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Understanding Health Studies and Risk Assessments Conducted in the Port Hope Community from the 1950s to the Present

April 2009

Canadian Nuclear Safety Commission
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Understanding Health Studies and Risk Assessments Conducted in the Port Hope Community from the 1950s to the Present.

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Synthesis Report

Understanding Health Studies and Risk Assessments
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from the 1950s to the Present

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EXTENDED EXECUTIVE SUMMARY

Many health studies have been performed throughout the years because of the historical and current presence of the nuclear industry in the community of Port Hope, Ontario and because some residents have expressed concerns about possible health effects in the community. The Commission Tribunal therefore requested that the Canadian Nuclear Safety Commission (CNSC) staff draft a report that would amalgamate the results and conclusions of all Port Hope health studies commissioned over the years. Based on the environmental and epidemiological studies conducted in Port Hope and the findings of research studies conducted in other countries, the CNSC concludes that no adverse health effects have occurred or are likely to occur in Port Hope, as a result of the operations of the nuclear industry in the community.

Two uranium processing facilities, both operated by Cameco Corporation, are situated within the town of Port Hope, Ontario. The Port Hope Conversion Facility (PHCF) was established in 1932, while the Cameco Fuel Manufacturing Inc. (previously Zircatec Precision Industries Inc.) was established in 1957. Both facilities are regulated through licenses issued by the Canadian Nuclear Safety Commission (CNSC).

From 1976 to 1981, a community clean-up project was undertaken, with the specific purpose to decrease the level of environmental contamination within the Port Hope area from previous operations. At the end of the clean-up, some wastes were left in place, and have continuously been monitored and managed since then. Currently, two projects - the Port Hope Area Initiative (PHAI) and Cameco's Vision 2010 - are undergoing environmental assessment and regulatory approval processes, to address the remaining low-level radioactive wastes and non-radioactive contamination.

The potential health impact of past and present uranium refining and processing industry in Port Hope has been the target of public controversy and misunderstanding. Some citizen groups have made various claims that the uranium processing industry has caused adverse health effects to Port Hope residents, while many other citizens believe Port Hope is a healthy place to live.

In response to public concerns, the provincial and federal government agencies conducted health and environmental studies in Port Hope, to measure the environmental levels of inorganic and organic contaminants and radiation, determine exposures and risks, and conduct epidemiological studies on members of the public and workers.

For this synthesis report, CNSC staff principally relied on findings from over thirty environmental studies and thirteen epidemiological studies. CNSC staff identified and summarized the scientific information needed to understand and assess the health effects of the past and present radium and uranium refining and processing activities in Port Hope. The CNSC evaluated the information, collected to assess effects on human health, along two lines of evidence:

- Environmental studies – which analyse and measure the environmental concentrations of contaminants attributable to the nuclear industry in Port Hope, and compares them with national and international benchmarks, to assess potential risks;

- Epidemiological studies – which compare the health status of the residents and nuclear workers of Port Hope with the general population (descriptive, ecological studies), and assess the relationship between occupational and residential exposures and adverse health effects (case-control and cohort studies).

The lines of evidence, based on the numerous studies conducted in Port Hope over five decades, support each other and reveal that the levels of exposure in local area residents and workers are low, and there is no evidence of health effects as a result of past and present activities of the radium and uranium refining and processing industry in the region. These findings are consistent with the international scientific understanding of the effects on human health of radiation, uranium toxicity and toxicity of other contaminants such as arsenic, fluoride and ammonia. The findings of every study conducted in Port Hope are also consistent with other studies examining similar populations worldwide.

In addition to presenting a brief history of the nuclear industry in Port Hope, the report summarizes the scientific understanding of the health effects of radiation, uranium, arsenic, ammonia and fluoride on humans and experimental animals, and lists the sources and levels of contaminants in the local environment. This information together with estimates of exposure and risk to residents of Port Hope and the results of the epidemiological studies conducted there, allowed CNSC staff to conduct a scientifically robust review, using the criteria developed by Bradford-Hill to establish causation (*i.e. does factor A cause disorder B?*). These criteria are: strength of association, the consistency of association, specificity, temporal relationship, biological gradient (dose response), biological plausibility, coherence, experimental evidence, and reasoning by analogy. It is clear from the scientific evidence that most of the contaminants found in Port Hope can cause harm to human health at high doses, and according to Bradford-Hill's criteria, a causal relationship exists between the contaminants and disease. Based on the experimental and epidemiological literature, the most plausible health effects of the radium and uranium refining and processing industry include cancers of the lung and bone, and kidney disease. However, when considering the biological gradient and experimental evidence criteria mentioned above, kidney disease and bone cancer are not plausible in Port Hope because uranium has not been found to cause kidney disease in humans, and radium has a threshold of 10 Sv for bone cancer. All other types of cancer and diseases, based on the dose-response criterion mentioned above, are not plausible in the case of Port Hope residents, because the environmental gamma ray doses, arsenic, ammonia, fluoride and other contaminant concentrations are very low, and their health effects can only be found at much higher levels.

The radiation levels in the air, soil, water and vegetation in Port Hope are very low and are situated at - or below - the CNSC public dose limit. The industrial sources of radiation contribute only a small fraction of the total radiation levels in the area. They are similar to those normal for southern Ontario, and are equivalent to the annual average dose to all Canadians. An increased risk of cancer would not be expected, because cumulative doses are so low. The CNSC *Radiation Protection Regulations*, with a requirement to keep doses As Low As Reasonably Achievable (ALARA), have also ensured that the level of emissions at the existing Port Hope facilities continues to be reduced.

Uranium has not been found to cause kidney disease in humans. Uranium concentrations in various environmental media in Port Hope do not represent a risk to the local residents. Uranium air concentrations in the municipality are significantly lower that the proposed Ontario guidelines for the protection of human health. Similarly, the uranium concentration in drinking water in Port Hope is significantly lower than the Ontario standard, and is similar to the uranium concentrations in drinking water reported by the province's water supply systems under the Ontario Drinking Water Surveillance Program. The uranium soil concentrations in Port Hope, while higher than the provincial background levels at some sites, due to the presence of historic waste, are not expected to result in adverse health consequences, since the values are generally below the guidelines set for the protection of human health. The few areas where uranium concentrations in soils are above the guidelines do not pose a health concern, thanks to their low human exposure, due to the fact that these areas are spatially-limited, and that uranium has been found to have low biological availability. Concentrations of uranium in locally-grown vegetation species have also been found to be very low (just slightly above the detection limit), and therefore do not represent a health risk to the Port Hope residents.

Levels of arsenic, ammonia and fluoride are at - or below - the current levels set to protect human health. The other environmental contaminants known to be associated with the uranium processing industry (antimony, nickel, copper, cadmium, chromium, cobalt, lead, selenium, bismuth, thorium, zinc) are also well below guideline levels set to protect human health.

The cancer incidence rates of Port Hope residents have been analysed over the last 30 years through five descriptive ecological epidemiological studies. Overall, the cancer incidence in the local residents – for all cancers combined – was comparable with the general population of Ontario and Canada, and other similar communities. The most common types of cancer in Port Hope were lung, colon and rectum, breast and prostate. This is consistent with the rest of the province and the entire country. Port Hope residents, especially women, had a significant excess of lung cancer. This pattern was seen for the whole Northumberland County, so it was not specific to the municipality. It is also consistent with the known main risk factor of lung cancer (tobacco smoking) within the community. There was no evidence of excess adult leukemia in Port Hope. The rate of all childhood cancers was comparable with the general Ontario population, as was childhood leukemia – which is a type of cancer particularly connected with high exposures of gamma radiation.

Five descriptive ecological studies also examined the residents' mortality over the last 50 years. The leading causes of death in Port Hope were circulatory disease, cancer and respiratory disease. This was consistent with the rest of Ontario and Canada. Port Hope residents had a statistically significant excess of circulatory disease, especially heart disease. This pattern was also seen for the whole Northumberland County – therefore, again, not specific to the municipality – and is consistent with the rest of the county and the known main risk factors of disease within the community. There was no evidence of excess kidney diseases – which was to be expected, since exposure to uranium has been found to be low, and because uranium has not been found to cause kidney disease in humans.

Mortality from all types of cancer was comparable to the general Ontario population. The leading causes of cancer death were cancers of the lung, colon and rectum, breast and prostate, which was consistent with the general trend in the provincial and national population, and with the rates of cancer incidence in Port Hope. All childhood cancer mortality was comparable with the general population of Ontario, as was mortality from congenital anomalies (birth defects).

One case-control study assessed the relationship between childhood leukemia and the father's radiation exposure. The study included workers from Cameco's Port Hope Conversion Facility. There was no evidence of an association between children's leukemia and their father's occupational radiation exposure, regardless of exposure period or type. A second case-control study assessed the relationship between lung cancer and residential radon in Port Hope. No conclusive evidence was found to link residential radon to lung cancer rates, even among people living in homes with high levels of radon exposure.

Finally, the most convincing evidence is derived from a very large cohort study, recently updated, which focused on approximately 3,000 radium and uranium workers from Port Hope. Detailed information on the workers' radiation exposure was collected from 1932 to the present day, and workers were followed in terms of their mortality (for 50 years) and cancer incidence (30 years). Overall, Port Hope workers were as healthy as the general male population of Canada. Their overall mortality from all types of cancers, particularly lung cancer and leukemia, was comparable to the general male population of the country. There was no evidence of excess mortality from kidney disease. Cancer incidence rates were comparable with the general male population of Canada.

The study also assessed the relationship between occupational exposure (radon exposure, gamma radiation dose) and mortality and cancer incidence. No relationship was found between Port Hope workers' radiation exposures and any cause of death, or incidence of cancer. This was largely because occupational exposures of these workers were so low.

The findings concerning Port Hope residents and workers (who also live in the community) are consistent with the findings of epidemiological studies conducted elsewhere in the world, analysing the health status of workers and the public exposed to the radium and uranium refining and processing industry. Descriptive epidemiological

studies have found no evidence of excess cancers among the populations residing near uranium milling, mining or processing facilities. The results of a large case-control study of workers at four uranium processing plants in the United States found no association with lung cancer, as a result of insoluble uranium compounds or radiation exposures. Finally, 14 international epidemiological studies conducted in other countries of more than 120,000 workers at various uranium processing, enrichment and metal fabrication facilities did not find the rate of any cancer to be significantly increased. Likewise, the available evidence suggests that there is little, if any, increase in kidney disease among humans exposed to uranium, even following high exposures.

The environmental and epidemiological studies conducted in Port Hope support each other, and overwhelmingly lead to the conclusion that the low levels of radiological and non-radiological environmental exposures within the town, resulting from the radium and uranium industry, have not caused any adverse effects on human health.

On this basis, the CNSC concludes that no adverse health effects have occurred or are likely to occur in Port Hope, as a result of the operations of the nuclear industry in the community.

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1.0 INTRODUCTION

Two uranium processing facilities, operated by Cameco Corporation, are situated within the town of Port Hope, Ontario. The Port Hope Conversion Facility (PHCF) was established in 1932, while Cameco Fuel Manufacturing Inc. (previously Zircatec Precision Industries Inc.), was established in 1957. Both facilities are regulated through licences issued by the Canadian Nuclear Safety Commission (CNSC).

Initially, the PHCF was established to extract radium from ore. The process was later adapted to extract and convert uranium into chemical forms that are used to produce fuel for nuclear reactors. The waste management practices during the early operations of the PHCF (mainly between 1932 and 1966) resulted in widespread radioactive and non-radioactive contamination throughout the community. A partial clean-up and stabilization of these materials was carried out from 1976 to 1981. Contaminants were removed to the Chalk River Waste Management Facility, to piles located in public areas in the community, or were left in place. Two projects to complete the clean-up and to establish long-term management facilities for these wastes are currently undergoing environmental assessment and regulatory approvals processes.

There has been much controversy and widespread misunderstanding among the general public regarding the health impact of the past and present uranium refining and processing industry in Port Hope. Over the years, over 30 environmental studies have been carried out to determine the extent of environmental contamination from past and present discharges from the two operating uranium refining and processing facilities in the community. Measurements of contamination in the community are used to estimate public exposures and assess the health risks to Port Hope residents. This process ensures the levels of contaminants are below the levels set to protect public health. Numerous epidemiological studies have monitored the health status of the community and assessed whether environmental and occupational exposures have resulted in elevated health risks to workers and the public.

The purpose of this document is to identify and summarize the wealth of scientific information used by the CNSC, Health Canada, and other government organizations, to understand and objectively assess the potential health impact of the past and current uranium refining and processing industry in Port Hope.

The document is divided into nine main chapters:

Chapter 1 provides the background, purpose and scope of this report.

Chapter 2 describes the sources of natural and man-made radiation.

Chapter 3 describes uranium refining and conversion, the history of the nuclear industry in Port Hope, the radioactive and non-radioactive contamination, and clean-up projects that are underway.

Chapter 4 describes uranium's chemical and radiological properties, low-level radioactive waste, and non-radioactive contaminants. It discusses the possible health effects of these contaminants; the limits, benchmarks and guidelines in place to protect health; and the actual levels of exposure in Port Hope.

Chapter 5 describes environmental studies that have been carried out in Port Hope. These studies provided the environmental concentrations of radioactive and non-radioactive contaminants attributable to the nuclear industry in Port Hope, and compared them with benchmarks protective of human health.

Chapter 6 describes epidemiological study methods, epidemiological studies conducted in Port Hope, and the international scientific understanding of the health effects of uranium refining and processing.

Chapter 7 discusses the scientific evidence presented in Chapters 2 to 6.

Chapter 8 provides the overall conclusions of the CNSC.

Chapter 9 provides a complete list of references.

Appendices I to III provide a glossary of terms and abbreviations, a background on radiation theory and the health effects of radiation, and the uranium decay chain.

2.0 RADIATION FROM NATURAL AND MAN-MADE SOURCES

Radiation is energy in the form of particles or waves. It can be either ionizing or non-ionizing. Ionizing radiation has sufficient energy to create ions by adding or removing electrical charges from neutral atoms or molecules in the material through which the radiation is travelling. Examples of ionizing radiation are alpha and beta particles, gamma-rays and X-rays. Non-ionizing radiation does not have sufficient energy to create ions - it includes sunlight, UV sources, artificial lighting and laser light. In this report, "radiation" refers to ionizing radiation. Appendix II discusses radiation theory in greater detail.

2.1 Natural (background) Ionizing Radiation Sources

Radiation has always existed and all life on earth is exposed to some level of ionizing radiation. Most of the radiation exposure to the public comes from naturally occurring ionizing radiation. The following are sources of natural ionizing radiation:

- *Cosmic rays* are particles with a broad energy spectrum that come from the sun and outer space. Their magnitude near the earth's surface varies with latitude and even more so with altitude. When cosmic rays interact with molecules in the upper atmosphere they can produce radioactive atoms such as carbon-14 and tritium.

- *Terrestrial radiation* is emitted by numerous radioactive elements in the earth's crust and the rocks of the earth's surface. These elements were present when the earth was first formed. Naturally occurring radionuclides (which primarily originate from the decay of uranium-238 . . . uranium series; thorium-232 . . . thorium series; and uranium-235 . . . actinium series) are present in low concentrations in all rock and soils (see Appendix III). The most common radionuclides in Canadian groundwater are uranium-238, uranium-234, radium-226, radon- 222 and lead-210 from the uranium series; radium-228 from the thorium series; and traces of uranium-235 from the actinium series. Uranium-238 and uranium-234, as well as all the other elements, are found in soils. (1).

- *Ambient (atmospheric) air* contains radon; a natural radioactive gas produced by the decay of uranium in the earth's crust. Radon and its short-lived decay products are the main source of natural ionizing radiation exposure to humans. Radon in this review refers to radon-222, a radionuclide in the uranium series. The thorium series also contains a radon isotope (radon-220) referred to as thoron. It has a short half-life (55 seconds) and contributes little to the normal radon exposure.

- *Food and drink* (internal sources) also contain radioactive elements such as potassium 40. Once ingested, these elements will be deposited in tissues, organs and bones. All humans have small amounts of radioactive elements naturally present in their bodies from the foods they eat and substances they drink. For

example, potassium is essential for the normal function of cells. Potassium-40 is a small fraction of all potassium and because it is radioactive, it delivers the largest annual internal radiation dose with the exception of radon.

Natural background radiation accounts for approximately 60% of the lifetime radiation exposure for an average Canadian. The average dose to Canadians from natural background radiation sources is approximately 2.4 mSv/year (2) (from 1.2 to 3.2 mSv, depending upon geographic location). A third of this is from terrestrial and cosmic radiation, and two-thirds is due to the inhalation and ingestion of radionuclides in air, water and food.

2.2 Man-made Ionizing Radiation Sources

People also receive radiation doses from man-made radiation sources. They include the following:

- *Medical irradiation:* After natural sources, the largest source of ionizing radiation to Canadians is from medical applications involving radiotherapy and nuclear medicine. In fact, medical diagnostic radiation is rapidly becoming equal to and perhaps exceeds natural radiation sources. This is because of an increase in the use of computed X-ray tomography (CT) scans and other nuclear medicine imaging techniques.

- *Technical and industrial uses*, such as uranium processing, nuclear power reactors, manufacturing processes, and research.

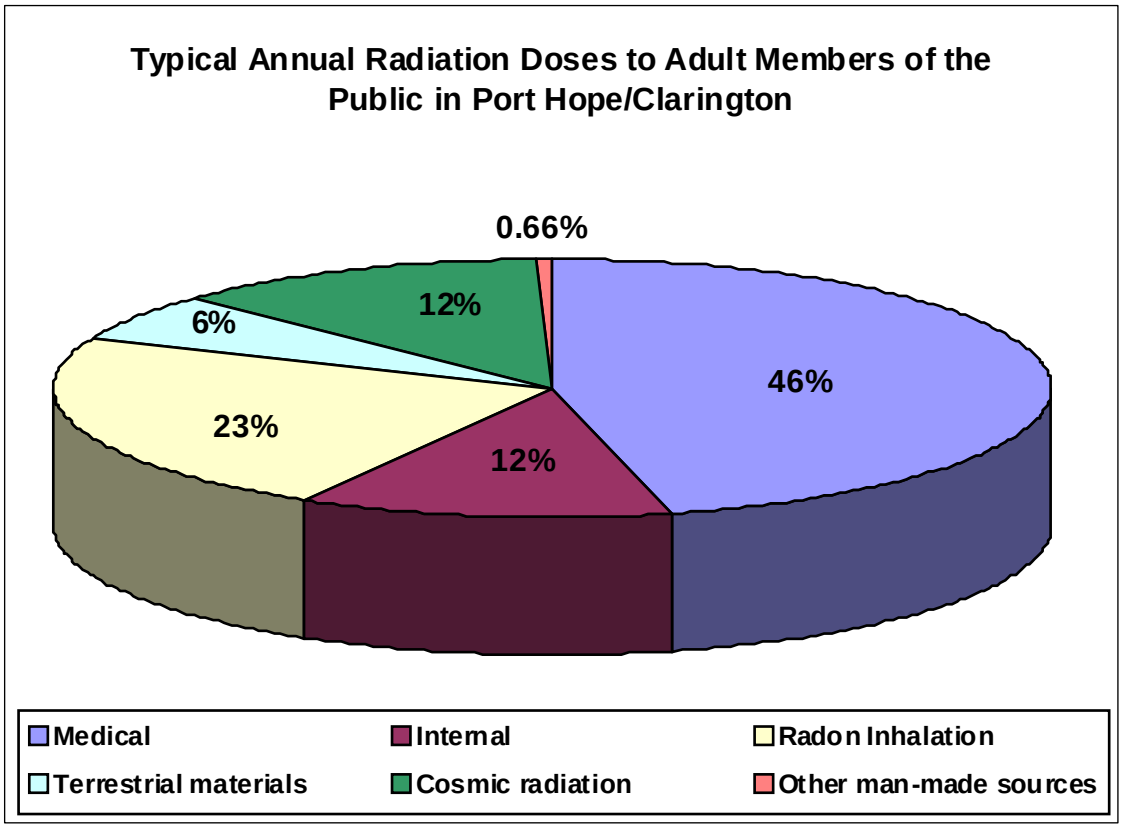
- *Fallout from atmospheric weapons testing:* Atmospheric testing largely ceased after the Nuclear Test Ban Treaty of 1963. Radioactive fallout such as tritium, carbon-14, strontium-90 and cesium-137 still persist in the environment; however, the total dose received by individuals in the North Temperate Zone (40-50 EN latitude), accumulated to the year 2000, for all tests conducted between 1945 to 1980 is estimated to be about 2.1 mSv (3).

Man-made sources represent approximately 40% of all radiation exposure for an average Canadian, the vast majority of which are from medical uses. The nuclear industry represents the smallest proportion of the total radiation to which humans are exposed (<1%).

2.3 Radiation Sources in Port Hope

The public in Port Hope is exposed to natural radiation (cosmic, terrestrial, radon and internal) and man-made radiation sources (medical radiation, radiation from low-level radioactive waste from the early operations of the radium and uranium refining and processing industries in Port Hope) and from controlled releases from currently operating

uranium conversion and fuel fabrication facilities in Port Hope (see Chapter 3). The following diagram shows the 2004 distribution of sources of radiation exposure to the public in Port Hope/Clarington [adapted from (4)].



3.0 THE HISTORY OF THE NUCLEAR INDUSTRY IN PORT HOPE

Over the years, discharges from the Port Hope Conversion Facility have resulted in the deposition of radiological and non-radiological contaminants throughout the community of Port Hope. This chapter describes uranium processing (mining, concentration, refining, conversion and fuel fabrication), the history of uranium processing in Port Hope, the resulting contamination and the contamination clean-up activities.

3.1 Uranium Refining and Conversion

Uranium is a common naturally occurring radioactive element and is found in concentrated deposits in many areas of Canada (including northern Saskatchewan, the Northwest Territories, Nunavut, Ontario, Quebec, and Labrador). It is also dispersed throughout the environment (in air, soil, rock, surface water, groundwater, plants, animals, and people). Natural uranium is composed of 0.7% uranium-235, 99.3% uranium-238 and a trace amount of uranium-234 by weight. Natural uranium can be enriched by adding uranium-235 to increase its proportion by weight. Natural uranium from which the uranium-235 was removed becomes depleted uranium. In Canada, uranium is primarily used as a fuel for nuclear power reactors. Depleted uranium was used as shielding for other radioactive sources and as a counterweight in aircraft. Elsewhere, enriched uranium has been used as a component of nuclear weapons and depleted uranium has been used for armour piercing projectiles.

Before uranium can be used as fuel, it must be extracted from the ground and processed into a usable form. Uranium ore is extracted from either an open pit or from underground mines. The ore is then transported to a mill where it is ground and treated in a chemical process that separates the uranium from other elements in the rock. Typically, mills are located at or close to mine sites. This process produces wastes (tailings) that contain low concentrations and normal concentrations of all the other radioactive decay products of natural uranium, non-radioactive contaminants in the minerals such as arsenic, cobalt, copper and nickel and processing chemicals. The volume of wastes is approximately the same as the volume of ore processed.

Concentrated uranium, the final product of milling, is a dry, fine powder referred to as “yellowcake”. Yellowcake is packed in steel drums and shipped to a refinery, such as the one in Blind River or the Port Hope Conversion Facility (PHCF) up until 1984. At the Blind River Refinery, yellowcake is further refined to remove any remaining impurities. During refining, uranium is converted to uranium trioxide (UO_3). Wastes from refining contain low levels of radioactive decay products, non-radioactive contaminants and processing chemicals. Uranium trioxide is packaged in totes and transported to the PHCF.

At the PHCF, uranium trioxide is converted into one of two substances. Uranium hexafluoride (UF_6), for export to other countries for further processing into light water and pressure water reactor fuel, and uranium dioxide powder (UO_2) which is used to fuel Canadian deuterium uranium (CANDU) reactors. Wastes from conversion contain decay products and processing chemicals. These wastes are recycled or disposed of at regulated waste management facilities located outside of the Port Hope area.

The ($UO_{2)}$ produced by the PHCF is fabricated into fuel pellets and bundles at Cameco Fuel Manufacturing Inc. (previously Zircatec Precision Industries Inc.).

3.2 Operations of Port Hope Nuclear Facilities

In 1932, Eldorado Gold Mines Limited (Eldorado) opened a radium extraction refinery on an existing industrial site situated adjacent to the Port Hope harbour. During the early years of refinery operations, high-grade pitchblende ores from the Port Radium mine in the Northwest Territories were shipped to Port Hope and processed for the recovery of radium.

In 1942, a Crown corporation of the federal government purchased Eldorado. The focus of Eldorado's processing shifted from radium to uranium due to the increasing demand for uranium (first military, then economic as power reactors were developed). Pitchblende was then processed for the recovery of uranium. Radium processing continued until 1954.

From 1955 to 1966, a new more efficient solvent extraction (SX) process was implemented for the recovery of pure uranium trioxide ($U0_3$) for the nuclear power industry. Between 1955 and 1966 some high-grade pitchblende ores were used as feed material in the SX plant. After 1966, only purified feed material, the concentrated uranium powder referred to as yellowcake was used.

Around 1968, operations were started to convert $U0_3$ from the SX plant into reactor-grade uranium dioxide ($U0_2$). Conversion operations to produce uranium hexafluoride (UF_6) from $U0_3$ began in 1970. In 1984, the SX operations were moved to the new Blind River Refinery. Since then the PHCF only receives $UO_{3.}$

The PHCF has also operated a small scale processing plant since 1959, to produce specialty uranium metal products used as shielding for other radioactive sources, and counterweights in aircraft.

In 1988, Eldorado Nuclear Limited was privatized and renamed Cameco (Canadian Mining and Energy Corporation). The name was later changed to "Cameco Corporation". The only processing carried out at the PHCF since 1984 and continuing today is the conversion of UO_3 to UO_2 or $UF_{6.}$

Finally, Zircatec Precision Industries Inc. was purchased by Cameco Corporation in 2008 and renamed Cameco Fuel Manufacturing Inc. Since the late 1950's the facility has processed uranium dioxide (UO_2) powder into ceramic grade uranium pellets. The pellets are loaded into zircalloy tubes which are then assembled into fuel bundles. The fuel bundles are shipped to Canadian nuclear power generating plants.

3.3 Waste Management in Port Hope

During the early years of radium and uranium refinery operations in Port Hope, high-grade pitchblende ores were processed. These ores contained uranium and its radioactive decay products, notably radium-226, (see Appendix III) along with other naturally occurring minerals containing chemical elements such as arsenic, cobalt, copper and nickel. These elements ended up in relatively high concentrations in wastes from the refining and processing operations. Trace amounts of antimony and lead were also present in the ore and ended up in lower concentrations in the waste products from this time period. Originally, wastes from the PHCF were located in the Port Hope landfill, at sites adjacent to the facility and in various locations throughout the community. The design of these storage sites and handling procedures did not fully limit the spread of the radiological and non-radiological contaminants to the adjacent environment. Materials from these sites were also used as construction materials in homes and buildings and as a source of fill material for construction and landscaping throughout the community. This practice was stopped when it was realized that it contributed to the spread of contamination within the community.

From 1948 to 1955, Eldorado placed wastes at the Welcome Waste Management Facility (WMF) in the Township of Hope. In 1955, the Welcome WMF closed and a new facility, the Port Granby WMF, opened near Port Granby in the Township of Clarington.

The majority of impurities (radioactive decay products and other metal elements) are removed during the production of yellowcake at uranium mine sites. Wastes generated in Port Hope after 1966 contained fewer and lower concentrations of radioactive contaminants and metal impurities. Wastes were primarily characterized by the presence of uranium and radium-226, with minor or trace levels of other non-radiological contaminants due to inefficiencies with the different refining processes.

Once the refining of yellowcake moved to Blind River in 1984, only UO_3 was received at the PHCF. The amount of low-level radioactive waste generated in Port Hope was again reduced since the radium-226, the other non-radiological contaminants, and most of the decay products had been removed.

Radioactive wastes from Cameco Fuel Manufacturing Inc. are returned to the PHCF for recycling. Contaminated wastes generated by current operations of the PHCF are recycled or disposed of at regulated facilities located outside of the Port Hope area.

3.4 Clean- up Activities in Port Hope

A clean-up project was initiated in Port Hope to decrease the levels of environmental contaminants within the community. Between 1976 and 1981, the Federal-Provincial Task Force on Radioactivity removed historic radioactive wastes from residential and commercial properties. Remedial action was taken in homes with radon levels above 0.02 WL (approximately 150 to 185 Bq/m^3). More than 100,000 tons of contaminated soils were transported to the Atomic Energy of Canada Limited (AECL) Chalk River

Waste Management Facility. Large deposits of contaminated soils located in vacant areas, along with sediments in the Port Hope harbour, were left in place for removal at a later date.

In 1982, the Low-Level Radioactive Waste Management Office (LLRWMO) was created and operated by AECL to monitor and manage the 600,000 tons of low-level radioactive wastes and contaminated soils that remained in the Port Hope area. In recent years, two projects, the Port Hope Area Initiative (PHAI) and Cameco's Vision 2010 have been initiated to consolidate these remaining low-level radioactive wastes, wastes stored in the Port Granby and Welcome waste management facilities, and historic wastes stored at the PHCF by placing them into two engineered long-term above-ground waste management facilities that will be located in Port Granby and Port Hope.

The projects are currently undergoing environmental assessment and licensing processes. Completion of these projects will further reduce the levels of environmental contamination to local natural background concentrations/levels.

4.0 POTENTIAL HEALTH EFFECTS OF ENVIRONMENTAL CONTAMINANTS

To assess the potential health effects of environmental contaminants within Port Hope, one must understand the chemical and radiological properties of uranium, the radiological properties of low-level radioactive waste, and the properties of other solid and liquid wastes from the uranium extraction and refining processes.

4.1 Health Effects of Uranium

Uranium can exist in a variety of chemical compounds in the natural environment and as a result of uranium refining and processing. Uranium dust particles can be deposited onto surface water, plant surfaces, and soil. This may occur by wet processes (rain, sleet, or snow) or dry processes (gravitation or wind turbulence). Uranium in surface water mostly comes from uranium dissolved from rocks and soil and, once dissolved, can be dispersed over large distances to lakes, rivers, and oceans. Plants usually do not readily take up uranium from the soil. Human exposure to uranium from the environment can occur through inhalation of air and dust and ingestion of water, soil, and vegetation. There is no evidence that uranium increases in concentration as it travels through the food chain, due to very low uptake of uranium by most organisms.

Uranium has both chemical toxicity and radiological properties (5). Naturally occurring uranium has low radioactivity and poses very little radiological danger because of the release of very small amounts of alpha radiation (6, 7, 8, 9, 10, 11). Uranium is not considered a human carcinogen (9, 10, 11) and genetic effects of radiation from uranium have not been observed at any level of exposure (6). No human cancer of any type has ever been seen as a result of exposure to natural or depleted uranium (6, 11).

The chemical toxicity of uranium is considered to have a greater potential to cause observable effects than its radioactive properties. Consequently, the chemical toxicity of uranium may be a greater health concern. None-the-less, uranium has never caused any health effects in humans even following high exposures. Uranium has low radioactivity so it is only considered for its heavy metal properties (6, 11, 12).

Uranium toxicity will have a more pronounced effect on the kidneys (6, 12) than on other organs or tissues (e.g., neurological, hematopoietic, hepatic, gastrointestinal, dermal and musculoskeletal effects). Therefore, the report focuses on a discussion of the effects of uranium on the kidneys.

4.1.1 Uranium's Chemical Toxicity

Uranium is well established as a metal toxic to kidneys, from laboratory animal studies. It exerts chemical action in the renal proximal tubules of the kidneys (12,13). Uranium may result in kidney dysfunction, cellular necrosis and atrophy of the tubular wall (6, 12). Kidney dysfunction is generally indicated by the presence of proteins, enzymes, or glucose in the urine (16, 12, 13).

Kidney toxicity at low doses has not been observed (5, 6, 7) and there is some evidence that impairment of kidney function may be temporary and kidney function may return to normal once the source of excessive uranium has been removed (7, 12). The evidence indicates that such transient cellular effects cannot be considered a health effect.

The toxicity of uranium depends on the route of exposure (inhaled or ingested) and the solubility of its chemical form (compounds). The most soluble and therefore readily absorbed uranium compounds are the most potent kidney toxicants (6, 12).

Experimental studies in rats found that acute respiratory effects may be limited to inflammation of the lungs, leading eventually to emphysema or pulmonary fibrosis (14). The acute respiratory effects of airborne uranium dust in uranium workers are consistent with the effects of inhaled dust (6). However, these effects are not likely to occur in environmental exposure conditions but only in rare accidental situations in occupational settings (5, 6). Large chronic concentrations of dissolved uranium were not chemically toxic to kidney tissue in a cohort of uranium mill workers. There was also no dose response, that is, no increase in risk with length of exposure (15).

Chronic inhalation of high levels of uranium compounds has consistently been found to cause kidney toxicity in experimental animals. In general, inhaled soluble uranium compounds are reported to be more toxic than inhaled insoluble compounds (6, 12). Chemically soluble forms of uranium, such as uranium trioxide (UO_3) and uranium hexafluoride (UF_6) are used in today's PHCF. These compounds are readily dissolved in lung fluids and transported rapidly through the body, in the bloodstream, and excreted through the kidneys (6, 12). The evidence indicates that there is little if any increase in kidney disease in workers involved in the processing of uranium ores or in uranium fabrication plants (6, 12).

The kidney toxicity associated with the oral ingestion of uranium compounds has also been observed in animal studies. In general the toxicity of uranium compounds is associated with the more soluble uranium compounds, causing systemic adverse effects on the kidney (6, 12). The effects on kidneys are also more consistently observed in association with higher doses of uranium exposure. Ingestion of uranium is unlikely to be associated with respiratory effects (6, 12).

In general, inhaled particles of insoluble uranium are more likely to be retained by the lungs compared to soluble uranium particles that are either absorbed or cleared by mechanical processes in the gastrointestinal tract. Insoluble uranium particles, such as uranium dioxide (UO_2) produced in today's PHCF can remain in the lung for a long period of time (6, 12), so may produce a larger radiation dose compared to readily soluble uranium compounds.

4.1.2 Uranium Guidelines and Concentrations Measured in Port Hope

Uranium limits and guidelines developed by various government agencies to protect human health are based on the chemical toxicity properties of uranium rather than its

radiological properties. This is because the concentrations of uranium that would pose a chemical risk are lower and more restrictive than the concentrations that would pose a radiological risk (5). Exposure to uranium concentrations at or below the guidelines would result in a radiation dose below the CNSC dose limit in the *Radiation Protection Regulations* of 1 mSv per year for members of the public (17).

The Table below shows the concentrations of uranium measured in various media in Port Hope and the uranium guidelines against which they are assessed. Note that soil uranium concentrations exceed the guidelines at some locations. It is explained in Chapter 5 that this is not a health concern because of the low biological availability of the uranium and the limited spatial extent of the elevated concentrations.

Environment	Uranium Benchmarks and Guidelines to Protect Human Health	Current Uranium Levels in Port Hope
Air	0.02 - 0.5 µg/m^3 – proposed range for a 24-hour Ontario wide Ambient Air Quality Criterion for uranium based on kidney effects from continuous inhalation exposure (5)	• 0.00028 µg/m^3 at Sports Complex (18) • 0.005 µg/m^3 at Waterworks (18)
Drinking Water	• 0.02 mg/L – Canadian Guidelines (19) • 0.02 mg/L – Ontario Standard (20)	• 0.00055 mg/L for 98th percentile (21)
Soil	• 23 mg/kg for agricultural land use (22) • 23 mg/kg for residential/ parkland use (22) • 33 mg/kg for commercial land use (22) • 300 mg/kg for industrial land use (22)	• from 0.25 to 51.3 mg/kg and median value of 3.1 mg/kg for the top 15-cm soil horizon (23)

4.2 Potential Health Effects of Low-Level Radioactive Wastes

Uranium is a natural radioactive element and predominantly emits alpha radiation (see Appendix III). Naturally occurring uranium has low radioactivity (6, 7, 8, 9, 10, 11) and poses very little radiological danger because of its very long half-life (4.5 billion years) and therefore the release of very small amounts of radiation (6, 11). Uranium is therefore not considered a human carcinogen (9, 10).

The extraction of uranium from ore produces both solid and liquid wastes. These wastes contain many radionuclides, as well as their decay products. Thus it is the radionuclides and their decay products present in the historic low-level radioactive waste (LLRW) products of the uranium refining and processing that are the radiological hazards of concern in Port Hope.

Because of its predominance in nature, the greatest man-made source of radiation from Port Hope, the LLRW, comes from the uranium (U-238) decay chain of natural uranium (see Appendix III) as it undergoes a series of transformations (or decays) to become a stable atom, lead-206. During the decay process eight alpha particles, six beta particles and gamma radiation are emitted. The most damaging radiation emitted from uranium and its decay products is alpha radiation. Alpha radiation will not penetrate skin, so it is only harmful when it is emitted within the body. The level of radiological harm is dependent on the rate (half-life) at which the alpha particles are emitted. The shorter the half-life of decay products, the more radioactive they are for the same number of atoms. U-238 decay products can be divided into two main groups: radon and its short-lived radon decay products, and long-lived radioactive dust.

Radon is the most important source of naturally occurring radiation exposure for humans and represents about half of the total exposure from natural background radiation (24). Ninety-five percent of radon exposure is from indoor air with about 1% exposure coming from drinking water (25). Radon and its decay products can accumulate in the air inside buildings and underground work areas. In Port Hope, historic low-level radioactive wastes (LLRW) located close to residential and commercial buildings were an additional source of radon and its decay products.

Radon gas (Rn-222) has a half-life of 3.8 days. It decays very quickly and emits alpha radiation very rapidly. It is an inert gas that is very mobile and readily passes from soil and water into the atmosphere, where it may be inhaled. Radon rapidly undergoes its decay in a series of five transitions to become stable lead-210. These transitions produce short-lived radon decay products (polonium-218, lead-214, bismuth-214, and polonium-214). During the Rn-222 decay process, three alpha particles are emitted. If inhaled, radon and its decay products may be deposited in the airway structures of the lungs and emit alpha radiation. Inhaled particles do not, however, deposit in the bronchial tree and pulmonary region with 100% efficiency. The alpha radiation can damage the sensitive tissue within the lung. Long-term exposures to high concentrations of inhaled radon gas and its radon decay products (RDP) can result in lung cancer (16, 26). There is no conclusive evidence of it causing any other type of cancer or any other cause of death (16, 27, 28, 29). No experimental or epidemiological studies have linked ingested radon with health effects in humans, and animal studies have concluded that the risk from ingestion is insignificant compared to the risk from inhalation (1).

Before processing, long-lived radioactive dust (LLRD) is made up of uranium and uranium decay products that have very long half-lives, meaning they emit alpha radiation at a very slow rate. The half-life of uranium-238 is 4.5 billion years. LLRD (uranium-238, uranium-234, thorium-230, radium-226 and polonium-210) is much less radioactive than radon gas and radon decay products. Dust from historic low-level radioactive waste in Port Hope would contain LLRD in conjunction with non-radioactive dust. As the uranium sent to Port Hope became more and more concentrated and had fewer impurities (milling and refining processes conducted elsewhere - see Chapter 3), the composition of the LLRD from more recent operations of the PHCF is composed primarily of uranium dust.

LLRD and uranium dust enter the human body primarily though inhalation, but also by ingestion (e.g., on leaves and surfaces of vegetables). The radiation hazard posed by alpha radiation emitted by LLRD and uranium dust depends on how long it is retained in the human body. This is determined by the size of the dust particles and the solubility of the chemical form of the radionuclides.

A small fraction of fine dusts are deposited and retained deep in the lung. Uranium dust can be either soluble or insoluble depending on its chemical form. Insoluble uranium particles (such as UO_2) will produce a larger radiation dose compared to rapidly soluble uranium. LLRD is insoluble. Insoluble dust clears slowly from the human body (i.e., lungs) over a period of years so even radiation emitted at a slow-rate can have a radiological impact that leads to respiratory effects. Soluble dust (such as UO_3 and UF_6) is dissolved in lung fluids and quickly cleared from the body. The radiological impact is low because of its short duration in the body and the slow rate of emission of radiation (see section 4.1.1).

Lead-214 and bismuth-214 are also part of the U-238 decay chain (Appendix III). Both emit proportionally large amounts of gamma radiation. Although it is known that high doses of gamma radiation can increase the risk of several cancers, including breast cancer, leukaemia, lung and other cancers (16, 24, 30), the gamma radiation dose from even extraordinary levels of decay products in the lung is very low and cannot be considered an additional carcinogen. Uranium miners never showed an excess of leukaemia, for example (16, 24, 26, 29, 31, 32, 33).

The PHCF stopped processing uranium ore for radium in the 1950s, thus radium (Ra-226, the precursor to radon) is no longer transported to Port Hope. Ingestion of radium would result in its deposition primarily in the bone; studies of people receiving high radium exposures strongly suggest an increased risk of bone cancer (16). Radium is the only exposure shown to result in bone cancer, with a threshold of about 10 Sv (16).

As indicated in Chapter 2, the radiation from these sources related to human industrial activities represents a minor proportion (less than 1%) of the total radiation dose received by Port Hope residents. Radiation dose limits and current doses in Port Hope are discussed in section 4.2.1.

4.2.1 Radiation Dose Limits and Current Doses in Port Hope

Ongoing monitoring of environmental and workplace radiation exposures ensure compliance with regulatory radiation dose limits used to protect members of the public and workers from radiation.

Radiation doses to workers are measured directly for each worker using instruments called dosimeters. The dose limit for Canadian nuclear energy workers is 50 mSv/year and 100 mSv over a five-year dosimetry period (34).

Various models are used to estimate radiation doses to the public. Environmental transfer models use measurements of radionuclides discharged from nuclear facilities and radionuclide concentrations in different environmental compartments. Biokinetic models indicate the transfer of the radionuclides between tissues and organs as they pass through the body. Dosimetric models are used to estimate dose.

For 2007, the radiation doses to the critical group or most exposed members of the public from the Cameco PHCF and Cameco Fuel Manufacturing Inc. were estimated to be 0.064 mSv/year (35) and 0.004 mSv/year (36), respectively. The major contributors to the dose are gamma exposure (0.061 mSv/year and 0.002 mSv/year, respectively) and uranium in the air (0.003 mSv/year and 0.002 mSv/year, respectively). These doses are very small and are much lower than the CNSC public dose limit of 1 mSv/year (17). This dose estimate is based on the lifestyle of a hypothetical resident (the critical group). Lifestyle characteristics have been chosen to overestimate dose (for example, individual breathing outdoor air for 24 hours a day).

The total estimated annual radiation dose to Port Hope area residents ranges from 1.4 mSv/year for adults to 2.6 mSv/year for infants (the most sensitive group). This dose is mainly from natural background radiation (see section 2), and includes a very small contribution from low-level radioactive waste, contaminated soils, and activities at current nuclear facilities. It does not include dose from medical and other man-made sources (37).

In 2007, the International Commission on Radiological Protection (ICRP) (38) determined the lifetime risk (probability) of fatal cancer, non-fatal cancer weighted for severity, and hereditary risks following a single low-dose, low-dose-rate exposure to be 5.7% per Sv. Because there is great uncertainty about the shape of the dose-response relationship at low doses, the current estimates for lifetime risk are presented for exposure at 1 Sv (16). The CNSC has set a dose limit of 1 mSv/year (17) for members of the public (for artificial sources over and above background radiation). Thus, an exposure to 1 mSv of radiation would give a lifetime increase in risk of approximately 1 in 10,000. The radiation doses in Port Hope from human industrial activities are far below the public dose limit and consequently the risk of cancer in Port Hope residents is expected to be indistinguishable from the level of spontaneous occurrence of cancer in the general Canadian population.

4.3 Potential Health Effects of Non-radioactive Contaminants

In 2004, an ecological risk assessment conducted by Cameco for the PHCF identified contaminants of potential concern based on a variety of criteria (39). This included compounds used in the conversion process, as well as those whose maximum predicted concentration exceeded one or several annual average criteria for air (40), groundwater (41), soil (42), water (43) and harbour sediment (44). This assessment indicated that the compounds of greatest interest are ammonia, arsenic and fluorides.

The main source of ammonia in Port Hope is from the PHCF stack emissions. Ammonia is toxic to humans if inhaled in high concentrations (for example, acute exposure to 1700 mg/m^3 can result in lung edema). It may be irritating to the eyes, nose and throat at lower concentrations (for example, 280 mg/m^3) (45).

The main source of arsenic in Port Hope is from the historic contamination. Naturally occurring minerals, containing chemical elements, were in the uranium ore (see Chapter 3). This included high concentrations of arsenic. Inorganic arsenic is a known human carcinogen: arsenic exposure may induce skin cancer, tumours of the urinary bladder and the lung, and potentially the liver, kidney and prostate (46). Inorganic arsenic is readily absorbed from the gastrointestinal tract and the skin (46). Chronic exposure to arsenic above 20 µg/kg body weight/day can lead to liver injury, peripheral neuropathy and vascular effects (47).

The main source of fluoride in Port Hope is from the PHCF stack emissions. In humans, chronic toxicity, the result of ingesting small amounts of fluoride over a long period of time, results in dental fluorosis (1-2 mg/L in drinking water) and skeletal fluorosis at higher doses (8-10 mg/L in drinking water). It is a condition caused by excessive intake of fluoride ions over an extended period of time during tooth development (before teeth erupt into the mouth), and can cause yellowing of teeth, hypothyroidism, or brittling of bones and teeth (48). Hydrogen fluoride and fluorine gases are irritating to the skin, eyes, and respiratory tract.

4.3.1 Current Levels of Non-radioactive Contaminants in Port Hope

In the last five years, no CNSC action levels for stack emissions of ammonia and fluorides from Cameco's PHCF were exceeded (see Table below) (35). Action Levels are derived from federal and provincial guidelines conservatively established to protect human health. Therefore, no health effects are expected from human exposure to ammonia and fluorides in Port Hope.

Port Hope Conversion Facility Air Emissions 2004-2008						
Parameter	**CNSC Action Level**	**2004**	**2005**	**2006**	**2007**	**2008**
		Annual Average	**Annual Average**	**Annual Average**	**Annual Average**	**Annual Average**
Ammonia (kg N/h)	29	2.2	2.4	2.2	3.1	3.2
Fluorides (g HF/h)	330	43.0	59.2	67.6	28.3	13.5

The current operations of Cameco's PHCF are not a source of arsenic in the environment (49). Elevated levels of arsenic in soils at various sites in Port Hope (up to 94 mg/kg in the top 15 cm soil horizon) are attributable to the historic wastes and historic releases from uranium and other industries in Port Hope (23, 50). Despite intensive clean-up activities, there remains at some locations exceedences of provincial (41) and federal (43) arsenic soil quality guidelines established to protect environmental and human receptors in the most restrictive residential/agricultural land use (20 mg/kg, and 12 mg/kg, respectively). Although these exceedences indicate that there is a potential for adverse

effects to human health, they are spatially limited. Average arsenic concentrations measured recently in Port Hope soil do not exceed 10 mg/kg (23) and are therefore below levels that have a potential to cause adverse health effects.

4.4 Summary

Based on the understanding of uranium's chemical and radiological properties, the radiological properties of low-level radioactive wastes, and the non-radioactive contaminants within Port Hope, the health effects of potential concern in relation to the uranium refining and processing industry could include the following:

Potential Risk Factors	Most Plausible Health Effects
UO_3, UF_6	Kidney disease[1]
Radon, RDP, LLRD	Lung cancer
Radium (Ra-226)	Bone cancer[2]

1 Found only in experimental animal studies at high exposures (6, 12)
2 Radium's bone cancer threshold is about 10 Sv (16). It therefore cannot be considered a plausible risk factor in Port Hope

The health effects found in atomic bomb survivors and others with high whole body gamma ray doses include leukaemia, breast cancer, lung cancer and other cancers (16, 24, 30). The very low gamma ray doses from decay products in the lungs from Port Hope public exposures cannot be considered a plausible risk factor for these cancers (16, 24, 26, 29, 31, 32, 33).

The health effects of arsenic, ammonia and fluoride are only seen at very high concentrations (45, 46, 47, 48). Therefore, their health effects are not considered plausible at Port Hope public exposure concentrations.

Chapters 5 and 6 present the level of exposure to these various environmental contaminants in Port Hope residents, an assessment of the predicted health risks from these exposures and the results of epidemiological studies.

5.0 ENVIRONMENTAL STUDIES IN PORT HOPE

Human exposure to contaminants is estimated by considering different environmental pathways. For example, soil contamination can be taken up by plants, which are then consumed by humans. Humans can also drink contaminated water and inhale contaminated air. Therefore, environmental data such as concentrations of contaminants in air, soil, water, vegetation and animals are used for calculations of exposure and subsequent assessment of risk in humans.

The following sections describe various environmental studies conducted in Port Hope to assess the levels of both the radiological and non-radiological sources of contamination within the town, and are used for assessment of risks to human health.

5.1 Levels of Contaminants in the Environment

The characterization of contaminant behaviour within and between environmental media (soil, water, plants or air) provides important data for calculations of human exposure to radiation and chemical substances, as well as human health risk assessment. These calculations and assessments depend on many factors such as contaminant concentrations, uptake levels, bioavailability, and transfer of contaminants from one environmental compartment to another (e.g., air to vegetation).

5.1.1 A Review of Phytotoxicology Investigations: 1974–2003 Cameco Corporation – Port Hope (49)

Scientists from the Ontario Ministry of the Environment (MOE) have monitored soil and vegetation in the vicinity of Cameco's PHCF since 1968, to determine the impact of Cameco's emissions on soil and vegetation in the Port Hope region. Contaminants of concern from atmospheric emissions from Cameco facilities result in deposition of contaminants to soil and vegetation. Emissions to the atmosphere from PHCF and its predecessors primarily included fluoride, uranium and arsenic.

Damage to the leaves of sensitive species of vegetation as a result of fluoride (in the form of hydrogen fluoride) has been observed in Port Hope since 1968. However, both the severity and extent of the damage has diminished substantially up to 1990. Since then, fluoride damage on vegetation has been observed only at trace levels in the most sensitive plant species at sites within 500 to 750 m of Cameco's PHCF.

Elemental uranium absorbed by tree foliage from the atmosphere is not toxic to plants, but will settle in the vicinity of the emission source and may accumulate in the soil. Investigations concluded that elevated concentrations of uranium in soil were caused by historical and recent atmospheric emissions and, at some sites, by the disposal of process waste containing uranium (see Chapter 3). Cameco continues to emit very small amounts of uranium to the atmosphere in accordance with their licence (total annual emissions of 69.9 kg in 2007) (35).

Arsenic originating from Cameco PHCF was measured in the leaves of plants in the mid-1980s, and in smaller amounts from 1989 to 2003. Levels of arsenic were marginally elevated in the 1980s, fell slightly through the 1990s, and are currently just above the analytical detection limits at the sample sites closest and most directly downwind of Cameco's PHCF. The arsenic concentration in the leaves of street trees in Port Hope in 2003 suggest that Cameco's PHCF was still a minor source of arsenic at that time, although these levels are very low and would not result in a measurable adverse effect to the trees.

The measurement of elevated soil uranium concentrations (and other elements) prompted the MOE to conduct a human health risk assessment in 1991 (51). The assessment concluded that "... exposures to the reported levels of uranium, antimony, chromium, copper, nickel, cadmium, cobalt, selenium, and zinc in Port Hope soils are not expected to result in adverse health consequences."

Re-sampling of the soil in 2000 from undisturbed sites within 500 m of Cameco's PHCF was done to determine if soil uranium and arsenic levels had increased at locations that were originally sampled in 1986 to 1987. It was concluded that soil uranium and arsenic concentrations have not increased since the mid-1980s suggesting that current emissions from the PHCF are not likely to result in further accumulation of uranium in surface soil in Port Hope. Some sites still had arsenic soil concentrations higher than the MOE upper range of background concentrations for soil in Ontario (17 mg/kg) and all sites exceeded the MOE Ontario Typical Range Soil Guideline for uranium (2.1 mg/kg). However, average concentrations of these potentially toxic substances were 13 mg/kg for arsenic and 20 mg/kg for uranium that is below the Ontario soil quality guideline for arsenic (20 mg/kg) and the most restrictive CCME soil quality guideline for uranium (23 mg/kg). Therefore, these concentrations are not expected to result in adverse health effects due to the limited spatial extent of the contaminated areas.

5.1.2 2002–2004 Uranium Concentrations in Port Hope Soils and Vegetation and Toxicological Effects on Soil Organisms (52)

The accuracy of any environmental risk assessment is highly dependent on the quality of the data used in models designed to predict contaminant behaviour in various environmental compartments. In order to understand major factors influencing the quality of Cameco's long-term predictions of uranium concentrations in Port Hope soils, CNSC staff undertook a review of existing information on uranium in Port Hope soils. As a result of this review, CNSC staff identified data gaps and areas of uncertainty that made the prediction of both the long-term behaviour and potential accumulation of uranium in Port Hope soils uncertain.

From 2002 to 2004, EcoMatters carried out a study for the CNSC to obtain information about uranium concentrations in Port Hope soils, its potential transfer to locally grown vegetation and its potential effect on soil organisms, as well as major soil parameters used to predict soil uranium concentrations. This provided important site-specific

information for prediction of the contaminant behaviour in Port Hope soil and in assessing the health risks of ingesting these contaminants.

Uranium and about 50 other elements were measured in soil and plant samples. The measured uranium soil concentrations were considerably lower than expected (18 mg/kg maximum), taking into consideration the previously obtained data in Port Hope (up to 135 mg/kg). Measured uranium concentrations in soil were lower than the soil guidelines for protection of human health (see section 4.1.2). The uranium concentration in about 70 locally grown vegetation species were very low (just slightly above the detection limit) and in general agreed with values measured previously in Port Hope, including the MOE results (49).

Based on these results, CNSC staff concluded that the long term behaviour and potential accumulation of uranium in soil and vegetation do not represent a risk to Port Hope residents.

It was found, however, that Cameco's approach to predict uranium soil concentrations using generic, rather than site-specific, soil characteristics and parameters did not appear to be conservative enough. Therefore, the CNSC staff requested that Cameco validate their soil model predictions and determine whether or not uranium would accumulate in Port Hope soil to levels that could pose a health or environmental risk in the future.

5.1.3 2008–Soil Characterization and Evaluation Study at Port Hope (23)

At the request of the CNSC in 2005, Cameco Corporation conducted a comprehensive soil characterization study in Port Hope. The study was requested to validate model parameters used for predicting soil uranium concentrations at locations exposed to the highest-expected air concentrations of uranium, and to confirm that concentrations of uranium in Port Hope soils would not reach levels that would pose risks to human health or the environment.

A soil and vegetation sampling program was carried out in 36 Port Hope locations including public properties such as parks. The measured soil uranium concentrations, the location-specific soil model parameters (for example, soil-water distribution coefficient, soil bulk density, soil moisture content) and modelled uranium air concentrations were used to estimate future long-term soil uranium concentrations.

Using the site-specific model parameter values, soil uranium concentrations were predicted to increase at a rate of around 0.2 mg/kg of dry soil per year at the location of highest expected deposition. These results are based on highly conservative estimates of air concentrations and air deposition rates and therefore future accumulation is overestimated. The model that makes these predictions still has to be validated in further studies. The CNSC is using this work to ensure that the operation of the PHCF will not result in accumulation of uranium in Port Hope soils to a level that would be a concern for human health or the environment.

The soil concentrations of uranium and arsenic, while elevated at some sites (up to 51.3 mg/kg and 94 mg/kg, respectively) in comparison to the provincial background levels (2.1 mg/kg and 17 mg/kg) due to the presence of historic wastes, are not expected to result in adverse health consequences due to low biological availability of uranium in Port Hope soils and the limited spatial extent of the contaminated areas. Recent measurements indicate that median levels of uranium and arsenic (3.1 mg/kg and 8 mg/kg, respectively) in soils are within the range of the Ontario background concentrations for these potentially toxic substances and, therefore, are below guidelines set for the protection of human health.

The site-specific and depth-specific soil and vegetation data obtained in this study were used to recalculate the radiation dose to human receptors (see table below). The recalculated doses did not differ significantly from original calculations by Cameco, with the exception of one case (Yacht Club), where the recalculated dose (0.006 mSv per year) was higher than the original dose of 0.00046 mSv per year calculated by Cameco. This is due to the greater contribution of the uranium component of the dose. However, in all cases, the calculated doses were well below the regulatory limit of 1 mSv/year for members of the public (17).

	Doses (mSv/year) calculated in SENES derived release limits report (53)	**Doses (mSv/year) recalculated in the soil characterization and evaluation study (23)**
Receptors at PHCF		
Resident		
Mill Street	0.041	0.040
Alexander	0.029	0.033
Recreational		
Fishing	0.101	0.109
Yacht Club	0.00046	0.006
Commercial		
Worker	0.011	0.016

Using the data obtained in this study, CNSC staff has estimated that it would take perhaps 160 years for uranium soil concentrations to reach the median level of 23 mg/kg assuming a highly conservative uranium concentration in air of 0.005 $\mu g/m^3$. The soil uranium guideline of 23 mg/kg was recently recommended for residential/agricultural land use to protect human health and the environment (22). The CNSC staff concluded, therefore , that operation of the Cameco PHCF will not result in accumulation of uranium in Port Hope soils to levels that would be a concern for human health or the environment, assuming a reasonable economic life span of 100 years.

5.1.4 1976–1996 – Past and Present Exposure to Uranium and Other Radionuclides in Port Hope (54, 55, 56, 57, 58)

Over the last 30 years, Health and Welfare Canada (HWC), now Health Canada, conducted several studies of ambient radiation levels in Port Hope, investigated past and present exposures to uranium and other radionuclides in air, water, soil, and garden vegetables.

In the summer of 1976, HWC investigated the uptake of radium-226 (Ra-226), lead-210 (Pb-210) and uranium by vegetables grown in contaminated Port Hope gardens. Highest concentrations were found in root and stem vegetables and, for radium, also leafy vegetables. Fruits generally showed the lowest values. The highest estimated dose to a person consuming all of their vegetables from a contaminated garden was about 0.0068 mSv/year from Ra-226 (55). This dose estimate is well below the public dose limit of 1 mSv/year for members of the public (17).

In 1981 and 1982, HWC monitored the concentrations of uranium in air in Port Hope (56). Air uranium concentrations ranged from 0.002-0.227 $\mu g/m^3$, with a geometric mean of 0.02 $\mu g/m^3$. The committed dose from the measured concentrations to a critical receptor for one year of refinery operation was estimated to be 0.16 mSv, well below the public dose limit of 1 mSv/year (17). These measured air concentrations are also well below the uranium air guideline proposed by the Ontario Ministry of the Environment (MOE) (5) to protect human health (see section 4.1.2). No significant health effects would be expected at these extremely low levels (see Chapter 4).

In 1988 and 1989, HWC undertook a follow-up environmental monitoring study at six sampling stations in Port Hope to assess the possible health impact of ongoing airborne uranium releases from Cameco's PHCF (57). In addition, thermoluminescent dosimeters (TLDs) were installed at each of the six sampling stations and at various points around the Cameco's PHCF fence and the waste storage area on Dorset Street East to obtain average gamma radiation doses. A gamma radiation survey in the vicinity of the sampling stations was also conducted using hand-held gamma monitoring equipment.

Average weekly uranium concentrations in air at each monitoring site were taken from July 1988 to September 1989. The air monitoring data at the six sampling stations varied from 0.00006 $\mu g/m^3$ to 0.0757 $\mu g/m^3$, with a geometric mean of 0.00105 $\mu g/m^3$. The average background concentration for uranium in air for southern Ontario was found to be about 0.0005 $\mu g/m^3$ in this study. Annual averages based on the 12-month period of monitoring ranged from 0.001-0.0158 $\mu g/m^3$. Average concentrations of uranium in air recorded during the 1988-1989 were generally a factor of 5-10 less than that observed during 1981-1982, verifying emissions had decreased as a result of operational changes implemented by Cameco at the PHCF, beginning in 1983. The closest air monitoring station to PHCF (the marina) had the highest annual average uranium concentration of 0.0158 $\mu g/m^3$. This would result in a 50-year committed dose of 0.044 mSv. This dose is well within the public dose limit of 1 mSv/year (17). The highest annual average uranium

concentration of 0.0158 μg/m^3 is also below guidelines proposed by the MOE (5) (see section 4.1.2).

Quarterly gamma radiation values recorded at the six monitoring stations during 1988-1989 ranged from 0.06-0.16 μSv/hour. Corresponding average values, taken over the five quarterly periods, ranged from 0.08-0.11 μSv/hour. These values were typical of background gamma exposures of 0.04-0.13 μSv/hour measured across Canada in 1988 (59). In addition, the gamma radiation survey yielded average gamma exposures similar to those obtained from quarterly TLD readings, verifying these results. The gamma radiation dose at the west fence of Cameco was used to estimate the gamma dose to a worker. For an average exposure rate of 0.30 μSv/hour to a worker spending a year at the west fence, the resulting radiation dose to the body would be 0.4 mSv/year. This is well within the regulatory dose limit of 50 mSv/year and 100 mSv over a five-year dosimetry period for occupational exposure (17).

Observed uranium concentrations during 1988-1989 were significantly lower than those observed during 1981-1982, verifying the decrease in emissions as a result of operational changes to PHCF. Resulting radiation doses from inhalation of airborne uranium particulate are well below regulatory guidelines, and represent a small fraction of normal background radiation (59). Doses to workers resulting from gamma fields within the Cameco PHCF are below occupational limits (17).

From 1973 to 1983, HWC monitored the levels of the natural radionuclides including uranium (U-238, Ra-226 and Pb-210) in the drinking water supplies of 17 communities across Canada, including Port Hope (58). Most of these communities utilized surface water supplies and the radionuclide concentrations were consistently low or non-detectable. The concentrations measured were as follows: uranium < 0.0001-0.001 mg/L; Ra-226 < 0.005-0.02 Bq/L; and Pb-210 < 0.005-0.02 Bq/L. These values were below the drinking water guidelines (19, 20).

From 1983 to 1996, the program was reduced to just three municipalities, including Port Hope (because of uranium refining and processing activities possibly impacting surface water supplies). Only radium and total uranium were monitored because the levels of lead had been shown to be consistently low or non-detectable in surface waters. Ra-226 levels in Port Hope drinking water remained non-detectable (less than the detection limit of 0.005 Bq/L) throughout this period, and uranium concentrations were within the normal range for surface water supplies (from 0.0004 mg/L to 0.001 mg/L). This was well below the maximum acceptable concentration of uranium in drinking water, which is 0.02 mg/L (19, 20).

5.1.5 1955–1993 – Average and Cumulative Exposures for Residents of Port Hope, Ontario Resulting from Historic Low-Level Radioactive Wastes in the Town (60)

In 1994, SENES Consultants Ltd. conducted a dose reconstruction study for Health Canada to estimate the average and cumulative exposures for typical residents of Port Hope, Ontario resulting from 40 years of exposure to products and by-products of

the uranium refining and processing industry and the historic low-level radioactive wastes in the town. Indoor and outdoor exposures to radon gas and radon decay products, gamma radiation, inhalation of airborne uranium and ingestion of uranium and radium from contaminated soils were assessed.

The largest doses to Port Hope residents were due to indoor radon and gamma radiation exposures. Indoor doses were ten-fold larger than outdoor doses because indoor concentrations are often higher and more time is spent indoors. Doses from inhalation of uranium and ingestion of both uranium and radium contributed less than 1% of the total dose. From 1955 to 1993, the annual average dose rate to Port Hope residents ranged from 0.27 mSv/year to 0.25 mSv/year for indoor gamma radiation and ranged from 0.99 mSv/year to 0.69 mSv/year for indoor radon. These radiation levels within Port Hope were similar to those in Cobourg and levels of radium and uranium in soil were typical of southern Ontario. These levels are considered to be equivalent to background doses because they are similar to the annual average dose to Canadians of 2.4 mSv/year (2) (see section 2.1). Based on the cumulative estimated exposures observed and existing knowledge of dose-response relationship of radiation risk, an increase in the risk of cancer relative to the Canadian population would not be expected because cumulative doses were so low (38, 61).

5.1.6 1994 – The Federal Assessment of Major Unlicensed Historic Waste Sites Town of Port Hope (62)

In 1994, Natural Resources Canada in cooperation with the Low Level Radioactive Management Office (LLRWMO) and the Siting Task Force Secretariat assessed nine main historic waste sites in Port Hope (Port Hope harbour; Port Hope landfill; Alexander Ravine; Waterworks area; Viaducts area; Mill Street site; Pine Street extension; Highland Drive roadbed; south groundwater discharge zone - Hunt's Pond area) in response to concerns about the environmental and health hazards associated with unremediated wastes in the Town of Port Hope. The assessment relied on both recent and historical environmental data, including measurements of radon concentrations and gamma radiation, analyses of surface and groundwater samples, soil samples, garden produce, and fish caught in the local waters. The assessment addressed both the on-site and off-site environmental impacts from the radiological waste, arsenic, and uranium in air, water and soil.

Incremental environmental impacts above normal background conditions were assessed in terms of their health implications. Both the off-site and on-site radiological dose levels for normal and non-routine activities at all nine sites ranged from 0.002 mSv/year to 0.048 mSv/year and were well within the natural variation in exposure to normal background radiation levels observed in Port Hope and below the CNSC public dose limit of 1 mSv per year (17). The federal department concluded that exposures that could result from remaining contamination or waste at the sites were well below public dose limits and did not pose any public health risks.

The assessment also took into consideration potential exposures to arsenic and uranium. Arsenic and uranium annual intakes (6.9 mg/L and 1.7 mg/L, respectively) estimated for normal and unlikely activities at the sites were also well below the annual intake limits for arsenic and uranium (18.3 mg/L and 73 mg/L, respectively) calculated based on maximum acceptable concentrations in Canadian Drinking Water Guidelines (19). Based on this, it is concluded that there are no health risks to Port Hope residents as a result of arsenic and uranium at the sites considered in this assessment.

5.1.7 1994- The Siting Task Force: Low-level Radioactive Waste Management (63, 64, 65)

The Siting Task Force was an independent group appointed by Natural Resources Canada to implement a five-phase co-operative siting process to find one or more sites for a long-term management facility for the Port Hope historic low-level radioactive wastes and contaminated soils. Part of this process was to describe the toxicological and epidemiological information on the potential health effects of selected soil contaminants associated with the low-level radioactive waste and contaminated soils (antimony, arsenic, cadmium, chromium, cobalt, lead and uranium) found at sites in the Town of Port Hope, the Port Granby Waste Management Facility and/or the Welcome Waste Management Facility. The report concluded that the contaminants were unlikely to pose a health risk to residents of Port Hope and its surroundings.

5.2 Summary

The uranium air concentrations in Port Hope are somewhat elevated (range 0.000028 $\mu g/m^3$ to 0.005 $\mu g/m^3$) compared with the provincial background concentrations, but are significantly lower than the proposed Ontario Ambient Air Quality Criteria developed to protect human health (range 0.02 $\mu g/m^3$ to 0.5 $\mu g/m^3$).

The uranium concentration in drinking water in Port Hope (0.00055 mg/L) is significantly lower than the Ontario standard for uranium in drinking water, and is consistent with uranium concentrations in drinking water reported by the province's water supply systems under the Ontario Drinking Water Surveillance Program (range 0.00005 mg/L to 0.004 mg/L).

The uranium and arsenic soil concentrations in Port Hope, while elevated at some sites (up to 51.3 mg/kg and 94 mg/kg, respectively) in comparison to the provincial background levels (2.1 mg/kg and 17 mg/kg, respectively) due to the presence of historic waste, are not expected to result in adverse health consequences due to low biological availability of uranium in Port Hope soils and the limited spatial extent of the contaminated areas. Recent measurements indicate that median levels of uranium and arsenic (3.1 mg/kg and 8 mg/kg, respectively) in soils are below guidelines set for the protection of human health (23 mg/kg and 20 mg/kg).

The assessments have also shown that radiation doses to members of the public from exposure to the radionuclides found in Port Hope as a result of uranium refining and processing are low and well below the public dose limit of 1 mSv per year established by the CNSC (17). The total radiation dose to Port Hope residents is similar to the average annual dose to Canadians of 2.4 mSv/year and therefore considered equivalent to background (2).

Overall, the environmental studies conducted over the years by Health Canada (previously Health and Welfare Canada), the Ontario Ministry of the Environment, the CNSC, the LLRWMO and Cameco indicate that contaminant concentrations and radiation levels remain well below guideline levels set to protect human health.

6.0 EPIDEMIOLOGICAL STUDIES IN PORT HOPE

This chapter describes the three main types of epidemiological studies conducted in Port Hope in the last 30 years, covering the time period from the 1950s to present day. These health studies have assessed the health status of the members of the public and radium and uranium refining and processing industry workers in Port Hope in several ecological studies, and have assessed the risk of radiation exposure and disease in several case-control and cohort studies. These studies have covered the time period prior to 1966, when the main sources of health concern were radiological, the time period of remediation of the LLRW within the town (1976 to 1981), the period of the SX plant (1967 to 1984) when uranium emissions were elevated, and current times (1984 to present) when the implementation of many mitigation measures significantly reduced uranium emissions (see Chapters 4 and 5 for sources and levels of exposure).

To set the context for the studies presented in this section of the report and help in their interpretation, it is important to note that the leading causes of death in Ontario and Canada are diseases of the circulatory system (heart disease and stroke), cancer, and diseases of the respiratory system (66, 67). Three types of cancer account for the majority of new cases for each sex: prostate, lung and colorectal in males and breast, lung and colorectal in females (68). Lung cancer remains the leading cause of cancer death for both men and women. Overall, colorectal cancer is the second leading cause of death from cancer (68).

Finally, epidemiological studies conducted in Port Hope are presented in sections 6.2, 6.3 and 6.4, from least to most reliable (ecological, case-control, cohort).

6.1 Types of Epidemiological Studies

A large number of epidemiological studies have been conducted in Port Hope. This section provides a basic definition of epidemiology and describes the purposes, strengths and limitations of various types of epidemiological studies. This will form the basis for an interpretation of the Port Hope studies presented in sections 6.2, 6.3, and 6.4 of this report.

Epidemiology is the study of the distribution and determinants of diseases in specified human populations, and the application of this study to control disease (16, 69). It is based on observation, not experiments, and so there is always varying degrees of bias. A well-designed study will try to minimize potential biases. There are three main types of epidemiological studies: cohort studies, case-control studies, and ecological correlation (descriptive) studies.

6.1.1 Cohort Studies

Cohort studies are the most robust type of epidemiological study. These studies start with a defined group of individuals (the cohort) who are free of the disease under consideration but who vary in exposure to a supposed noxious factor (for example, occupational radon and its decay products, gamma radiation exposure). Detailed information is gathered on each individual's exposure (i.e., date of first and last exposure, time since exposure, annual and cumulative exposure, dose rate) and each member of the cohort is followed over time in order to determine differences in the rate at which disease (for example, cause of death, cancer diagnosis) develops in relation to his/her exposure to the noxious factor (16, 70). Individuals with different exposures (or levels of exposure) are compared to assess differences in their probability (or risk) of developing or dying from a given disease (16, 70). Cohort studies can produce useful information on the incidence of disease and death rates and can assess the risk (or probability) of developing a disease effect as a result of the exposure of interest. They are efficient in studying rare exposures (i.e., occupational radiation exposure) and are not as prone to bias as case-control studies. However, they are very costly and require a large number of subjects and a long follow-up period. There may be problems in loss of cohort members, problems in disease ascertainment, and changes in criteria and methods over time (i.e., reporting of occupational radiation requirements; monitoring of work areas versus individual radon monitoring).

6.1.2 Case-control Studies

Unlike cohort studies, case-control studies tend to focus on a single disease. People recently diagnosed as having a disease (cases) are compared with people who do not have the disease (controls). The purpose is to determine if the two groups differ in the proportion of persons who have been exposed to a specific factor or factors, with the aim to establish a relationship between the disease and factor. It compares cases and controls with regard to the presence of some element in their past. Data on individuals (the "cases") with a recently diagnosed specified disease (for example, lung cancer or childhood leukaemia) are assembled and are matched with data on a suitable set of "control" (comparison/reference) individuals. The control individuals are otherwise similar to the cases (for example, the same age or sex, the same opportunity of having the exposure) but do not have the specified disease (16, 70). Detailed information on exposures (for example, residence at birth, residence at death, father's occupation, father's occupational radiation exposure; number of years living in a home, number of hours at home per day, residential radon exposure) and other information (for example, smoking history, diet, exercise, genetic factors, other occupational exposures) are collected on both the cases and controls. The relationship between the exposure and the disease is examined by comparing the cases (diseased) and controls (non-diseased) with regard to the distribution of a number of exposures between the two groups (16, 70). The advantage of a case-control study is that detailed histories of exposure and other information can be collected relatively easily, they are relatively inexpensive to carry out, the number of subjects can be small (especially for rare diseases), results can be obtained relatively quickly and they can identify more than one risk factor. However, case-control

studies are prone to bias (i.e., selection of appropriate controls, recall bias of past exposures), information from the past may be incomplete and there can be problems in selecting an appropriate control group and matching on variables (16, 70). Thus, cohort studies are regarded as more reliable than case-control studies.

6.1.3 Descriptive Ecological (Correlation) Studies

In ecological studies, the occurrence of a specific disease within a defined population (for example, Port Hope), time and geographical area is compared with the (expected) occurrence of the disease, based on a stable reference population (i.e., the general population of Ontario or Canada).

These studies analyze populations or groups of people, rather than individuals. An association observed between variables on a group level does not mean the same association will exist at the individual level. These are the weakest type of epidemiological study since they operate at a group level, not an individual level, and data are averaged over groups (16, 70). Mortality and morbidity statistics, reflecting the frequency of occurrence of disease in a population, are often routinely collected so ecological studies provide a readily available indicator of the frequency of disease in a population. Descriptive ecological studies are useful monitoring tools for epidemiologists to identify high and low rates of disease in a population, which may warrant further study. They can identify trends over time or within groups. These studies are also relatively simple, easy and inexpensive to conduct in comparison with case-control and cohort studies. However, because ecological studies do not examine individual exposures, they are limited in that it is not possible to make conclusions or draw inferences on the risk factor (for example, radiation increased the risk of disease in the population) in relation to the disease incidence or mortality.

Information on exposures to individuals is not known, and no consideration is given to the multiple risk factors of disease (for example, diet, exercise, tobacco, alcohol or obesity) (16, 70). On occasion, assignment of cumulative exposures is made to groups (for example, average residential radon levels in areas of a town); however, many assumptions still exist and individual exposures are still unknown. This becomes especially important when a risk factor (i.e., tobacco smoking) is known to be strongly associated with the disease (i.e., heart disease, lung cancer). Mortality figures do not reflect the frequency of illnesses that are successfully treated. Morbidity figures do not accurately reflect the incidence or prevalence of illnesses that are not diagnosed by a medical professional or are not severe enough to require treatment or hospitalization. Errors in the assignment of place of residence are known to occur and are often not specific, particularly in rural communities. Population mobility and daily activities also impacts on the assignment of environmental exposures. Finally, the precision of the statistics is often limited because of the small numbers of observed and expected cases or deaths in small populations (16, 70), which makes interpretation of results extremely difficult.

Standardized mortality ratios (SMR) and standardized incidence ratios (SIR) are commonly used for comparison in ecological studies. They represent the ratio of the observed divided by the expected number of deaths (SMR) or cases (SIR). A ratio of 1.0 indicates that the observed (i.e., number of cases, deaths) for the specified area was the same as expected when compared with the reference population. A ratio of 1.4 indicates a rate of 40% higher than the reference population; a ratio of 0.7 indicates a rate that is only 70% of that of the reference population (16, 70). The 95% confidence interval evaluates how likely it was that chance could explain the difference in rates seen between the study population and the reference population. If the confidence interval is wide, the ratio is based on few observed cases or deaths. If it is narrow, the ratio is based on many observed cases or deaths. If the confidence interval does not include 1.0 (1.0 means no difference in risk), it is considered statistically significant at the 95% confidence level.

Interpreting SIRs and SMRs must be done with a good deal of caution and departures from 1.0 must be viewed extremely cautiously. When a statistically significant elevated risk for a particular disease, such as cancer, is observed in an area, it is only an indication that there may be an elevated risk in the area associated with environmental, social, behavioral or genetic factors. Port Hope's population (approximately 15,000) is a relatively small population in which to observe rare disease. Mortality and incidence rates calculated for small populations are unstable (in a statistical sense), especially for less common diseases or cancer sites, even if studied over extended periods of time. Therefore, excess rare diseases in such areas should be interpreted with greatest caution; the stability of the rate must be carefully examined. Thus, a high cancer rate in a given region is not sufficient evidence to implicate specific risk factors, or require more epidemiological investigation to assess the relative importance of various factors. The more rare the disease/birth defect/cancer site and the smaller the population, the more important the role of chance (natural random variation in disease) and the less dependable (unstable, variable) the risk estimate is (16, 70).

6.2 Descriptive Ecological (Correlation) Studies of the General Population of Port Hope

At least nine ecological studies were conducted in Port Hope to compare the overall health status of Port Hope residents with that of other communities or the general population. These studies cover the time period from the 1950s to present day. The rates of all causes of death (mortality), newly diagnosed cancers (incidence), and birth defects among Port Hope residents were compared with the general population of Ontario, other similar communities, and Canada. The strengths and limitations of these studies are discussed in Section 6.1.3.

6.2.1 1954–1978 – Standardized Mortality Ratios (SMRs) in Selected Urban Areas in Ontario (71)

In 1984, the Ontario Ministry of Labour published a report that compared the mortality rates of 40 different urban areas, including Port Hope, with the mortality rates of the general Ontario population. Mortality data for Ontario residents was provided by the

Ontario Registrar General by sex, cause of death and age group. Statistics Canada provided population counts by sex, age group and place of residence. The study looked at 33 different causes of death and covered the period from 1954 to 1978. Table 1 provides the observed and expected deaths and standardized mortality ratios for 20 causes with greater that 5 deaths for each sex, in the time period of study. The results of the study show that the mortality rates for the 33 causes of death were not significantly different in Port Hope residents compared with the general Ontario population.

TABLE 1

Observed and Expected Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Selected Causes of Death in Port Hope, Ontario Compared with Canadian Mortality Rates, 1954-1978

Port Hope Residents, Males and Females, All Ages

Cause of Death	Sex	Observed Deaths	Expected Deaths	SMR	95% Confidence Interval
All Causes	M	978	892.9	1.10	1.0, 1.2
	F	870	886.5	0.98	0.9, 1.0
All Causes except Accidents/Poisonings/Violence	M	906	915.7	1.11	0.9, 1.1
	F	822	943.8	0.97	0.8, 0.9
All Cancers	M	154	161.0	0.96	0.8, 1.1
	F	149	160.7	0.93	0.8, 1.1
Stomach	M	7	14.5	0.49	0.2, 1.0
	F	8	10.7	0.75	0.3, 1.5
Intestine except Rectum	M	13	16.2	0.80	0.4, 1.4
	F	26	24.6	1.06	0.7, 1.5
Trachea, Bronchus and Lung	M	42	42.2	0.99	0.7, 1.3
	F	13	9.5	1.37	0.7, 2.3
Breast	F	34	32.0	1.06	0.7, 1.5
Prostate	M	18	15.8	1.14	0.7, 1.8
Leukaemia	M	6	7.0	0.86	0.3, 1.9
	F	8	6.1	1.30	0.6, 2.6
Diabetes Mellitus	M	6	11.2	0.53	0.2, 1.2
	F	6	17.8	0.34	0.1, 0.7
Diseases of the Circulatory System	M	553	474.2	1.17	1.0, 1.3
	F	516	517.6	1.00	0.9, 1.1
Ischemic Heart Disease	M	392	333.4	1.18	1.0, 1.3
	F	303	306.6	0.99	0.9, 1.1
Cerebrovascular Disease	M	90	82.0	1.10	0.9, 1.3
	F	124	130.4	0.95	0.8, 1.1
Diseases of Arteries, Arterioles and Capillaries	M	25	25.8	0.97	0.6, 1.4
	F	33	33.9	0.97	0.7, 1.4
Diseases of the Respiratory System	M	71	62.9	1.13	0.9, 1.4
	F	55	50.5	1.09	0.8, 1.4
Pneumonia	M	38	31.5	1.20	0.9, 1.7
	F	42	35.9	1.17	0.8, 1.6
Diseases of the Digestive System	M	28	32.9	0.85	0.6, 1.2
	F	32	29.9	1.07	0.7, 1.5
Cirrhosis of the Liver	M	20	12.1	1.65	1.0, 2.6
	F	7	7.1	0.98	0.4, 2.0
Congenital Anomalies	M	6	8.4	0.72	0.3, 1.6
	F	4	7.5	0.53	0.1, 1.4
Accidents, Poisonings and Violence	M	72	77.2	0.93	0.7, 1.2
	F	48	42.6	1.13	0.8, 1.5

Expected values are based on Canadian rates.

6.2.2 1973–1979 – Mortality Atlas of Canada, Volume 3: Urban Mortality (72)

In 1984, Health and Welfare Canada compared mortality rates of various Canadian urban communities with the general Canadian population from 1973 to 1979. Causes of death and population data from the 1976 population census were provided by Statistics Canada. Urban localities with a 1976 population over 5,000 were selected for analysis. The cancer mortality rates for Port Hope, including Hope Township, were not significantly different compared with the general Canadian population. Table 2 illustrates that for the population aged 35-69, Port Hope had statistically significant elevated ($p<0.05$) mortality rates for males for coronary heart disease and cirrhosis of the liver. Similar findings for all causes were found in many of the less populated urban areas of Ontario. Similarly, significantly high rates of coronary heart disease were found mostly in eastern Ontario and along the eastern shore of Lake Ontario.

TABLE 2

Observed and Expected Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Selected Causes of Death in Port Hope, Ontario Compared with Canadian Mortality Rates, 1973-1979

Port Hope Residents, Males and Females					
Cause of Death	**Sex**	**Observed Deaths**	**Expected Deaths**	**SMR**	**95% Confidence Interval**
All Causes (all ages)	M	486	443	1.1	1.0, 1.2
	F	420	398	1.1	1.0, 1.2
Coronary heart disease (ages 35-69)	M	90	66	1.4+	1.1, 1.7
	F	28	22	1.3	0.8, 1.8
All Cancer (ages 35-69)	M	41	47	0.9	0.9, 0.6
	F	42	38	1.1	0.8, 1.5
Lung Cancer (ages 35-69)	M	12	17	0.7	0.4, 1.2
	F	6	4	1.5	0.5, 3.3
Chronic obstructive lung disease (ages 35-69)	M	6	6	1.0	0.4, 2.2
	F	3	2	1.5	0.3, 4.4
Cirrhosis of the liver (ages 35-69)	M	14	7	2.0+	1.1, 3.4
	F	3	3	1.0	0.2, 2.9
Motor Vehicle Traffic Accidents (ages 15-34)	M	8	9	0.9	0.4, 1.8
	F	1	2	0.5	0.0, 2.8
Infant Mortality (ages less than 1 year)	B[1]	13	19	0.7	0.4, 1.2

Expected values are based on Canadian rates.
[1]Infant Mortality is for both sexes combined.
+ Statistical significance at the $p<0.05$ level.

6.2.3 1950–1987 (1964–1986) – A Study of Childhood Leukaemia around Canadian Nuclear Facilities (73, 74, 75)

In 1987, researchers from the Ontario Cancer Treatment and Research Foundation and the University of British Columbia examined mortality and incidence of childhood leukaemia in the vicinity of Ontario nuclear facilities, including the PHCF. They

conducted a two-phase study of childhood leukaemia incidence rates from 1964 to 1986 and childhood leukaemia mortality rates from 1950 to 1987 in children aged 0-4 years and 0-14 years, respectively.

The objective of the study was to compare the rates of leukaemia in children born to mothers living within 25 km of a nuclear facility with the rates of childhood leukaemia of the general population of Ontario. The study included people living in Northumberland County, located near the PHCF. Residence was obtained from birth certificates, cancer registry or death certificates.

No evidence was found of any statistically significant excess childhood leukaemia incidence or mortality, whether assessed by residence at death, residence at birth, at the county level or "nearby" the PHCF (including Port Hope, Cobourg, Newcastle, Hope Township, Hamilton Township and Haldimand Township). The findings were similar in children aged 0–4 years (Table 3) and aged 0–14 years (Table 4), respectively.

In summary, the standardized mortality ratios (SMR= observed deaths/expected deaths) and standardized incidence ratios (SIR= observed cases/expected cases) were not statistically significantly different compared with the general population of Ontario. The 95% confidence intervals around the SMR and SIR included the null value (1.00) (i.e., observed equals expected) and were generally wide. The statistical power of these studies was limited due to the rarity of childhood leukaemia and the small number of observed and expected deaths and cases.

TABLE 3

Leukaemia mortality (SMR) and incidence (SIR) in children aged 0–4 years in Northumberland County (Port Hope - uranium refinery) by residence at time of death and birth, and by geographic region

Mortality (1950–1986)	Observed Deaths	Expected Deaths	SMR	95% Confidence Interval
By residence at death				
Northumberland County *	9	9.3	0.97	0.44, 1.84
Nearby PHCF*	6	4.3	1.38	0.5, 3.01
By residence at birth				
Northumberland County *	12	8.1	1.49	0.77, 2.6
Nearby PHCF*	7	4.5	1.57	0.63, 3.23
Incidence (1964–1985)	**Observed Cases**	**Expected Cases**	**SIR**	**95% Confidence Interval**
By residence at birth				
Northumberland County *	14	9.8	1.43	0.78, 2.39
Nearby PHCF*	9	5.2	1.72	0.78, 3.26

* Rows do not constitute independent analyses. A total of 20 individuals contribute to observed frequencies.

SMR – Standardized Mortality Ratio: ratio of observed over expected (O/E) number of deaths.
SIR – Standardized Incidence Ratio: ratio of observed over expected (O/E) number of cancer cases.

E. A. Clarke, J. McLaughlin, and T. W. Anderson. Childhood Leukaemia Around Canadian Nuclear Facilities – Phase I. Final Report. AECB Report INFO-0300.1. Atomic Energy Control Board. Ottawa, Canada (1989).

TABLE 4

Leukaemia mortality (SMR) and incidence (SIR) in children aged 0–14 years in Northumberland County (Port Hope - uranium refinery) by residence at time of death and birth, and by geographic region

Mortality (1950–1987)	**Observed Deaths**	**Expected Deaths**	**SMR**	**95% Confidence Interval**
By residence at death				
Northumberland County*	22	23.0	0.95	0.6, 1.45
Nearby PHCF*	14	10.6	1.32	0.72, 2.21
By residence at birth				
Northumberland County*	20	17.5	1.14	0.7, 1.76
Nearby PHCF*	12	10.0	1.20	0.62, 2.09
Incidence (1964–1986)	**Observed Cases**	**Expected Cases**	**SIR**	**95% Confidence Interval**
By residence at birth				
Northumberland County*	21	18.8	1.11	0.69, 1.7
Nearby PHCF*	13	11.7	1.11	0.59, 1.91

* Rows do not constitute independent analyses. A total of 34 individuals contribute to observed frequencies.

SMR – Standardized Mortality Ratio: ratio of observed over expected (O/E) number of deaths.
SIR – Standardized Incidence Ratio: ratio of observed over expected (O/E) number of cancer cases.

E. A. Clarke, J. McLaughlin and T. W. Anderson. Childhood Leukaemia around Canadian Nuclear Facilities – Phase II. Final Report. AECB Report INFO-0300.2. Atomic Energy Control Board. Ottawa, Canada (1991).

6.2.4 1984–1988 – Great Lakes Health Effects Program, Atlas II: Cancer Incidence in the Great Lakes Region, Ontario (76)

In 1992, Health Canada's Great Lakes Health Effects Program compared cancer incidence rates in various Ontario groups of municipalities with populations with at least 10,000 persons, with the general Ontario population, for the 5-year period, 1984 to 1988. Newly diagnosed cancer patients were provided by the Ontario Cancer Registry, which is approximately 95% complete. Residence was according to the Ontario Residence Code, which codes at the municipality level. The atlas focused on all cancers and eight different types of cancer related to drinking water contaminants (stomach, colorectal, digestive tract, lung, breast, prostate, bladder, and leukaemia). Table 5 summarizes the results of this study. Northumberland County-Cobourg (which included Port Hope) only had statistically significant higher male lung cancer compared with the general male population of Ontario. No excess cancer incidence was found among any other cancer site, or among women. The grouped municipality level (Northumberland County-Cobourg) was not specific enough to allow conclusions to be drawn for the town of Port Hope.

TABLE 5

Observed and Expected Number of Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers for Northumberland County-Cobourg, Ontario Compared with Ontario Cancer Incidence Rates, 1984-1988

Northumberland County Residents, Males and Females, All Ages

Cancer Site	Sex	Observed Cases	Expected Cases	SIR	95% Confidence Interval
Population		34,685			
All	M	414	394	1.05	0.95, 1.16
	F	397	377	1.05	0.95, 1.16
Stomach	M	15	14	1.07	0.60, 1.77
	F	5	9	0.58	0.19, 1.35
Colorectal	M	65	58	1.11	0.86, 1.42
	F	68	58	1.16	0.90, 1.47
Digestive Tract	M	111	98	1.13	0.93, 1.36
	F	99	90	1.10	0.89, 1.34
Lung	M	109	84	1.30+	1.06, 1.56
	F	45	40	1.14	0.83, 1.52
Breast	M	-	-	-	-
	F	100	97	1.03	0.84, 1.25
Prostate	M	64	69	0.93	0.72, 1.19
	F	-	-	-	-
Bladder	M	25	29	0.87	0.56, 1.29
	F	7	10	070	0.28, 1.43
Leukaemia	M	15	14	1.11	0.62, 1.82
	F	12	11	1.09	0.56, 1.90

+Statistical significance at the $p<0.05$ level.

6.2.5 1978–1988 – Great Lakes Health Effects Program: Atlas I: Birth Defects Atlas of Ontario (77)

In 1992, Health Canada's Great Lakes Health Effects Program compared the rates of 23 different birth defect categories in infants and stillborns in 51 Ontario counties and 50 of the larger municipalities with those of the general Ontario population from 1978 to 1988. Birth defects were ascertained for stillbirths, newborns and infants during the first year of life through computer searches. The Ontario Ministry of Health provided birth defect data, the Hospital Medical Records Institute provided hospital data, and Statistics Canada provided vital status data for 1973 to 1977, respectively. The Canadian Congenital Anomalies Surveillance System (CCASS) of Health Canada provided data from 1978 to 1988. There were 9,654 births in Northumberland County over the 10-year time period. The overall birth defect ratio was 0.77, based on 388 counts (95% CI: 0.70, 0.85) (see Table 6); thus, the overall rate of birth defects in the county was significantly low compared with the population of Ontario. Eye anomalies, pulmonary artery anomalies, and limb reduction anomalies had birth prevalence ratios of over 1.5, but none were statistically significantly different from 1.0 so were comparable with the rest of the population of Ontario. The results of this study are for the entire county and are not specific enough to draw conclusions specifically for Port Hope.

TABLE 6

Observed Case Counts, Birth Defect Birth Prevalence Ratios, and 95% Confidence Intervals for Northumberland County, Ontario Compared with Ontario Rates, 1978-1988[1]

Birth Defect	Observed Cases	Birth Prevalence Ratio	95% Confidence Interval
Births (1978-1988)	9,654		
Stillbirths	63	0.95	0.73. 1.22
Cases	293	0.79	0.70, 0.88
Overall Birth Defects	388	0.77	0.70, 0.85
Central Nervous System Defects	31	0.95	0.65, 1.35
Anencephalus & Similar Anomalies	3	0.72	0.15, 2.08
Spina Bifida	9	1.06	0.48, 2.00
Microcephalus & Brain Reduction	5	0.81	0.26, 1.88
Congenital Hydrocephalus	10	1.08	0.52, 1.98
Eye Anomalies	11	1.62	0.81, 2.90
Congenital Heart Defects	39	0.67	0.48, 0.92
Ventricular Septal Defect	15	0.65	0.37, 1.08
Atrial Septal Defect	6	0.51	0.19, 1.10
Circulatory System Anomalies	29	1.08	0.73, 1.56
Pulmonary Artery Anomalies	16	1.55	0.88, 2.51
Respiratory System Anomalies	7	0.62	0.25, 1.27
Digestive System Anomalies	53	1.02	0.76, 1.33
Urinary System Anomalies	11	0.74	0.37, 1.32
Renal Agenesis & Dysgenesis	4	1.04	0.28, 2.64
Hypospadias, Epispadias	19	0.77	0.46, 1.20
Cleft Lip and/or Cleft Palate	16	0.98	0.56, 1.59
Limb Reduction Anomalies	7	1.71	0.68, 3.51
Clubfoot	28	0.59	0.39, 0.86
Polydactyly, Syndactyly	8	0.53	0.23, 1.05
Down Syndrome[2]	9	0.72	0.33, 1.36
Infant Deaths Caused by Birth Defects	19	0.80	0.48, 1.25

[1] Ascertainment in the first year of life.
[2] Adjusted for Mother's Age.

6.2.6 1986–1992 – Great Lakes Health Effects Program: Port Hope Harbour Area of Concern: Health Data and Statistics for the Population of the Region (78)

In 1998, Health Canada's Great Lakes Health Effects Program published several documents on the health status of various communities, including Port Hope and Hope Township. The rates of various health indicators (mortality rates, rates of hospitalization, cancer incidence rates, and birth outcomes) were compared with the general population of Ontario from 1986 to 1992.

The SMR for all causes of death in Port Hope was comparable with that of the general Ontario population. A statistically significant excess mortality was found for circulatory disease (such as heart disease) in males and females, male pneumonia and influenza, and female skin infections (based on 2 cases) compared with the general Ontario population (Table 7).

The cancer incidence for all cancers in Port Hope was comparable with the general Ontario population. In fact, the SIR for all cancers for men in Port Hope was statistically significantly lower compared with the general Ontario male population. However, Port Hope women had a statistically significant excess of cancers of the pharynx compared with the general Ontario female population, based on <5 cases. The precision of the statistics is likely limited by the small number of observed and expected cases which makes interpretation of the results extremely difficult (16, 70). The SIR for all childhood cancers (based on ages 0-24 years) was comparable with the general Ontario population (Table 8).

Finally, the rates of congenital birth defects were not significantly different compared with the Ontario population. In fact, the incidence ratio of all birth defects for female infants in Port Hope was statistically significant low (incidence ratio=0.53; 95% CI: 0.31, 0.84) compared with that of the general Ontario population, based on 17 cases.

TABLE 7

Observed Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Selected Causes of Death for the Port Hope Harbour Area of Concern, Ontario Compared with Ontario Mortality Rates, 1986-1992

Port Hope Residents, Males and Females, All Ages (0–85+)

	Males			Females		
Causes of Death	**Observed Deaths**	**SMR**	**95% Confidence Interval**	**Observed Deaths**	**SMR**	**95% Confidence Interval**
All Causes	470	0.99	0.9, 1.08	511	1.10+	1.01, 1.20
Hereditary and Degenerative Diseases of the Central Nervous System	9	1.11	0.51, 2.10	7	0.68	0.27, 1.39
Parkinson's Disease	6	2.52	0.92, 5.41	1	0.51	0.01, 2.83
Ischemic Heart Disease	117	0.91	0.75, 1.09	129	1.09	0.91, 1.29
Disease of Pulmonary Circulation	5	2.56	0.83, 5.89	7	2.93+	1.18, 5.98
Other Forms of Heart Disease	22	1.33	0.83, 2.01	30	1.55+	1.05, 2.21
Diseases of Arteries, Arterioles and Capillaries	23	1.64+	1.04, 2.45	46	2.59++	1.90, 3.45
Artherosclerosis	13	2.54++	1.36, 4.34	36	3.09++	2.17, 4.28
Pneumonia and Influenza	26	1.59+	1.04, 2.33	26	1.30	0.85, 1.90
Chronic Obstructive Pulmonary Disease	22	0.99	0.62, 1.50	14	1.10	0.60, 1.84
Emphysema	5	1.69	0.55, 3.87	1	0.76	0.02, 4.22
Other Diseases of the Digestive System	6	0.56	0.20, 1.20	6	0.79	0.29, 1.70
Other Diseases of Urinary System	1	0.65	0.02, 3.62	6	2.67	0.98, 5.75
Infections of Skin and Subcutaneous Tissue				2	9.20+	1.11, 33.22
Malignant Neoplasms of the Digestive Organs and Peritoneum	42	1.16	0.84, 1.52	28	0.87	0.58, 1.26
Cancer of Colon and Rectum	21	1.45	0.90, 2.21	17	1.21	0.71, 1.94
Cancer of the Trachea, Bronchus and Lung	40	1.00	0.71, 1.36	20	1.06	0.65, 1.63
Cancer of the Prostate	18	1.2	0.71, 1.89	-	-	-
Cancer of the Kidney, Other and Unspecified	2	0.70	0.08, 2.52	3	1.58	0.32, 4.46
Leukaemia	2	0.42	0.05, 1.5	2	0.50	0.06, 1.81

+ Statistical significance at the $p<0.05$ level.
++ Statistical significance at the $p<0.01$ level.

TABLE 8

Observed Number of Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers for the Port Hope Harbour Area of Concern, Ontario Compared with Ontario Cancer Incidence Rates, 1986-1992

Port Hope Residents, Males and Females, All Ages (0–85+)

	Males			Females		
Cancer Site	**Observed Cases**	**SIR**	**95% Confidence Interval**	**Observed Cases**	**SIR**	**95% Confidence Interval**
All Cancers	214	0.85-	0.74, 0.97	229	0.98	0.86, 1.12
Cancer of the Pharynx				<5	>1+	1.15, 10.67
Cancer of the Trachea, Bronchus and Lung	47	0.97	0.71, 1.28	33	1.33	0.91, 1.86
Cancer of the Genitourinary Organs	60	0.76	0.58, 0.98	29	0.70	0.47, 1.00

Port Hope Residents, Males and Females, Ages 0–24.

	Males			Females		
Cancer Site	**Observed Cases**	**SIR**	**95% Confidence Interval**	**Observed Cases**	**SIR**	**95% Confidence Interval**
All Childhood Cancers	5	1.27	0.41, 2.92	<5	<1	

+ Statistical significance high at the p<0.05 level.
- Statistical significance low at the p<0.05 level.

6.2.7 1971–1996 – Cancer Incidence in Port Hope (79)

In 2000, Health Canada prepared a report on cancer incidence in Port Hope for the CNSC. The study compared Port Hope's cancer incidence with that of the general population of Ontario and four other municipalities in the same region (Cobourg, Lindsay, Belleville and Orillia) from 1971 to 1996. Residence was based on the Ministry of Health (MOH) code and postal code. Since postal code information was not available for the whole study period, this summary is based on MOH code, unless otherwise specified. Cancer incidence data routinely collected by the Ontario Cancer Registry (OCR) were obtained and summarized for the Town of Port Hope and for the four municipalities. A second objective was to describe cancer incidence by residence for areas of the town reported to have had higher radiation exposures before remedial work (1967-1976) than others and some areas reported to have higher soil levels of metals and radionuclides (60).

Comparisons with the four other municipalities found no cancer trends to suggest cancer incidence was unusual within Port Hope. For example, from 1986 to 1996 for selected types of cancer (all ages and sexes combined), the SIR was significantly elevated for all cancers in Orillia, lung cancer and leukaemia in Belleville, and childhood leukaemia in Lindsay.

Overall, the incidence of all cancers in Port Hope was comparable with the general Ontario population (see Table 9). A total of 1,208 cancers were diagnosed in Port Hope from 1971 to 1996, compared with 1,173 expected. The most common cancers were cancer of the lung, colon and rectum, breast, and prostate, which was consistent with the pattern of leading cancer incidence in Canada and Ontario (68, 80).

TABLE 9

Observed Number of Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers for Port Hope, Ontario Compared with Ontario Cancer Incidence Rates, 1971–1996

Port Hope Residents, Both Sexes Combined, All Ages (0–85+)

Cancer Site	Observed Cases	SIR	SIR Flag	95% Confidence Interval
All Cancers	1208	1.03		0.97, 1.09
Lip	12	1.39		0.72, 2.42
Tongue, gum and mouth	14	1.01		0.56, 1.7
Pharynx	N/A	<1.00		
Cancer of the esophagus	N/A	>1.00		
Stomach	33	0.89		0.61, 1.25
Colon and rectum	188	1.07		0.93, 1.24
Gallbladder	N/A	<1.00		
Pancreas	34	1.05		0.73, 1.46
Nose/sinuses	N/A	>1.00	+	
Larynx	N/A	<1.00		
Trachea, bronchus and lung	195	1.17	+	1.01, 1.34
Bone	5	1.38		0.45, 3.18
Connective tissue	N/A	>1.00		
Malignant melanoma of skin	29	1.04		0.7, 1.5
Breast	169	1.08		0.92, 1.25
Cervix uteri	9	0.48	-	0.22, 0.91
Uterus excluding cervix	39	1.05		0.75, 1.43
Ovary	17	0.69		0.4, 1.1
Other female genital organs	7	1.17		0.47, 2.39
Prostate	106	0.94		0.77, 1.13
Bladder	N/A	<1.00		
Kidney	33	1.17		0.81, 1.65
Brain and other nervous system	N/A	>1.00		
Thyroid	6	0.52		0.19, 1.12
Ill-defined and unknown sites	53	1.24		0.93, 1.63
Hodgkin's disease	7	0.86		0.35, 1.76
Non-Hodgkin's lymphoma	31	0.76		0.52, 1.09
Multiple myeloma	N/A	<1.00		
Leukaemia	34	0.91		0.63, 1.27

+ Statistical significance high at the $p<0.05$ level.
- Statistical significance low at the $p<0.05$ level.

Standardized Incidence Ratios (SIRs), 1971 to 1996, Standard Population Canada 1991
-area Port Hope T/CA, based on Ministry of Health (MOH) residence code

NA indicates a total suppressed because a component is less than 5

Health Canada. *Cancer Incidence in Port Hope 1971–1996*. CNSC, INFO 0716. August 2000.

There was a statistically significant excess of lung cancer (trachea, bronchus and lung) in Port Hope compared with the general Ontario population from 1971 to 1996. This was dominated by a statistically significant excess of lung cancer among women from 1986 to 1996 (Table 10). There was no excess lung cancer from 1971 to 1985 or in men. From 1971 to 1996, cancers of the nose and sinuses were significantly high in Port Hope. This was dominated by excess nose and sinus cancer from 1971 to 1985 among men (Table 10), based on only 5 cases. No excess was seen from 1986 to 1996 or in women.

While there were some increases and some decreases in cancer occurrence when data were divided into smaller groups by time period, sex, and residence coding (Table 10), the overall observed patterns were similar to those of the other communities in Ontario.

TABLE 10

Observed Number of Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers for Port Hope, Ontario Compared with Ontario Cancer Incidence Rates

Cancer site	Time Period	Sex	Observed Cases	SIR	95% Confidence Interval
Colon & rectum (PC)	1986-1996	F	61	1.42+	1.09, 1.82
Lung	1986-1996	F	47	1.44+	1.06, 1.91
Brain & other nervous system	1986-1996	F	11	2.21+	1.11, 3.94
Esophagus	1971-1985	M	10	2.41+	1.16, 4.41
Lip (PC)	1986-1996	M	7	2.75+	1.10, 5.60
Nose & Sinus	1971-1985	M	5	5.61++	1.81, 12.88
Pharynx	1986-1996	F	5	4.17+	1.35, 9.58
Ovary	1986-1996	F	5	0.43-	0.14, 0.99
Liver (PC)	1986-1996	M&F	<5		

+/- Statistical significance (high/low) at the $p<0.05$ level.
++/-- Statistical significance (high/low) at the $p<0.01$ level.
(PC) residence based on postal code, not MOH code.

Finally, incidence of all childhood cancers (ages 0-19) in Port Hope from 1971 to 1996 was comparable with the general Ontario population (SIR=1.41; 95% CI: 0.85, 2.19), based on 19 cases. There was no evidence of excess childhood leukaemia, a cancer very sensitive to radiation exposure (SIR=1.41; 95% CI: 0.45, 3.29), based on 5 cases. There was a statistically significant excess of childhood brain and nervous system cancer from 1971 to 1985, based on 5 cases (SIR=4.17; 95% CI: 1.35, 9.57; $p<0.05$). No excess was seen from 1986 to 1996. In Port Hope, there were 5 or fewer cases for each type of childhood cancer over the 26-year period studied, making interpretation extremely difficult because of the rarity of childhood cancers and the great instability in rates.

Increased exposure to residential radon and its decay products is known to be an important risk factor for lung cancer (16, 24, 81, 82). When areas within the town were grouped by exposure, a statistically significant trend with exposure was found for lung cancer (Table 11). However, since the radon levels reported in Port Hope were all close to the public dose limit of 1 mSv/year (range of 0.9-1.46 mSv/year), the lack of individual residential radon exposures or information on other known risk factors of lung cancer, especially tobacco smoking, makes interpretation of these findings extremely difficult (refer to section 6.1.3). The new Health Canada radon guidelines for indoor air is

200 Bq/m^3 (83). This is equivalent to approximately 3.4 mSv, thus the levels found within Port Hope were below Health Canada's recommendations. Assigning cumulative radon exposure even to groups is based on few measurements and many assumptions exist about residential occupancy and constancy of exposures over decades.

TABLE 11

Observed Lung Cancer Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals by Radiation Exposure Group[1] for Port Hope, Ontario Compared with Ontario Cancer Incidence Rates, 1986–1996

Port Hope Residents, Both Sexes Combined, All Ages (0-85+)				
Exposure Group	**Lung Cancer Cases**	**SIR**	**P-value Trend**	**95% Confidence Interval**
			<0.001	
Group 1 (0.90-1.10 mSv/y)	15	0.55-		0.31, 0.91
Group 2 (1.16-1.17 mSv/y)	22	0.98		0.62, 1.49
Group 3 (1.27-1.46 mSv/y)	55	1.48++		1.11, 1.92

[1] Based on population weighed block values from 1967-1976 from SENES (1995) Report for 1996 Enumeration Areas (60).
+/- Statistical significance (high/low) at the p<0.05 level.
++/-- Statistical significance (high/low) at the p<0.01 level.

In summary, the observed cancer pattern was similar to variations in cancer incidence seen in similar communities in Ontario. Overall, the study found no unusual cancer trends to suggest cancer incidence was unusual within Port Hope. In most cases, a statistically significant excess cancer incidence was based on very small numbers of observed and expected cases and the confidence intervals were wide. Despite that the study covered an extended period of time (1971-1996), the Port Hope population is small so the study only has sufficient power to detect large variations in cancer incidence.

6.2.8 1956–1997 – Cancer and General Mortality in Port Hope (84)

In 2002, Health Canada prepared a second report for the CNSC, comparing Port Hope mortality for all major causes of death with the general Ontario population from 1956 to 1997. The study also compared the cancer mortality results with the 1971 to 1996 cancer incidence report discussed in section 6.2.7 (79).

Overall, mortality from all causes of death in Port Hope was statistically significantly higher compared with the general Ontario population (see Table 12). A total of 4,299 deaths occurred among Port Hope residents, compared with 3,985 expected. The SMR was 1.08 (95% CI: 1.05, 1.11). The leading causes of death in Port Hope were circulatory disease, cancer and respiratory disease. This is consistent with the leading causes of death in Ontario and Canada (66, 67). The statistically significant excess mortality from circulatory disease (such as heart disease and stroke) dominated this. From 1956 to 1997, circulatory disease was the leading cause of death in Port Hope, based on 2,301 observed deaths (2,000 expected). Northumberland County (1986-1997) also had high circulatory disease mortality (SMR=1.11; 95% CI: 1.07, 1.14) so the excess deaths were not specific to Port Hope. The main risk factors for circulatory disease include high blood pressure, high cholesterol, diabetes, smoking, stress, excessive alcohol consumption, physical

inactivity and being overweight (85). Therefore, it is highly unlikely that the high circulatory disease mortality was related to the nuclear industry in the town. The town also had statistically significant high mortality from respiratory disease, mainly from pneumonia. Pneumonia is an infection of the lungs caused by bacteria, viruses, and fungi. Similar findings for circulatory diseases and pneumonia were noted in an earlier study of Port Hope (78) (see section 6.2.6), not surprisingly since the period 1986 to 1992 was also included in the present study. Mortality for diabetes and Alzheimer's disease were statistically significant lower in Port Hope, compared with the general Ontario population. For all other major causes of death, mortality in Port Hope was comparable with the general population of Ontario.

Mortality for all cancers in Port Hope was comparable with Ontario's general population (see Table 13). A total of 836 cancer deaths occurred among Port Hope residents from 1956 to 1997, compared with 845 expected (SMR=0.99; 95% CI: 0.92, 1.06). The leading cancer deaths were lung cancer, colon and rectum cancer, breast cancer and prostate cancer, which were consistent with the general Ontario population, the leading causes of cancer deaths in Canada (68) and the cancer incidence report (79) discussed in section 6.2.7.

From 1956 to 1997, mortality due to brain and nervous system cancers in Port Hope was comparable with the general Ontario population. However, statistically significant high mortality from brain and nervous system cancers in women (SMR=2.39; 95% CI: 1.03, 4.67) were seen from 1986 to 1997, based on 8 deaths compared with 3.3 expected. There was no excess in other time periods or in men. As indicated in Chapter 4, the relationship between ionizing radiation and brain and central nervous system cancers is not strong and most of the radiation-associated tumour risk occurs for tumours that are benign (16). Great caution is critical in interpreting these findings because of the rarity of this cancer, the small number of observed deaths, the wide 95% confidence intervals, and the many limits of ecological studies.

Overall, all childhood cancer mortality in Port Hope was comparable with the general Ontario population, based on a total 11 deaths compared with 7.4 expected (SMR=1.48; 95% CI: 0.74, 2.65) for the 42-year period of study. Mortality from childhood leukaemia was comparable with the general Ontario population, based on 5 deaths compared with 3.1 expected (SMR=1.63; 95% CI: 0.53, 3.81). There was no evidence of excess mortality from brain and nervous system cancers or non-Hodgkin's lymphoma in Port Hope children, each with less than five deaths over the study period. Mortality from congenital anomalies (birth defects) was comparable with the general Ontario population. In fact, the SMR for female infants in Port Hope was lower than that for Ontario. Similar findings were found in two previous studies (77, 78) (as discussed in sections 6.2.5 and 6.2.6).

TABLE 12

Observed and Expected Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Selected Causes of Death for Port Hope, Ontario Compared with Ontario Mortality Rates, 1956–1997

Port Hope Residents, Both Sexes Combined, All Ages (0–85+)

Causes of Death	Observed Deaths	Expected Deaths	SMR	SMR Flags	95% Confidence Interval
All Causes	**4,299**	**3,984.7**	**1.08**	++	**1.05, 1.11**
Infectious and parasitic diseases	22	31.8	0.69		0.43, 1.05
All cancers	836	844.6	0.99		0.92, 1.06
Diabetes	42	77.0	0.55	--	0.39, 0.74
Alzheimer's disease (senile and pre-senile)	22	37.5	0.59	--	0.37, 0.89
Circulatory disease	2,301	1,999.5	1.15	++	1.1, 1.20
Hypertensive disease (1969–1997)	38	20.9	1.82	++	1.29, 2.49
Ischemic heart disease	1,390	1,220.2	1.14	++	1.08, 1.20
Cerebrovascular disease	450	413.1	1.09		0.99, 1.19
Diseases of arteries, arterioles and capillaries	199	132.9	1.50	++	1.3, 1.72
Respiratory disease	356	294.9	1.21	++	1.09, 1.34
Influenza	13	9.1	1.43		0.76, 2.43
Pneumonia	202	150.8	1.34	++	1.16, 1.54
Chronic obstructive pulmonary disease	110	103.7	1.06		0.87, 1.28
Ulcers	12	18.6	0.65		0.33, 1.12
Cirrhosis of liver	51	42.1	1.20		0.9, 1.59
Kidney diseases	42	44.1	0.95		0.69, 1.29
Congenital anomalies	26	29.6	0.88		0.57, 1.29
Perinatal mortality	51	48.3	1.06		0.79, 1.39
Accidents/poisonings/violence	232	237.8	0.98		0.85, 1.11
Motor vehicle traffic accidents	70	67.0	1.04		0.81, 1.32
Accidental falls	58	53.0	1.09		0.83, 1.41
Fires	6	9.4	0.64		0.23, 1.37
Suicide	38	45.0	0.85		0.6, 1.16
Homicide	6	6.3	0.95		0.35, 2.04

++ /-- Statistical significance high/low at the $p<0.01$ level.

Deaths for 1966–1997 presented to avoid residual disclosure of a table cell less than 5.
N/A indicates total suppressed because a component is less than 5.

Health Canada. *Cancer and General Mortality in Port Hope, 1956–1997.* Prepared for the Canadian Nuclear Safety Commission, INFO-0734. June 2002.

TABLE 13

Observed and Expected Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Cancer Deaths for Port Hope, Ontario Compared with Ontario Mortality Rates, 1956–1997

Port Hope Residents, Both Sexes Combined, All Ages (0–85+)

Cancer Site	Observed Deaths	Expected Deaths	SMR	95% Confidence Interval
All Cancers	836	844.6	0.99	0.92, 1.06
Tongue, Gum, Mouth, Pharynx (excluding Nasopharynx)	N/A		<1.00	
Cancer of the Esophagus	23	17	1.35	0.86, 2.03
Stomach	35	48.6	0.72	0.5, 1.00
Colon And rectum	136	132.8	1.02	0.86, 1.21
Gallbladder	N/A		<1.00	
Pancreas	40	43.7	0.91	0.65, 1.25
Larynx	N/A		<1.00	
Trachea, bronchus and lung (1966–1977)[1]	158	154.2	1.02	0.87, 1.20
Malignant melanoma of skin	N/A		>1.00	
Breast[2]	80	80.3	1.00	0.79, 1.24
Cervix uteri	6	12.8	0.47	0.17, 1.01
Uterus excluding cervix	16	11.5	1.39	0.79, 2.25
Ovary	19	24.1	0.79	0.48, 1.23
Prostate	54	47.3	1.14	0.86, 1.49
Bladder	27	24.3	1.11	0.73, 1.62
Kidney	19	15.7	1.21	0.73, 1.88
Brain and other nervous system	24	19.5	1.23	0.79, 1.83
Ill-defined and unknown sites	48	41.6	1.15	0.85, 1.53
Non-Hodgkin's lymphoma	27	25.5	1.06	0.70, 1.54
Multiple myeloma	8	13.2	0.61	0.26, 1.19
Leukaemia	24	32.7	0.73	0.47, 1.09

[1]Deaths for 1966–1997 presented to avoid residual disclosure of a table cell less than 5.
N/A indicates total suppressed because a component is less than 5.
[2] based on female breast cancer deaths only.

Health Canada. *Cancer and General Mortality in Port Hope, 1956–1997*. Prepared for the Canadian Nuclear Safety Commission, INFO-0734. June 2002.

6.2.9 1986-2004 Haliburton, Kawartha, Pine Ridge (HKPR) District Health Unit, Cancer in the HKPR District (86)

The Haliburton, Kawartha, Pine Ridge (HKPR) District Health Unit (which includes Port Hope) routinely monitors the prevalence of known risk factors and the health status of residents within the health district. Existing provincial cancer incidence, mortality and risk factor databases are used for disease and risk factor surveillance and health planning. This area includes Northumberland and Haliburton Counties, and the City of Kawartha Lakes.

The health unit recently published a report on cancer in the HKPR District (86). This report focuses on cancers occurring in the area that have modifiable risk factors, including eating too few fruits and vegetables, a sedentary lifestyle, drinking too much alcohol, tobacco use, and exposure to ultraviolet (UV) radiation. According to the 2006 Rapid Risk Factor Surveillance System (RRFSS) for HKPR District (87), 21.1% adults (aged 18+) consumed fruits and vegetables less than three times daily, 35.2 % had low or inactive physical activity levels, and 22.1% were daily smokers.

In the HKPR District, (1986-2004) incidence of all cancers combined was higher for males than for females, which was consistent with the trend observed for Ontario. Among males, the three most commonly diagnosed cancers were prostate cancer, followed by lung cancer and colorectal cancer. Among females, the three most commonly diagnosed cancers were breast cancer, followed by lung cancer and colorectal cancer. This was consistent with, the three leading cancer sites for each sex in 2004 in Ontario and Canada (68, 80).

In Ontario, the breast cancer incidence increased from the 1980s and has remained stable since the early 1990s. The breast cancer incidence in HKPR District has followed a similar pattern and is not statistically different relative to Ontario (Table 10). The incidence of colorectal cancer in the HKPR District decreased from the mid-1980s to the mid 1990s, following the same pattern observed for Ontario. For females, the colorectal cancer incidence was significantly greater in the HKRP District relative to Ontario. In the HKPR district, the incidence of lung cancer was increasing among women while remaining steady among men. This was also apparent in Ontario. Lung cancer incidence among both males and females was significantly greater in the HKPR District compared with Ontario. Higher lung cancer rates relative to Ontario are likely attributed to historically higher smoking rates among HKPR District residents. The most common cause of lung cancer is tobacco smoking, accounting for approximately 90% of all lung cancers (88). Oral cancer includes cancers of the lip, tongue, salivary glands, gum, floor of the mouth and pharynx. The main risk factor associated with oral cancer is tobacco use (89). Oral cancer incidence is significantly greater in the HKPR district relative to Ontario for males. The incidence rates of melanoma, a skin cancer caused by UV radiation, have been increasing in Canada and the HKRP District rates show a similar increase over time. For both sexes combined, the incidence of melanoma is significantly higher in the HKPR District relative to Ontario.

The HKPR District Health Unit is not specific enough to draw conclusions for Port Hope. However, the report indicates that high rates of lung and oral cancers, colorectal cancer and melanoma as well as high prevalence of modifiable risk factors are an important health concern within the HKPR District Health Unit, which includes Port Hope.

TABLE 14

Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers for HKPR District, Ontario Compared with Ontario Cancer Incidence Rates, 1986-2004

HKPR District, by Sex, All Ages

	Males		Females	
Cancer Site	**SIR**	**95% Confidence Interval**	**SIR**	**95% Confidence Interval**
Prostate	0.94	0.87, 1.01		
Oral Cancer[1]	1.54	1.26, 1.82	0.55	0.31, 0.79
Lung	1.15	1.05, 1.26	1.37	1.23, 1.50
Colorectal Cancer	0.98	0.88, 1.08	1.15	1.03, 1.27
Female Breast Cancer			0.96	0.89, 1.04
Melanoma (both sexes combined)	1.27	1.09, 1.45		

[1] including cancer of the lip, tongue, salivary glands, gum, floor of mouth and pharynx

6.3 Case Control Studies in Port Hope

Two case-control studies were conducted in Port Hope. The first assessed the likelihood of developing lung cancer as a result of residential radon exposure (90, 91). The second assessed the likelihood of offspring developing childhood leukaemia as a result of a father's occupational radiation exposure (92, 93).

6.3.1 1980 – A Case-control Study of Lung Cancer Relative to Domestic Radon Exposure (90, 91)

In 1980, researchers at Queen's University conducted a case-control study of lung cancer in Port Hope relative to residential radiation exposure. Approximately 550 homes in Port Hope were identified as a "problem home" (annual level over 0.229 Working Level Months (WLM) which is approximately 1.15 mSv/year) based on dose reconstruction data conducted in 1976 for remedial action (60). The study investigated the relationship between lung cancer and residential radon exposure in homes as a result of disposal of radioactive waste in Port Hope.

Twenty-seven lung cancer cases, obtained from the Ontario Cancer Registry (OCR), were identified between 1969 and 1979. Cases lived for at least seven years in Port Hope prior to diagnosis (to account for the latency period between radiation exposure and cancer diagnosis). These were compared with forty-nine control individuals without lung cancer, matched on age, sex and duration of residence in Port Hope. Case and control individuals (or proxies) were interviewed to obtain information on residence, smoking and drinking habits and employment history. Residential radon exposures were estimated for each home occupied.

When the analysis was separated into "lived in a problem home" and "did not live in a problem home", a strong confounding factor was observed between exposure and smoking. The four cases exposed to radon (i.e., living in a problem home) were smokers and the two controls exposed to radon (i.e., living in a problem home) were not. When smoking was controlled for, a marginally significant (p=0.050) positive association was observed between exposure in a problem home and lung cancer. After adjusting for smoking, the odds ratio (OR) was 6.81 with 95% confidence interval of 0.51-90.6. The excessively large confidence interval was due to the large variance of the estimated odds ratio caused by the extreme confounding between smoking and radon exposure.

The small numbers of subjects together with the low levels of cumulative radiation exposure experienced by the residents made it impossible to draw unambiguous, clear cut conclusions. Regarding exposure resulting from radioactive contamination, the statistical analyses could not find conclusive results and the research team did not feel that they provided evidence of an identifiable, increased risk of lung cancer from elevated alpha radiation levels in some Port Hope homes. Statistical analysis showed a very strong association between cigarette smoking and lung cancer in the study with 90% of the cases attributable to smoking. It was not possible to distinguish the roles of smoking and radon gas in causing lung cancer in this study; however, in studies of uranium miners the interaction between smoking and radon exposure on risk of lung cancer was intermediate between multiplicative and additive (26). Finally, this study had a limited statistical power of 0.28 to detect a relative risk as large as 2.0 because of the small number of cases.

6.3.2 1992 – Paternal Radiation Exposure and Leukaemia in offspring: the Ontario Case-Control Study (92, 93)

In 1992, researchers at the Ontario Cancer Treatment and Research Foundation (OCTRF) and the University of Toronto conducted a case-control study to determine if there was an association between childhood leukaemia in offspring and the father's occupational exposure to ionizing radiation before a child's conception.

Cases were children (aged 0–14 years) that died from or were diagnosed with leukaemia between 1950 and 1988 and who were born to mothers who, at the time of the child's birth, lived in the vicinity of an operating Ontario nuclear facility. Eight control children (without leukaemia) per leukaemia case were identified from birth certificates and matched by date of birth and mother's place of residence at birth. There were 112 cases and 890 controls, including children of fathers employed at the PHCF. Father's individual preconception radiation exposures (including external whole body dose of gamma radiation, internal radon exposure) for cases and controls were obtained from the Canadian National Dose Registry and analysis of employer records.

The study found no evidence of an association between childhood leukaemia in offspring and the fathers' exposure to ionizing radiation before a child's conception. There was no statistically significant association between childhood leukaemia and the father's occupational ionizing radiation exposures occurring prior to a child's conception.

Specifically, there was no evidence of an elevated leukaemia risk in relation to any exposure period (lifetime, six months or three months prior to conception) or exposure type (total external whole body dose of gamma radiation or radon exposure). The odds ratio for any lifetime exposure of the father prior to conception was 0.87 (95% CI: 0.32, 2.34) based on 6 cases and 53 controls. The odds ratio for whole body exposure of the father during the six-months prior to conception was 0.96 (95% CI: 0.34, 1.77) based on 5 cases and 41 controls. There was also no apparent increase in effect with increasing paternal radiation dose.

The results did not demonstrate an association between childhood leukaemia and occupational exposures of fathers before the child's conception. The case-control design and the existence of the dose and cancer registries made it possible to study the relationship between a rare disease and rare exposure over a forty-year period. However, the study lacked information on other known risk factors of leukaemia. Despite the study having covered a long period of time and referring to a large population, it had sufficient power to detect only large relative risks. These findings were consistent with a recent large case-control study, as part of the United Kingdom Childhood Cancer Study that did not find any evidence of a relationship between parental occupational exposures to ionizing radiation and childhood leukaemia (94).

6.4 Cohort Studies of Radium and Uranium Workers in Port Hope

The "Eldorado Study" is a cohort study of Eldorado Nuclear Ltd. employees who worked anytime between 1932 and 1980. This included underground uranium miners and surface workers at the Beaverlodge mine in Northern Saskatchewan and the Port Radium mine in the Northwest Territories, the radium and uranium refining and processing facility workers at Port Hope, Ontario, as well as workers at "other sites" (such as aviation, research and development, exploration, and head office). The whole original Eldorado cohort included 18,424 male employees with employees' mortality experience was followed up from 1950 to 1980 (95). The Beaverlodge and Port Radium miners cohorts studies were previously published (31, 32, 33) and are internationally recognized for their role in the understanding of the relationship between radon progeny exposure and lung cancer risk (16, 24. 26, 29, 96).

The original (95) and recently updated Eldorado study (97) provides follow-up data on approximately 50 years of mortality and 30 years of cancer incidence for Port Hope's radium and uranium refining and processing facility workers. The main objective of the study was to assess the relationship between occupational radiation exposures and lung cancer. However, all causes of death and all cancer incidences were assessed.

6.4.1 1950–1980 – Mortality in the Original Eldorado Study (95)

The original Eldorado cohort was composed of the original Beaverlodge cohort (31, 32), the original Port Radium cohort (33), a third cohort of 1,831 Port Hope radium and uranium refinery and processing facility workers (95), and workers at "other sites". Detailed information was collected on each employee, including worker histories and

radon exposures from company records. Each individual's records were linked to the Canadian mortality database from 1950 to 1980 to determine cause of death.

Overall, Port Hope workers (see Table 15) showed an overall "healthy worker effect" (98) with lower mortality rates for all causes of death (SMR=0.84, $p<0.05$). Mortality for all cancers (SMR=0.84) and lung cancer (SMR=0.82) were comparable with the general male population of Canada. Mortality from lymphoma, leukaemia and cancers of the lung, kidney, liver and bone, skin, bladder, and prostate were also comparable with the general male Canadian population. The only cancer death, which showed statistically significant excess, was cancer of the rectum, based on 7 deaths. There is little or no information on radiation-related risk of rectal cancer at doses less than about 1 Gy (16). The small number of observed deaths limits interpretation of these findings.

The relationship between radon exposure and lung cancer mortality was not assessed for Port Hope workers, as it was for their uranium miner counterparts at the Beaverlodge and Port Radium mines (31, 32, 33) because there were too few lung cancer deaths among Port Hope workers (14 observed deaths) for any meaningful analysis.

TABLE 15

Observed and Expected Number of Deaths and Standardized Mortality Ratios (SMR) for Selected Causes of Death Compared with Canadian Mortality Rates, 1950–1980

Port Hope Eldorado Employees, Males

Causes of Death	Observed Deaths	Expected Deaths	SMR	SMR Flag
All Causes	249	295.71	0.84	-
All cancers	59	70.96	0.83	
Stomach	5	5.87	0.85	
Rectum	7	2.61	2.68	+
Trachea, bronchus, lung	14	17.16	0.82	
Pancreas	5	3.48	1.44	
Lymphatic and hematopoietic	9	15.53	0.58	
Hodgkin's disease	7	10.87	0.64	
Other malignant neoplasms	6	4.73	1.27	
Ischaemic heart disease	98	98.63	0.99	
Other heart disease	5	9.93	0.5	
Cerebrovascular disease	14	18.61	0.75	
Cirrhosis	8	6.42	1.25	
External causes	28	33.70	0.83	
Motor vehicle accidents	12	11.34	1.06	
Other external	7	12.96	0.54	
All other causes	20	28.41	0.7	

+/- Statistical significance (low/high) at the 5% level.

C. Nair, J. D. Abbatt, G. R. Howe, H. B. Newcombe, S. E. Frost. *Mortality experience among workers in the uranium industry*. In *Occupational Radiation Safety in Mining*, Toronto, Canadian Nuclear Association Proceedings of the International Conference, Vol. 1, 354-364 (1984).

6.4.2 1950–1999 – Mortality and Cancer Incidence (1969–1999) in the Updated Eldorado Uranium Workers Cohort Study (97)

A recent update of the original Eldorado study (95) evaluated the relationship between workers radiation exposures and lung cancer mortality and cancer incidence. This study included workers from the Port Radium and Beaverlodge mines and the Port Hope radium and uranium refinery and processing facility and "other sites". Further detailed information was collected on a total of 3,003 Port Hope radium and uranium processing workers, including those in the original study (95). Updated radiation exposures (radon progeny exposure, gamma dose) to 1999 were collected from individual company records and the National Dose Registry. Each worker's records were linked to the Canadian national mortality (1950 to 1999) and cancer incidence (1969 to 1999) databases, providing death and cancer follow-up for 50 and 30 years, respectively.

Several analyses were conducted including a) a comparison of mortality and cancer incidence with the general Canadian male population; b) an internal dose-response analysis of the relationship between lung cancer and radon progeny exposure, and c) an internal analysis of the relationship between all causes of death and all cancers related to radon decay product (progeny) exposure and gamma ray dose.

Port Hope workers had the highest average cumulative gamma ray doses (101.5 mSv; SD=257.3 mSv)) and the lowest average cumulative radon progeny exposures measured in Working Level Months (10.4 WLM[1], SD=43.0 WLM; approximately 52 mSv, SD=215 mSv). Port Hope workers were also exposed to relatively concentrated forms of uranium with greater solubility than that found in uranium ore.

As illustrated in Table 16, Port Hope workers mortality for all causes of death, and all cancers were comparable with the general Canadian male population. No statistically significant increase in cancers of the lung, kidney, liver, bone, skin, bladder, prostate, lymphoma, leukaemia, non-malignant respiratory disease, renal disease or liver disease was found. There was a slight excess of cancer of the rectum, similar to workers in the original study, however this was no longer statistically significant.

Port Hope workers had higher mortality than expected for high blood pressure (hypertensive disease). Hypertensive disease was elevated, despite that ischaemic heart disease, stroke and other cardiovascular disease were not. A more detailed examination was done of Port Hope workers' death certificates for hypertensive disease. No autopsies had been performed on any of these individuals. One case was clearly a coding error when the data from the death certificate was entered into the mortality database. In several cases, hypertension was mentioned among as many as five other causes of death listed, including diabetes and stroke. For at least two of the cases, it was reasonable that hypertension could equally well have been coded as diabetes or stroke. In most of the remaining cases there was insufficient information to clearly implicate anything else. The net effect of this examination was to reduce the number of deaths from hypertensive disease by as much as half, eliminating the statistical significance of the elevated SMR.

[1] 1 WLM equals approximately 5 mSv.

Table 17 illustrates that the incidence of all cancers among Port Hope workers was also less than that expected compared with the Canadian male population. There was no statistically significant departure from 1.0 and in general, Port Hope workers had cancer incidence very similar compared with the general male population.

A relationship between lung cancer and radon progeny exposure was not found in Port Hope workers (Table 18), as there was for their uranium-mining counterparts. The excess relative risk (ERR) estimate for lung cancer mortality was 0.18 (95% CI: -0.10, 1.49, p=0.59) and the ERR for lung cancer incidence was 0.68 (95% CI: -0.23, 3.07, p=0.173). Port Hope workers' average cumulative radon progeny exposures were very low (10.4 WLM or 52 mSv). Radon progeny exposures were not related to any other cause of death or cancer incidence.

Although Port Hope workers' average cumulative gamma ray doses (101.5 mSv) were the highest among Eldorado workers, there was no evidence of a relationship between occupational gamma ray dose and increased risk of leukaemia, any other cancers, or any other causes of death. Clearly, there was no evidence of increased risks between radium and uranium refining processing facility workers' occupational exposures and lung cancer, leukaemia, other cancers and other causes of death.

TABLE 16

Observed and Expected Number of Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals for Selected Causes of Death in Port Hope Eldorado Male Employees Compared with Canadian Male Mortality Rates, 1950–1999

Causes of Death	Observed Deaths	Expected Deaths	SMR[1]	95% Confidence Interval	SMR Flag
All causes	1104	1077.5	1.0	1.0, 1.1	
All cancers	272	283.8	1.0	0.8, 1.1	
Stomach cancer	14	18.4	0.8	0.4, 1.3	
Colon cancer	22	26.9	0.8	0.5, 1.2	
Rectal cancer	16	9.4	1.7	1.0, 2.8	
Pancreatic cancer	11	14.7	0.7	0.4, 1.3	
Lung cancer	101	92	1.1	0.9, 1.3	
Prostate cancer	21	25.9	0.8	0.5, 1.2	
Bladder and other urinary cancer	11	8.3	1.3	0.7, 2.4	
Diabetes mellitus	14	19.5	0.7	0.4, 1.2	
All nervous system diseases	12	17.6	0.7	0.4, 1.2	
Hypertensive disease	13	4.9	2.7	1.4, 4.5	++
Ischemic heart disease	345	324.2	1.1	1.0, 1.2	
Stroke	72	69.3	1.0	0.8, 1.3	
All other cardiovascular disease	85	79.4	1.1	0.9, 1.3	
Chronic obstructive lung disease	25	43.4	0.6	0.4, 0.9	--
Pneumonia	29	26.8	1.1	0.7, 1.6	
All digestive diseases	41	43.9	0.9	0.7, 1.3	
Genitourinary diseases	19	16	1.2	0.7, 1.9	
Motor vehicle accidents	24	23.2	1.0	0.7, 1.5	
Suicide	18	20.2	0.9	0.5, 1.4	
Other external causes	28	38.4	0.7	0.5, 1.1	

[1] Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals).
-/+ Statistical significance low/high at $p<0.05$ level.
--/++ Statistical significance low/high at $p<0.01$ level.

G.R. Howe. *Eldorado Nuclear Epidemiology Study Update - Eldorado Uranium Miners' Cohort: Part I of the Saskatchewan Uranium Miners' Cohort Study.* Canadian Nuclear Safety Commission. RSP-0205 (2006).

TABLE 17

Observed and Expected Number of Cases, Standardized Incidence Ratios (SIR) and 95% Confidence Intervals for Selected Cancers in Port Hope Eldorado Male Employees Compared with Canadian Male Cancer Incidence Rates, 1969–1999

Type of Cancer	Observed Cases	Expected Cases	SIR[1]	95% Confidence Interval	SIR Flag
All cancers	426	455.3	0.9	0.8, 1.0	
Stomach cancer	13	17.5	0.7	0.4, 1.3	
Colon cancer	34	39.9	0.9	0.6, 1.2	
Rectal cancer	23	24	1.0	0.6, 1.4	
Pancreatic cancer	10	12.4	0.8	0.4, 1.5	
Laryngeal cancer	11	9.6	1.1	0.6, 2.1	
Lung cancer	110	100.3	1.1	0.9, 1.3	
Malignant melanoma	11	8.3	1.3	0.7, 2.4	
Prostate cancer	91	94.7	1.0	0.8, 1.2	
Bladder and other urinary cancer	27	29.7	0.9	0.6, 1.3	
Non-Hodgkin's lymphoma	15	15.4	1.0	0.5, 1.6	
Leukaemia	10	12.2	0.8	0.4, 1.5	

[1] Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals).
-/+ Statistical significance low/high at $p<0.05$ level.
--/++ Statistical significance low/high at $p<0.01$ level.

G. R. Howe. *Eldorado Nuclear Epidemiology Study Update - Eldorado Uranium Miners' Cohort: Part I of the Saskatchewan Uranium Miners' Cohort Study*. Canadian Nuclear Safety Commission. RSP-0205 (2006).

TABLE 18

Excess Relative Risk Estimates for Radon Decay Products (per 100 WLMs) for Lung Cancer Mortality and Incidence in Port Hope Eldorado Male Employees, 1950-1999[1]

Lung Cancer	Observed	ERR Estimate	95% Confidence Interval	X^2 (1)	p-value
Mortality (1950-1999)	101	0.18	-0.10, 1.49	0.29	0.59
Incidence (1969-1999)	110	0.68	-0.23, 3.07	1.85	0.17

[1] All models adjusted for age at risk (five-year intervals), calendar year at risk (five-year intervals) and duration of employment (< than six months vs. > than six months) stratification.

G. R. Howe. *Eldorado Nuclear Epidemiology Study Update - Eldorado Uranium Miners' Cohort: Part I of the Saskatchewan Uranium Miners' Cohort Study*. Canadian Nuclear Safety Commission. RSP-0205 (2006).

6.5 International Scientific Understanding

The 2006 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) summarizes the international understanding of the health effects from exposure of workers and the public to uranium (16). A review of the findings from epidemiological studies of non-mining uranium workers was carefully evaluated and provided no evidence of increased cancer risks or uranium-related disease among occupationally exposed workers (9, 10, 11, 12, 99, 100, 101). Fourteen epidemiological studies of more than 120,000 uranium workers at uranium processing, enrichment, metal fabrication and milling facilities did not find the rate of any cancer to be significantly increased. The total risk for all cancers taken together was close to that expected (11). There was reasonable consistency among the findings for the 14 epidemiological studies of workers employed throughout the world (11). These results were consistent with a large-scale case-control study of uranium dust exposure (primarily insoluble uranium compounds) and lung cancer mortality among workers at four uranium-processing operations located in Missouri, Ohio, and Tennessee; that is, no association between increasing lung cancer risk and increasing dose was found among 787 lung cancer cases with at least 30 years of potential follow-up (102). Further analyses for cumulative external doses and exposures to thorium, radium, and radon did not reveal any clear association between exposure and increased risk (102). Similarly two recent cohort studies of uranium processing workers reveal no significant increases in leukaemia or other cancer risks (103, 104), although the number of people studied was small.

A study of workers in Colorado Plateau uranium mills reported increased numbers of deaths from non-malignant-respiratory diseases, lung cancer, lymphoma, and kidney disease. However, the authors were unable to determine if these deaths resulted from working in the mills (15). Most recently, a cohort study among uranium mill workers with follow-up up to 50 years found no statistically significant elevation in any cause of death in the 904 non-miners employed at the Grants uranium mill, New Mexico (105). Among 718 mill workers with the greatest potential exposure to uranium ore, no statistically significant increase in any cause of death from cancers of the lung, kidney, liver or bone, lymphoma, non-malignant respiratory disease, renal disease or liver disease was seen. Thus, there was no clear evidence of uranium related disease for uranium mill workers exposed to uranium dusts and mill products (105).

Descriptive ecological studies of populations residing near uranium milling, mining or processing facilities, (27, 28, 101, 106, 107), found no excess cancers. Studies of populations exposed to increased concentrations of uranium and other radionuclides in drinking water found no association with any cancers or overt kidney disease (108, 109, 110, 111, 112).

There are several explanations as to why uranium is not conclusively found to cause cancer in humans and why it is not considered a human carcinogen (9, 10). As indicated in Chapter 4, uranium is not very radioactive and its chemical properties are such that any ingested insoluble uranium is excreted rather quickly from the body (primarily as feces) without being absorbed (99). The mechanisms by which uranium affects the kidneys are well understood (6, 13, 113).There is little or no epidemiological evidence for an association between uranium and any cancer.

Overall, epidemiological studies assessing uranium risk have not found the rate of any cancer to be significantly increased by exposure to uranium (9, 11, 12, 16, 99, 105).

7.0 DISCUSSION

This document describes the history of the radium and uranium refining and processing industry in Port Hope since 1932. It discusses the main sources of ionizing radiation to humans: 60% of ionizing radiation comes from natural sources, 40% comes from medical sources and less than 1% comes from the nuclear industry. The main past and present sources and levels of ionizing radiation, uranium and various other non-radiological contaminants within the town of Port Hope are also presented. This document discusses the most plausible health effects known to be associated with these occupational and environmental exposures. Finally, it describes the many environmental and epidemiological studies that have looked extensively at the sources and levels of contaminants within the town and the health status of the residents and workers of Port Hope, and assessed the relationship between radiation exposure and adverse health outcomes. To put this into context, the findings in Port Hope were compared with the findings of other similar communities living near uranium processing facilities and other workers exposed to these occupational exposures in other countries.

In 1965, Sir Bradford-Hill (114) established the following nine criteria for causation (Does factor A cause disorder B?). These criteria can be used in Port Hope: Strength of association, the consistency of the association, specificity, temporal relationship, biological gradient (dose-response), biological plausibility, coherence, experimental evidence, and reasoning by analogy. It is clear from the scientific understanding that most of the contaminants found in Port Hope can cause harm to human health at high doses, and based on Hill's criteria, a causal relationship exists between the contaminants and disease. Based on the experimental and epidemiological literature the most plausible health effects of the radium and uranium refining and processing industry include cancers of the lung and bone, and kidney disease. However, uranium has not been found to cause kidney disease in humans (9, 10, 11, 12, 99, 100) and radium has a threshold of 10 Sv for bone cancer (16). All other types of cancer and other diseases are not plausible in Port Hope residents because the environmental gamma ray doses, arsenic, ammonia, fluoride and other contaminant concentrations are very low and health effects are only found at much higher levels (16, 24, 30, 45, 46, 47, 48).

Radiation dose limits, benchmarks and guidelines for uranium and other contaminants have been put in place by government agencies to protect human health from the contaminants in Port Hope. These protective levels are generally based on exposure levels that do not result in adverse health effects and are conservative, to maximize human protection. Likewise, it is clear that the actual levels of exposure in Port Hope are well within or well below the levels that are protective of human health.

Many environmental studies performed in Port Hope have not identified any significant adverse environmental effects associated with radioactive and the other environmental contamination in Port Hope. Uranium concentrations in air observed during 1988-1989 (range from 0.001-0.0158 $\mu g/m^3$) (57) were significantly lower than those observed during 1981-1982 (range from 0.002-0.227 $\mu g/m^3$) (56) as a result of the implementation of mitigation measures and have since remained relatively stable. The concentrations of

uranium and other contaminants currently found in soil, garden vegetation and urban street foliage are not anticipated to result in measurable adverse effects. The current and past environmental studies indicate that the levels of exposure of Port Hope residents to radioactive contaminants, ammonia, antimony, arsenic, barium, benzo(a)pyrene, beryllium, cadmium, cobalt, copper, fluoride, lead, manganese, molybdenum, nickel, nitrate, polychlorinated debenzo-furans (PCDF), silver, strontium, suspended solids, uranium, and zirconium are low and are unlikely to cause any adverse health effects.

Many epidemiological studies of the residents and workers of Port Hope have studied the health status of the community and workers over time, and have assessed the health risks from their exposures. These studies have included descriptive ecological studies, case-control studies, and cohort studies and have included the entire period of exposure (1932 to present) and have studied health outcomes in Port Hope over the time period from the 1950s to present day, a period that covers both before and after the implementation of mitigation measures that significantly reduced the levels of exposures within the town from the historic waste and current emissions. All causes of death, all cancers and birth defects have been assessed. These epidemiological studies are essential to evaluate whether the protective measures in place in Port Hope are appropriately protecting human health: put together, the evidence from these epidemiological studies suggests that no effects are likely to occur because the levels of exposures are too low to cause harm.

There was no evidence of excess cancer incidence or mortality for all cancers in Port Hope for the entire time period studied (71, 72, 76, 78, 79, 84). Although there was a statistically significant excess of lung cancer incidence in women in one time period (1986 to 1996) there was no excess in other time periods or in men. Similar findings were also found in Northumberland County and the HKPR District, which indicates that high rates of lung cancer were not specific to Port Hope. This excess lung cancer incidence is unlikely to be related to environmental exposures since there was no significant excess during the earlier time period (71, 72) when environmental exposures were highest. Even with a long latency period between exposure and disease outcome, there was ample time before the 1980s for statistically significantly high lung cancer mortality rates to emerge (no temporal relationship). No conclusive results were found linking residential radon with lung cancer in the case control study in Port Hope (90, 91) and it was not possible to distinguish the roles of smoking and radon gas in causing lung cancer in this study. Residential radon exposures in Port Hope (90, 91) were much lower than those of occupational radon exposures among the Port Hope radium and uranium refining and processing facility workers (95, 97). The Port Hope radium and uranium workers did not have excess lung cancer incidence or mortality (95, 97). Their lung cancers were not significantly associated with either their occupational radon progeny or gamma radiation exposures (97). The lack of a relationship between lung cancer and occupational exposures in these workers (no strength of association and no dose response relationship) provides another line of evidence that environmental radon levels in Port Hope are unlikely to cause lung cancer. Tobacco smoking is, of course, the primary cause of lung cancer with relative risks for current smokers being greater than 10-fold higher than that of non-smokers (115, 116, 117). It is essential to consider the potential confounding impact of smoking on risk estimates. However, without individual information on

smoking status, it is difficult to interpret the female lung cancer rates in Port Hope. None-the-less, based on the weight of evidence, the higher lung cancer rates in Port Hope are most likely attributable to historically higher smoking rates among residents, as indicated in the Rapid Risk Factor Surveillance System (87).

There was no evidence of statistically significant excess cancer incidence or mortality from breast, kidney, liver, bone, prostate, urinary bladder, skin, lymphoma or leukaemia in residents of Port Hope compared with the general population (71, 72, 73, 74, 75, 78, 79, 84). Likewise, no relationship between these types of cancer and worker's occupational radiation exposures was found (95, 97). The experimental evidence indicates that only very high exposures to the main radiological and non-radiological contaminants found in Port Hope (see Chapter 4) are related to these cancers. It was highly implausible that the very low public exposures to gamma radiation, uranium, arsenic, ammonia, fluoride and other contaminants would increase the risk of these diseases in Port Hope residents. The scientific understanding was confirmed by the lack of these cancers in Port Hope. The findings that there are no excesses of these types of cancers in Port Hope residents and workers are reassuring and indicate that exposures have been low.

All childhood cancer incidence and mortality in Port Hope was also comparable with the general population. Several studies looked at childhood leukaemia. Overall, incidence and mortality of childhood leukaemia were comparable with the general population of Ontario (73, 74, 75, 79, 84). A case-control study found no evidence that childhood leukaemia in offspring was associated with father's occupational ionizing radiation exposures (92, 93). Likewise, there was no evidence of excess birth defects in Port Hope. In fact, the mortality for female infants in Port Hope was lower than that for Ontario (77, 78, 84). This is not surprising since no hereditary effects of radiation have been observed in humans (118).

Although there were some increases in some cancers when findings were broken down by age group, sex and time period, and residence coding (such as cancers of the colon and rectum, brain and other nervous system cancer, esophagus, lip, pharynx, nose/sinuses), it was unlikely these cancers were related to the nuclear industry within the town, because of their lack of biological plausibility and the lack of experimental evidence linking them to Port Hope contaminants (see Chapter 4). They were more likely due to the natural variation in the occurrence of disease. The small number of observed and expected cases and deaths for most of these cancers, and the wide confidence intervals makes any interpretation of findings uncertain. The available evidence indicates that colon cancer is inducible by whole body gamma ray radiation; however, there is little or no information on radiation-related risk of rectal cancer at gamm ray doses less than about 1 Gy (equivalent to about 1 Sv) (16, 38, 61). Ionizing radiation can induce tumours of the brain and central nervous system although more data is needed to better understand the dose response relationship. The relationship is not as strong as for several other tumours, for example breast or leukaemia, and most of the radiation-associated tumour risk occurs for tumors that are benign (16). Cancers of the esophagus, lip, pharynx, nose/sinuses are most likely linked to tobacco smoking (87, 88, 89), as is lung cancer. These types of

cancer (except lung cancer) are not known to be associated with exposures to the environmental contaminants found in Port Hope (see Chapter 4.) and not surprisingly, no relationship between these cancers and worker's occupational radiation exposures were found (95, 97).

The excess overall mortality for all causes in Port Hope from 1956 to 1997 was dominated by the excess of circulatory disease within the town, which represented over 50% of all deaths in Port Hope (84). Circulatory system disease is the leading cause of death in Ontario and Canada (66, 67). The scientific data are not at present sufficient to conclude that there is a causal relationship between exposure to ionizing radiation and the incidence of cardiovascular disease for doses of less than about 1-2 Gy (about 1-2 Sv) (16). Consequently, given the level of radiation exposure in Port Hope residents, ranging from 0.9-1.46 mSv/year from 1967-1976 (60); and 0.004 to 0.064 mSv/year in 2007 (35, 36), it is highly unlikely that this disease is associated with radiation in the town and most likely reflects other risk factors of this disease, such as smoking, obesity, physical inactivity, high blood pressure, diabetes, stress and alcohol consumption (85). Excess circulatory disease was also found for all Northumberland County so these findings are not specific to Port Hope. Similarly, the Rapid Risk Factor Surveillance System (RRFSS) suggests that the overall community (HKPR District) has a high prevalence of important risk factors for circulatory disease, including poor diet, physical inactivity and high rates of cigarette smoking (87). The Eldorado radium and uranium workers' overall mortality for all causes of death was comparable with the general male population of Canada. Although they did have a statistically significant excess of hypertensive disease, it was based on only 13 deaths. More detailed examination of these deaths indicated errors in coding of death certificates. None-the-less, heart disease, stroke and other cardiovascular disease were not found to be in excess among workers compared with the general male population of Canada and no relationship between workers' radiation exposures and risk of circulatory disease was found. There was no evidence of excess kidney disease in Port Hope residents or workers.

The many Port Hope epidemiology studies provided no evidence that the nuclear industry in Port Hope has caused any adverse health effects to residents or nuclear workers in the town over the time period the nuclear industry has been in operation in Port Hope. The most plausible health effects were not in excess in Port Hope. No relationships between exposure and disease were found in the two case-control studies, or the two cohort studies of Port Hope workers. This was because the exposures were too low to result in any adverse health effects. Likewise, environmental studies consistently showed that the levels of radiation and various metals contaminants within the town are too low to expect increases in cancer or other health effects. The findings from these two lines of evidence are consistent with each other. These findings are consistent with the scientific understanding of the effects of radiation and uranium toxicity on human health and are consistent with other epidemiological studies examining similar populations worldwide.

8.0 CONCLUSION

The CNSC reviewed and synthesized a large number of studies grouped in two lines of evidence to assess the potential health effects of the past and present radium and uranium refining and processing industry in the town of Port Hope.

- Environmental studies – which analyse and measure the environmental concentrations of contaminants attributable to the nuclear industry in Port Hope, and compares them with national and international benchmarks, to assess potential risks;

- Epidemiological studies – which compare the health status of the residents and nuclear workers of Port Hope with the general population (descriptive, ecological studies), and assess the relationship between occupational and residential exposures and adverse health effects (case-control and cohort studies).

Many studies were carried out in Port Hope; there were at least thirty environmental studies and thirteen epidemiological studies. This clearly shows that there has been a tremendous effort expended to measure environmental levels of contaminants, estimate exposures and risks and conduct epidemiological studies. The lines of evidence, based on numerous studies conducted in Port Hope, spanning a period of over five decades, support each other and reveal that the levels of exposure in Port Hope are low and there is no evidence of health effects as a result of past and present activities of the radium and uranium refining and processing industry in Port Hope.

These findings are consistent with the international scientific understanding of the effects on human health of radiation, uranium toxicity, and toxicity of other contaminants measured in Port Hope.

The findings of all studies conducted in Port Hope are also consistent with other studies examining similar populations worldwide.

On the basis of this comprehensive review and synthesis of the levels of exposure to radiation and to other contaminants associated with the radium and uranium refining and processing industry in Port Hope, comparisons of estimated exposures with criteria and benchmarks protective of human health, and the results of 13 epidemiological studies on Port Hope residents and PHCF workers, the CNSC concludes that no adverse health effects have occurred or are likely to occur in Port Hope as a result of the operations of the nuclear industry in the town.

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APPENDIX I

Glossary and Acronyms

Age-adjusted Rate: The summary rate of disease or death in a population where the age-specific rates are weighted by the age structure of standard population. This allows rates to be compared over time as the population age distribution changes.

***Alpha Particles*:** Positively charged particles consisting of two protons and two neutrons that are emitted by the nuclei of radioactive (unstable) elements as they decay. Alpha particles are relatively large and can be stopped by skin or a sheet of paper. An alpha particle is a helium nucleus.

Atom: Unit of matter consisting of a single nucleus surrounded by a number of electrons equal to the number of protons in the nucleus. The atom is the smallest portion of an element that can combine chemically with other atoms.

***Attribution*:** The process of ascribing an effect to a particular cause. If an exposure is not the only known cause of a particular effect, it is only possible to assign a probability that the effect was caused by the exposure. Even though a vast scientific literature can be used to support attribution, each effect must be examined on its own merits. Varying degrees of confidence will be associated with the judgment. For example, for populations with average radiation doses of below 100 mSv (16), it cannot be determined if an adverse health effect was caused by exposures from the nuclear industry, background radiation, or other causes. It is only possible to assess the probability of causation.

Background Radiation: The ionizing radiation emitted from a variety of natural and artificial radiation sources.

***Beta Particles*:** High-energy negatively charged electrons ejected by radioactive (unstable) element as they decay. A beta particle is identical in mass and charge to an electron. Beta particles are relatively small and can penetrate up to two centimetres of water or human flesh. A sheet of aluminum a few millimetres thick can stop them.

Case-control Study: A study designed to determine whether people with a disease or condition (cases) differ in exposure to certain agents and factors than do a similar group of people who do not have the disease (controls).

Cohort Study: A study designed to follow a group of people (a cohort) over time to determine whether their exposures to certain factors, as measured at the beginning and over time, influence whether or not they develop a certain disease or condition.

Collective Dose: The total radiation dose incurred by a population.

Committed Dose: A dose of radiation, received by an organ or tissue from a nuclear substance during the 50 years after the substance is taken into the body of a person 18 years old or older or during the period beginning at intake and ending at age 70, after it is taken into the body of a person less than 18 years old.

Confidence Interval: A range of values for a variable of interest; for example, a rate, constructed so this range has a specified probability (usually 95%) of including the true value of the variable (69). The specified probability is called the confidence level, and the end points of the confidence interval are called the confidence limits.

Confounder: Some factor associated with both the disease and exposure.

Decay (radioactive decay): The process of spontaneous transformation of a radionuclide or the decrease in the activity of a radioactive substance.

***Decay Product*:** A nuclide or radionuclide produced by decay, sometimes referred to as progeny. It may be formed directly from decay of a radionuclide or as a result of a series of decays through several radionuclides.

Descriptive Study: A study concerned with and designed only to describe the existing distribution of variables, such as health status, without regard to causation (69). Unlike analytic studies, which usually attempt to identify risk factors that cause disease, descriptive studies do not test hypotheses.

Deterministic Effects: "Direct" changes in cells that are certain to occur after an acute dose of radiation – in excess of a threshold level (1000 mSv) – below which radiation effects are not detected. (16). A specific set of clinical and laboratory findings occur in a particular time sequence. The severity of health effects increase with the radiation exposure received, ranging from a temporary change of the blood count without any clinical signs to a lethal dose beyond therapy.

***Dose*:** A general term for a measure of the energy deposited by radiation in a target. See the more specific terms absorbed dose, equivalent dose, effective dose and collective dose.

Ecological Study: A study in which the units of analysis are populations or groups of people, rather than individuals (69). An ecological study is a descriptive epidemiological study that generates hypotheses but does not establish cause and effect.

Effective Dose: A measure of dose designed to reflect the amount of radiation detriment likely to result from the dose. It represents the sum of the equivalent doses received by different tissues of the human body; each multiplied by a "tissue-weighting factor" (w_T). Unit: sievert (Sv).

Electron: A stable elementary particle having a negative electric charge of 1.6×10^{-19} C and a mass of 9.1×10^{-31}kg.

Element: A substance with atoms of all of the same atomic number.

***Emissions*:** Gases, particles, and materials released or emitted into the environment often due to combustion or burning of a fuel.

***Enriched Uranium*:** Is uranium that has been processed to increase the concentration of fissionable uranium-235 isotope to prepare it for use in some types of reactors. Natural uranium is about 0.7 percent U-235. Enriched uranium is about three percent U-235.

Epidemiology: The study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control health problems in populations.

Equivalent Dose: A measure of the dose to a tissue or organ designed to reflect the amount of harm caused to the tissue or organ. Obtained by multiplying the absorbed dose by a radiation- weighting factor to allow for the different effectiveness of the various types of radiation in causing harm to tissue. Unit sievert (Sv).

Excess Relative Risk: Another way of expressing risk is excess relative risk. This is figured by subtracting 1 (that is, the background risk) from the relative risk.

***Gamma Rays*:** penetrating electromagnetic radiation emitted by an atomic nucleus during radioactive decay; a wave form of ionizing radiation.

***Genes*:** The biological units of hereditary. They are arranged along the length of the chromosomes.

***Gray* (Gy):** Radiation damage is dependent on the absorption of radiation energy and is approximately proportional to the concentration of absorbed energy in tissue. The gray is the SI unit of absorbed radiation dose corresponding to the absorption of 1 joule of radiation energy per kilogram of material. For gamma and beta radiations, the gray is numerically equal to the sievert.

Half Life: For a radionuclide, the time required for the activity to decrease, by a radioactive decay, by half. A shorter life means a more radioactive substance.

Incidence: The number of new cases of disease appearing in a population in a specific time period, usually within a year.

Incidence Rate: the number of new cases of disease appearing in a time period divided by the number of people at risk of developing that disease.

Ion: An atom, molecule or fragment of a molecule that has acquired an electric charge through the loss of capture of electrons.

***Ionization*:** The process by which an atom or molecule acquires or loses an electric charge (i.e., the production of ions).

***Ionizing Radiation*:** For the purpose of radiation protection, radiation capable of producing ion. Examples are alpha particles, gamma rays, X rays and neutrons.

***Irradiation*:** The use of ionizing forms of radiation to kill bacteria and pests in human food and animal feed or to slow the ripening process in fruits and vegetables after harvest.

***Isotopes*:** Various forms of atoms of the same chemical element, which are distinguished by the number of neutrons in the nucleus. The number of protons remains the same. Uranium has 16 different isotopes.

Licence Limit: Release limit for a given substance as specified in a licence. The limit shall be below or equivalent to provincial or federal standards or guidelines.

***Molecule*:** A group of atoms bonded to each other chemically. It is the smallest portion of a substance that can exist by itself and retain the properties of the substance.

Mortality Rate: Number of deaths occurring in a specific time period (usually a year) divided by the population at risk.

Natural Uranium: refers to uranium with the same isotopic composition as found in nature. It contains approximately 0.7% uranium-235, 99.3% uranium-238, and a trace of uranium-234 by weight. In terms of amount of radioactivity approximately 2.2 % comes from uranium-235, 48.9% uranium-238, and 48.9% uranium-234.

***Neutron*:** An elementary particle found in the nucleus of atoms with no electrical charge. Neutrons are released from the nuclei of Uranium-235 atoms during fission. Neutrons have a mass of about 1.6×10^{-27} kg and a mean lifetime of about 1000 seconds.

***Non-ionizing Radiation*:** Radiation that is not ionizing radiation, that is, it does not possess sufficient energy to produce ions. Examples are ultraviolet radiation, visible light, infrared radiation, and radiofrequency radiation.

Nucleus (of an atom): The positively charged central portion of an atom. It contains the protons and the neutrons.

Nuclide: A species of atom characterized by the number of protons and neutrons and the energy state of the nucleus.

***Proton*:** A stable elementary particle found in the nucleus of atoms with a positive electric charge of 1.6×10^{-19} kg.

***Radiation*:** Energy travelling through space in the form of waves or particles. Ionizing radiation (e.g., alpha particles, beta particles, gamma rays, x-rays and neutrons) has the ability to remove electrons from the matter it encounters. The term radiation, as used in this report implies ionizing radiation. The type of radiation emitted by uranium is ionizing radiation.

Radioactive (adjective)**:** Exhibiting radioactivity; emitting or relating to the emission of ionizing radiation or particles.

***Radioisotope*:** Unstable atoms of various chemical elements that emit radiation. They are widely used in medicine to treat diseases such as cancer or to diagnose injuries and disease. Radioisotopes can also be used to kill pests and bacteria in food and to slow the ripening process in fruits and vegetables after harvest.

***Radionuclide*:** A radioactive nuclide.

Radon Decay Products *(also Radon Progeny)**:*** A term used to refer collectively to the immediate products of the radon decay chain. These include Po-218, Pb-214, Bi-214, and Po-214. They have an average combined half-life of about 30 minutes.

Relative Risk: Comparison of the risk of developing cancer (or any other disease) for exposed persons with the risk for unexposed persons. Results of health studies are often expressed as a relative risk. This is because many harmful agents are thought to increase the risk for disease by multiplying background risk by a certain amount per unit of dose (the relative risk), rather than by adding a fixed excess risk per unit of dose.

While absolute risk is expressed as a number between 0 and 1 or as a percentage, relative risk is a ratio that can be expressed with any real number. Relative risk is usually estimated by taking the risk of the disease in the exposed group and dividing it by the background risk, that is, the risk of the disease in the unexposed group.

A relative risk will be greater than 1.0 if the radiation dose increases the risk of the disease. This is because a relative risk of 1.0 indicates that the risk of disease in the exposed group equals the risk of the unexposed group. For example, if the risk is 2 percent (2 in 100) in the exposed group and 2 percent in the unexposed group, the relative risk is 1.0 (2 divided by 2 = 1). A relative risk of 2.0 suggests that, compared with the unexposed group, the exposed group has twice the risk of developing the disease in question. A relative risk of 3.0 suggests that the exposed group has three times the risk, and so on.

Regulatory Body: An organization designated by the government as having legal authority for regulating nuclear radiation, radioactive waste, and transport safety.

Risk: The probability of a specified health effect occurring in a person or group as a result of exposure to radiation.

Sievert (Sv): The sievert is the unit of radiation equivalent dose, H, which is used for radiation protection purposes, for engineering design criteria, and for legal and administrative purposes. The sievert is the SI unit of absorbed radiation dose in living organisms modified by radiation type and tissue weighting factors. The sievert is the unit of dose measuring the "equivalent dose" and "effective dose". It replaces the classical radiation unit the rem. Multiples of sievert (Sv) used in this document include millisieverts (mSv) and microsieverts (μSv).

Standardized Incidence Ratio (SIR): The ratio of the observed number of new cases of a disease or condition in a population to the expected number of new cases (observed/expected). The expected number is determined by applying the sex and age-specific incidence rates of a standard population, such as Ontario or Canada, to a population of the study population, such as Port Hope. An SIR of 1.0 indicates that there is no difference between the study and standard population. An SIR greater than 1.0 means that there are more new cases of disease in the study population than in the standard population. An SIR less than 1.0 means that incidence is lower in the study population.

Standardized Mortality Ratio (SMR): The ratio of the observed number of deaths from a disease or condition in a population to the expected number of deaths (Observed/ Expected). The expected number is determined by applying the sex and age-specific death rates of a standard population, such as Ontario or Canada, to a population of the study population, such as Port Hope for the study period. An SMR of 1.0 indicates that there is no difference between the study and standard population. An SMR greater than 1.0 means that there are more deaths from the disease in the study population than in the standard population. An SMR less than 1.0 means that there are fewer deaths in the study population.

Statistical Power: The probability that an epidemiological study will detect a given level of elevated risk with a specific degree of confidence. The statistical power of a study is greatly affected by the sample size, the dose level(s) of the exposed group and the magnitude of the risk coefficient (16).

Stochastic Effects (cancer and hereditary effects): Radiation acts on DNA molecules in particular and may result in delayed pathological effects such as cancer or genetic alterations. These effects are random; thus they do not appear systematically. It is usually considered that the probability of their occurrence increases proportionally to the radiation dose received: the lower the dose, the lower the probability of occurring.

Thermoluminescent Dosimeters (TLDs): A type of radiation dosimeter which measures gamma and beta radiation dose by measuring the amount of visible light emitted from a crystal in the detector when the crystal is heated. The amount of light emitted is dependent upon the radiation exposure.

Uranium Decay Chain: Decay chain refers to the radioactive decay of different discrete radioactive decay products as a chained series of transformations. Most radioactive elements do not decay directly to a stable state, but rather undergo a series of decays until eventually a stable isotope is reached.

***Uranium Ore*:** Rock that contains unusually high concentrations of natural uranium which can be extracted through the mining and milling process.

Working Level Months (WLM): The calculation of radiation dose due to the inhalation of radon and its decay products is very complex and the common practice is simply to record radon decay product exposure. The concentration of radon decay products in workplace air is generally expressed in Working Levels (WL), where 1 WL is that concentration of radon decay products per liter of air that would result in the ultimate release of 1.3×10^5 MeV of potential alpha energy. Occupational exposure to radon decay products is the product of time in the workplace and the concentration of radon decay products in the workplace air, measured in Working Level Months (WLM), where 1 WLM is equivalent to one working month (170 hours) in a concentration of 1 WL

X-ray: Penetrating electromagnetic radiation emitted by an atom when electrons in the atom loose energy, and having wavelengths shorter than those of visible light.

Yellowcake: The name given to the final product of most uranium mills including those in Canada. Yellowcake is a coarse powder which is insoluble in water. In the past, the chemical composition of yellowcake was variable and depended upon leachent and subsequent precipitating conditions. Modern yellowcake typically contains 70 to 90% triuranium octoxide (U_3O_8) by weight.

AECB: Atomic Energy Control Board (now the CNSC)
AECL: Atomic Energy of Canada Limited
CANDU: CANadian Deuterium Uranium reactors
CCO: Cancer Care Ontario
CCDB: Canadian Cancer Database
CI: see Confidence Interval
CMDB: Canadian Mortality Database
CNSC: Canadian Nuclear Safety Commission (previously the AECB)
COPC: Contaminants of potential concern
EA: Environmental Assessment
ERR: see Excess Relative Risk
FARE: Families Against Radiation Exposure

HWC: Health and Welfare Canada (now Health Canada)
HC: Health Canada (previously HWC)
HKPR: Haliburton Kawartha Pine Ridge District Health Unit
LLRWMO: Low Level Radioactive Waste Management Office
LNT: Linear no-threshold model
NRCan: Natural Resources Canada
NDR: National Dose Registry
MOE: Ontario Ministry of the Environment
MOH: Ministry of Health residence code
OCR: Ontario Cancer Registry
OR: Odds Ratio
PHAI: Port Hope Area Initiative
PHCF: Port Hope Conversion Facility
PHCHCC: Port Hope Community Health Concerns Committee
RR: Relative Risk
SX: solvent extraction
SIR: see Standardized Incidence Ratio
SMR: see Standardized Mortality Ratio
TLDs: thermoluminescent dosimeters
UNSCEAR: United Nations Scientific Committee on the Effects of Atomic Radiation
UMRC: Uranium Medical Research Centre
WMF: Waste Management Facility

APPENDIX II

Radiation Theory and Health Effects of Radiation

Radiation Theory

A basic understanding of radiation theory is necessary to understand radioactivity and how uranium and its decay products can potentially cause health effects, as outlined in Chapter 4.

All matter is made up of elements that are composed of atoms. Atoms have a small core called a nucleus, which is orbited by even smaller electrons. The nucleus contains particles called protons and neutrons. Most atoms are stable and will never change, but certain atoms have an uneven balance of neutrons and protons and are unstable. To become stable, they will undergo spontaneous disintegration, releasing nuclear material and energy. The atom they next become may be stable or may not. Uranium is an example. It will naturally turn into lead after billions of years. As an unstable atom decays, its atomic structure changes releasing radiation in the form of alpha particles and beta particles and gamma rays and neutrons.

Alpha, beta and gamma radiation are referred to as ionizing radiation, because when they react with surrounding matter they have enough energy to affect the atomic structure of the molecules or cells of which they are part. Molecules located in their path can lose one or more electrons, possibly breaking apart the molecule. These then transform into electrically charged "ions" which may in turn disrupt the structure of the molecules or cells of which they are part or they may capture another electron or molecule and become stable again. This is why radiation is said to be ionizing. This ionization phenomenon is the main mechanism by which radiation interacts with matter.

An important characteristic of ionizing radiation is how deeply it can penetrate body tissues. This penetration depends on the radiation's energy, electrical charge, and mass.

- ***Alpha particles*** are produced when positively charged helium nucleus, consisting of two protons and two neutrons, is expelled by a radioactive element as it decays. Alpha particles are relatively large, energetic particles that transfer their energy over a short distance but can be stopped by skin or a sheet of paper. They have low penetration. Therefore, radioactive elements that emit alpha radiation are not harmful to humans unless they are taken into the body either by inhalation (breathing in), ingestion (eating and drinking), or through an open wound. Radon and some of its decay products emit alpha radiation.

- ***Beta particles*** are produced when an electron is expelled by radioactive elements as they decay. These particles are relatively small. Their range in the air is a few metres at most. They can penetrate the surface layer of human flesh, although many are

absorbed after a few millimetres. Therefore, radioactive elements that emit beta particles are hazardous to skin or the lenses of eyes. They can also harm internal tissue when emitted from radionuclides that have been taken into the body. A sheet of aluminum foil, plastic, or a glass pane can stop beta particles.

- ***Gamma rays*** are a photon, or packet of energy that is emitted from the nuclei of some radioactive elements as they decay. Photons are a wave form of ionizing radiation and they travel at the speed of light. Because they have no charge or mass, they can deeply penetrate matter, possibly reaching all tissues of the body Thick, dense materials, such as concrete or lead are used for shielding for gamma rays.

While the decay of uranium emits an alpha particle, it does so at a very slow rate (U238 half-life of 4.5 billion years, U235 half-life is 700 million years, U234 half-life is 240,000 years).

Health Effects of Radiation

Exposure to radiation from all sources can result in changes in sensitive biological structures. Since the most sensitive structure in the cell is the DNA molecule, exposure to radiation may damage the DNA, causing the cells to die or to fail to reproduce. This can result in the loss of tissue or organ function, or in the development of cancer (16). For the purposes of this report, exposure to radiation, termed radiation dose, is measured using a unit called the millisievert (mSv).

There are two broad classes of radiation effects on the human body. The first class involves "deterministic effects", which do not occur until the dose reaches a certain threshold level. Above this level, the effect will definitely occur, and the severity of harm will increase with dose. Following a very high dose of radiation (more than 1000 mSv) delivered over a short period of time (hours to days), serious health effects can occur, usually within days to weeks after the exposure. The effects of such high doses include nausea, vomiting, diarrhoea, hair loss, haemorrhage, immune function loss, nervous system damage, radiation burns, and even death. Fortunately, such doses are extremely rare and do not arise from environmental exposures, such as those found in Port Hope.

The second class of effects is termed "stochastic effects", which means that the likelihood of the effect increases with the amount of radiation received. The main stochastic effects in humans are cancer in the exposed individual and possible genetic effects in the offspring. Following a lower acute dose (above 100 mSv), no effects are seen immediately, but there is an increased likelihood of developing cancer depending on as the amount of radiation received. Several other factors also affect the likelihood of developing cancer including individual sensitivities to radiation exposure, the types of radionuclide(s) an individual has been exposed to, and the dose rate. The latency period between exposure and recognition of a cancer can range from 5 years to several decades. The cancers most frequently associated with radiation exposure are leukaemia and solid tumours of the lung, breast, thyroid, bone, digestive organs, and skin (16). However,

radiation-induced cancers are indistinguishable from those that occur from other causes, so the relationship between radiation and cancer can only be shown in large populations of irradiated individuals as an increase of cancers over the background incidence. The main sources of epidemiological evidence on radiation risks have come from studies of the atomic bomb survivors at Hiroshima and Nagasaki (30), patients who received high radiation doses for diagnostic or treatment purposes, and occupational exposed workers, including uranium mine and processing workers. There is established evidence that radiation exposures above 100 mSv increase cancer incidence and mortality (16, 119). Radiation exposure has not been demonstrated to cause risks to offspring (hereditary effects) in human populations (16, 24, 118). However, these health effects have been found in experimental studies of plants and animals (118) and are accounted for in the risk estimates used to set dose limits.

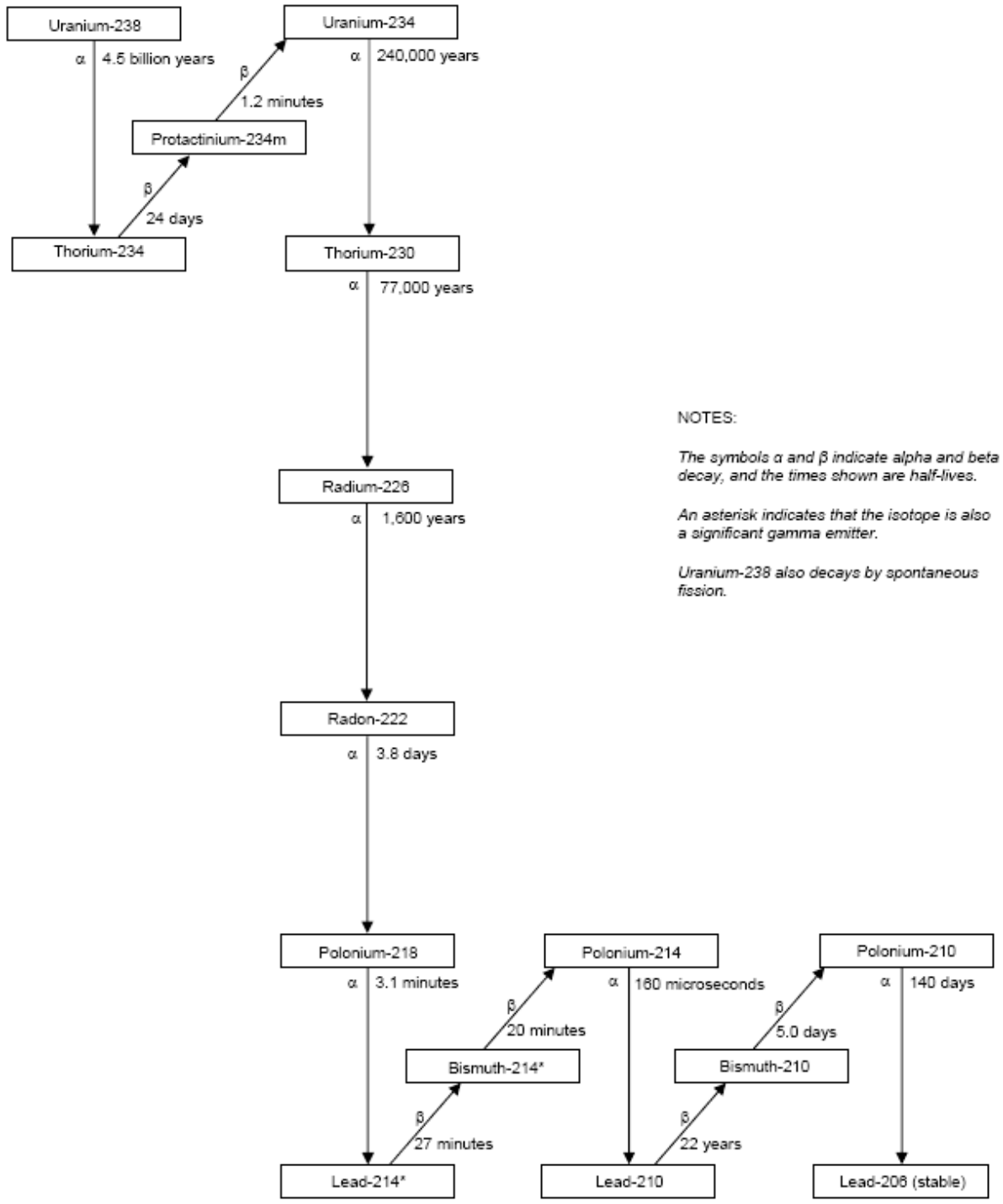
Health effects at moderate and low radiation exposures (that is, below 100 mSv) are less clear. So far, neither the study of the survivors of the Hiroshima and Nagasaki atomic bomb nor any other studies have provided conclusive evidence of the increased incidence of cancer caused by radiation at much smaller doses (16, 30). This is in part due to the fluctuations of the natural incidence of cancers. At low doses (below 100 mSv), the explanation of the development of health effects must rely on the science of radiation biology which studies the mechanisms of radiation interaction with human cells. Although much is known about the initiating mechanisms of radiation at the cellular level, there are uncertainties in the understanding of how low dose radiation can cause cancer in tissues or organisms. Therefore, to be prudent and to account for the scientific uncertainties, it is generally assumed that the likelihood of effects diminishes proportionally with dose down to zero; that is, the linear no-threshold (LNT) model The LNT model is also commonly used to assess the risks from chemical carcinogens.

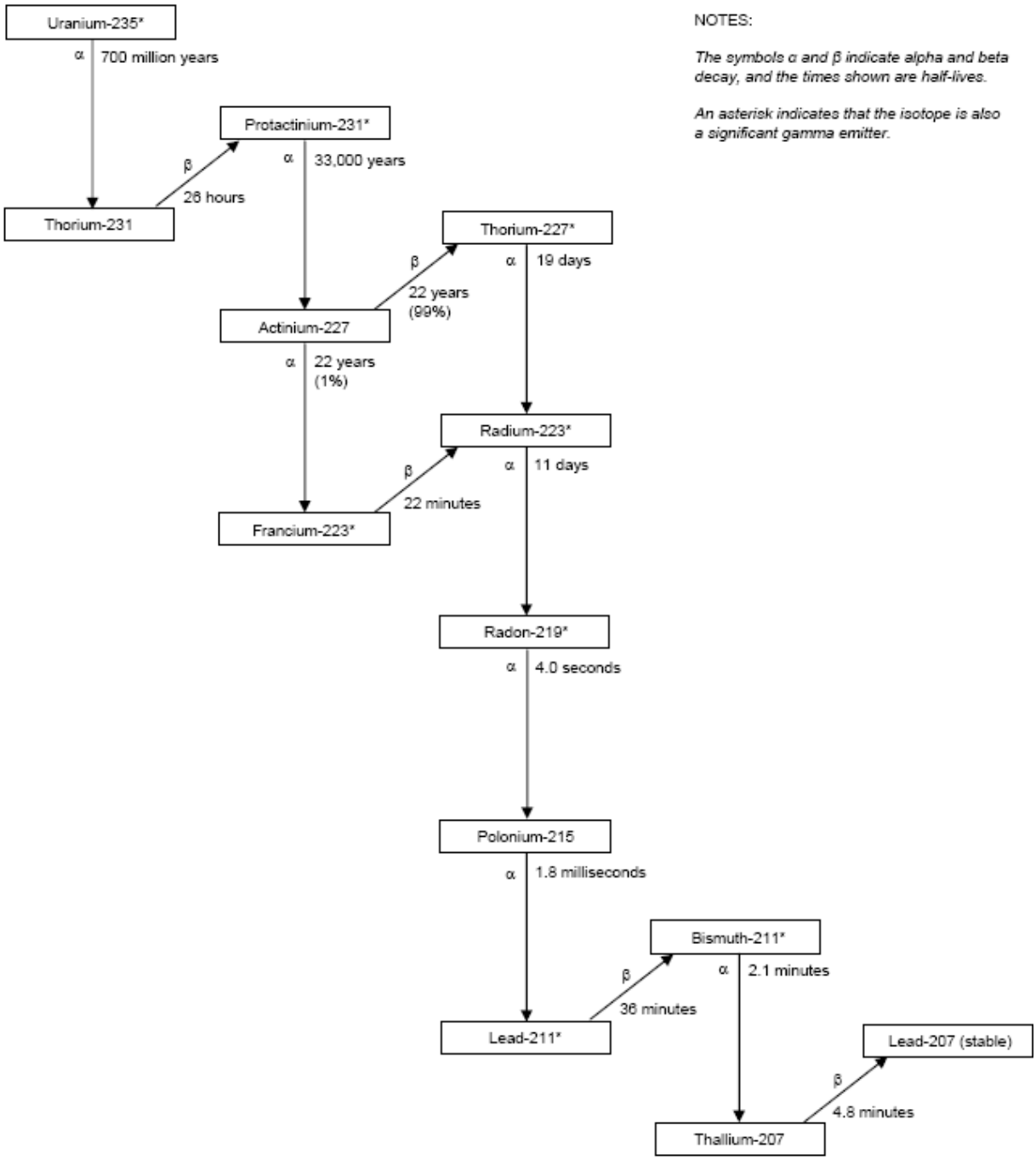
An increased risk of cardiovascular and other diseases following radiation exposure may be of concern. At very high levels of radiation exposure received during radiotherapy to the area around the heart, it has long been recognized that radiation-induced heart disease can occur at doses to the heart greater than a few thousand mSv. At high doses, between 1000 and 2000 mSv, increased cardiovascular disease has only been seen in the atomic bombing survivors in Hiroshima and Nagasaki (30). At doses between 100 mSv and 1000 mSv there is no clear evidence of systematic increases in cardiovascular or other non-cancer diseases (16, 30).

APPENDIX III

Uranium-238 Decay Chain

Taken from Argonne National Laboratory, EVS Human Health Fact Sheet, August 2005 (120).




Uranium-235*
α
700 million years
Protactinium-231*
β
26 hours
α
33,000 years
Thorium-231
Thorium-227*
β
22 years
(99%)
α
19 days
Actinium-227
α
22 years
(1%)
Radium-223*
β
22 minutes
α
11 days
Francium-223*
Radon-219*
α
4.0 seconds
Polonium-215
α
1.8 milliseconds
Bismuth-211*
β
36 minutes
α
2.1 minutes
Lead-211*
Lead-207 (stable)
β
4.8 minutes
Thallium-207
NOTES:
The symbols α and β indicate alpha and beta decay, and the times shown are half-lives.
An asterisk indicates that the isotope is also a significant gamma emitter.