclides are derived based on a multi-pathway approach. Currently, the legal dose limit governing all radioactive emissions from nuclear facilities in Canada is 5 mSv per year. The AECB is currently in the process of incorporating the 1990 ICRP recommendations of 1 mSv per year into their regulations.

In practice, the AECB specifies that annual releases from nuclear generating stations must be a small fraction of the maximum annual release limits for each of several groups of radionuclides. The dose to an individual in the critical group from all radioactive emissions must be less than 0.05 mSv per year for each of several groups of radioactive substances, with a total of about 0.3 mSv per year for all radionuclides combined. The actual maximum doses to the most exposed population from nuclear generating stations are about 30 times lower. Such operational limits are first referenced in AECB News Release 73-1 [AECB1973], which mentions "... the intention of the major licensee to take any steps necessary to keep effluents below 1 percent of the license limit". While justification for the target was based on the ALARA principle, no cost-benefit analysis was undertaken. Rather the target was set by reviewing the operating records for the Pickering-A generating station. According to these records radioactive releases were generally below 1% of the limit, so that 0.05 mSv per year for each group of radionuclides seemed to be readily achievable and was therefore selected as the target. Later, a requirement of 0.05 mSv per year was set as an operating target on emissions, and has since become a *de facto* limit [AECB 1994b]. The system of licensing requirements which are set at a fraction of the legal dose limit is supported by the most recent recommendations of the ICRP [1991] on dose constraints.

Monitoring to demonstrate compliance with the *Act* and license conditions is the responsibility of the licensee. Emissions are continually monitored. Should those for a given week or month exceed the specified operating emission levels, examination of procedures and facility design by the licensee is required to determine what actions, if any, are necessary to ensure that the annual emission requirements can be achieved. Independent monitoring is performed by other agencies including provincial ministries and Health Canada. In estimating doses to demonstrate compliance with their license conditions, licensees must consider and sum all potential pathways of exposure to a radioactive material in the environment (e.g. inhalation from air, absorption through skin, ingestion from food or water), based on assumptions that likely over- estimate actual exposures. The dose received from each of the different radioactive materials is summed to obtain the total individual dose or collective dose to members of the public.

In addition to the management of risk associated with the normal operations of nuclear power plants, the AECB requires that the public be adequately protected in the event of a radiological emergency. Nuclear power plants are equipped with special safety systems whose sole function is to prevent or mitigate serious accidents that could result in radiological releases from the plants. The design of these and other safety related systems is based on a defence-in-depth approach. The AECB requires that the performance of these systems during serious accidents be analyzed on a conservative basis during the design of the facility to demonstrate with a high degree of confidence that the resulting doses to the public will be at acceptable levels [AECB 1995d]. These analyses must be updated as required by new information during the life of the facility.

The major licensees are also required to have an effective on-site emergency response plan and coordination with the province, in conformity with provincial requirements. Off-site nuclear emergency plans are a provincial responsibility as outlined, for example, in Ontario's *Nuclear Emergency Plan* and the *Ontario Emergency Plans Act*. These are supported by the *Federal Nuclear Emergency Response Plan*, administered by Health Canada.

Finally, licensees are required to conform to all other relevant federal and provincial regulations with regard to non-radioactive emissions. For example, in Ontario, the main authorities are the *Ontario Water Resources Act* and Regulations, and the *Ontario Environmental Protection Act* and Regulations. These *Acts* do not apply to radioactive emissions from facilities regulated by the AECB; however, emission limits or ambient environmental quality limits for non- radioactive substances under these *Acts* are included when appropriate in AECB licences, and are therefore under the authority of the AECB.

The Radiation Emitting Devices Act

The *Radiation Emitting Devices Act* (*RED Act*) applies to all devices that emit X-rays or non-ionizing radiation in occupational or clinical settings, or in personal use. Regulations written under the *RED Act* specify minimum safety standards for the design, construction, labelling, and advertisement of the devices or their components. The standards apply to devices at the point-of-sale, and are concerned with the performance of a device with regard to its intended function and manner of operation.

Provincial Legislation

The responsibility for controlling the use of radiation emitting devices belongs to the provinces. Provinces regulate and monitor exposure that may result from radiation emitting devices (but not radioactive material), as well as non-nuclear fuel cycle activities which give rise to occupational exposure to radionuclides. Some provinces, such as Saskatchewan, have prepared their own legislation for the control of ionizing radiation exposure [AECB 1995b].

The provinces set general environmental quality standards for radiation which are not used to regulate emissions from federally-regulated facilities. For example, Ontario Drinking Water Objectives for radionuclides are used to evaluate the acceptability of water supplied to the Ontario public and are legally enforceable on agencies supplying communal water. These drinking water objectives cannot be used to control emissions from federally licensed facilities.

The *Ontario Health Protection and Promotion Act* gives Medical Officers of Health (MOHs) the authority to close water supplies immediately when public health is threatened. MOHs use Ontario Drinking Water Objectives to evaluate public health risks. The Ontario Ministry of Health under the same *Act* also controls patient therapeutic exposures to radiation.

The AECB regulates allowable radiation exposures for miners in uranium mines, while the provinces regulate radiation exposures (primarily radon and its progeny) to miners in non-uranium mines (e.g., gold mines). The allowable limits for non-uranium miners in Ontario are roughly one quarter of those allowed for uranium miners under AECB regulations, or approximately one-third of the dose limit recommended by a Canadian federal-provincial committee as an allowable limit for radon in the air in homes [ICRP 1993]. Some other non-nuclear industries have the potential for radiation exposure to workers from naturally-occurring radioactive materials, such as the manufacturing of phosphate fertilizer. Under the provisions of the *Ontario Occupational Health and Safety Act*, employers are required to prescribe what precautions will be taken to protect workers from harm. In practice, exposures are low.

4.1.3 Public Exposures

Background levels of radiation exposure from natural sources have been extensively documented by various scientific committees, including UNSCEAR, BEIR, and the NCRP. In Canada the average dose from natural background radiation is about 2 mSv per year which includes a population-weighted average for the inhalation dose from radon gas. However, doses from background radiation vary extensively. This is partly due to the wide range of radon levels in homes measured across Canada, which give doses from 0.2 – 3.5 mSv or more per year from exposure to radon and radon progeny [UNSCEAR 1982, NCRP 1987]. There are also some data indicating that some individuals living in northern Canada may receive higher total doses from natural radioactivity due to elevated levels of polonium-210 in foods such as caribou meat.

A radon guideline for homeowners was established in 1988 by a federal-provincial working group under the Conference of Deputy Ministers of Health. The guideline recommends that remedial measures be taken where the level of radon in a home is found to exceed 800 Bq/m^3 (or about 14 mSv per year) as the annual average concentration in the normal living area. This would be equivalent to a risk of fatal cancer of about 1 in 15 for lifetime exposure at this level, using recent ICRP estimates [ICRP 1993]. As there is some theoretical risk at any level of radon exposure, the guideline suggests that homeowners may wish to reduce levels of radon as low as practicable. The guideline was reviewed and re-adopted in 1995. Interpretation of measurements in homes and advice to homeowners is generally the responsibility of the provinces.

Radiation doses from medical diagnoses and therapy, which are of considerable benefit to patients, may represent a significant source of exposure for the individual and appear to average about 1 mSv per year [ACRP 1996a]. Such exposures when averaged across the population, are less than background, and considerably greater than those from industrial sources, as will be noted later in this report.

On average, radiation exposures of the public from regulated sources represent a minor increase above exposures from natural sources, and are substantially less than the variations in the background dose across the country. The major contributors to the total dose arising from CANDU reactors are radioactive noble gases (krypton and xenon) and iodines emitted into the air, carbon-14 emitted into the air and subsequently incorporated into food, and tritium emitted into air and water. Based on environmental models and actual monitoring data, estimates of maximum annual doses received by members of the public living near Ontario nuclear generating stations in 1994 were on the order of 0.01 mSv [Ontario Hydro 1995]. Because of the cautious nature of the assumptions used in these models, actual doses received will be lower, and those received by populations living further from reactor sites will be substantially less.

Doses to the most exposed individual members of the public near AECB-licensed facilities have been calculated using radionuclide concentrations in various environmental media obtained either directly from monitoring data or from environmental transfer models. In general, estimated maximum annual doses to a hypothetical individual residing near various nuclear fuel cycle facilities, based on conservative environmental transfer models, are in the range of:

- 0.0025 – 0.2 mSv for uranium refining and conversion facilities
- 0 – 0.17 mSv for fuel fabrication plants
- 0.002 – 0.02 mSv for nuclear power plants.

Estimates of calculated maximum doses to members of the public living near uranium refining and fuel fabrication plants [AECB 1995] may be too high, since no measurements of actual maximum doses are available, as they are for nuclear power plants in Ontario. Although dose information is not available for people living in the vicinity of uranium mines, the AECB requires mine operators to impose limits on effluent contaminant concentrations and implement adequate environmental monitoring programs.

Based on ICRP risk coefficients, a theoretical fatal cancer risk can be calculated for the various exposure levels experienced by the public. For example, the hypothetical number of fatal cancers associated with the estimated maximum annual dose from nuclear power plant emissions, 0.01 mSv, is about 1 in two million. As another example, a natural background radiation dose of 2 mSv per year is about 1 in 10,000 per year, or 7 in 1,000 for a 70 year lifetime exposure. This is 2.5% of the total risk of fatal cancer observed in the Canadian population in 1991 and 1992 [Statistics Canada 1993, 1995].

4.1.4 Summary

In summary, the risks associated with ionizing radiation exposure from regulated practices are limited through the system of radiological protection recommended by the ICRP, implemented by the licensee and regulated by the AECB. All regulated practices must produce a net benefit to society, must be optimized with respect to benefits versus risks, and must include a system of individual dose limitation. Dose limits recommended by the ICRP and AECB are viewed as the lower limit of unacceptable levels. They must not be exceeded under normal circumstances, and actual doses should be as low as reasonably achievable, economic and social factors taken into consideration. Public dose limits apply to the sum of all exposures from all regulated practices, and are based on both a level of risk and on variations in natural background radiation. In practice, maximum doses to individuals in the general public from nuclear generating stations in Ontario are about 0.01 mSv per year or about 100 times lower than the recommended legal limit, which in turn, is lower than the variation in background radiation levels across Canada.

4.2 Chemical Hazards

4.2.1 Philosophy

Chemical risk management began with an assumption that public health could be completely protected. This assumption developed in the U.S. early in the century for food additives. By the 1960s, an approach which balances costs and benefits became well established for chemicals, although, in retrospect, it was aimed at reducing risk to levels that would be considered low by almost any criterion. Currently, the presence of natural sources of carcinogens is being given increasing attention in risk management strategies, although for many synthetic chemicals, significant natural sources are absent.

The idea that the dose-response characteristics for some chemicals might have no threshold resulted in a 1958 amendment to the U.S. *Food, Drug, and Cosmetics Act* prohibiting the addition of any chemical that can cause cancer in humans or animals to the human food supply. Almost immediately, however, it was realized that assuring the complete absence of carcinogens from the food supply was impossible, particularly in view of the rapidly advancing ability to detect ever lower levels of chemicals in food, and the abundance of naturally occurring carcinogens. As a result, the U.S. Food and Drug Administration proposed that if risks calculated under the no-threshold assumption were below some small value, the carcinogen was effectively absent in the food.

The first U.S. proposal for a *virtually safe dose* was to limit cancer risk to one in one hundred million (10^{-8}) over a lifetime of exposure [cf. Rodricks *et al* 1987]. This idea was tied to the notion that if the entire United States population was exposed at or near the virtually safe dose, only one or two of the then-current U.S. population of approximately 150 million would be affected. Shortly thereafter, it was realized that this criterion was an almost impossible burden on regulators for assuring the safety of food additives with considerable benefits. It was then proposed that a lifetime risk of one in a million would be considered negligible by most people. At this level, only about three excess cancer cases per year would result if everyone in the U.S. were exposed.

The *one in a million* criterion for *acceptable* risk became institutionalized over the next several years and, when cancer risks from environmental exposures became recognized in the late 1960s and early 1970s, the concept of negligible lifetime risk at one in a million (10^{-6}) was often applied, predominantly in the U.S. [Kelly and Cardon 1994]. Initially of greatest concern were widespread risks such as exposures to PCBs or pesticide residues in the environment. Later the same risk criterion began to be applied to much less widespread risks such as those which existed in the vicinity of industrial facilities or hazardous waste disposal areas.

Eventually, it became evident that one in a million (10^{-6}) was a very stringent criterion when relatively few people were exposed [US EPA SAB 1992]. Risks levels at or above one in ten thousand are accepted in setting U.S. Environmental Protection Agency (EPA) Maximum Contaminant Levels for carcinogens in drinking water when further limitation is not technically or economically feasible. In general, however, risk levels above one in ten thousand, even to very few individuals, are viewed as excessive and therefore require action to reduce exposure and risk [US EPA SAB 1992].

The U.S. EPA has set a lifetime risk goal of one per million (10^{-6}) for the regulation of individual genotoxic chemicals, particularly when the exposed population is large. Prior to the establishment of the *Clean Air Act Amendments* of 1990, Section 112 of the *Clean Air Act* required the EPA to set emission standards for hazardous air pollutants “to protect the public health with an ample margin of safety”. This was interpreted to mean that the EPA must first determine a *safe* emissions level (representing an *acceptable* degree of risk), and then add a margin of safety in view of uncertainties in scientific knowledge about the pollutant in question. Thus, the EPA adopted a general policy that a lifetime cancer risk of one in ten thousand (10^{-4}) for the most exposed person may constitute *acceptable* risk and that the margin of safety should reduce the risk for the greatest possible number of persons to an individual lifetime risk no higher than one in one million (10^{-6}) [NRC 1994].

Review of relevant decisions by the U.S. EPA and other U.S. government agencies shows that the levels of lifetime risks deemed *acceptable* for the public by different U.S. agencies under different circumstances vary over a range of ten thousand fold from about one in a million to one in a hundred [Sadowitz and Graham 1994]. The levels of lifetime risk associated with Health Canada guidelines for drinking water vary under different circumstances from about one in ten million for dichloromethane to about one in one thousand for arsenic (see Table 3).

The U.S. Office of Management and Budget reviewed the cost of compliance with EPA regulations. The Office found that the cost in millions of 1990 U.S. dollars per potential premature death avoided varied considerably as a result of compliance with EPA regulations [U.S. Office of Management and Budget 1991]. For example, the cost of setting drinking water standards for trichloromethane (chloroform) was about $200,000, while that for disposal of wood preserving chemicals as hazardous waste was about $5.7 trillion ($10^{12}$). The Office concluded that further consideration of the balance between health risks and benefits, in terms of lives lost and lives saved in society, would be appropriate before the promulgation of such regulations. When total societal resources are limited, excessive societal expenditures on reduction of minimal risks, rather than on more severe risks, are expected to be detrimental to societal health. By way of comparison, the AECB has suggested that total expenditures to reduce industrial radiation exposures should not exceed about $2 million 1994 Canadian dollars per fatal cancer avoided [derived from AECB 1994].

Regulatory authorities in Canada do not recommend any single legal dose limit or level of *acceptable* risk at which to regulate chemical carcinogens. Risk management decisions concerning control are made following consultation with affected parties, and involve judicious balancing of the estimated risks against the associated costs, feasibility of controls, and benefits to society. For example, management strategies pertaining to exposure from *Priority Substances* found to be toxic under the *Canadian Environmental Protection Act (CEPA)* vary as a function of different cost/ benefit profiles, based on best-available control technologies economically achievable.

In establishing point-of-consumption standards for chemicals, regulatory agencies generally set generic values based on the risk-reduction potential in terms of costs, benefits, achievability, existing background sources, and societal values. In general, the majority of regulatory controls are for synthetic chemicals. However, in those cases where the contaminants occur naturally, as for example trace metals in drinking water, the extent of exposure from natural sources is often considered in the development of standards or controls (e.g. arsenic in drinking water).

Exposure to a single type of chemical through many pathways has, in the past, often not been taken into account in the development of controls for chemical contaminants; it is, however, being increasingly considered in this regard. For example, for *Priority Substances* under *CEPA*, the relative magnitude of the contribution of each pathway of exposure (e.g. air, food, water and consumer products) to total intake is estimated for each of five age groups in the population. These contributions are then considered in the risk management process to ensure that the total estimated risk from all sources is controlled. Probably the greatest barrier to consideration of multimedia exposure in establishing risk management strategies results from jurisdictional constraints. At present, different agencies effectively deal with different media.

In addition to controlling industrial emissions of chemicals into the environment, point-of-source risk management includes controls on occupational exposures. A number of chemicals and industrial processes have been identified as carcinogenic in occupationally exposed workers [Doll and Peto 1981; IARC 1995]. However, cancer risks associated with particular chemicals in the workplace are difficult to assess due to the lack of an adequate database on both chemical hazards and exposure levels. Though it is possible to estimate the risk for some substances (e.g. benzene, arsenic and asbestos) these represent only a small proportion of chemicals commonly used in industry.

The estimated risks associated with occupational exposure limits for different carcinogens differ considerably, due to the weighting of various risk-management factors. Gold *et al* [1987] compared the legally permissible dose limits for workers in the U.S. to the chronic dose level that induces cancer in 50% of laboratory animals. For 41 chemicals on which reasonable data existed, this ratio differed by more than 100 000 fold. Although this ratio does not take into account actual exposure levels or the number of exposed workers, it nevertheless suggests that more attention should be given in risk reduction strategies for occupational exposures to chemical substances that appear most hazardous to animals.

In Ontario, occupational exposure levels are set by the Ontario Ministry of Labour based both on health studies, and impact studies in terms of cost and benefit. For specific chemical carcinogens, the ALARA approach is used to establish regulatory limits. Actual limits exist only for those chemicals that are in most common usage, or for which health effects are known. However, all reasonable precautions must be taken by an employer to protect the health and safety of workers from all potential exposures.

4.2.2 Regulatory Control

Risk management for chemicals is carried out under a number of different Acts and Regulations, most notably: the *Food and Drugs Act* and associated *Regulations*, the *Canadian Environmental Protection Act (CEPA)*, the *Pest Control Products Act*, the *Drinking Water Materials Safety Act*, and the *Hazardous Products A*ct. Control mechanisms for chemicals are complex, involving the responsibility of several levels of government. Consequently, the information provided below is not exhaustive, but is intended to highlight some of the major legislation. Some further information is provided in Appendix C.

In assessing and managing chemical risks, Canada interacts with many international organizations such as the International Agency for Research on Cancer (IARC), the Food

and Agricultural Organization (FAO), the International Program on Chemical Safety (IPCS), the World Health Organization (WHO), the Organization for Economic and Co-operative Development (OECD), and the Codex Alimentarius Commission. However, in general, there are no international bodies that recommend standard chemical risk management approaches. In North America, the International Joint Commission of Canada and the U.S. serves as an advisory body to both national governments on pollution management in trans-national boundary waters.

Source Control

The management of chemical contaminants in the environment generally involves the use of source controls.

Canadian Environmental Protection Act (CEPA)

The assessment and management of chemical risks is carried out at the federal level under the *Canadian Environmental Protection Act (CEPA)*. The *Act* sets out broad federal policies on pollution prevention and control, and contains provisions for dealing with toxic substances, nutrients, ocean dumping, environmental research, guidelines and codes of practice, as well as agreements with provinces and territories. In general, controls are implemented where substances are not addressed under other federal legislation and, in particular, where there are international or transboundary implications. For substances deemed to present a risk to health or the environment, controls may be instituted in consultation with the provinces, and polluters can be fined for failure to comply with the regulations. Twenty-five regulations have been enacted under *CEPA*.

CEPA's mandate covers toxic substances throughout the ecosystem and may control any stage of a product's life cycle. The primary focus of the *Act* is the prevention of environmental problems before they occur. Preventive measures include regulation and enforcement mechanisms, non-regulatory approaches such as incentives with industry, as well as the development and transfer of pollution measurement and control technologies. Environment Canada and Health Canada develop *CEPA* regulations and guidelines, and Environment Canada administers the *Act* on behalf of the federal government.

The *Toxic Substances Management Policy (TSMP)* is a new federal policy for managing toxic substances. Under the *TSMP*, any substance that results from human activity, takes a long time to break down, accumulates in biological tissues, and is *CEPA toxic* or equivalent will be designated as a Track I substance and targeted for virtual elimination. For substances that meet some but not all of these criteria (Track II substances), the objective is to prevent or minimize their release throughout their life cycles (during their manufacture, use, transport and disposal), using pollution prevention approaches [Environment Canada 1995c].

Fisheries Act

The protection of waters frequented by fish is covered under the *Fisheries Act*, which is the legal responsibility of the federal Department of Fisheries and Oceans. Contained in the *Act* are provisions related to the implementation of pollution prevention, inspections, enforcement, and civil remedies. Environment Canada is also responsible for the administration of these pollution prevention provisions.

Example of Source Control

In addition to provisions contained under *CEPA* and the *Fisheries Act*, the control and regulation of industries producing or using chemicals is subject to provincial regulation, although actual regulatory approaches may vary between provinces and territories. The following describes the situation in Ontario.

The Ontario Ministry of Environment and Energy (OMEE) has the legal mandate under the *Ontario Water Resources Act* and the *Ontario Environmental Protection Act* to regulate industrial discharges to water, air, and land which may be harmful to human health, non-human biota, and commercial or private uses of water and air. The powers under these *Acts* include the requirement for Certificates of Approval prior to construction and operation of industrial facilities. Such certificates include limits on the amount of allowable discharges of chemicals and other harmful agents into the environment.

The Ministry has established standards, guidelines and objectives for assessing and setting emission limits and ambient environmental quality for contaminants in such media as air, surface water, drinking water, soil, and hazardous wastes. Criteria for general environmental quality are established to protect against the most sensitive effects in the most sensitive populations. Regulated criteria, such as for air and hazardous waste, are directly enforceable. Other criteria become legally-enforceable when included in a legal instrument such as a Certificate of Approval or control order.

Under the *Ontario Water Resources Act*, the discharge of non-radioactive substances from industrial and municipal sources into provincial waterways is controlled using a two-pronged approach. These are the Treatment Technology-Based Effluent Requirements and the Receiving Water-Based Effluent Requirements [OMEE 1994]. Treatment-based water effluent requirements were developed under the *Municipal Industrial Strategy for Abatement* (MISA) program for several industrial sectors, including the electric power generation sector. The goal of MISA is to protect the environment through the elimination of persistent toxic substances from wastewater discharged into Ontario's waterways. Receiving water-based effluent requirements are developed on a site-specific basis, and are based on OMEE's surface water quality objectives. OMEE policy requires that the more stringent of the two approaches be applied.

Under the MISA program, industrial discharges cannot be lethally toxic to aquatic life before dilution. After dilution in the watercourse, contaminant levels must meet provincial water quality objectives for both toxic and carcinogenic chemicals. Objectives are set to prevent toxic effects in aquatic life at

all stages of development. In rare cases where bioaccumulation of a specific contaminant, such as PCBs or dioxins, may occur in species consumed by humans (e.g. sport fish), objectives for that contaminant will be based on human health.

The MISA effluent requirements have been promulgated as regulations and will be legally enforceable in 1998 for the electric power generating sector [OMEE 1995]. The regulations require that industries comply with discharge limits that have been set based on both loading (i.e. kilograms discharged per day) and effluent concentration for specific substances. These limits were developed based on both the results of an effluent monitoring program and the best available technology economically achievable (BATEA) for pollutant reduction. BATEA is defined as the combination of demonstrated treatment technologies and industrial process changes that can reduce or eliminate the discharge of contaminants and that the industry can afford.

Total daily loading and monthly-average loading and concentration limits have been established for several substances for the electric power generation sector. To show compliance with these limits, facilities are required to: establish sampling points for effluent collection; monitor the effluent on a daily and weekly basis; and calculate the loading and concentration values for the two time periods. Furthermore, the facilities must measure the pH of the effluent; conduct acute lethality tests for rainbow trout and *Daphnia magna*; conduct chronic toxicity testing using fathead minnow (i.e., 7-day growth inhibition test) and *Ceriodaphnia dubia* (i.e., 7-day reproduction inhibition and survivability test); conduct quality control tests; and determine the volume of the effluent. Finally, the facilities are required to keep records of the data and analytic procedures, and prepare and submit reports to the OMEE. These reports will also be available to the public [OMEE 1995].

The Ministry has not established a generic policy on acceptable risk levels for carcinogens; rather they are evaluated on a case-by-case basis taking into account the scientific information and the implementation implications. Releases of carcinogens in liquid effluent must meet OMEE surface water quality objectives after dilution in the watercourse. Emissions of carcinogens into air must not exceed Point of Impact (POI) standards. These POI standards apply to short-term (30 minutes) releases and are set at a factor of 15 times the annual ambient air quality criteria (AAQC) for the specific contaminant. In setting AAQCs for individual carcinogens, a lifetime risk of ten per million to one per million (10^{-5} to 10^{-6}) is generally used for specific chemicals, in the absence of significant technical and economic limitations, but is case-specific.

As a condition of licensing, facilities are responsible for monitoring and reporting to the responsible authority to ensure compliance with their licensing conditions, and are subject to compliance inspection and monitoring. The regional offices of the OMEE are responsible for working in conjunction with the industry when abatement actions are required to address potential or actual harmful effects. Non-compliance with MISA regulations is a violation and is grounds for enforcement and prosecution. The non-compliance event is documented and investigated further by OMEE. Non-compliance does not automatically lead to prosecution, as abatement activity is also considered to bring about compliance [OMEE 1994]. In situations where serious harm or breaches of conditions have occurred, investigations and legal prosecution are pursued by the Investigations and Prosecutions Branch of the OMEE. For infractions under *CEPA*, options range from negotiations with the licensee, to prosecution.

Finally, as a requirement for licensing, industries are required to have effective emergency response plans in conformity with provincial requirements. The Major Industrial Accidents Council of Canada, a non-governmental organization, sets standards and guidelines to aid industry in emergency preparedness.

Point-of-Use Control

In general, chemical risk management strategies involving food and drinking water rely heavily on point-of-use controls. Major legislation relating to the control of food additives and contaminants, pesticides and herbicides and drinking water is discussed below. The *Guidelines for Canadian Drinking Water Quality* are discussed in section 4.4.

Food and Drugs Act and Associated Regulations

Risks from food additives and food contaminants are managed through the *Food and Drugs Act* and associated *Regulations*, the *Pest Control Products Act*, the *Fisheries Act*, and the *Meat Inspection Act*. Food additives are chemical substances deliberately added to food with a view to achieving an intended beneficial effect. Food contaminants are chemical substances that are found in food but not deliberately added. They can occur as a result of human activity, industrial or otherwise, or because of their natural occurrence in the environment. The approach to evaluating the risk to humans from deliberately-added or not-deliberately-added chemicals in food is similar, but the management of risk in each of these instances is different.

The evaluation of food additives is based on complete toxicological data supplied by a petitioner before their usage is approved. If a food additive is shown to be a carcinogen in any species, it will not be approved; if previously approved, it would be removed from the Food Additive Tables. In all cases, the use of such additives must be justified, and the minimum level which achieves the desirable effect must be established. If, in taking into account all existing uses of an additive, a proposed additional use does not cause the estimated or probable daily intake to exceed the acceptable daily intake, then that extension of use can be approved and written into the regulations. Monitoring and compliance activities ensure that the level of *acceptable* risk established at the time of evaluation is not exceeded.

Sodium and potassium salts of nitrite are used to cure meats and fish. Nitrite has several effects on food including colour preservation, flavour enhancement and antioxidant effects; however, the most important function is inhibition of

the growth of bacteria, particularly *Clostridium botulinum*. Growth of *C. botulinum* results in production of botulinum neurotoxin and leads to foodborne botulism, a potentially fatal intoxication.

While nitrite itself is not carcinogenic, use of nitrite in foods may lead to formation of nitrosamines by reaction of nitrous acid with secondary amines [Kim and Foegeding 1993]. The carcinogenic and mutagenic properties of nitrosamines have been well documented [Lijinsky 1976], and the occurrence of nitrosamines in foods has been demonstrated [Gray and Randall 1979].

The risk of botulism arising from removal of nitrite as a preservative in cured meats has been suggested to be in the same range as that calculated for cancer deaths resulting from its inclusion in foods [Miller 1980]. Any changes in the regulations reducing permitted levels of nitrite in cured meats are likely to affect microbiological stability of the product [Gibson *et al* 1984]. A reduction in levels of nitrite in foods would require substitution with another suitable preservative to keep the risk of botulism poisoning within acceptable levels.

Unlike food additives, chemical contaminants are usually evaluated after their potential or actual presence in food is recognized. Since no proponent submits toxicological data as in the case of food additives, the necessary data are obtained from published scientific literature. The toxicological database for chemical contaminants is therefore often incomplete. The probable daily intake of the contaminant is then estimated based on the identification of all foods that may contain the contaminant, the intake of those foods by general and target populations, and consideration of other routes of exposure, for example in air or water. If the probable daily intake exceeds the tolerable daily intake derived from the database, various risk management options may be considered. Options include establishing guidelines or legally-binding tolerances for the contaminant, restricting the sale or distribution of foods obtained from the source locality, and recommending or issuing advisory notices about consumption of contaminated foods. Consideration is also given to whether the nutritional benefit of a food outweighs any measure to restrict consumption of a staple in the diet. If the contaminant is proven to be a carcinogen, exposures would be reduced to a level as low as reasonably achievable, social and economic factors being taken into account.

Pest Control Products Act

The *Food and Drugs Act* and the *Pest Control Products (PCP) Act* are the relevant federal instruments for the assessment and management of risks from pesticides. Under the *PCP Act*, safety, merit and value have to be considered in the assessment of potential risks from pesticides. This fundamental principle focuses specifically on the protection of human health and the environment, and product performance. Standard risk assessment procedures as outlined in the Health Protection Branch risk management booklet [HPB 1990] are followed.

The maximum legally permitted residue levels for pesticides which have undergone detailed risk assessment typically range from 1-5 parts per million (ppm). Other pesticides are subject to a maximum residue limit of 0.1 ppm. Actual residue levels of pesticides in food are generally lower than the regulatory limit. Food market-basket surveys indicate that most pesticides are generally not detected.

Monitoring for compliance of pesticide residues in food under the *Food and Drugs Act* and associated *Regulations* is conducted by laboratories within Health Canada including the newly formed Canadian Food Inspection Agency.

Drinking Water Material Safety Act

In Canada, it is the responsibility of municipal water authorities to decide how to adapt treatment processes in order to implement provincial-territorial drinking water limits. To assist municipalities — and individuals who rely on private water supplies — the federal Minister of Health introduced the *Drinking Water Materials Safety Act* in December 1996. The purpose of the *Act* is to protect the health of Canadians by preventing unsafe drinking water materials from being sold or imported into Canada. The *Act* would provide for the certification (by accredited third-party certification organizations) of water treatment devices, water treatment additives and water system components to which health-based performance standards have been established. For example, chemical additives such as chlorine-based disinfectants and fluoride would be regulated, as well as materials that come in contact with treated drinking water and household drinking water treatment devices [Bureau of Chemical Hazards 1995; Health Protection Branch 1995a]. In 1996 and 1997, Health Canada held a series of public consultations designed to elicit feedback on this initiative.

Canadian Environmental Protection Act (CEPA)

In addition to a number of source control measures, *CEPA* includes measures for point-of-use control, including environmental guidelines and codes of practice (further information about *CEPA* may be found in the "Source Control" section and in Appendix C).

4.2.3 Public Exposures

Some carcinogenic chemicals exist naturally in the environment; exposure to these substances varies primarily as a function of proximity to the sources. For example, although the level of arsenic in drinking water supplies is generally less than 5 micrograms per litre (µg/L), levels range from 50–500 µg/L in the vicinity of natural sources. The interim maximum acceptable concentration of arsenic in drinking water supplies is set at 25 µg/L, on the basis primarily of achievability at reasonable cost. It has been designated as *interim* to be reviewed periodically in light of developments of treatment technology and additional data on health risks primarily due to the high estimated lifetime skin cancer risk of one in one thousand at this level.

Ames *et al* [1990] have pointed out that natural sources of carcinogens can be significant. They have suggested that the public is ingesting, on average, about 10,000 times more pesticides from natural sources than from industrial sources, and have argued that the carcinogenic risks of these may be greater than those of synthetic pesticide residues in food.

Natural carcinogenic pesticides and chemicals found in fruit and vegetables include methoxypsoralen, limonene, caffeic acid, and aflatoxin. A recent review by the U.S. National Research Council [1996] supported the hypothesis that risks from natural carcinogens found in the food supply may outweigh the risks from synthetic chemical contaminants, although additional research was called for to establish support for this conclusion. The potential health effects associated with ingested food contaminants does not imply that individuals should avoid certain foods. Rather, it is known that natural foods also contain protective factors which tend to decrease the carcinogenic effects of chemicals from natural and industrial sources [Doll 1992].

In general, exposure levels to synthetic chemicals are well below regulatory standards and guidelines. Doll and Peto [1981] have calculated that about 80% of all cancer deaths in North America are due to factors such as dietary habits, cigarette smoking, infections, and reproductive or sexual behaviour (Table 2). Although the values in Table 2 are uncertain, industrial products and food additives together are believed to account for less than 2% of all fatal cancers in the general public. The corresponding values calculated by Travis *et al* [1991] and by Gough [1990] are in the range of 0.25% to 2%. In general, the values calculated by Doll and Peto [1981], listed in Table 2, have stood up remarkably well in light of more recent scientific reviews of available evidence [Krewski 1987, Henderson *et al* 1991, Ames *et al* 1995, Trichopoulus *et al* 1996, Willett *et al* 1996]. Miller [1992] has suggested that the proportion of cancers attributed to occupation might be underestimated by a factor of about 2.

It is important to recognize that although there has been no evidence of cancer associated with exposure to chemical contaminants in the general environment, there have been serious non-cancer effects resulting from releases of pollutants to the environment. Studies have linked hospitalizations due to respiratory illness to summertime concentrations of ozone and particulate matter as well as elevated ambient levels of carbon monoxide and the coefficient of haze in regions across Canada [Burnett *et al* 1995; Thurston *et al* 1994; Burnett *et al* 1996; Stieb *et al* 1996; Delfino *et al*, 1996]. In addition, particulate matter and carbon monoxide have been linked to cardiac disease and cardiovascular mortality [Burnett *et al* 1996; Ozkanyak *et al* 1995]. Based on the results from these studies and a number of similar investigations conducted worldwide, it now appears clear that more adverse cardio-respiratory health events occur on days when ambient air pollution is elevated. However, none of these studies has been able to demonstrate a statistically significant association between ambient concentrations of ozone and either deaths or hospitalizations for cardiac diseases.

Table 2. Estimated Proportion of U.S. Cancer Deaths Attributed to Various Factors

Factor	Best Estimate of Percent of all Cancer Deaths
Diet (including fat intake, meat intake, obesity)	35
Tobacco (primarily cigarette smoking)	30
Infection (including certain viruses)	~10
Reproductive and sexual behaviour (including number of sexual partners, number of children)	7
Occupation	4
Alcohol	3
Geophysical factors: ultraviolet light	1 – 2*
Geophysical factors: ionizing radiation	2.5*
Pollution (including combustion products in air, chlorinated water supplies)	2
Medicine and medical procedures	1
Industrial products	<1
Food additives	<1

Table 2 derived from Table 20 in Doll and Peto (1981)

* Data on the ultraviolet component of sunlight are derived from the text in Doll and Peto (1981); data on the ionizing radiation component were increased on the basis of recent ICRP 1991-1993 risk estimates and recent data on background exposures to radiation from natural sources (UNSCEAR 1993).

Considerable efforts have been made in Western countries to reduce pollution from the combustion of fossil fuels. Several countries, including Canada and the United States, established new stringent guidelines and standards for air pollutants such as sulphur dioxide, nitrogen dioxide, carbon dioxide, ozone, and particulate matter. In Canada, the National Ambient Air Quality Objectives [Environment Canada 1994] for these pollutants are rarely exceeded.

4.2.4 Summary

In summary, the risks associated with chemical hazards are controlled primarily through implementation of various federal and provincial regulations and standards. Source controls are used primarily for environmental contaminants, while point-of-use standards are used for contamination in food and drinking water. Point-of-use controls are also implemented through, for example, the *Guidelines for Drinking Water Quality* and the *Food and Drugs Act*. Regulation of industries producing or

using chemicals is the responsibility of each province. Criteria and standards governing industrial discharges to water are based on the prevention of toxic effects in aquatic life; emissions to air are based on the prevention of harmful effects in humans and vegetation. Limits for releases of carcinogens into the environment are established on a case-specific basis. In Ontario, short-term (30 minutes) atmospheric emissions of carcinogens must not exceed by more than a factor of 15 the ambient air quality criteria for specific contaminants, which are generally based on a lifetime cancer risk of one in a hundred thousand (10^{-5}) to one in a million (10^{-6}). Regulations and standards are legally enforceable by the province.

4.3 Microbiological Hazards

In Canada, regulations for microbiological contamination in food are established under the *Food and Drugs Act*. Unlike chemical or radiological hazards, microbiological hazards are highly sensitive to environmental conditions such as temperature changes. Thus, they may increase or decrease in municipal water supplies, or during production, processing, storage, retailing, and home preparation of foods. Therefore, risk management strategies do not usually aim at achieving a defined level of risk. Instead, risks are reduced by first subjecting water or food to treatments to bring about a specific number of ten-fold reductions of specific target organisms, and then preventing recontamination by, for example, the use of appropriate packaging, limiting the growth of organisms by such means as refrigeration, dehydration or curing. Examples of treated food include pasteurized milk, canned goods, and refrigerated foods.

Point-of-consumption approaches are used in the management of microbiological hazards, and are carried out at the both the federal and provincial levels. These strategies have been developed on an *ad hoc* basis, and have not yet been derived from microbiological risk assessments. However, it is expected that the basic control procedures for pathogens will apply. In addition, microbiological hazards are managed by surveillance of human infections and disease, monitoring of microbiological pathogens and their indicator organisms in the environment (i.e. food, water, soil, feed, farm animals), voluntary guidelines for industry, training of employees, and education of both those responsible for manufacturing or selling safe products, and the general population.

4.4 Drinking Water

The *Guidelines for Canadian Drinking Water Quality* are described in this report as an example of how radiological, chemical, and microbiological risk assessment and management practices are combined within a flexible risk reduction strategy. In Canada, the quality of drinking water is primarily the responsibility of the provinces and municipalities. Health Canada works in collaboration with provincial health and environment ministries to establish national guidelines for drinking water quality under the auspices of the Federal-Provincial-Territorial Committee on Environmental and Occupational Health. The *Guidelines* are intended to facilitate the delivery of high quality drinking water to Canadians [Health Canada 1995, Krewski *et al* 1996].

Development of the drinking water guidelines is a flexible process designed to accommodate the needs of the various jurisdictions involved. The steps of risk assessment and risk management are clearly delineated in the development of Canadian Drinking Water Guidelines, with Health Canada recommendations for genotoxic carcinogens being as low as possible. Maximum acceptable concentrations for these compounds are then established by a Federal-Provincial Subcommittee, taking into account feasibility and costs. These include identification of substances to be reviewed, assessment, evaluation, decision making and approval, announcement and publication of decisions, and re-evaluation of findings as required. Certain steps may be modified in order to satisfy the needs of the jurisdictions involved. Through this consensus-based development process, a guideline is established, and the associated health risk assessment is modified to create a criteria summary that reflects the risk management decisions involved in the guideline's development.

Although not mandatory, the *Guidelines* may be used by the provinces and territories as a basis for setting maximum permissible levels for radionuclides, chemicals, and microbiological hazards. Provinces may adopt the *Guidelines* in whole or in part, or may establish their own criteria.

Radionuclides

Guidelines for radionuclides in drinking water conform to international radiation protection methodologies, including recommendations of the World Health Organization [WHO 1993]. As a result of the method of dose limitation recommended by the WHO, 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, the guideline dose for radionuclides applies to the total dose received from all radionuclides in the water supply.

Maximum acceptable concentrations (MAC) for radionuclides in drinking water are based on a committed effective dose of 0.1 mSv from one year's consumption of drinking water, consumed at the rate of two litres per day, or one-tenth of the ICRP's recommendation on total public exposure from regulated sources. The guideline reference dose is based on the total activity in a water sample, whether the radionuclides appear singly or in combination, and includes the dose due to both natural and artificial 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.

Because radionuclide guidelines are based on a reference dose, rather than actual concentrations in water, MACs are orders of magnitude greater than concentrations currently observed in, for example, the Great Lakes [Ahier and Tracy

1995]. The estimated average annual dose from drinking water from all radionuclides in the Great Lakes is on the order of 0.001 mSv per year, which corresponds to a 70-year lifetime risk of fatal and weighted non-fatal cancer of about four per million. This dose represents about 1% of the Health Canada guideline for radionuclides in drinking water or about 0.05% of the average annual dose attributable to radiation from natural sources. Specific cases where doses are higher occur as a result of natural radionuclides, such as radium-226, in ground and well water.

Water supplies which would result in a total radiation dose below the reference level are considered acceptable for consumption based on radiological considerations. However, treatment of water supplies for radionuclides should be governed by the ALARA principle of keeping exposures as low as reasonably achievable, economic and social factors taken into consideration, and levels may be reduced further if justified. In cases where a single sample does not meet the guideline, the reference dose would be exceeded only if exposure to the same measured concentration were continued for a full year. Hence, such a sample does not in itself imply that the water is unsuitable for consumption, and should be regarded only as a level at which further investigation, including additional sampling, is needed. Guidelines do not constitute an approval to permit the increase of radionuclide concentrations to the MAC; any facility contributing radionuclides to a drinking water source must meet the regulatory requirements of the AECB.

As noted in the introduction, the Ontario Ministry of Environment and Energy has established an interim objective of 7000 Bq/L for tritium in drinking water. Ontario Hydro Nuclear Plants, the only significant industrial source of tritium in Ontario, have agreed to keep average annual concentrations of tritium in drinking water at nearby pumping stations to less than 100 Bq/L. It might be noted that the average 1994 concentration of tritium in drinking water at the Ajax pumping station was 15 Bq/l, and about half of this concentration was due to the nearby Ontario Hydro Pickering Nuclear Generating Station with the remainder due to residual fallout from nuclear weapons testing and to natural sources. Tritium in drinking water accounted for about one percent of the total industrial radiation dose received by all persons living within 30 km of the Pickering station in 1994 [Ontario Hydro Nuclear 1995].

Chemicals

Different approaches to guidelines are adopted for carcinogenic versus non-carcinogenic chemical contaminants. In the case of non-carcinogenic chemicals, it is generally assumed that the dose-response relationship demonstrates a threshold below which no adverse health effects are observed. For carcinogenic chemicals, it is generally assumed that carcinogenesis is a non-threshold phenomenon. Consequently, carcinogenic chemicals should ideally be absent from drinking water. However, the incremental risks associated with exposure to low levels of these chemicals in drinking water may be sufficiently small so as to be essentially negligible compared with other risks commonly encountered in society.

Maximum acceptable concentrations (MACs) for substances not known to be carcinogenic are based on a tolerable daily intake (TDI) for organ-specific neurological/behavioural, reproductive or teratological effects. Where possible, the TDI is derived by dividing the lowest no-observed-adverse-effect level (NOAEL) obtained from long-term ingestion studies by an uncertainty factor. Uncertainty factors are derived on a case-by-case basis; in general, however, a factor of 1 to 10 times is used to account for various elements of uncertainty including intra-species variation, inter-species variation, nature and severity of the effect, adequacy of the study and the use of a lowest-observed-adverse-effect-level (LOAEL) versus a NOAEL. An additional factor of 1 to 5 times can be incorporated where there is information indicating a potential for interaction with other chemicals. If the chemical in question is an essential nutrient at low concentrations, dietary requirements can also be taken into consideration in deriving an uncertainty factor. Finally, in certain cases, an additional factor of 1 to 10 times can be applied to account for limited evidence of carcinogenicity.

Where appropriate, the MAC is based on intake in the most sensitive subpopulation (e.g. pregnant women, children). When appropriate data exist on other sources of exposure (e.g. air, food, soil), a proportion of the TDI can be allocated to drinking water in the calculation of the MAC. In the absence of such data, a default allocation of 20% is used. When a MAC is less than levels considered to be reliably measurable or achievable, an interim MAC (IMAC) is established, and improvements in methods of measurement or treatment are recommended.

MACs are set as close to zero as reasonably practical, on the basis of consideration of the following factors:

- the MAC must be achievable by available water treatment methods at reasonable cost;
- where possible, the upper 95% confidence limit for the lifetime cancer risk associated with the MAC is less than ten in a million to one in a million (10^{-5} to 10^{-6}), a range that is generally considered to be *essentially negligible*. In cases where intake from sources other than drinking water is significant, the upper 95 % confidence limit for the lifetime cancer risk associated with the MAC is less than or equal to one in a million (10^{-6});
- the MAC must be reliably measurable by available analytical methods.

The guidelines recommend that where estimated lifetime cancer risks associated with the MAC are greater than one in a hundred thousand (10^{-5}) to one in a million (10^{-6}), an interim MAC be established, and improvements be made in methods of measurement and/or treatment.

The levels of lifetime risk associated with MAC levels of various chemical carcinogens vary under different circumstances from about one in ten million for dichloromethane in drinking water to about one in one thousand for arsenic in drinking water (Table 3). In general, guidelines exceeding risks of one-in-a-hundred thousand (10^{-5}) to one-in-a-million (10^{-6}) are associated only with natural chemical contaminants that

may have a significant background, such as arsenic. In cases where actual exposure levels approach or exceed the guideline, usually only a small population is exposed.

Microbiological Hazards

Microbiological considerations are based on disease-causing or pathogenic microorganisms that commonly occur in polluted water. Pathogenic microorganisms present in surface water include certain protozoa, bacteria and viruses; protozoa are not commonly found in ground water. The most common illnesses attributable to waterborne pathogenic microorganisms are gastrointestinal illness and diarrhea, although more serious health effects may occur, including death. For some waterborne pathogenic microorganisms, notably the protozoan *Giardia lamblia* or hepatitis A virus, one infectious unit of virus or a single protozoan can cause illness.

While the desired goal in terms of public health protection is zero risk of illness from waterborne pathogens, it is rarely technically and economically feasible. Instead, *acceptable* microbial risks are derived and used in risk assessment. The MAC for total coliform bacteria in drinking water is zero organisms per standard sample, although some samples may contain very low numbers to account for the non-uniform distribution of coliforms in water. No consecutive samples from the same site should contain coliform organisms. The Federal-Provincial Subcommittee on Drinking Water is considering the U.S. Surface Water Treatment Rule which has set a risk of one infection per 10,000 people per year as a health goal for exposure to one particular type of protozoa in treated drinking water. Thus most public water systems must disinfect as well as provide filtration. Using this approach, treatment must achieve at least 99.9% removal or inactivation, or both, of Giardia, and 99.99% removal or inactivation, or both, of viruses.

Disinfection and other treatment methods are recommended to prevent waterborne diseases and ensure good quality drinking water. The Federal-Provincial Subcommittee on Drinking Water emphasizes that the health risk, such as carcinogenicity, associated with the use of disinfectants must also be considered.

Summary

In summary, exposure limits based on lifetime risk for most individual chemical carcinogens are more stringent than corresponding limits for all radionuclides combined. With the exception of arsenic, the lifetime cancer risks associated with the MACs for individual chemical carcinogens in drinking water are significantly lower than the risk for all radionuclides (Table 3). However, according to internationally accepted practice, the total dose from all radionuclides is evaluated and compared with the guideline reference dose. No attempt is made to evaluate the potential risk of all chemical contaminants combined because of the large number of chemical contaminants that may be present in drinking water, not all of which have been assessed. Unlike radionuclides, background exposures are generally not considered in establishing chemical exposure guidelines. In addition, many of these act by different mechanisms leading to the development of different types of tumours. Information on the potential interactions between chemical contaminants in drinking water, or between radionuclides and chemicals [UNSCEAR 1982], is also generally lacking.

Harmonizing drinking water guidelines for chemicals and radionuclides would require consideration of a number of fundamental risk assessment and risk management issues. Guidelines for radionuclides are set to protect public health at a given risk level; actual exposures are generally much lower. Chemical exposure guidelines are set at the lowest achievable level that is both protective of human health and cost effective. To achieve harmonization, there are technical, regulatory and jurisdictional issues that would have to be resolved, as well as the basic question of whether harmonization would result in public health benefits. Future discussions on harmonization should take place in a broader context in which all relevant public health concerns are addressed. For example, in addition to chemicals and radionuclides in drinking water, the impact of microbial agents on public health needs to be assessed.

Table 3. Estimated Lifetime Cancer Risks from Selected Carcinogens in Canadian Drinking Water*

Agent	Risk per Million People based on Continuous Exposure at Maximum or Interim Maximum Acceptable Concentrations
Arsenic	890
Benzene	3.1 – 34
Benzo(a)pyrene	0.5
Carbon tetrachloride	1.7 – 5.2
1,2-Dichloroethane	8
1,4-Dichlorobenzene	0.6 – 2.2
Dichloromethane	0.085
2,4,6-Trichlorophenol	2.2
Trihalomethanes (chloroform)	3.6
Vinyl chloride	10
All radioactive materials combined in drinking water**	400

* As derived from Guidelines for Canadian Drinking Water Quality, Supporting Documentation, 1995 Revision, developed jointly by Health Canada and the provinces.

** Because radionuclide guidelines are based on a reference dose, rather than actual concentrations in water, Maximum Acceptable Concentrations are orders of magnitude greater than concentrations currently observed in, for example, the Great Lakes.

5. Discussion

Complete elimination of exposures to carcinogens in the environment, synthetic or natural, is not technically feasible. If cancer can potentially occur at any level of exposure (i.e. the linear no-threshold hypothesis) then complete elimination of potential risk is not possible. Consequently, it is important to have an operational concept of safety which is more practical than that of zero risk. Such an approach uses the concept of *acceptable* or *essentially negligible* risk to determine the exposure levels at which carcinogens are regulated. Risk assessments for ionizing radiation and genotoxic chemicals assist in establishing a basis on which to recommend permissible limits.

The degree to which environmental carcinogens should be controlled requires consideration of the risks of health effects and their severity, the benefits of the associated practice or industry, the costs of mitigation, and societal priorities, all in accordance with legal statutes underlying carcinogen regulation. An operational definition of safety which is consistent with the linear no-threshold assumption of cancer risk is that of very low, but non-zero, levels of exposure which reduce risk to the greatest extent feasible, with consideration given to economic and social factors. This introduces options for risk management other than complete elimination of exposure [Hrudey and Krewski 1995].

The goal of radiation risk management strategies has been to reduce public exposures arising from regulated practices to levels that are small in comparison with the unavoidable natural radiation background, based on a balancing of costs and benefits. For chemical carcinogens, the objective has been to reduce exposures to the extent possible, based on cost of mitigation or prevention and the best available economic technologies.

The disparity between risk management practices and risk levels deemed *acceptable* for ionizing radiation and genotoxic chemicals in the environment is not easily resolved. Although principles for risk management are similar, significant differences exist as a result of differences in behaviour between ionizing radiation and genotoxic chemicals, the primary sources of data used for risk assessment, the range over which dose-response relationships have been characterized, and the degree to which organ- and species-specific differences to carcinogenic effects have been characterized and incorporated into assessment methodologies [NCRP 1989]. However, the NCRP 1989 report cited does not deal with exposure limits or *acceptability* of risks and as such is of limited value for the present report.

There are also significant differences in the consideration of background levels from natural sources, with which the effects of regulated sources of ionizing radiation and genotoxic chemicals are often compared. All radiation exposures, whether natural or artificial in origin, are treated collectively when compared with natural background exposures. In contrast, individual chemicals are compared with the same chemicals when a natural background exists, rather than with the entire background of natural carcinogens since it is generally assumed that exposures from different chemicals are not additive; rather, effects may depend on interactions between specific contaminants.

In addition, the end-point of concern in setting criteria for chemical releases to the environment is sometimes based on ecological health, rather than human health, when it is assumed that other species are more sensitive to health effects. For example, in Ontario, release limits for emissions to waterways are generally based on toxic effects in aquatic life. In setting criteria for radiation practices, the AECB considers primarily human health, on the basis that for carcinogenic effects, the protection of human health implies the protection of environmental quality [ICRP 1977, 1991]. Criticism of this assumption however was sufficient to result in the inclusion of "releases of radionuclides from nuclear facilities (impact on non-human biota)" on the most recent *Priority Substances* List of *CEPA*. As a result, releases of radionuclides are currently undergoing an ecological risk assessment under *CEPA* to determine whether or not they are toxic according to the definition of "toxic' found in Section 11 of *CEPA*. If found to be *CEPA-toxic* then releases of radionuclides from nuclear facilities would be managed in accordance with the federal government's *Toxic Substances Management Policy*.

Radiation protection is based on a well-established system of priorities that has been accepted for many years. The first priority is the prevention of immediate health effects from ionizing radiation exposure, among both workers and the general public. Subsequent priorities are concerned with controlling long-term health effects resulting from exposure to low

levels of radiation consistent with social and economic factors. Although no immediate effects arise from such exposure to low doses at low dose rates, the potential for the appearance of a cancer at some later date is assumed to depend on the cumulative dose received over an extended period of time. As a result, long-term averages, most commonly on a yearly basis, are emphasized in regulating radiation exposures.

Since the interactions of radiation and tissues are basically the same, regardless of the type of radiation or tissue, the risk from all radiations and radionuclides can be assessed collectively by employing a set of average weighting factors that take into account the effectiveness of the radiation in causing harm, the sensitivities of the various tissues to cancer induction, and the severity of the types of cancer. This contrasts with chemical risk assessment which treats all cancers more or less equally.

The management of radiation risks is based on a system of dose limitation as recommended by the International Commission on Radiological Protection. The ICRP has recommended public dose limits based on the boundary between *unacceptable* and *tolerable* risk. These limits are derived from both a judgement on *acceptable* risk, and the variations in dose from unavoidable background radiation. Man-made practices resulting in doses which are small in comparison to background, while not necessarily justified, do imply that the radiation risk situation of the exposed individual is not significantly changed by the new practice. The ICRP has indicated that continued exposure of the public from deliberate practices at or near the limit for many years is not acceptable. Actual exposures are to be kept below the legal dose limit by application of the ALARA principle, requiring that exposures be kept as low as reasonably achievable, economic and social factors being taken into account.

Public exposures to ionizing radiation have been controlled to low levels through attention to this principle. The AECB requires that a licensee demonstrate that a nuclear power plant operates in a manner which results in annual doses no greater than 0.05 mSv per group of radionuclides to individuals in the population group at greatest risk. The AECB considers that such demonstration is equivalent to satisfying an ALARA process. Emissions are continually monitored and should those for a given week exceed the specified operating emission levels, examination of procedures and facility design by the licensee is required to determine what actions, if any, are necessary to ensure that the annual emission targets can be achieved. Maximum annual public exposures from nuclear generating stations in Canada in 1994 were about 0.01 mSv to a hypothetical individual living outdoors near the plant boundary 24 hours per day all year. Doses to populations situated further from these stations were significantly less. These doses are well below both the current legal limit of 5 mSv per year specified in licences issued by the AECB, and the proposed public limit of 1 mSv per year, corresponding to background radiation, which the AECB is in the process of adopting. The theoretical lifetime risk associated with an exposure level of 0.01 mSv is less than five in one hundred thousand, or about 0.5 % of the risk associated with natural background radiation.

Risk management practices for chemical exposures arose initially out of concern about carcinogenic additives and contaminants in food, eventually extending in the 1960s and 1970s to the regulation of industries releasing chemicals into the environment. In regulating exposures arising from industries producing or using chemicals, limits on allowable emissions for individual chemicals are usually set to the lowest achievable level considering both the environmental and health risk, and the best technology economically available.

Consideration of natural background is implicitly incorporated in the concept of best economically available technology (i.e. that technologies are often not available at reasonable costs to reduce high naturally occurring levels), however, this is done on a chemical-specific basis, rather than as a comparison of all chemical carcinogens with the total background of natural carcinogens. Synthetic chemicals are often regulated to very low levels since they do not exist naturally in the environment.

Based on the absence of a general model of carcinogenesis for all chemicals, it is not possible to establish an absolute maximum exposure limit equivalent to the ICRP dose limit for radiation. Rather, limits for releases of carcinogens are governed on a case-by-case basis. For example, Ontario has implemented a limit on short-term (30 minutes) releases of carcinogens into the air of no more than 15 times the ambient air quality criteria for specific contaminants, which are generally based on a lifetime risk of one in a hundred thousand to one in a million (10^{-5} to 10^{-6}). Health Canada does not, in general, recommend levels of risk for chemical carcinogens, as it is considered more appropriate to base management options on consultation with affected parties and on a risk-benefit approach in terms of cost and feasibility. For example, in establishing guidelines for drinking water quality, maximum acceptable concentrations have been based on an *essentially negligible* lifetime risk level of one in one hundred thousand to one in a million (10^{-5} to 10^{-6}), where feasible. However, actual risk levels associated with carcinogen guidelines range from about one in a thousand to one in ten million (10^{-3} to 10^{-7}), based on practical considerations. (Values are derived from Table 3).

As with radiation exposure, long-term cumulative exposures to chemical carcinogens are emphasized. If limits or guidelines are exceeded, a number of actions may be taken by the regulating authority, depending on the nature of the offence, and whether or not the limit is legally enforceable. These may include recording the number of days in a year in which recommended limits are exceeded, issuing public health advisory statements, temporarily closing the source of the contaminant (e.g. a municipal water supply), or instituting action against the responsible party. By contrast, short-term exposures are important in the regulation of microbiological hazards, where health effects may be observed within a few days or weeks.

The balancing of risks and benefits finds appreciable application in the regulation of both ionizing radiation and genotoxic chemicals, even though the formal application of this principle is not as fully developed for the latter. This approach is being considered in the development of risk management strategies for substances found to be toxic under the *Canadian Environmental Protection Act*, and that are *Track II* in the *Toxic Substances Management Policy*. Health risks and benefits are also evident with respect to management of microbiological risks in drinking water. In this case, there is a need to balance the health effects of disinfecting chlorinated and carcinogenic organic chemicals in municipal water supplies against the need to protect the public against infectious disease.

The disparity in potential health risks associated with the legal dose limits for ionizing radiation and genotoxic chemicals has often been a source of contention amongst various groups in society. In such cases, the basic principles involved in regulation need to be emphasized. The legal limits for radiation exposure apply to all regulated practices collectively, and represent a maximum value below which actual exposures must be maintained and reduced to the degree feasible. Legal limits for chemicals are contaminant-specific, and since they are based on a concentration level that is achievable, they are generally closer to actual exposure levels. In both cases, the excess risk associated with regulated practices does not appreciably alter the total risk environment to which humans are exposed.

For example, a critical review of the risk of fatalities from all causes resulting from the generation of electricity in Canada has been made by the Advisory Committee on Nuclear Safety [ACNS-10 1991(Rev.)]. This review consisted of estimates of risk from normal operations and anticipated accidents at the major electrical energy systems in Canada, including risks associated with fuel supply and transport, power plant construction, plant operation and waste management. The ACNS concluded that the risk of public fatalities per gigawatt-year of electricity produced in Canada using state-of-the-art technology is in the range of 4.5 to 7 for coal-fired plants, 0.06 to 0.3 for nuclear plants and 0.003 to 0.04 for hydroelectric plants.

The U.S. EPA Science Advisory Board has considered the merits of continuing to regulate ionizing radiation and chemical carcinogens separately as compared to regulating them in a similar manner [US EPA SAB 1992]. In the latter case, options may range from requiring the same levels of lifetime risk to be used as a basis for remediation or regulation (e.g. one in ten thousand, or one in one thousand where remediation is not easily achievable), or simply using the same policy framework without requiring that the same levels of lifetime risk be used. The EPA Science Advisory Board made no recommendation on harmonization in this report. The topic of harmonizing regulatory approaches to radiation and chemical hazards has also been discussed by Kocher and Hoffman [1991] and Overy and Richardson [1995].

Table 4 summarizes values derived from a number of different sources cited in the text, and provides a useful comparison of cancer risks to the public from ionizing radiation and from chemicals. Although the specific details of various risk management strategies have not been stressed in this report, Table 4 can be considered as a summary of the results of risk management (i.e. the risk remaining following implementation of risk management strategies). With the exception of cigarette smoking, which is a well characterized hazard [Illing and Kaiserman 1995], all listed values are theoretical. All data in this table are upper-limit estimates based on the conservative assumption of a linear no-dose threshold model. Although the risks associated with exposures to individual chemicals present in the environment are believed to be low, the large number of such chemicals may lead to cumulative risk which potentially exceeds the cumulative risk from all exposures to environmental radionuclides. There may also be synergism or antagonisms between radiation and chemical exposure [UNSCEAR 1982].

Table 4. Estimated Lifetime Cancer Risks from Chemicals and Radionuclides (All Numbers Given Are Rough Approximations)

Agents	Risk per Million People based on Continuous Exposure at Average Environmental Concentrations
Industrial sources: • All chemicals combined • All radionuclides combined	about 1,400 [(a)] < 1 [(b)]
Drinking water: industrial, natural and other sources combined: • All chemicals combined • All radionuclides combined	unknown < 1 [(c)]
Natural sources • Radon in homes • Other sources of radioactivity • Chemicals from natural sources in food	2,500 [(d)] 4,000 [(e)] unknown [(f)]
Cigarette smoking	93,500 [(g)]

Note: Overall cancer mortality in Canada is 280,000 per million. [Statistics Canada 1993, 1995]

(a) U.S. data derived from Doll and Peto [1981], Gough [1990] and Travis and Hester [1991]. Comparable Canadian data are not readily available. The range is 700 to 5,600 per million people exposed. Although these values for chemicals were generally derived using standard methods, they may be very conservative (i.e. too high) as pointed out in the text of this report.

(b) Data derived from ICRP [1991] risk estimates and estimates of collective dose to i) persons living within 30 km of nuclear generating stations in Canada [Myers et al 1994], and ii) the public from uranium refining and conversion facilities and fuel fabrication plants, as derived from AECB data. The total collective dose to the Canadian population from these sources was about 3 person-Sv per year. The total collective dose in Canada from the global distribution of long-lived radionuclides from nuclear power is negligible by comparison [UNSCEAR 1993].

(c) Value based on the assumption that the major source of radioactivity in drinking water for most Canadians is tritium from nuclear weapons testing, at a current level of about 10 Bq/L (the guideline value of 7000 Bq/L of water is equivalent to 0.1 mSv per year).

(d) Value based on the risk estimates given in ICRP Publication 65 [1993] and an average measured value of about 30 Bq radon/m^3 of air in homes in Canada [Létourneau et al, 1992].

(e) Value based on ICRP [1991] risk estimates and an average exposure of 1 mSv/year to Canadians from natural radiation sources other than radon.

(f) No reliable value has been published. An estimate of about 10,000 might be given assuming that about 10% of the total cancer hazard attributed by Doll and Peto [1981] to diet (see Table 2) might be due to natural carcinogens in food [Ames et al 1990]. It is further assumed that 28% of all deaths in Canada are due to cancer at current rates [Statistics Canada 1993, 1995]; if non-fatal cancers are weighted according to ICRP [1991], the total harm from all cancers would be about 18% higher than that from fatal cancers alone.

(g) Data derived by attributing 33% of all cancer deaths to tobacco, primarily cigarette smoking [Peto *et al* 1994] (see Table 2), and assuming that 28% of all deaths in Canada are currently due to cancer.

This report has attempted to provide an overview of the methods and approaches used for the assessment and management of ionizing radiation and genotoxic chemicals subject to regulation. While there are differences, it appears that both systems offer effective protection of public health at actual environmental exposure levels, based on a lack of observable health effects using current epidemiological methodology. A summary of important similarities and differences, is provided in Table 5.

Table 5. Comparison of Risk Assessment and Management Aspects for Ionizing Radiation and Carcinogenic Chemicals

Risk Assessment	Ionizing Radiation	Carcinogenic Chemicals
Sources of contaminants	Natural and artificial.	
Number of potential carcinogens	Relatively stable and known.	Number continues to increase.
Types of effects at environmental exposure levels	Long-term carcinogenic effects which are indistinguishable from cancers caused for other reasons.	
	Hereditary effects questionable.	Effects such as immunological hypersensitivity.
Sources of risk data	Primarily epidemiological studies on humans.	Primarily toxicological studies on animals.
Risk assessment approach	All exposures are assessed using a single unifying approach.	Different approaches are used for different agents.
Risk extrapolation	Linear no-threshold extrapolation from high dose data. Some evidence for practical threshold effects for specific radionuclides.	Linear no-threshold extrapolation from high dose data for genotoxic carcinogens. Evidence of threshold effects for non-genotoxic carcinogens.
Risk estimates	Includes risk of fatal cancer, plus an allowance for non-fatal cancers weighted for severity of type and ease of curing, length of life lost or impaired, risk of serious hereditary disorders.	Different types of cancer are generally treated equally, without weighting.
Uncertainties in risk estimates	Generally less uncertainty due to reliance on human data.	Generally greater uncertainty due to reliance on animal data. Lack of data for many chemicals.

Table 5. Continued

Risk Management	Ionizing Radiation	Carcinogenic Chemicals
Goal	To minimize risk, recognizing economic and social factors.	
Sources of recommendations on exposure limits	Internationally recommended system of radiation protection.	Fewer international recommendations; general guidance from national and international organizations. Limits generally set as low as reasonably achievable, social and economic factors being considered. Limits for carcinogens in drinking water may vary 10,000 fold in theoretical risk.
Principles for controlling exposure	Limit based on acceptable risk, and variations in unavoidable natural background radiation. Limit is based on human-health considerations. Actual exposures to be maintained as low as reasonably achievable, economic and social factors taken into consideration. Limit covers all exposures from all regulated practices. Total risk of all health effects is readily calculated on the basis of international recommendations.	Limits for individual carcinogens aim for a lifetime risk of 10^{-5} to 10^{-6} with the limit dependent on best available technology economically achievable, background levels, etc. Individual limits are not compared against the total background of natural carcinogens. Although human health is generally the critical factor, limits are sometimes based on ecological considerations. Limits apply to individual chemicals, often via only one route of exposure. No attempt to calculate total risk associated with all individual limits.
Manner of implementation	Dose limitation: public dose limits lower than occupational limits. Optimization of risk-benefit. Control at source for regulated practices, and at point-of-use.	
Public dose limits for industry	Operational limits for nuclear generating facilities based on achievable levels, significantly lower than the legal dose limit.	Operational limits for industry based on achievable levels, and are similar to actual exposure levels.
Risk-benefit approach	Although applied inconsistently, given significant consideration in the optimization of health protection. A monetary limit on cost of preventing a premature death by reduction in radiation exposures from industrial sources has been recommended by AECB.	Traditionally given lesser consideration, although is being used under *CEPA*, and in context of best available technology economically feasible. No set limit on cost of preventing a premature death by reduction in exposure to chemical carcinogens.

6. Conclusions

Carcinogens in the environment cannot be completely eliminated. Because there is a possibility that carcinogens may pose some risk at any level of exposure, there is a need to assess and manage the potential risks associated with human exposure to these agents.

In Canada, regulatory practices invoke legal exposure limits for ionizing radiation and chemical carcinogens. However, there is a lack of consensus on *acceptable* levels of risk for these hazards. Rather, the *acceptable* risk levels associated with legal limits and established guidelines vary up to a million-fold. These guidelines take into account to varying degrees the specific application and agent or process being regulated, the economic and social costs and benefits and technology factors.

Risk assessment methods for both ionizing radiation and genotoxic chemicals are well developed and generally similar in principle. Risk assessment begins with the identification of a hazardous agent, and given that sufficient information is available, is followed by establishment of a dose-response relationship. This allows adverse health effects to be estimated for various levels of human exposure to the agent. The dose-response relationship for both types of hazard is assumed to be linear with no threshold dose below which deleterious effects are absent, implying that there is some probability of an adverse health effect at any level of exposure, no matter how low.

Epidemiological and toxicological data are used for both radiation and chemical risk assessments. However, radiation risks are based mainly on epidemiological studies involving atomic bomb survivors in Japan, while chemical risks are based largely on animal toxicological studies. Although epidemiological data have the advantage of avoiding extrapolation from animals to humans, toxicological data from animal studies can be used to assess potential risks in advance of human exposure. Where past working conditions and medical practices have led to appreciable levels of human exposure, this experience has been useful in identifying carcinogenic agents and in establishing dose-response relationships.

The combined risk for exposures to different radionuclides via different pathways is routinely calculated, while aggregation of the risks of different genotoxic chemical hazards is not generally attempted, given the varying nature and virtually unlimited number of the latter and the potential for synergistic and antagonistic effects among them. Another difference arises from the ability to establish the relative biological effectiveness of radiation in different body tissues, but not typically for chemical carcinogens that are effective at multiple sites.

Estimates of risk can be subject to considerable relative uncertainty at low levels of exposure, particularly when extrapolation is necessary beyond the conditions under which the original data were collected. These uncertainties are believed to be smaller for estimates of radiation-induced cancer than for estimates of chemical-induced cancer, mainly because of the nature of the data generally used in each case and because of the relatively well-known mechanisms of radiation carcinogenesis compared to a number of less well-known mechanisms for genotoxic chemicals. The Joint Working Group recognizes that it is important to characterize uncertainties in all risk estimates and urges that this be done to the extent possible.

Risk management strategies for both ionizing radiation and genotoxic chemicals are well developed and similar in principle: both set legal limits to exposures and endorse the ALARA principle (i.e. that risk should be as low as reasonably achievable taking into account social and economic factors). There are, however, differences in the application of these principles for the two types of hazards. The basis for the setting of legal exposure limits is different for radiological and genotoxic chemical hazards, including consideration of background exposures from natural sources, the manner in which exposures of species other than humans are taken into account, and the ability to account for the effects of exposures from more than one source and for long-term hazards from ingestion. The formal application of the ALARA principle is more fully developed in radiation protection, but it is not applied in a completely systematic manner in either area. Risk management strategies for both radiation and genotoxic chemical hazards employ approaches such as source controls, point-of-use controls, and educational approaches, although there are some differences as noted in Table 5.

It is not possible to determine whether or not environmental exposures to ionizing radiation or genotoxic chemicals pose the greater risk of cancer at this time. Although the risks associated with exposure to individual chemicals in the

environment are generally low, the large number of chemicals may lead to a cumulative risk which exceeds that of all exposures to radionuclides released into the environment by regulated activities. Also, uncertainties arise because of the differences in the levels of knowledge of the mechanisms of harm for the two types of hazards. Difficulties in anticipating and managing the synergistic or antagonistic effects, which sometimes occur with genotoxic chemicals, complicate the issue. Uncertainties associated with predictions of risks at low levels of exposure based on extrapolations from high levels of exposure further complicate such comparisons.

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, based on the absence of observable health effects using epidemiological methodology.

The consensus of the Joint Working Group is that it does not appear fruitful at this time to consider harmonizing the regulation of ionizing radiation and genotoxic chemicals. Future opportunities for harmonization should, however, be considered. In doing so, consideration must be given as to whether public health benefits would be derived from harmonization. Further, discussions should take place in a broader context in which all relevant public health concerns are addressed. For example, in addition to ionizing radiation and genotoxic chemicals, the impact of microbiological agents on public health should be considered.

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AECB, Advisory Committee on Nuclear Safety
AECB, Advisory Committee on Radiation Protection
AECB, Group of Medical Advisors
Federal-Provincial-Territorial Committee on Environmental and Occupational Health

P. Ayotte, CSP-Québec
M. Bélanger, Ministère de la Santé et des Services sociaux, Québec
R.H. Clarke, National Radiological Protection Board, England
A.G. Croff, Oak Ridge National Laboratory
N.E. Gentner, Chalk River Laboratories
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K. Krishnan, Université de Montréal, Montréal
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A.A. Pilt, Emergency Measures Ontario, Toronto
N. Rubin, Energy Probe
A. Scott, Ontario Ministry of Labour
L. Smith, Ontario Ministry of Health, Toronto
E. Worona, Human Resources Development Canada
W. F. Young, Water Research Center, United Kingdom

Appendix A: Joint Working Group Members

D. Krewski (*Co-Chairman*)	Health Canada
D.K. Myers (*Co-Chairman*)	Advisory Committee on Radiological Protection (ACRP)
B. Ahier	Health Canada
R.J. Atchison (*Scientific Secretary*)	Atomic Energy Control Board (AECB)
P.L. Birkwood	Health Canada
A. Boisset	Advisory Committee on Nuclear Safety (ACNS)
D. Chambers	Advisory Committee on Radiological Protection (ACRP)
M. Gaudry	Advisory Committee on Nuclear Safety (ACNS)
D.J. Gorman	Advisory Committee on Radiological Protection (ACRP)
J. Harwig	Health Canada
B. Huston	Health Canada
M.W. Lupien (*Scientific Secretary*)	Atomic Energy Control Board (AECB)
B. Meek	Health Canada
D. Meyerhof	Advisory Committee on Radiological Protection (ACRP)
A. Nahas	Health Canada
J.T. Rogers	Advisory Committee on Nuclear Safety (ACNS)
B. Thomas	Health Canada
P.A. Thompson	Atomic Energy Control Board (AECB)
B. Hanna Thorpe	Ontario Ministry of Environment and Energy
S. Vlahovich	Group of Medical Advisors (GMA)

Health Canada Correspondence Working Group:

J. Austin	P. Ghadarian
S. Bartlett	J. Salminen
R. Burnett	P.J. Waight
G.R. Douglas	J.M. Zielinski
C. Franklin	

Appendix B: Terms of Reference

Purpose

To examine the similarities, disparities and inconsistencies between the levels of risk considered *acceptable* for regulating ionizing radiation and those considered *acceptable* for regulating chemical and microbiological hazards.

Means

- Briefly review scientific approaches for assessing and managing chemical, radiological, and microbiological risks.
- Compare levels of risk associated with exposure guidelines for chemical, radiological, and microbiological agents.
- Identify similarities and differences in the risk assessment and management approaches used for the three types of risks.
- Explore the feasibility of harmonizing these approaches.

Outputs

The joint working group will submit a report to the Assistant Deputy Minister of the Health Protection Branch, Health Canada, and to the President of the Atomic Energy Control Board.

Appendix C: Overview of Risk Management Responsibilities

Atomic Energy Control Board

The *Atomic Energy Control Act*[1] regulates, among other things, the use of radioactive materials and fissile material or processes which could be used in a nuclear chain reaction. The *Act* also extends to the import and export of nuclear items, and involves Canadian participation in the activities of the International Atomic Energy Agency, as well as compliance with the requirements of the *Treaty on the Non-Proliferation of Nuclear Weapons* and other bilateral and multilateral agreements [AECB 1995].

Operators of nuclear facilities, as well as those who use or possess unusual amounts of radioactive materials in Canada, must comply with the *Act* and all regulations made pursuant to it. The *Act* covers occupational and public exposures and also requires major licensees to have an effective emergency response plan coordinated with the province and in conformity with provincial requirements. Radioactivity associated with industries and processes not covered by the *Atomic Energy Control Act* is generally the responsibility of the provinces.

The Atomic Energy Control Board (AECB) administers the *Act* and has the lead role in the regulation of nuclear facilities and users of nuclear materials. Regulatory control of nuclear facilities and nuclear materials is achieved through a comprehensive licensing system. Monitoring to demonstrate compliance with the *Act* and license conditions is the responsibility of the licensee. Independent monitoring is performed by other agencies such as provincial ministries, or Health Canada.

The AECB's licensing system is administered with the cooperation of federal and provincial government departments in such areas as health, environment, transport and labour. The concerns and responsibilities of these departments are taken into account before licenses are issued by the AECB. Emission limits or ambient environmental quality limits for non-radioactive substances under relevant federal or provincial legislation are included when appropriate in AECB licences.

Nuclear facilities that are regulated by the AECB include power and research reactors, uranium mines, mills, and refineries, nuclear fuel fabrication plants, particle accelerators, heavy water plants, and radioactive waste management facilities. The AECB also issues radioisotope licenses and permits for radioactive sources used in medical diagnosis and in radiation therapy, and for sources used in industry, research, and consumer products.

Applications for new facilities or modifications to existing facilities are reviewed to the extent necessary under the Canadian Environmental Assessment Act. If a comprehensive assessment is necessary under the *Act*, the AECB will provide comments along with other departments and members of the public to the Environmental Assessment Panel. The AECB licensing process begins once the project receives approval.

License applicants must submit comprehensive details on the design of a proposed facility, its effect on the local environment, and the manner in which it is expected to operate. The AECB reviews these submissions in detail, using existing legislation, appropriate codes of practice, and experience. Submitted designs must meet strict limits on the emissions that occur in operations and under commonly occurring upset conditions.

Once a license is issued, the AECB carries out compliance inspections to ensure that its requirements are continually met and, if necessary, prosecutes licensees who violate regulations or licensing conditions. In 1992, there were approximately 3,800 licenses distributed all across Canada and 13 prosecutions for violations of licensing requirements, primarily for minor over-exposures of an employee, were initiated by the AECB [AECB 1995b].

Health Canada

Health Canada's primary goal is to protect and improve the health of Canadians, and to ensure that health risks are minimized to the extent possible and practicable. In doing so, the Department assesses the risks associated with contaminants in food and water; the manufacture, sale and use of drugs; medical

1. A new act to replace the Atomic Energy Control Act has been passed by Parliament. The new Nuclear Safety and Control Act, which recognizes the many changes since 1946 when the existing Act was passed, had not yet come into force at the time of publication of this report.

devices; pesticides; the home and work environment; consumer products; radiation in the environment; radiation-emitting devices; tobacco; disease threats; and natural and civil disasters. The Department also develops strategies for managing these risks. Within Health Canada, the primary responsibility for health protection rests with the Health Protection Branch.

In assessing and managing health risks, the Branch conducts pre- and post-market product evaluations, conducts research, oversees marketplace removal of unsafe products, conducts regulatory field visits to food and drug manufacturing plants, negotiates agreements concerning hazardous materials in the workplace, undertakes research, provides national reference and diagnostic laboratory services, carries out national surveillance programs, investigates and manages disease outbreaks, and participates in international committees. The Branch works with industry, scientists, professional associations, workers and the public to carry out these risk management activities, and to protect and inform them.

The broad public health role of Health Canada is mandated by federal legislation and federal/ provincial/territorial agreements, and influenced by international agreements. Many programs within the Branch exist by virtue of a specific piece of legislation. Key legislation pertaining to radiation, chemicals, and microbiological hazards include the: *Food and Drugs Act; Radiation Emitting Devices Act; Canadian Environmental Protection Act; Pest Control Products Act; Drinking Water Materials Safety Act;* and *Canadian Environmental Assessment Act*. Other federal statutes that cover some aspects of the assessment, use or management of chemicals include the: *Canada Labour Code*; *Fisheries Act*; *Hazardous Products Act*; and the *Transportation of Dangerous Goods Act*.

Health Canada is the principal health advisor to the AECB and other bodies concerned with the management of radioactivity, and contributes to the risk management decisions taken by other departments through the provision of advice on health-related issues. In many instances, the federal government shares or transfers sections of regulatory control responsibilities to the provinces in areas such as health and environment.

The *Food and Drugs Act* and associated *Regulations* provides broad powers to Health Canada which allow it to impose restrictions on the manufacture, sale and advertising of foods, cosmetics, drugs, and therapeutic devices, to ensure safety and prevent deception.

The *Radiation Emitting Devices Act* (*RED Act*) is administered by Health Canada, and applies to all devices that emit X-rays or non-ionizing radiation in occupational or clinical settings (e.g. X-ray equipment, lasers, ultrasound therapy devices), or in personal use (e.g. microwave ovens, television receivers). Regulations written under the *RED Act* specify minimum safety standards for the design, construction, labelling, and advertisement of the devices or their components. The standards apply to devices at the point-of-sale, and are concerned with the performance of a device with regard to its intended function and manner of operation. The responsibility for controlling the use of radiation emitting devices belongs to the provinces. Provinces regulate and monitor exposure that may result from radiation emitting devices (but not radioactive material), as well as non-nuclear fuel cycle activities which give rise to occupational exposure to radionuclides.

The *Canadian Environmental Protection Act (CEPA)* aims to protect human health and the environment by reducing or eliminating *toxic* substances, which currently exist in Canadian commerce, from the environment, and controlling the entry of *new* substances into Canada that may adversely affect human health or the environment. Under *CEPA*, *substances* include radiation, chemicals and biotechnology products. The *Act* is administered jointly by Environment Canada and Health Canada. Health Canada's role is to evaluate the health risks of contaminants currently in the environment, and assess the potential health risks of new substances, including those created through biotechnology. The Department also develops risk management strategies to effectively control human exposure to toxic substances.

Actual control and regulation of industries producing or using chemicals is provincial responsibility. Monitoring for compliance is the responsibility of the industries with additional independent compliance monitoring performed by the provinces. As a requirement of licensing, industries emitting chemicals to the environment are responsible for emergency preparedness through the Major Industrial Accidents Council of Canada. Uncontrolled or willful discharges of chemicals to the environment may be subject to specific pieces of legislation at the federal and provincial levels, including not only the *Canadian Environmental Protection Act*, but also the *Fisheries Act*, and the *Transportation of Dangerous Goods Act*.

The *Toxic Substances Management Policy* is a new federal policy for managing toxic substances. Under the *Policy*, any substance that results from human activity, takes a long time to break down, accumulates in biological tissues, and is *CEPA toxic* or equivalent will be designated as a *Track I* substance and targeted for virtual elimination. For substances that meet some but not all of these criteria (*Track II* substances), the objective is to prevent or minimize their release throughout their life cycles (during their manufacture, use, transport and disposal), using pollution prevention approaches. Public accountability for the *Policy's* implementation will be through the Commissioner of the Environment and Sustainable Development. Federal departments including Health Canada must comply.

The *Pest Control Products Act and Regulations*, are the principal federal legislation for the control of pesticides in Canada. Pesticides include insecticides, herbicides, and fungicides. Any pesticide imported, sold or used in Canada must first be registered under the *Act*, which is administered by the Pest Management Regulatory Agency of Health Canada. A pesticide cannot be registered under the *Act* unless the Agency determines that any associated risks to people and the environment are acceptable, and that the product serves a useful purpose. The *Act* also includes the authority to ensure that the risks and value of registered pesticides remain acceptable. Although the registration of pesticides is a federal responsibility, provinces have jurisdiction over the use of pesticides within their borders. Monitoring for compliance of pesticide residues in food under

the *Food and Drugs Act* and associated *Regulations* is conducted by laboratories within Health Canada including the newly formed Canadian Food Inspection Agency.

The *Canadian Environmental Assessment Act* requires federal departments and agencies to assess the environmental implications of projects for which they have decision-making authority, whether as proponents, land administrators, funding sources or regulators. The *Act* provides a transparent and effective means of integrating environmental, health and economic factors as well as public concerns into decisions regarding projects involving the federal government. Health Canada has two major responsibilities under the *Act*: to ensure that for all Health Canada projects, environmental assessments are conducted as early as possible and before irrevocable decisions are made; and, upon request, to give departmental specialist/expert information or knowledge to other federal departments, public review panels or mediators conducting environmental assessments.

The control of drinking water quality is a provincial responsibility. National guidelines for physical, chemical, microbiological, and radiological characteristics are developed through a federal/provincial committee structure which advises the Assistant Deputy Minister of the Health Protection Branch, and for which the Secretariat is provided by Health Canada. The Provinces may adopt these guidelines as regulations or enact stricter regulations, as they see fit. Monitoring for compliance is the responsibility of the provincial or municipal drinking water supply operator, and in the case of private wells, the responsibility of the home owner.

To assist municipalities, and individuals who rely on private water supplies, the federal Minister of Health introduced the *Drinking Water Materials Safety Act* in December 1996. The purpose of the *Act* is to protect the health of Canadians by preventing unsafe drinking water materials from being sold or imported into Canada. The *Act* would provide for the certification (by accredited third-party certification organizations) of water treatment devices, water treatment additives and water system components to which health-based performance standards have been established. For example, chemical additives such as chlorine-based disinfectants and fluoride would be regulated, as well as materials that come in contact with treated drinking water and household drinking water treatment devices. In 1996 and 1997, Health Canada held a series of public consultations designed to elicit feedback on this initiative.

Regulations for microbiological contamination in food are established under the *Food and Drugs Act* and associated *Regulations*. In addition, microbiological hazards are managed by surveillance of human infections and disease, monitoring of microbiological pathogens and their indicator organisms in the environment (i.e. food, water, feed, soil, farm animals), voluntary guidelines for industry, training of employees, and education of both those responsible for manufacturing or selling safe products, and the general population.

Management of public exposures through "point-of-use" contact (e.g. food consumption) is shared by Health Canada and the provinces. The *Food and Drugs Act* and associated *Regulations* apply nationally to all food for sale in Canada, and is supported by the *Fisheries Act* and the *Meat Inspection Act*. Monitoring for compliance is conducted by Health Canada through regional offices of the Health Protection Branch. Provinces manage some aspects of local food production and inspection.

Interactions with International Organizations

Canada interacts with many international organizations in an effort to achieve a degree of international consistency in its risk assessment and management methodologies and actions. International agencies are strictly advisory bodies, having no regulatory mandate; the federal government has broad powers in the regulation of health and environmental hazards.

In the field of radiation protection, Canada relies heavily on international reviews and compilations of scientific information such as the reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), and national organizations such as the United States Committee on the Biological Effects of Ionizing Radiation (BEIR) for radiation risk assessments. Health Canada and the Atomic Energy Control Board contribute to this area through support and direct conduct of research projects.

In managing radiation risks, Canada again relies heavily on international recommendations, particularly those of the International Commission on Radiological Protection (ICRP). Canada also actively participates in the activities on the International Atomic Energy Agency (IAEA), the Nuclear Energy Agency of the Organization for Economic Cooperation and Development (OECD), and the World Health Organization (WHO) for the achievement of international consensus and harmonization of standards and approaches. Additional scientific information is obtained from national organizations such as the U.S. Environmental Protection Agency, and the American Conference of Governmental and Industrial Hygienists.

In assessing and managing chemical risks, Canada interacts with many international organizations such as the International Agency for Research on Cancer (IARC), the Food and Agricultural Organization (FAO), the International Program on Chemical Safety (IPCS), the World Health Organization (WHO), the Organization for Economic and Co-operative Development (OECD), and the Codex Alimentarius Commission. However, in general, there are no international bodies that recommend standard chemical risk management approaches. In North America, the International Joint Commission of Canada and the U.S. serves as an advisory body to both national governments on pollution management in trans-national boundary waters.

Glossary

absorbed dose	The mean energy deposited by ionizing radiation per unit mass of the body or organ or tissue of the body. Unit: gray (Gy), 1 Gy = 1 joule/kilogram.
activity	The rate of disintegration of a radioactive substance, i.e., the average number of nuclear transformations per unit time. Unit: becquerel (Bq), 1 Bq = 1 disintegration per second.
ALARA Principle	A principle of risk management according to which exposures are kept as low as reasonably achievable, social and economic factors being taken into account.
carcinogen	Chemical, physical or biological agents which can cause cancer in humans or experimental animals.
carcinogenic potency	The capability of a substance to induce tumours. It is generally expressed as a dose or a concentration associated with a specified increase in tumour incidence.
clonal expansion	A process by which a single cell with a given mutation, or change in the genetic material, may be expanded in number by cell division to form a clone of cells all with the same mutation.
carcinogenicity	The potential to cause cancer.
collective dose	The sum of the individual doses received by all persons exposed to a given source of radiation. Unit: person-sievert (p-Sv).
committed dose	The total dose received from a radioactive substance in the body during the remainder of a person's life (assumed as 50 years for adults, 70 years for children) following intake of the substance.
critical group	A fairly homogeneous group of people whose location, age, habits, diet, etc., cause them to receive an average dose from a given source, or sources, of radiation that is greater than the average dose received by any other group exposed to that source or sources.
de minimis risk	A risk that is so low that it is generally accepted as being of no significance to an individual or to society. The ACNS and ACRP recommend that an individual risk of 5×10^{-7} per year be considered a de minimis risk.
deoxyribonucleic acid (DNA)	The genetic material present in all living cells that carries coded instructions for all life processes.

dose (of radiation)	Either an absorbed dose, equivalent dose, or effective dose, depending on the context.
dose (of chemical)	The amount or concentration of a substance taken into the body.
dose coefficients	Frequently used to refer to the tissue weighting factors and radiation weighting factors recommended by the International Commission on Radiological Protection. The tissue weighting factors are used to convert absorbed dose to a given tissue into its equivalent in terms of biological harm in absorbed dose to the whole body. The radiation weighting factors are used to convert absorbed dose from one type of ionizing radiation (e.g., alpha particles) into its equivalent in terms of biological harm from another type of ionizing radiation (e.g., X-rays).
effective dose	The sum of the equivalent doses received by different tissues of the human body each multiplied by a "tissue weighting factor".
environmental exposure	Exposures to both man-made and naturally occurring substances found in the environment.
epidemology	The study of the distribution and determinants of disease and deaths in human populations.
equivalent dose	The absorbed dose multiplied by a "radiation weighting factor" which accounts for the different potential for adverse effects of the different types of radiation. Unit: sievert (Sv). Note: Since the radiation weighting factors are dimensionless, the units of the sievert are also joules/kilogram.
genotoxic	Agents that can damage DNA, thus possessing the ability to induce cancers, heritable disorders and abnormalities in a developing embryo.
Guillian-Barré Syndrome	A type of acute idiopathic polyneuritis.
ionizing radiation	Radiation that has sufficient energy to remove the orbital electrons from atoms.
infectious unit	The smallest number of virus particles that, in theory, can cause infection. For most viruses (those viral particles which are intact and contain the full viral genome) one viral particle may be considered infectious and can infect a susceptible cell. "Infectious unit" is usually only used when referring to virus particles, and not to protozoans or bacteria.
initiated cell	A living cell within the body whose genetic material has been altered in such a way that it could lead, after a subsequent series of further changes, to development of cancer.
linear no-threshold hypothesis (LNTH)	The assumption that the risk of cancer or genetic effect is linearly proportional to dose and that there is no threshold below which there is no risk of adverse effects.
LOAEL	Lowest observed adverse effect level.
maximum tolerated dose	The highest dose of a chemical substance administered in carcinogenicity bioassay, generally selected to cause minimal chronic effects.
NOAEL	No observed adverse effect level.
congenital toxoplasmosis	A parasitic infection of the newborn resulting from transplacental passage from an infected mother. The newborn is usually asymptomatic at birth but later manifests a wide range of signs and symptoms, particularly infection of the retina which can lead to blindness.

radiation weighting factor	A value recommended by the International Commission on Radiological Protection, and usually adopted by national regulatory agencies, to convert absorbed dose from various types of ionizing radiation into its equivalent in terms of biological harm from β, x, or γ radiation.
reactive arthritides	Acute joint inflamation which is triggered by an infection elsewhere in the body (arthridites = plural of arthritis)
regulated practices	Human activities that are regulated by law by the appropriate authorities at the national, provincial, or municipal level
risk	The measure of the likelihood and severity of an adverse effect. It is calculated as the product of the consequences of an event and the probability of its occurrence.
risk assessment	A process involving the identification of hazards and the estimation of associated risks.
risk coefficient	The increase in the annual incidence or mortality rate per unit dose from exposure to a hazard.
risk management	A process involving the consideration of the results of risk assessment and other factors to identify and implement strategies to control risk.
stem cell	A small group of living cells within the tissues of the body with the ability to divide and form all the other cells of that tissue.
threshold dose	A dose or exposure to a chemical agent, ionizing radiation, or microbiological agent below which no harmful biological effects are produced.
tissue weighting factor	A value recommended by the International Commission on Radiological Protection, and usually adopted by national regulatory agencies, to indicate the probability that a unit dose of radiation to the tissue will result in a cancer in the exposed person or a serious genetic disorder in his/her descendants.
toxicology	The study of the adverse effects of agents on living organisms, including individual, and groups of, humans.
urticaria	A skin disease characterized by transient eruptions resembling wheals and attended with itching, as in hives or nettle rash.

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