Tritium Hazard Report: Pollution and Radiation Risk from Canadian Nuclear Facilities

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GREENPEACE

PREFACE

This report on tritium releases in Canada is in two parts. Part 1 discusses tritium discharges from nuclear facilities in Canada and compares them with those from reactors in other countries. It examines the resulting tritium concentrations in drinking water, air and in food near Canadian nuclear stations. Although tritium releases from Candu facilities are very large, radiation protection regulators continue to maintain that these releases are of little concern because tritium's radiation doses and its resulting hazards are small.

Part 2 examines these contentions in considerable detail. It shows that tritium's radiation "doses" are, questionably, estimated to be several hundreds of times lower than most other radioactive elements.

Radiation and radioactivity (including risks, doses, biology and epidemiology) are complex matters which are often difficult to grasp. Therefore Part 2 is designed to be read primarily by health physicists and radiation protection scientists. However, efforts have been made to make this report more accessible to the wider public. In particular, technical terms have been explained and scientific jargon has been avoided.

The report concludes that official attitudes on tritium are unscientific and incorrect, that tritium's hazardous nature should be fully acknowledged by radiation protection agencies in Canada, and that tritium's dose coefficient should be increased substantially.

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Any mistakes, of course, remain the sole responsibility of the author.

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EXECUTIVE SUMMARY

Part 1 examines the high tritium discharges from Canadian nuclear power stations, which are considerably larger than those in other countries. Canadian tritium discharge limits are much less stringent than those in other countries. Canadian tritium limits in drinking water are considerably more lax than those in force in Europe and the US.

Those Great Lakes with nuclear reactors on their shores have tritium levels 2 to 5 times greater than those of Lake Superior, which has no reactors. The tritium level in Lake Ontario is increasing each year, due to discharges and to major tritium leaks in past years from Candu stations.

Tritium concentrations in drinking water, in air, and in vegetation and food near Candu stations are all significantly increased. These result in high tritium intakes in residents living within 5 to 10 km of Candu reactors and very high tritium intakes in residents who live within 1 to 2 km. However, because of tritium's very low dose factors, the radiation "doses" to those exposed are considered insignificant and are declared "within safety limits" by nuclear regulators.

Part 2 examines the science on tritium's doses. It finds that significant objections have been made in the past to tritium's official dosimetry and the official models used to estimate tritium doses, especially from organically bound tritium. A number of recent UK and US reports continue to raise questions about tritium's official doses.

The report concludes that scientific concerns about tritium's hazards are inadequately recognised by Canada's nuclear regulators. It therefore recommends that a precautionary approach to tritium discharges should be adopted in Canada. In particular, it recommends that:

1) the Ontario and Federal governments should establish a committee (whose members should include scientist representatives from environmental groups) to examine tritium's dosimetry and risks. In particular, the committee should examine recent authoritative reports which raise questions about currently-accepted views on tritium's dosimetry and risks;
2) case-control and cohort epidemiology studies should be commissioned to examine possible adverse health effects in tritium-contaminated areas;
3) pregnant women and young (less than 4 years old) children and their mothers should be advised not to live near tritium-emitting facilities (i.e., within 10 km);
4) people who live very near (i.e., within 5 km) tritium-emitting facilities should be advised not to consume food from their own gardens, bee hives and orchards, and not to consume wild foods, e.g., blackberries and mushrooms, growing very near the facilities;
5) because tritium reduction facilities themselves release large quantities of tritium, nuclear reactor operators should be requested to examine the option of long-term storage of tritiated water from moderator circuits in decay tanks as a way of reducing tritium discharges; and
6) operators of tritium-emitting facilities should give further consideration to other ways and means of reducing tritium releases.

PART 1: TRITIUM RELEASES IN CANADA

Chapter 1 Introduction

What is tritium?

Tritium is the radioactive isotope of hydrogen, the smallest and lightest element. It has a radiological half-life of 12.3 years and decays to the stable isotope helium (^{3}He), emitting a beta particle (and a neutrino). The beta particle has a maximum energy of 18.6 kilo electron volts (keV) (average energy of 5.7 keV) with a short range—a few centimetres in air, 0.9 micrometers (μm) in water, and about 0.6μm in tissue. This means that tritium is not dangerous externally, but it is an internal radiation hazard when inhaled, or ingested via food or water, or absorbed through the skin. Tritium is the most commonly encountered and important beta-emitting radionuclide.

Tritium has always kindled much interest among scientists and has been widely studied in the past: see Appendix 1 to Part 2 of this report, NCRP (1979), NEA (1980), ACRP (1991), and CCNR, http://www.ccnr.org/tritium_1.html. More recent studies on tritium can be found in various reports and websites. For example, see the following:

- US Environmental Protection Agency website on tritium, http://www.epa.gov/radiation/radionuclides/tritium.htm (accessed February 26, 2007)
- The report by Richard Osborne (2002), *Tritium in the Canadian Environment: Levels and Health Effects*. Report RSP-0153-1. Prepared for the Canadian Nuclear Safety Commission under CNSC contract no. 87055-01-0184 by Ranasara Consultants and Richard Osborne
- A tritium report by the US Agency for Toxic Substances and Disease Registry http://www.atsdr.cdc.gov/hac/PHA/livermore4/lms_toc.html
- A forthcoming report on tritium by the UK Government's Advisory Group on Ionising Radiation http://www.hpa.org.uk/radiation/advisory_groups/agir/index.htm

Tritium is discharged in two main forms. In its elemental form (HT), it is an invisible, odourless gas chemically identical to hydrogen gas. In its water form (i.e., tritiated water, or HTO), it is practically indistinguishable from ordinary water. In fact, it is useful to think of tritium as radioactive water because this is by far its most common form. Tritium releases from Canadian nuclear plants are mostly tritiated water; the tritium recovery facility at Darlington emits tritium gas (HT).

Both forms of tritium are very radioactive (in technical terms, they have high specific activities). One gram of HT contains about 360 terabecquerels[1] (TBq) of radioactivity, and one gram of HTO contains about 55 TBq[2] of radioactivity. Both forms of tritium are very pervasive; HT permeates most materials, rubber and many grades of steel with relative ease, and HTO—being chemically identical and physically similar to ordinary water—very rapidly mixes throughout the atmosphere, hydrosphere, lithosphere and biosphere, in short, everywhere. HT is converted to HTO in dry indoor conditions at the rate of about 1% per hour—faster in humid conditions. It is readily converted to HTO by bacteria in soil.

Tritium has a number of unusual properties apart from its rapid distribution throughout the environment. One is its property of exchanging rapidly with other H atoms in the environment—

[1] 9,800 curies (Ci) in old units
[2] 1,500 Ci in old units

including in humans. Another is its propensity to form strong bonds with carbon to form organically bound tritium (OBT) during metabolic reactions and in cell reproduction (see box below).

Organically bound tritium (OBT)
OBT is tritium which has become chemically attached to carbon atoms[3] in organic molecules. Organic binding is tritium's most significant property, but, unfortunately, official dose models for OBT underestimate its hazards.

Humans can accumulate OBT in two ways. The first is by consuming OBT in food, e.g., vegetables, wheat, honey, milk, that has been grown and harvested in areas near Candu reactors contaminated by tritiated water vapour. The second is by drinking/eating, breathing, and absorbing tritiated water that then is then both metabolised into organic molecules needed by the body, and incorporated into new cells.

OBT is more problematic than HTO for two reasons. First because OBT's residence time (i.e., half-life) in humans is much longer (20 to 50 times) than HTO's residence time (see Part 2 of this report). And second because OBT must by definition be located near organic molecules (such as DNA) more often than HTO. As stated by Taylor et al (1990) "... the concentration of OBT in tissues of interest, are greater by up to an order of magnitude after ingestion of OBT than after HTO ingestion...". This means that radiation exposures from OBT are much larger than that from HTO.

Like most radionuclides, tritium is a carcinogen, mutagen and teratogen. Many scientists have expressed concerns about tritium's radiotoxicity (see Appendix 1 of Part 2). However, some radiation authorities continue to assert, rather misleadingly, that tritium is a "weak" radionuclide because the energy of its decay particle is very low. However, paradoxically, in radiation biology the weaker a particle the more effective it becomes. Unfortunately this remains unrecognised in official circles and tritium's official dose factor (i.e., the dose given by the disintegration of one atom of tritium) is currently very small. Indeed, it is the lowest among common radionuclides by some margin. There are many questions about tritium's official radiation doses: these are considered in detail in Part 2.

Tritium is naturally formed in the upper atmosphere by cosmic rays. The quantity of tritium thus produced each year is ~ 7.4×10^4 TBq (Luykx and Fraser, 1986). In Western developed countries in the early 1980s, the annual amounts released by **civil** nuclear power and reprocessing facilities (Masschelein and Genot, 1983; NEA/OECD, 1980) were about the same or higher. These will have increased since then to exceed tritium's natural production rate.

In addition, much larger amounts of tritium are released from **military** activities. Between 1954 and 1962, atmospheric weapons testing released extremely large amounts of tritium—1.6×10^8 TBq, almost all in the northern hemisphere (UNSCEAR, 1988). By 2007, this will have decayed about 16-fold. In addition, at the height of the cold war in the 1970s and 1980s, annual tritium discharges from nuclear weapons manufacturing amounted to 2.8×10^6 TBq (Jaworowski, 1982). This included the US nuclear weapons plants at Savannah River and

[3] Another form of OBT is tritium that attaches to O, P, N and S atoms. These bonds are weaker than C-H bonds and their average half-life is shorter (about 30 days) than that of tritium bound to C atoms (1 to 2 years). This is exchangeable OBT and is discussed in Part 2.

Hanford, which annually emitted on average 1.1 x 10^5 TBq (NCRP, 1979) in the 1950s to the 1980s.

Chapter 2 Tritium Releases In Canada

Most tritium releases in Canada are from Candu nuclear power generating stations, as shown in the following tables. In addition, substantial tritium releases occur from the SRB Technologies facility in Pembroke, Ontario, which are reported in Annex 3 below. This facility manufactures tritium-containing emergency lighting equipment.

Tables 2.1 to 2.4 reproduce recent releases of tritium from Candu nuclear stations.

Table 2.1 Recent emissions of tritium oxide (HTO) to air (TBq/a)

	2001	2002	2003	2004	2005
Bruce A + B	650	580	560	864	731
Pickering A + B	580	510	480	620	500
Darlington	240	190	170	280	130
Gentilly-2	190	180	150	260*	180*
Point Lepreau	140	130	100	100*	180*
TOTALS	1800	1590	1460	2120	1720

*estimated reading from graphs

Table 2.2 Recent discharges of tritium oxide (HTO) to water (TBq/a)

	2001	2002	2003	2004	2005
Bruce A + B	163	414	860	585	426
Pickering A + B	280	427	258	290	260
Darlington	94	69	100	160	220
Gentilly-2	450	500	350	120*	360*
Point Lepreau	150	140	81	100*	220*
TOTALS	1187	1410	1649	1250	1490

*estimated reading from graphs

Table 2.3 Air releases of elemental tritium (HT) (TBq/a)

	2001	2002	2003	2004	2005
Darlington	108	56	66	750	790

Table 2.4 Canada—Total tritium released (TBq/a, rounded to 3 figs)

	2001	2002	2003	2004	2005
HT	110	56	66	750	790
HTO to water	1190	1410	1650	1250	1490
HTO to air	1800	1590	1460	2120	1720
TOTALS	3100	3100	3200	4120	4000

Sources: NPRI, 2004, 2003, 2002.
OPG and Bruce Power annual environmental reports—OPG (2006) and Bruce Power (2006).

In Table 2.4, the figures for HT and HTO have been added together to arrive at total tritium releases, although their dosimetric impacts are different. This matter is discussed further below.

From the above tables, it can be seen that the total amount of tritium released by major nuclear facilities each year in Canada is of the order of ~3,000 TBq. This is about 10% of the natural production of tritium in the northern hemisphere of ~30,000 TBq per year. (Relatively little mixing occurs between the atmospheres of the northern and southern hemispheres).

Tritium releases from reactor types compared

Tables 2.5 and 2.6 below compare tritium releases from various reactor types. It can be seen that heavy water reactors emit considerably more tritium than other reactors, whether total discharges or normalised discharges (i.e., per GW year of electricity produced) are compared.

Table 2.5 Tritium (HTO) releases (gaseous + liquid) from various reactor types (TBq/a)

Reactor Type	**UNSCEAR 1993 (for year 1985)**	**Reactor Type**	**UNSCEAR 2000 (for year 1997)**
HWR (Bruce 1-4, Canada)	600	HWR (Bruce 1-4, Canada)	660
PWR (Diablo Canyon 2, US)	17	PWR (Diablo Canyon 1+2 US)	55
AGR (Hinkley Point B, UK)	12	AGR (Hinkley Point B, UK)	18.7
BWR (Wurgassen, Germany)	2	BWR (Philippsburg, Germany)	1.6

Table 2.6 Normalised tritium (HTO) releases (gaseous + liquid) from various reactor types (TBq per GW year)

	OECD/NEA 1980	**UNSCEAR 2000 Table 37 for years 1995–1997**	**European Commission for year 1999**	**Sood et al, 1990[4]**
HWR	750	670	-	650
PWR	37	21.4	20	-
BWR	7	1.7	1.3	-

Sources: OECD/NEA (1980); UNSCEAR (2000); EC (2000).

Why do heavy water reactors discharge large amounts of tritium?

Heavy water reactors discharge far more tritium than other reactor types because—as their name suggests—they use heavy water (deuterium) as coolant and moderator. During reactor operation, deuterium is activated by fission neutrons to form tritium via the following reaction:

$$^{2}H + \text{neutron} \rightarrow {}^{3}H$$

[4] Sood et al (1990) stated that 1.35 GWe in Ontario nuclear power stations produce 0.88×10^{4} TBq of tritium on average per year.

Other reactor types use different materials as coolant and moderator; for example, PWRs and BWRs use ordinary water and AGRs use CO_2 and graphite as coolant and moderator respectively. In these other reactor types, tritium is formed mostly as a tertiary fission product; i.e., it is split off from U-235 and Pu-239 when they undergo fission. This occurs within Candu reactors as well: the H-3 activation rate in Candu reactors is about 1,000 times greater than the H-3 fission product rate.

Tritiated water vapour emissions to air

When a reactor is in operation, tritium is continuously formed and continuously released to the atmosphere in the form of radioactive water vapour. Contrary to what many people assume, few tritium air emissions are via a stack or chimney; most are via the continuous leakage of tritiated water vapour from machines, pumps, seals, pipes, reactor walls, etc. That is, tritiated water vapour literally oozes out of practically every surface, nook and cranny of the reactor building.

The volumes of heavy water in Candu nuclear reactors are very large, more than 3 million litres (3,000 tonnes) each at Pickering, Darlington and Bruce stations. In addition, considerable efforts have to be expended to keep water (whether tritiated or not) at high temperatures and pressures from escaping from the pipe work, especially of the cooling circuits. The result is that heavy water losses from the cooling water circuits in Candu reactors are very large—about 3% of their inventory per year. Heavy water losses from the moderator circuits inside the reactor are lower, at about 0.1% of their inventory per year, mainly because the circuits are smaller and are mostly inside the reactor, i.e., leaks are potentially recoverable (data from Song et al, 1995).

Although most *heavy water* releases come from the coolant, most (~2/3rds) of the *tritium* releases are from the moderator because its tritium concentrations are considerably higher (~20–30 times) than those in the moderator.

Tritium—liquid water discharges

Liquid water discharges to the Great Lakes from Candu stations largely result from maintenance operations, that is, from opening pipes, valves, seals, etc., which inevitably result in spillages and discharges. With both vapour emissions and liquid water discharges, efforts are made to trap and retrieve the lost amounts and re-use them in their respective circuits, but large amounts are still lost and released as emissions and discharges.

The amount of heavy water which escapes from a single Candu 6 reactor is surprisingly large —about 4,000 litres per year (Song et al, 1995). About 90% leaks from the coolant and ~10% from the moderator. Therefore, about 32,000 litres of heavy water escape (via vapour emissions) each year from Pickering when its eight 500 MW reactors are all operating. This means that every day about 100 kg of heavy water in vapour form is released from the Pickering station on average, though there are fluctuations. By extrapolation, it is estimated that the Bruce station, with eight 750 MW reactors, will emit about 150 kg each day, and that the Darlington station, with four 850 MW reactors, will also release about 100 kg of heavy water each day when all its reactors are operating.

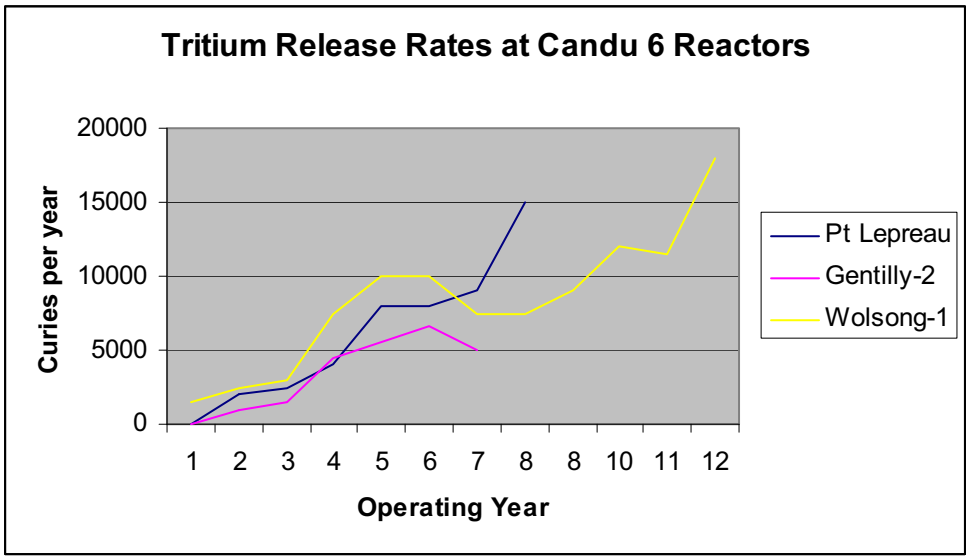
Tritium—HT gaseous emissions

The Darlington Tritium Recovery Facility and the SRB Technologies plant at Pembroke Ontario (see Annex 3) emit very large quantities of tritium in the form of hydrogen gas or HT. HT has very different dosimetric properties from tritiated water. The International Commission on Radiation Protection (ICRP) considers that HTO's doses from inhalation and ingestion are 25,000 times greater than those for HT (ICRP, 1989)[5]. This is because the body is not thought to absorb or metabolise hydrogen gas, whereas water is a vital component of all body tissues and metabolic processes. Therefore the important factor is how fast HT is converted (oxidized) to HTO. This had been the subject of differing views in the past (Sweet and Murphy 1984, and Spencer and Dunstall 1986). However, nowadays it is understood that HT dispersed into the atmosphere diffuses readily into the soil and is converted to HTO by enzymatic reactions, the rate of which depends on the porosity, water content and microbial activity of the soil. The converted HT is subsequently transported as HTO (Ichimasa, 1995). Generally, for conservatism in assessments, released tritium is often assumed to be in the form of HTO, as eventually almost all HT will be converted to HTO over longer time periods. (Davis et al, 1997).

History of tritium inventories of Candu reactor circuits[6]

Because of continuous neutron activation, tritium concentrations in both moderator and coolant steadily increase during each year of operation. This results in tritium releases increasing each year after start-up, as shown in Figure 2.1 below, reproduced from Song et al (1995).

Figure 2.1



Tritium concentrations continue to rise in each year of operation in both moderator and coolant circuits until equilibrium levels are reached (where possible). Equilibrium means the

[5] More precisely, the maximum derived air concentration for HTO is 25,000 times greater than that for HT.

[6] This section on tritium reactor inventories is partly based on information contained in an article discussing the Candu 6 reactor in Wolsong, South Korea (Song et al, 1995). The article states that, as all Candu 6 reactors are generically similar, their tritium characteristics during operating periods are also very similar.

rate of creation is equal to the rate of elimination. However, until 1990 there were only two mechanisms which decreased tritium concentrations: one was tritium's radioactive decay, which reduces its concentration by about 5% per year, and the other was by leakage (in other words, discharges and emissions) and by the replacement of leaked amounts with non-radioactive heavy water. The latter mechanism was more important. However these two mechanisms were slow in comparison with tritium activation rates in moderator and coolant circuits. The result was that, in the case of the coolant, equilibrium was only approached after about 30 years' operation (90% of equilibrium level reached after ~27 years (Song et al, 1995)). In the case of the moderator, the tritium activation rate was so high that equilibrium was never reached.

This meant that the tritium production rates in Candu reactors were extremely large. Indeed, Ontario Hydro engineers (Sood et al, 1990) stated that Ontario Hydro's (as it was then called) 20 reactors would produce 8,800 TBq of tritium per year on average over the next 30 years. The continuous increase in tritium inventories results in continuous increases in tritium discharges and emissions and this can be seen from the following table of tritium releases from Ontario Power Generation (formerly Ontario Hydro) nuclear plants.

Table 2. 7 Tritium releases from Candu reactors after start-ups (TBq/a)

YEAR	HTO (to air)	HTO (to lakes)	TOTAL
1972 {1}	530	40	570
1973	1,400	190	1,590
1974	920	540	1,460
1975	760	390	1,150
1976 {2}	940	221	1,161
1977	1,944	736	2,680
1978	1,460	1,353	2,813
1979	2,550	1,950	4,500
1980	2,280	1,370	4,650
1981	3,980	1,020	5,000
1982	2,180	1,340	3,520
1983 {3}	4,504	1,966	6,470
1984 {4}	2,979	1,018	3,997
1985	2,108	2,815	4,923
1986	2,506	2,397	4,903
1987	3,428	3,343	6,771
1988	3,758	3,425	7,183
1989	4,436	2,638	7,074
1990	3,311	1,980	5,291

Sources: AECB (1990), Ontario Hydro (1990), Ontario Hydro (1991).
{1} Pickering A started.
{2} Bruce A started.
{3} Pickering B started.
{4} Bruce B started.

Problems with high tritium concentrations

These increasing tritium concentrations caused considerable problems in the past. An important problem was the increasing exposures to radiation to operators, from ingestion and

inhalation of tritiated water vapour. There also were problems with radiation degeneration of seals, resins and filters, resulting in increased shutdowns and lower capacity factors. In addition, in the late 1980s, the emissions caused high levels of tritium contamination at the (then) Ontario Hydro laboratories adjacent to the Pickering plant. This resulted in incorrect tritium measurements at the laboratory. In the end, the labs had to be closed and moved further away from the reactors.

There were environmental concerns too: in the early 1980s, Environment Canada grew alarmed at the large and growing discharges of tritium (see Finlay et al, 1982). Their studies showed that, without a facility for reducing tritium concentrations (then in design stage), tritium release rates would increase each year of operation (Environment Canada, 1986: see pages 195–197). The resulting code of practice (Environment Canada, 1986) pointed to several technologies for reducing tritium emissions in addition to a tritium reduction facility. This included proposals for the withdrawal and storage in tanks of moderator heavy water to permit the decay of tritium. However, the draft code's proposals were opposed by the nuclear industry and the AECB, and were not implemented (Ottawa Citizen, 1984; Toronto Star, 1984).

The nub of the problem was the very high tritium concentrations in the coolant and particularly the moderator. Before 1990, the only active way to reduce these was to release contaminated heavy water and replace it with tritium-free heavy water, but this meant higher tritium discharges. To allow the continued operation of the reactors, in early 1990 a tritium reduction facility (TRF) at Darlington was started up which extracts tritium from tritiated heavy water. This was licensed from the French Atomic Energy Commission and stems from a Swiss design. In this facility, tritium atoms on water molecules are transferred to deuterium gas molecules by means of catalytic exchange at 200°C. Then the tritiated deuterium (i.e., tritium gas) is separated from deuterium gas by cryogenic distillation at -250°C and then stored.

Ontario's reactors contain ~10,000 tonnes of moderator and coolant heavy water. It takes a number of years for this large inventory to be treated once through at the TRF. The theoretical maximum design flow rate of the TRF is 360 kg per hour (Sood et al, 1990 page 366). Assuming this maximum rate, 24 hours per day for 365 days, it would take over 3 years to put the 10,000 tonnes at Bruce, Pickering and Darlington through the TRF once. Each treatment reduces the tritium concentration by about 2/3rds. Under realistic batch-operating conditions, with shutdowns for maintenance and repairs, it is estimated to take about 4 to 5 years to put the 10,000 tonnes of heavy water at Bruce, Pickering and Darlington once through the TRF. During this time, more tritium would be created, therefore reducing the tritium concentrations in Ontario reactors is a slow process, and the TRF has to be run continuously to keep tritium concentrations at Bruce, Pickering and Darlington from increasing.

Other problems are the estimated 4,000 truckloads per year to transport heavy water to and from Darlington, and the TRF's own substantial high tritium emissions (790 TBq in 2005). In fact, the TRF's net contribution to reducing tritium discharges to the Ontario public is relatively small. Its main function is to keep tritium concentrations in Ontario reactors low enough to permit their continued operation.

Can tritium releases be reduced?

In the 1980s, Environment Canada tried to persuade the then-OH and AECB to undertake a number of technical steps to reduce tritium emissions. It is recommended that Environment Canada's suggestions in 1986 should now be reconsidered.

Chapter 3 Tritium Discharge Limits

Derived release limits (DRLs) are the amounts of a nuclide whose discharges are estimated to result in the public dose limit of 1 millisievert (mSv) per year not being exceeded, to members of the most exposed group (critical group) living near nuclear stations. This is a representative group of people thought to receive the highest doses: in some cases it will be a theoretical group. The dose is estimated by using computer models for environmental transport and nuclide intakes in humans. However, there can be considerable uncertainty about these dose estimates, as shown by the CERRIE (2004) report.

The limit applies to the sum of all man-made radiation exposures to the critical group, but the DRL calculation is done on each nuclide individually, as if it were the only one released.

The Canadian Nuclear Safety Commission (CNSC, 2005) has stated "… to ensure that the public dose limit is not exceeded, CNSC licences **restrict** the amount of radioactive materials that may be released in effluents from nuclear generating stations (emphasis added). These effluent limits are derived from the public dose limit and are referred to as derived release limits (DRLs). In addition, the nuclear generating stations set operating targets that are a small percentage of the derived release limits".

However, as can be seen in the tables below, the DRLs for tritium are extremely lax. They are in the range of hundreds of thousands of TBq of tritium per annum, that is, hundreds of times higher than actual discharges. Contrary to what the CNSC has stated above, its DRLs do not "restrict" the amounts actually discharged. In practice, nuclear stations are operated so that they actually discharge only a few percent of the DRLs.

Table 3.1 Derived release limits (DRLs) for tritium air and water emissions

Nuclear station	Tritium (HTO) emissions to air (TBq/a)	Tritium (HTO) discharges to lakes (TBq/a)
Bruce-A*	88,000	45,000
Bruce-B	93,000	600,000
Darlington	460,000 (HTO)	880,000
Darlington TRF	4,600,000 (HT)	NA
Pickering-A	70,000	170,000
Pickering-B	70,000	170,000
Gentilly-2	440,000	1,200,000
Point Lepreau**	430,000	16,000,000

Data source: CNSC (2005).
*Liquid DRLs for Bruce A are based on one condenser cooling water pump operating.
Higher liquid DRLs apply for 2, 3, or 12 pumps operating.
** DRL for tritium in liquid releases at Point Lepreau is higher than for the other nuclear generating stations, as the effluent is discharged to sea, i.e., there is no drinking water pathway.

TABLE 3.2 Combined limits for HTO (liquid and vapour) (TBq /year)

Country	Site	TBq /Year
Canada	Point Lepreau	16,400,000
Canada	Gentilly	1,640,000
Canada	Darlington	846,000
Canada	Bruce	826,000
Canada	Pickering	480,000
France	per site	1,800
Belgium	Doel	100
Germany	per site	70
Netherlands	Borssele	30
UK	Torness AGR	20
UK	Sizewell PWR	8

Sources: Belot et al, 1996; Commission of the European Communities, 1988; FSA, 2006.

The central point revealed by this table is that the CNSC's limits permit much greater discharges of tritium in Canada than is permitted in Europe. For example, Canadian limits are two to four orders of magnitude more lax than limits for nuclear reactors in European countries. It may be argued that the above table does not permit proper comparisons because electricity throughputs are not equalised, but we are looking at regulatory limits here and these are based on health considerations, i.e., the total amounts released.

Given the disparity between European and Canadian limits, it is difficult to see how health considerations have been applied in the two cases. These figures raise questions about Canadian DRL limits.

Chapter 4 Tritium Limit in Drinking Water

Canada Health has had a long history of adopting soft standards on radioactive pollutants compared to other pollutants, especially for tritium. For example, its former 1978 guidelines (Health and Welfare Canada, 1979) formerly recommended a very lax tritium limit of 40,000 becquerels per litre (Bq/L) for drinking water. Even then, it acknowledged that its limits for radionuclides were based on contested health risk estimates instead of the tighter concept of minimum detection levels used for most other pollutants.

In 1980, the Porter Commission (1980) on electric power in Ontario requested a Federal-Provincial Working Party to examine the question of radiological limits for drinking water. The Working Party recommended a chronic (lifetime) maximum concentration of 4,000 Bq/L. However, Canada Health and Welfare waited for another 14 years before it tightened the limit to 4,000 Bq/L in 1994.

The current Canadian Federal limit for tritium in drinking water is 7,000 Bq//L, which is very lax compared with the limits set by the European Commission and the US EPA. The current US limit[7] is 740 Bq/L, based on a maximum dose to the public of 40 μSv per year from drinking water.

In the US, the state of Colorado has set a stricter standard[8] for tritium in surface water, of 18.5 Bq/L[9]. The US state of California uses a limit[10] of 15 Bq/L. Both are based on a one-in-a-million lifetime risk of a fatal cancer, which is the goal of cleanup under the US Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), more commonly known as the Superfund.

Table 4.1 Tritium limit in drinking water (Bq/L)

Agency	Tritium limit (Bq/L)
Canada Health and Welfare	7,000
US EPA*	740
European Union**	100
Recommended by Ontario Government's ACES*** in 1994 but not implemented	100 (initially) 20 (after 5 years)
US State of Colorado	18
US State of California	15

*EPA (1999).
**European Commission (1998).
*** Advisory Committee on Environmental Standards.

Only one year's exposure assumed

The current Canadian Federal limit for tritium corresponds to a risk of 350 excess fatal cancers per million people. On the other hand, the Canadian Federal drinking water objectives for chemicals are set at levels that provide a lifetime risk of 1–10 excess fatal cancers per million

[7] 20,000 picocuries per litre.
[8] 500 picocuries per litre.
[9] For example, the US Department of Energy has specified the Colorado state action level for tritium in surface water in its clean-up program at the Rocky Flats plutonium plant in Colorado.
[10] 400 picocuries per liter.

people. The primary reason for the difference is that the excess cancers predicted from radiation exposure are calculated by assuming *one year's* consumption of drinking water: the lifetime risk is calculated as if that year of consumption were the only consumption. With chemicals, the assumption is that people consume the affected drinking water for their whole lifetime—commonly set at a 70-year exposure. Why such a difference should exist is strange—it's another example of the apparently favoured status of radiation in Canada.

Ontario Government's ACES report (1994): A sorry episode

In 1992, the Ontario Government requested its Advisory Committee on Environmental Standards (ACES) to enquire into this issue for setting an Ontario drinking water standard for tritium. It reviewed the above differences in approach to standard setting, and its report (ACES, 1994) concluded that it was inappropriate to assume only one year's consumption in calculating the fatal cancer risk.

At the time, 7000 becquerels per litre (Bq/L) for tritium had been proposed as a Federal drinking water guideline. ACES advised that in order to estimate the risk rationally and make it comparable with the approach used for chemicals, the 7000 figure should be divided by 70 years.

In addition, ACES recommended that the 7000 Bq/L limit should be further divided by 5 because the limit had relied on a risk of 5 fatal cancers per million persons being acceptable, when the more acceptable risk was 1 in a million. ACES thus arrived at its recommended figure of 20 Bq/L (i.e., 7000/350). However, ACES acknowledged that Ontario Hydro would find that meeting this limit immediately was not possible without shutting down the reactors, and it therefore suggested a less strict limit of 100 Bq/L for a 5-year period for Ontario Hydro to prepare for the new limit.

ACES also reviewed the history of creation of tritium exposure from human activities. It noted that routine releases of tritium from CANDU nuclear generating stations are among the world's largest sources of tritium. ACES agreed that tritium exposures should be reduced to background levels and therefore advised that the drinking water standard should be set at 100 Bq/L immediately, with a five-year phase-in period to 20 Bq/L. This was to give Ontario Hydro time to put into place any additional measures needed to reach a 20 Bq/L standard. ACES noted that neither standard should be a problem in any event, given that the average levels at drinking water intakes nearest the nuclear generating stations were generally below that level. ACES also acknowledged that one water intake—Port Elgin—was a problem.

The ACES report in May 1994 was welcomed with over 2,000 letters of support, but Ontario Hydro alleged that the ACES-recommended standard would cost billions to implement. In December 1994, the Provincial Government deferred to Hydro's views and announced a standard of 7,000 Bq/L, to widespread criticisms by environmental groups. This sorry episode for Ontario's environment is described in more detail by McMullan and Eyles (1999).

Chapter 5 Tritium Concentrations In The Great Lakes

As explained in Chapter 1, tritium is formed naturally in the upper atmosphere from the bombardment of cosmic rays. In addition, residual concentrations of tritium remain from atmospheric bomb testing in the 1950s and 1960s. The resulting background tritium level[11] in Lake Superior (which has no nuclear facilities) was 2 Bq/L in 1997/1998 (King et al, 1998). This is commensurate with background levels throughout Canada (i.e., very remote from nuclear facilities). Therefore this can be taken as a background level[12] for all the Great Lakes. However, the four Great Lakes other than Lake Superior have higher tritium levels resulting from nuclear discharges, as shown in Table 5.1. It can be deduced from this table that, on average, a third of present tritium levels in Lakes Huron, Erie and Ontario result from background and two-thirds from nuclear discharges.

Table 5.1 Average tritium concentrations in the Great Lakes in 1997/98

	Average tritium concentration (Bq/L)
Lake Superior	2
Lake Michigan	3
Lake Huron	7
Lake Erie	5.5
Lake Ontario	7.1

Sources: King et al (1998, 1999).

As the Canadian background tritium water concentration is 2 Bq/L, tritium levels in Lakes Huron, Erie and Ontario have been elevated 2 to 5–fold by tritium discharges, mostly from Canadian reactors.

The same is true of tritium levels in Ontario drinking water; these have also been increased 2 to 5 times by tritium discharges. From Ontario Ministry of Labour and Ministry of the Environment data (reported in Health Canada, 2006), the **average** tritium concentration in over 3,000 drinking water samples across Ontario was between 5 and 10 Bq/L during 2000–2006. Many of these samples will have been from lake supplies but some samples will have come from wells[13].

Near the Candu stations on Lake Ontario and Lake Huron, lake tritium concentrations are higher than the above averages. For example, the Ontario Ministry of Labour and Ministry of the Environment has observed tritium values of 120 Bq/L in drinking water at Southampton and 24 Bq/L at Port Elgin, both near the outflow of the Bruce nuclear station. A high value of 18 Bq/L was observed at the R.C. Harris water treatment plant near the outflow of the Pickering nuclear station in Toronto. (data from Health Canada, 2006) (No dates, tables or further explanations given).

[11] Tritium levels in the Great Lakes increased to about 20–30 Bq/L in the late 1950s and early 1960s as a result of atmospheric atomic bomb testing. This has now mostly decayed to about 1 Bq/L and constitutes a part—perhaps as much as a half—of background tritium levels.

[12] It appears that there are no published *average* levels more recent than 1998.

[13] It should be recalled that tritium in most wells will originate from tritium emissions to air and not tritium discharges to lake water.

As a result of discharges from nuclear facilities, tritium concentrations in the Great Lakes are gradually increasing. Tritium is removed from the Great Lakes by radioactive decay, sedimentation, and flushing (i.e., down the St Lawrence Seaway), but the annual amounts added are greater than the annual amounts removed. Chant et al (1993) estimated that the average tritium concentration in Lake Ontario was increasing by 0.12 Bq/L each year from Candu operations. Since Lake Ontario contains 1,640 km³ (1.64×10^{15} litres) of water, this means a net increase of ~200 TBq each year, which is a large amount.

History of tritium leaks into the Great Lakes

In addition to routine releases to surface waters, significant groundwater and lake contamination has occurred at Canadian nuclear facilities in the past.

- In 1979, tritium groundwater concentration reached 2.15 MBq/L following a release of 666 TBq at Pickering.
- In August 1983, a pressure tube at Pickering-2 ruptured due to embrittlement, dumping an unspecified amount of coolant into the reactor building.
- In September 1983, a leak of 222 TBq of tritium occurred from the Douglas Point reactor on Lake Huron. The prevailing counter-clockwise circulation pattern in the lake carried the tritium plume northeast to Port Elgin, where drinking water levels reached 1,600 Bq/L during a 2-day period.
- In January 1990, a loss-of-coolant accident at Bruce-4 resulted in a 12,000 kg leak of heavy water into Lake Huron.
- In June 1991, following a leak from Chalk River Nuclear Laboratories into the Ottawa River, the tritium concentration in drinking water at Petawawa was about 400 Bq/L. At Ottawa (200 km downstream), the tritium level was ~150 Bq/L.
- In August 1992, a tube break at Pickering-1 caused the release of 2,300 TBq of tritium into Lake Ontario. A nearby drinking water plant was shut down and elevated levels of tritium (up to 195 Bq/L) were found in Toronto drinking water.
- In May 1994, Ontario Hydro found a tritium groundwater concentration of 0.7 MBq/L following a leak at Pickering.
- In December 1994, a valve failure at Pickering-2 led to 140 tonnes of heavy water being discharged into Lake Ontario.
- In May 1995, a valve failure at Bruce-5 caused a 25-tonne leak of radioactive heavy water.
- In April 1996, a heavy water leak at Pickering-4 released 50 GBq of tritium into Lake Ontario: tritium levels in local drinking water reached 100 times background levels.
- In July 1997, it was revealed that Ontario Hydro (the predecessor to OPG) had failed to report tritium contamination of groundwater on the Pickering site for a period of 20 years.

The raised tritium concentrations in drinking water, the continued rise in tritium levels in most Great Lakes, and the many spills of large quantities of tritium are all matters of concern. This is particularly the case for the health of women (see Bertell, 2005). As a matter of public health policy, it is unfortunate that discharges of tritium are made near drinking water intake pipes for large populations in Ontario. At the least a more precautionary approach should be adopted. Operators of tritium-emitting facilities should give further consideration to other ways and means of reducing tritium emissions.

Chapter 6 Tritium-In-Air Concentrations Near Nuclear Stations

Raised tritium levels in drinking water are a serious matter, but raised tritium levels in air are even more serious. This is because, near Candu stations, tritium exposures from skin absorption, inhalation, swimming, and food ingestion (all contaminated by tritium water vapour) are larger than tritium exposures from drinking water. The same is likely to be true for collective doses from tritium in air and in water. For this reason, it is important to pay particular attention to tritium-in-air concentrations near nuclear reactors.

The process of tritium intake

Tritium air emissions from Candu reactors are in the form of tritiated water vapour. This water vapour travels downwind of the reactor and, via the (not widely known) process of atomic exchange, tritium rapidly enters all material containing hydrogen. This includes all biota, including all plants, animals and of course, people. For example, it enters fruit and vegetables in market stalls and shops, including supermarkets (Inoue et al, 1993). Put succinctly, all biota including humans become tritiated to the ambient levels in the environment (as shown in Figure 6.1). The level of tritium contamination depends on tritium concentrations in water vapour emissions from the reactors.

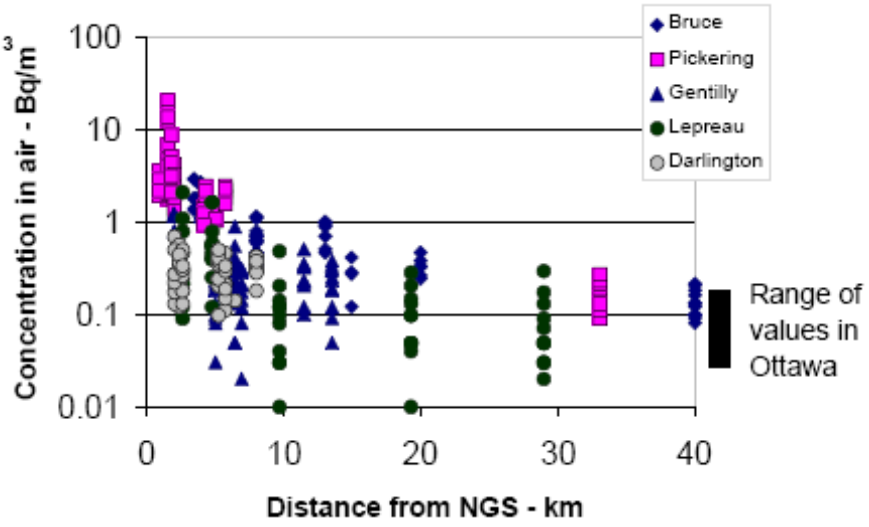
More worryingly, some of this absorbed tritium is fixed into carbon molecules during the growing season in plants, and in animals during cell reproduction and cell metabolism. This is called organically bound tritium (OBT) and is an important component of tritium exposures. When there are repeated (i.e., chronic) exposures to tritium, as occur near nuclear reactors, OBT concentrations gradually increase in all biota. After a few years, average OBT levels are a significant fraction of tritiated water levels in Ontario sites near Candu reactors (Osborne, 2002). Unfortunately, the ICRP and CNSC refuse to fully recognise these increases in OBT concentrations from repeated exposures, in their dose models for tritium (see Part 2).

Tritium levels in water vapour in air

Tritium air concentrations are often expressed in Bq in *air*. However, we need to know the amount of tritium in the air's *water vapour*. For example, if the air concentration is given as 1 Bq/m^3 of air and the water vapour content in local air is ~10 grams per m^3 (a representative value, see Davis et al, 1996) then people in the locality are breathing in and absorbing water vapour with a tritium concentration of 100 B/L. **This latter figure (i.e., the tritium concentration in air's water vapour) is the crucial parameter in estimating doses to people living near Candu reactors.**

Tritium concentrations in water vapour near nuclear stations depend on the distance from the station. This is shown in Figure 6.1, reproduced with the permission of the CNSC from Osborne (2002), using data from Health Canada (2001).

Figure 6.1 Annual averages of tritium concentrations in air measured at distances from nuclear power stations in Canada, 1985–1999.



Sources: *Tritium in the Canadian Environment: Levels and Health Effects*. Report RSP-0153-1. Prepared for the Canadian Nuclear Safety Commission under CNSC contract no. 87055-01-0184 by Ranasara Consultants and Richard Osborne. Data from Health Canada (2001).

A number of points need to be made here. The first is that the logarithmic scale of the Y- axis: compresses the data range. Although this is needed for a table of manageable size, it is slightly misleading in that the range of concentrations appears not to be large. This is not the case; for example, the highest air concentration here (30 Bq per cubic metre) is 3000 times greater than the lowest (0.01 Bq per cubic metre).

A second point is that, as explained above, we need to know the tritium concentration in *water vapour* and not in air. If we assume a reasonable value of 10 grams of water per cubic metre of air, then the tritium water vapour concentration 1 to 2 km from Pickering is 100 to 3,000 Bq/L. The latter is a high value.

Third is that the suggested background range (0.04 to 0.13 Bq/m^3) at Ottawa is not free of man-made tritium. Although Ottawa is 190 km from AECL Chalk River, the nearest reactor, it still is subject to raised tritium levels because it is downstream from it. For example, AECL (Neimi et al, 2002) has reported that discharges from Chalk River Laboratories to the Ottawa River increased the tritium concentration from about 3 Bq/L to 10 Bq/L, 10–30 km downriver: there would have been slightly raised levels all the way to Ottawa. Also in June 1991, Health Canada monitored the levels of tritium in the Ottawa River following a spill from Chalk River Nuclear Laboratories. The concentration in drinking water at Ottawa (downstream) was about 150 Bq/L.

Fourth, Figure 6.1 indicates tritium concentrations in *selected* materials, and of course, ALL environmental materials are tritiated near Candu reactors. This includes water in rivers, streams and drinking water in wells. Osborne (2002) has reported tritium concentrations ranging up to 30 Bq/L in one well close to the Pickering station and about 60 Bq/L in seasonal wells close to the Bruce station. Concentrations of tritium in drinking water wells close to an

unnamed “small tritium-handling facility” ranged up to 230 Bq/L, with most measurements being less than 50 Bq/L.

Fifth it is useful to multiply the Y axis values by 100 and label it as tritium in water vapour in air. This shows that these values can rise to 3,000 Bq/L close to the Pickering station.

Finally, yet importantly, the data points are *annual averages*. Actual air concentrations will vary considerably, which is worrying as large pulses of tritium emissions may occur but these are obscured by the publication only of average concentrations (and annual ones, as well). Pulsed tritium concentrations could in theory result in heavy labeling of cells being formed in the embryos and fetuses of pregnant women at that particular moment. This fear was expressed by Professor E. Radford in his 1979 testimony to the Ontario Government’s Select Committee on Ontario Hydro Affairs (Hearings on The Safety of Ontario's Nuclear Reactors, Tuesday, July 10, 1979, http://www.ccnr.org/tritium_2.html#scoha).

The real background level of airborne tritiated water vapour

It is important to know the true background concentration of airborne tritiated water vapour in Canada, so that we can deduct this value from observed values near nuclear stations to establish what concentrations are due to the discharges. The difficulty is that most tritium measurements are made near nuclear stations and one has to be very remote, at least >300 km away, in order to escape from low levels of tritium contamination from nuclear stations. Only a few tritium-in-air measurements are made very remote from nuclear facilities. One is in Table 3.3 of the Bruce Power Group’s *Annual Summary and Assessment of Environmental Radiological Data for 2005* (Bruce Power, 2006). This shows that the average tritium concentrations in precipitation at Calgary AB, Saskatoon SA, and Fredericton NB are all below the limit of detection of the instruments used. This limit was 3.7 Bq/L, which points to the air concentration[14] being lower than 0.037 Bq/m^3.

However, remote measurements in water (see Table 4.2) and in vegetation (see Table 7.1), both of 2 Bq/L of water, give support to the view that the background level for tritium in atmospheric moisture in Canada is about 2 Bq/L of water, which translates to ~0.02 Bq/m^3 of air.

The value of 0.02 Bq/m^3 is very near the bottom line of Figure 6.1. Therefore it can be seen that most of the data points in Figure 6.1 are 10 to 100 times higher than background levels. In particular, the tritium-in-air moisture levels 1 to 2 km from Pickering as reported by Health Canada (2002) are **50 to 1,500 times higher than the Canadian background level**.

Historical trends

Given the importance of tritium-in-air concentrations, it is relevant to examine their historical trends and it is reassuring to note that at Ontario reactors these have declined since ~1989. See figures below from OPG (2006) and Bruce Power (2006). These declines are probably due to the commencement of the TRF at Darlington in 1990. Before 1990, stocks of heavy water were withdrawn from OH reactors to supply the initial feed tanks at the TRF.

[14] The real figure will depend on the amount of water in each cubic meter of air at the place of measurement. This varies but an average figure of 10 grams per cubic meter is often used (Davis et al, 1996)

Figure 6.2 Pickering nuclear site tritium-in-air trend

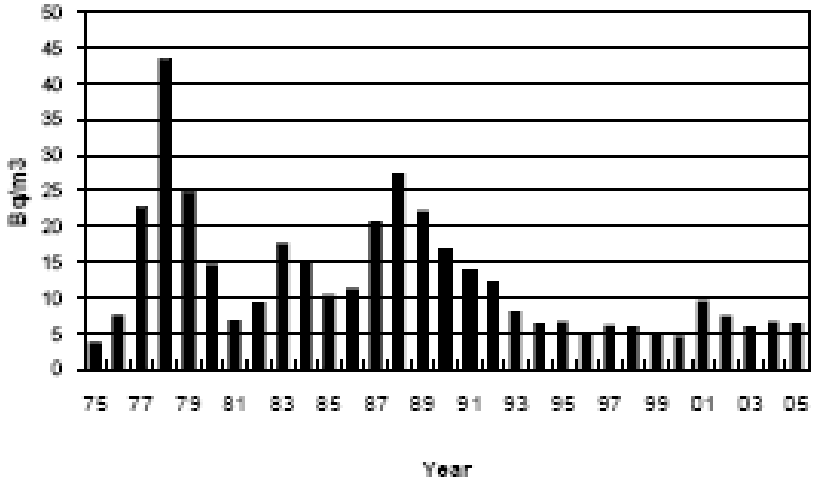


Figure 6.3 Darlington nuclear site tritium-in-air trend

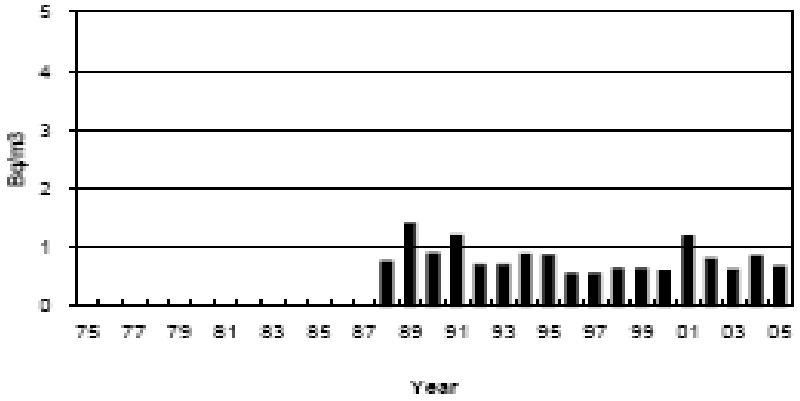
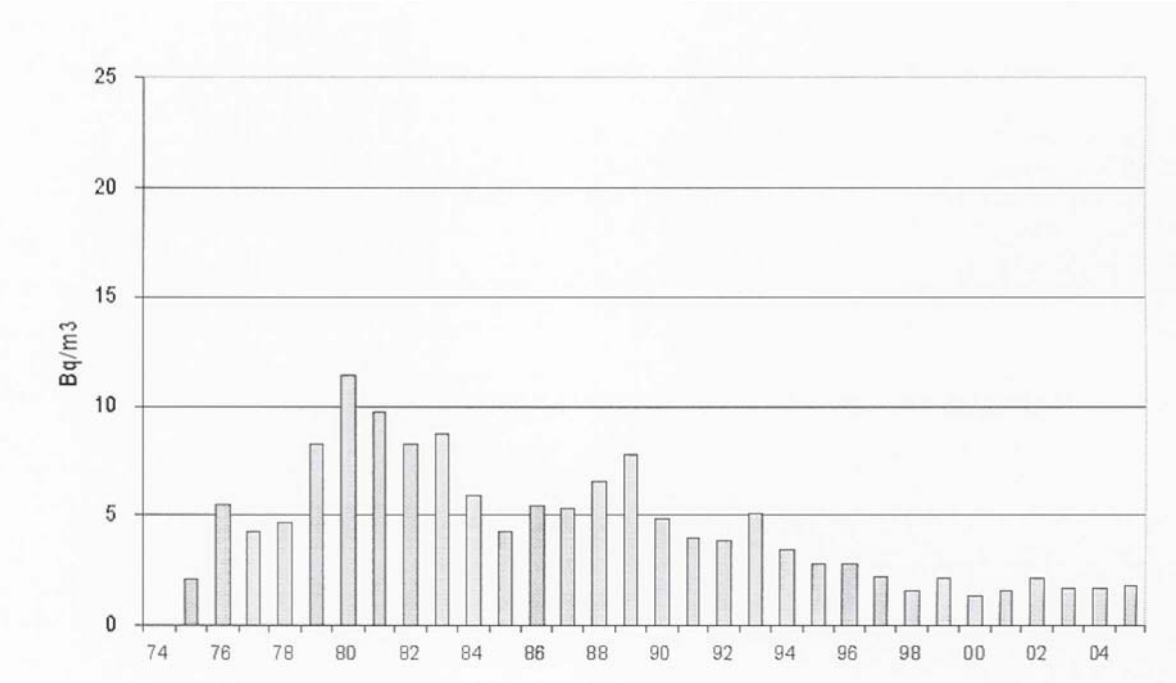


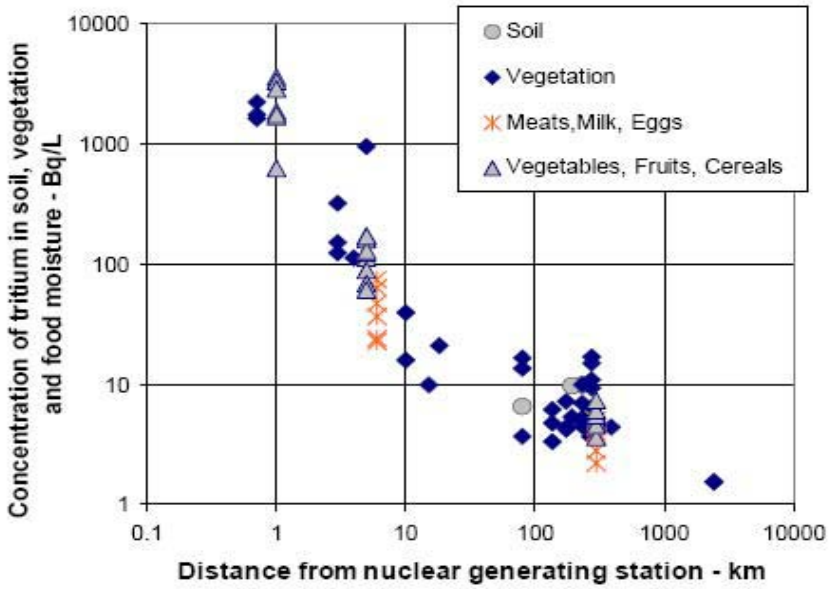
Figure 6.4 Tritium-in-air at Bruce Power indicator sites—annual average



Chapter 7 Tritium Concentrations in Food

The tritium-in-air described in the previous chapter is discharged continuously from Candu stations. As a result, the moisture in vegetation and foods in areas near them become tritiated to levels similar to the tritium concentrations in the moisture of downwind air. Figure 7.1 shows that as we go further way from the station, the concentrations in air and therefore in vegetation decline.

Figure 7.1 Tritium concentrations in the moisture of vegetation and foods



Sources: Reproduced with permission of the CNSC from *Tritium in the Canadian Environment: Levels and Health Effects*. Report RSP-0153-1. Prepared for the Canadian Nuclear Safety Commission under CNSC contract no. 87055-01-0184 by Ranasara Consultants and Richard Osborne. Data from Health Canada (2001).

This figure shows tritium concentrations in the moisture of vegetation, soil and locally produced foods at different distances from a nuclear generating station. The **vegetation and soil** data are for samples collected in 1997 and 1998 near the Pickering reactors (Kotzer et al, 1999). The **food** data are for locally produced samples collected at the Pickering station, Ajax, and Perth, in 1990 (Brown, 1995).

It is interesting to compare these data with the annual averages of tritium-in-air concentrations shown in Figure 6.1. There is a good fit between the data in the two figures, showing that tritium concentrations in food and vegetation moisture are directly the result of tritium-in-air concentrations.

All of these data show that tritium concentrations in food and vegetation in Canada, even hundreds of kilometres from nuclear power stations, are elevated above the background of readings even further distant from nuclear stations. The benchmark level is that set by the data point to the extreme lower left in figure 7.1, i.e., 2 Bq/L found about 3,000 km from the nearest nuclear station. This is the background level from which all other concentrations need to be measured.

Chapter 8 How Much Tritium Do People Take In?

In previous chapters, we have examined the high levels of tritium discharges to the Great Lakes and to the atmosphere from Candu reactors. Next, we examined the resulting high tritium concentrations in areas near Candu reactors. Now we turn to the question: do these high tritium concentrations in air and water result in high tritium concentrations in people living near Candu reactors?

To answer this requires us to add up the tritium that people take into their bodies from each source every year. There are four main sources:

- from breathing tritium-contaminated air,
- from eating tritium-contaminated food,
- from drinking tritium-contaminated water, and
- from skin absorption of tritium-contaminated water vapour.

In order to calculate these amounts we need to know dietary habits and breathing and eating rates etc. These have been compiled by Health Canada (1994) from a national habit and diet survey[15]. These values, together values for air and drinking water intakes from Health Canada (2001), allow one to estimate intakes. The values for adults are shown in Table 8.1.

Table 8.1 Annual food, water and air intakes by adult Canadians

Source of tritium	**Annual intake**
Vegetables	90 kg/yr
Fruit	70 kg/yr
Meat, poultry, fish and eggs	70 kg/yr
Dairy products	100 kg/yr
Cereals	90 kg/yr
Miscellaneous	70 kg/yr
Total foods	490 kg/yr
Drinking water and made-up drinks	550 litres per year
Air	8,400 cubic metres per year

Source: Health Canada (1994)

If we multiply together the above average rates by the measured tritium concentrations shown in Figure 6.1, we arrive at annual tritium intakes. We do this (similar to the examples used by Osborne, 2002) for three types of tritium exposures:

(a) areas very remote (i.e., >300km) from any nuclear facility,
(b) areas near (5–10 km) to Candu facilities, and
(c) areas very close (1–2 km) to Candu facilities.

Table 8.1 below shows the intake of tritium for a Canadian adult living very remote (>300 km) from nuclear facilities. It shows average annual intakes are 2,200 Bq of HTO and 200 of OBT.

[15] Daily rates in Health Canada (2001) are here multiplied by 365 days per year.

Most comes from drinking water (50%) and food (39%). These estimates are similar to those by Osborne (2002) who, using different assumptions, estimated that adults living in areas "close to the general Canadian background" would have annual intakes of 5,900 Bq of HTO and 350 Bq of OBT.

In the tables below, all totals are rounded to two significant figures.

Table 8.1 Annual Intake of tritium for **Canadians living far away from nuclear facilities** (i.e., background tritium intakes)

Source of water tritium (HTO)	**Intake per annum**	**HTO concentration**	**HTO Bq/year**
Water in drinks	550 l	2 Bq/L	1,100
Water in food	500 kg x 0.85 = 425 litres	2 Bq/L	850
Air inhalation	8,400 m^3	0.02 Bq/m^3	170
Skin absorption	60% of inhalation	0.02 Bq/m^3	100
TOTAL			**2,200**

Source of organic Tritium (OBT)	**Intake per annum**	**OBT Concentration**	**OBT Bq/year**
OBT in food	500 kg x 0.15 = 75 kg	~2.6 Bq/kg	~200
TOTAL			**~200**

Sources: Data from Osborne (2002), Trivedi et al (1997).
Assumptions:

- background tritium level is 2 Bq per litre
- 1 kg water = 1 litre
- average specific activity of OBT = 1.3 x average specific activity of HTO (Osborne, 2002)
- 2 litres of water consumed per day in drinks
- on average, 85% of food is water, 15% is organic matter
- annual skin absorption is 60% of inhalation intake (Osborne, 1966)

If we compare these estimates with the annual tritium intake of an adult living in the area further away but still relatively **near** (i.e., **within 5 to 10 km**) to a Candu facility, the tritium uptakes are appreciably higher, as seen in Table 8.3.

Table 8.3 Annual Intake of tritium for **Canadians living near a nuclear facility (within approximately 5 to 10 km)**.

Source of tritium (HTO)	**Intake per annum**	**HTO concentration**	**HTO Bq/year**
Water in drinks	550 litres	30 Bq/L, from Osborne (2002)	16,500
Water in food	500 kg x 0.85 = 425 l	100 Bq/L, from Osborne (2002)	42,500
Air inhalation	8,400 m^3	1 Bq/m^3, from Osborne (2002)	8,400
Skin absorption	60% of inhalation intake	1 Bq/m^3	5,040
Swimming	0.024 l per hour x 100 hours = 2.4 l	30 Bq/L	70
TOTAL			**73,000**

Source of tritium (OBT)	**Intake per annum**	**OBT concentration**	**OBT Bq/year**
OBT in food	500 kg x 0.15 = 75 kg	80 Bq/g estimated from figs 2 and 3 of Osborne, 2002	6,000
TOTAL			**6,000**

Sources: Data from Osborne (2002) and checked with data in Trivedi et al (1997).
Assumptions: See Table 8.2.

The main point from this table is that tritium intakes for those living near (5 to 10 km) to Candu reactors are 30 to 40 times greater than background intakes. Here water in food (58%) is a more important source than drinking water (23%). Note that in this exercise, drinking water is from a well, i.e., contaminated by tritium emissions to air and not tritium discharges to lakes.

These estimates are not too different from estimates by Osborne (2002) of 67,000 Bq for HTO and 7,000 for OBT per year. Trivedi et al (1997) calculated that the annual uptakes in adults living in Deep River, Ontario (10 km from the AECL Chalk River reactor) were about 20,000 Bq of tritium as HTO and 800 Bq of tritium as OBT. The above estimates are not inconsistent with those from Trivedi et al, taking into account that tritium discharges from Chalk River laboratory are considerably lower than those from the larger Pickering, Darlington and Bruce nuclear power stations.

Finally, if we compare these estimates with the annual tritium intake of an adult who lives very near a Candu reactor, i.e., within 1 to 2 km, and who eats produce from his/her own garden, the tritium intakes are much higher, as shown in Table 8.4.

Table 8.4 Annual Intake of tritium for **those living very close to a nuclear facility (1 to 2 km)** who eat produce from a highly contaminated garden

Source of tritium (HTO)	**Intake per annum**	**HTO concentration**	**HTO (Bq/year)**
Water in food	500 kg x 0.85 = 425 l	~2,000 Bq/L read from Fig 7.1	850,000
Air inhalation	8,400 m^3	10 Bq/m^3	84,000
Water in drinks	550 litres	100 Bq/L from Osborne, 2002	55,000
Skin absorption	60% of inhalation intake	10 Bq/m^3	33,000
Swimming	0.024 l per hour x 100 hours/year = 2.4 litres	100 Bq/L	2,400
TOTAL			**~1,000,000**
Source of tritium (OBT)	**Intake per annum**	**OBT concentration**	**OBT (Bq/year)**
OBT in food	500 kg x 0.15 = 75 kg	~700 Bq/kg estimated from figs. 2 and 3 in Osborne, 2002	~53,000
TOTAL			**~53,000**

Sources: Data from Osborne (2002), Trivedi et al (1997).
Assumptions: See Table 8.2.

This table indicates that people living very near Candu reactors can receive large amounts of tritium, with annual intakes of 1 million Bq per year of HTO and a third of a million Bq per year of OBT. These values are 500 to 1,400 times greater than Canadian background levels. They indicate that people living very near Candu reactor sites who eat produce from their gardens are likely to be highly contaminated with tritium. In comparison, Osborne (2002) estimated that the intakes of those living very close to Candu reactors were two thirds of a million Bq per year of HTO and 42,000 Bq of OBT. These are not too different from the above estimates. Notice that about 90% of their tritium (water) intake is from food and if the OBT from food is added, it can be seen that almost all their tritium intake is from food. These results are summarised in tables 8.5 and 8.6.

Table 8.5 Annual HTO tritium intakes at different distances from a Candu facility (Bq per year)

Location km away from Candu	**This study Bq HTO per year**	**Osborne, 2002 Bq HTO per year**
>300 km	2,200	5,900
5–10 km	73,000	67,000
1–2 km	1,400,000	640,000

Table 8.6 Annual OBT tritium intakes at different distances from a Candu facility (Bq per year)

Location km away from Candu	**This study Bq OBT per year**	**Osborne, 2002 Bq OBT per year**
>300 km	200	350
5–10 km	6,000	7,000
1-2 km	53,000	42,000

From tables 8.5 and 8.6, it can be seen that the closer people live to Candu facilities, the greater their tritium contamination and tritium intakes. It can also be seen that the relationship between distance from reactor and intake is not linear but exponential, so that people living close to the reactors who eat produce from their own gardens are very highly contaminated indeed.

Are these levels hazardous?

To answer this we have to define a level of risk we are prepared to accept. Then we have to calculate how much tritium a member of the public could ingest/inhale/absorb without exceeding this risk.

It is usually agreed that an annual[16] risk of about one in a million of dying from cancer from a radiation exposure is acceptable. Using this risk and assuming an average adult consumes 730 litres of drinking water per year, the European Union (1998) has estimated that the maximum concentration of tritium in drinking water is 100 Bq per litre - see table 4.1. This means 730 litres per year x 100 Bq per litre = **73,000 Bq per year**. Notice that this limit is for the total for tritium intake in a year, and no more (man-made) radiation would be allowed, otherwise the maximum risk would be exceeded.

Applying this suggested maximum to table 8.5, reveals that most people living within 5 to 10 km of Candu stations would have this level of intake. Those living very near (1 to 2 km) would have ten to twenty times that amount. And this does not take into account OBT intakes which will occur simultaneously. Therefore the answer to the question “Are these levels hazardous?” is yes, by European standards.

Another, simpler, way of looking at the matter is as follows. Scientists working repeatedly with radionuclides tend to develop “rules of thumb” for dealing quickly with radioactivity quantities. Although rough and ready, they are not unscientific. One of these is that intakes (of beta-gamma emitters) greater than 100,000 Bq would trigger the need for some kind of action or investigation. And this is what would strike most radiation protection scientists about the intake estimates for those living close to Candu stations discussed in this chapter. However official agencies state that because these intakes are of tritium (and not any other nuclide), the “doses” are low and their intakes are therefore below public health limits. We shall discuss how “safe” these tritium levels are in the next chapter.

[16] lifetime risks were discussed in chapter 4.

Chapter 9 Are High Tritium Levels Hazardous to Health?

In previous chapters, we examined the high tritium discharges to the Great Lakes and to the atmosphere from Candu reactors. Next, we examined the resulting high tritium concentrations in vegetation and water near Candu reactors. In the previous chapter, we examined the resulting high tritium intakes in people living near Candu reactors. Now we examine how hazardous these levels of tritium intake are.

To do this is a seemingly simple step—one multiplies the totals in tables 8.3 and 8.4 by the official ICRP "dose factors" for HTO and OBT. A dose factor converts each Bq of a nuclide to a radiation "dose" expressed in sieverts (Sv). One then compares this calculated dose to the present Canadian public dose limit of 1,000 microsieverts (μSv) per year.

The problem, and here we get to the nub of the matter, is that the official dose factors for HTO and OBT are the smallest (by considerable margins) of all common radionuclides. For example, the ingestion dose coefficient for tritiated water (1.8×10^{-11} Sv per Bq) is ~700 times lower than that for Cs-137, a common man-made radionuclide. When tritium's tiny dose factors are used to calculate doses, the resulting doses are miniscule. For example, according to official dose factors, the highest exposed persons in Table 8.4 above only receives about 20 μSv per year from tritium (i.e., 50 times lower than the annual 1000 μSv safety limit). Therefore Canadian health agencies and radiation regulators consider tritium discharges not to be dangerous.

But these official considerations depend crucially on tritium's dose factors' being correct. Are they correct? The answer is so important that we have devoted Part 2 of this report to it. The box below contains a brief précis of Part 2.

Summary of Part 2: The Radiation Hazards of Tritium

Many scientists have expressed concerns about tritium's low dose factors and its radiotoxicity: indeed, demurring views about tritium's official doses have existed for decades (for more recent, see Fairlie, 2007; Harrison et al, 2003, and CERRIE, 2004).

Part 2 reveals that a number of radiation protection precepts and procedures are deficient or inappropriate when determining tritium's hazards. The report's main arguments about tritium are summarised below.

1. Tritium's unusual properties of extremely high mobility, exchangeability, and binding with organic materials are not properly recognised by official dose models.
2. Because of the short range of tritium's beta particle, tritium's damage depends on its exact location in the cell. For example, tritium next to a DNA molecule exerts more damage than tritium in, say, extracellular water. At present, it is not possible to model where tritium goes in the body with any accuracy. Official models assume that tritium (HTO) is distributed equally throughout the body, but we don't know that. Some scientists think we should use safer models, in case equal distribution turns out to be wrong.
3. Tritium is often described as a "weak" beta-emitter, but in radiation biology, so-called "weak" beta particles are more effective (i.e., dangerous) than energetic ones. This is especially the case with tritium, but this is not acknowledged in setting its dose factor.
4. Much evidence indicates that tritium's effectiveness (in radiation biology experiments

comparing tritium with gamma rays) is two or three times that recognised by the ICRP.

5. Little official recognition is given to tritium's ability to incorporate in organic molecules to high levels as a result of chronic exposures that occur near Candu reactors.
6. Official dose models for organically bound tritium therefore significantly underestimate its doses.

From these points, examining tritium's doses tends to turn into a critique of current official radiation protection precepts and practices. This is unfortunate, because few people outside of the nuclear industry and its regulators understand these. However, it is important that an effort is made, in order to appreciate the true degree of tritium's hazards.

In a nutshell, the second part of this report concludes official attitudes on tritium are unscientific and incorrect. It concludes that the recent evidence of tritium's hazards should be acknowledged by radiation protection agencies in Canada, that a precautionary approach should be adopted with dose factors for HTO and OBT being significantly increased.

Recent reports

Very recently, a number of reports have been published which also raise questions about tritium's dosimetry. These include the US IEER report (Makhijani et al, 2006—see Chapter 7), which recommends a much stricter US EPA standard for tritium in drinking water. In addition, the reports of the UK Government's CERRIE Committee (2004) and AGIR (2007) Committee both examine questions surrounding current methods of determining tritium doses. Also, in a very recent article, the author (Fairlie, 2007) examines objections to ICRP's current approach to the effectiveness of tritium's beta decay particle. These recent articles and reports should be studied by the Federal Canada and Ontario Governments and by the CNSC.

The recent history of the CERRIE Committee in the UK is instructive on the debate about tritium. This Committee was set up by the UK Government in 2001 because of concerns about the risks of internal radiation. Tritium is an example, par excellence, of internal radiation. Because its external radiation is considered harmless, it is only an internal hazard. The Committee spent a great deal of time considering tritium's risks and examining proposals for increasing tenfold HTO's dose factor, and for increasing OBT's dose factor to five times greater than that for HTO. In the end, the Committee could not reach a consensus on tritium's risks, despite much constructive discussion. (See the documents on tritium in www.cerrie.org.) Most official representatives voted for current values, while representatives of environment groups voted for increased values. Nevertheless, the UK Government's permanent COMARE Committee in its response to the CERRIE Report recommended yet more investigation into tritium's dosimetry. The outcome has been the recent report of the UK's AGIR Committee (2007) which recommended (at least) doubling HTO's dose factor.

Would increasing tritium's dose factors make a difference?

If we increased tritium's dose factors significantly (e.g., for HTO's dose factor to be increased 10-fold and for OBT's dose factor to be 5 times greater than HTO's dose factor), would this result in different conclusions? The answer is yes. For example, the revised radiation dose to the most exposed persons in Table 8.4 would be well over the public dose limit of 1,000 μSv per year.

Occupational doses

We have not considered the matter of occupational doses mainly because of the lack of available data, but there may be a problem with members of the public who live with a worker who is occupationally exposed to tritium. Workman et al (1998) showed that the indoor air of such homes had 70-fold elevated tritium levels (compared with outdoor concentrations), and that their daily tritium intake was 18 times higher than adults living in a non-occupationally exposed home. (See also Part 2, Appendix VII.)

Epidemiology

An obvious question is, can we see any adverse health effects at locations with high tritium concentrations? Health effects can sometimes be spotted by means of epidemiology studies, particularly among those who are most exposed. But radiation health effects are notoriously difficult to pick up because cancers caused by radiation are not different from naturally-occurring cancers, and there are many such cancer cases; indeed in the UK about 25% of all deaths are from cancer[17]. This requires us to look for small increases in cancer rates among exposed populations which already suffer many cancers. It is difficult to pick up small increases in radiation-induced cancers (the signal) among the many natural cancers (the noise). Very large expensive epidemiology studies are required to get a big enough signal to satisfy scientists that that there really is an effect and that any increase has not occurred by chance or from statistical blips.

In any event, relatively few such studies have been carried out. (See Appendix VII to Part 2.) These have shown increases in childhood leukemia but these studies are first-stage studies (often termed “ecologic”) which may be subject to bias such as social class, or to confounding factors such as smoking. The result is that their findings are indicative, not conclusive. What are needed now are second-stage (i.e., case-control or cohort studies) to match cases of cancer with people who do not have cancer, to obtain more conclusive results. These should have been carried out after the first-stage studies in the 1980s and early 1990s showed signs of increased incidences of leukemia in affected areas.

[17] The figure for Canada is thought to be similar.

Chapter 10 Conclusion and Recommendations

This report has examined tritium discharges from Canadian nuclear power stations, which are much greater than from nuclear power stations in other countries. Canadian tritium discharge limits are considerably less stringent than those in other countries. Canadian tritium limits in drinking water are also considerably more lax than those in Europe and the US.

The report also shows that tritium levels in those Great Lakes with nuclear reactors on their shores are 2 to 5 times greater than in Lake Superior, which has no reactors. The tritium level in Lake Ontario is also increasing each year, mainly due to Candu discharges and to major tritium leaks in past years. Tritium concentrations in drinking water, in air, and in vegetation and food near Candu stations are all significantly increased. These result in turn in high tritium intakes in residents living within 5 to 10 km of Candu stations and very high tritium annual intakes in residents who live within 1 to 2 km of the Candu stations.

However, because of tritium's very low dose factors, the radiation "doses" which result from tritium exposures are very small and are considered safe or within health limits by Canada's nuclear regulators. But the report points to significant objections to tritium's dosimetry and to the models used to estimate tritium doses, especially from organically bound tritium. It notes a number of recent reports in the UK (AGIR, 2007; Fairlie, 2007; CERRIE, 2004) and the US (Makhijani et al, 2006) which raise questions about tritium's official dosimetry.

The report concludes that scientific concerns about tritium's hazards are inadequately recognised by the Canada's nuclear regulators. It therefore recommends that a precautionary approach to tritium discharges should be adopted in Canada. In particular, it recommends that:

1) the Ontario and Federal governments should establish a committee (whose members should include scientist representatives from environmental groups) to examine tritium's dosimetry and risks. In particular, the committee should examine recent authoritative reports which raise questions about currently-accepted views on tritium's dosimetry and risks;
2) case-control and cohort epidemiology studies should be commissioned to examine possible adverse health effects in tritium-contaminated areas;
3) pregnant women and young (less than 4 years old) children and their mothers should be advised not to live near tritium-emitting facilities (i.e., within 10 km);
4) people who live very near (i.e., within 5 km) tritium-emitting facilities should be advised not to consume food from their own gardens, bee hives and orchards, and not to consume wild foods, e.g., blackberries and mushrooms, growing very near the facilities;
5) because tritium reduction facilities themselves release large quantities of tritium, nuclear reactor operators should be requested to examine the option of long-term storage of tritiated water from moderator circuits in decay tanks as a way of reducing tritium discharges; and
6) operators of tritium-emitting facilities should give further consideration to other ways and means of reducing tritium releases.

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Annex 1 Carbon-14

Canada's reactors also discharge relatively high amounts of carbon-14. Carbon-14 and tritium have similar properties; for example, both bind with organic molecules in plants and animals.

A major difference between them is that carbon-14's half-life is 5,730 years, which is ~460 times greater than tritium's half-life of 12.3 years. This means that C-14 is a potent contributor of radiation doses to future populations—not just in Canada but in the rest of the world, as C-14 is distributed globally. The estimation of collective doses to Ontario and to Canadian populations from Candu releases is a task for the future.

The important point is that tritium's discharges are 100 to 1,000 times greater than C-14 discharges. For this reason, the report has examined tritium discharges. However, any future epidemiological studies will need to take into account exposures to C-14 as well as to tritium.

The following tables report C-14 releases and DRLs at Canadian nuclear plants.

Table A Air releases of carbon-14 from nuclear power plant facilities (TBq/a)

Facility	2001	2002	2003	2004	2005
Bruce A + B	3.09	2.49	4.81	3.81	10.44
Pickering A + B	6.46	1.99	3.7	2.7	4.8
Darlington	3.5	2.8	3.5	1.9	1.6
Gentilly-2	0.40	0.37	0.39		
Point Lepreau	0.22	0.29	0.21		
TOTALS	13.67	7.94	12.61		

Sources: NPRI, 2004, 2003, 2002.

Table B Water releases of carbon-14 from nuclear power plant facilities (GBq/a)

Facility	2001	2002	2003	2004	2005
Bruce A + B	9.5	8.5	8.2	14	14
Pickering B	3.3	1.5	0.1	4	5.5
Darlington	3	1.7	1.2	0.4	0.28
Gentilly-2	0.3	0.3	0.3		
Point Lepreau	2.8	3.4	1.8		
TOTALS	18.9	15.4	11.6		

Sources: NPRI, 2004, 2003, 2002.

Annex 2 Wind Frequencies for Darlington and Pickering

The table below indicates that wind frequencies are fairly well spread around the compass at both locations, with a slight preponderance of winds from the West.

Table A Wind Frequency by Direction (at 10 m height)

Direction from	**Darlington nuclear wind frequency (%)**	**Pickering nuclear wind frequency (%)**
N	3.64	7.8
NNE	4.03	4.56
NE	8.22	4.6
ENE	8.67	6.17
E	4.65	5.94
ESE	3.22	3.32
SE	2.15	1.77
SSE	1.94	2.56
S	2.36	8.9
SSW	6.97	9.17
SW	9.22	4.91
WSW	7.13	4.9
W	9.09	7.29
WNW	9.69	9.27
NW	12.42	9.39
NNW	6.59	9.43
Total	100	100

Annex 3 Tritium Emissions from SRB Technologies, Pembroke, Ontario

In the past, the SRB Technologies facility at Pembroke, Ontario emitted very large quantities of HT gas and HTO water vapour (see table below.) In recent years these discharges have declined, due to licence restrictions, but these restrictions have recently been relaxed.

HT has very different dosimetric properties from tritiated water. The ICRP considers that HTO's doses from inhalation and ingestion are 25,000 times greater than those for HT (ICRP, 1989)[18]. This is because the body is not thought to absorb or metabolise hydrogen gas, whereas water is a vital component of all body tissues and metabolic processes. Therefore the important factor is how fast HT is converted (oxidized) to HTO. This had been the subject of differing views in the past (Sweet and Murphy, 1984, and Spencer and Dunstall, 1986). However nowadays it is understood that HT dispersed into the atmosphere diffuses readily into the soil and is converted to HTO by enzymatic reactions, the rate of which depends on the porosity, water content and microbial activity of the soil. The converted HT is subsequently transported as HTO. (Ichimasa, 1995). Generally, for conservatism in assessments, released tritium is often assumed to be in the form of HTO, as eventually almost all HT will be converted to HTO over longer time periods. (Davis et al, 1997).

For these reasons, HT and HTO discharges are summed in the table below to indicate total tritium releases.

Table A Annual tritium emissions from SRB Technologies (TBq per year)

Year	HTO (vapour and liquid)	HT (gas)	Total
1996	572	2,319	2,891
1997	1,024	8,679	9,703
1998	1,362	12,890	14,252
1999	671	6,624	7,295
2000	1,388	16,598	17,986
2001	993	12,875	13,868
2002	868	8,398	9,266
2003	420	6,338	6,758
2004	342	3,973	4,315
2005*	247	935	1,224
2006**	72	213	285

Sources: Pre-2000 data: Dr. Patsy Thompson, from presentation to the Pembroke Municipal Council, November 19, 2002, Canadian Nuclear Safety Commission. 2000–2006: SRBT Annual Compliance Reports (http://www.betalight.com/pip/relations.htm).

NB: SRBT subsequently reported in a letter to CNSC staff dated August 24, 2005 that it had found an error in the calculation of the quantity of gaseous emissions per week, which resulted in a 10-fold underestimation in the reported values of the gaseous emissions from all previous years (Canadian Nuclear Safety Commission, Report CMD 05-H26.B (http://www.nuclearsafety.gc.ca/eng/commission/pdf/05-H22A-UpdatedAgenda.pdf). Data for years prior to 2005 have been corrected for this error.

*In a letter dated November 17, 2005, and orally on November 18, 2005, SRBT informed the CNSC staff that "the tritium emission monitoring system may not be providing reliable measurements of the concentration and quantity of tritium released to the environment" (Canadian Nuclear Safety Commission, document CMD 05-H26.C, http://www.nuclearsafety.gc.ca/eng/commission/pdf/2005-11-30-H34C-UpdatedAgenda.pdf).

[18] More precisely, the maximum derived air concentration for HTO is 25,000 times greater than that for HT.

This may have affected measurements during the 5-year licensing period, 2001–2005.

** During 2006, SRBT's Nuclear Substance Processing Facility Operating Licence NSPFOL-13.00/2006 restricted the company to use only one unit—the reclamation unit or a single betalight production filling rig to process tritium—at any given time. Prior to 2006, SRBT operated many units simultaneously.

In a comment, SRB tritium discharges, particularly in the years 1997 to 2003, were extremely high and they merit considerable CNSC attention and concern. In the author's view, it may be worthwhile to carry out OBT measurements in flora and fauna downwind and downstream of the SRB plant. Consideration should also be given to tritium measurements among those living near the SRB facility and SRB employees.

PART 2: The Radiation Hazards Of Tritium

Chapter 11 Introduction

1. Tritium (H-3) is the radioactive isotope of hydrogen. It is a beta-emitter with a maximum decay energy of 18 keV (average 5.7 keV), and a half-life of 12.33 years. Tritium is formed naturally through cosmic ray interaction with H in the upper atmosphere and is transferred to the troposphere. Tritium emission rates from civil and military nuclear facilities considerably exceed natural production rates—indeed by over two orders of magnitude. Tritium is created in nuclear fuel through activation of ^{1}H and ^{2}H and as a tertiary fission product. In comparison with other nuclides, large amounts of tritium are released from heavy water reactors, nuclear fuel reprocessing plants and pharmaceutical plants. Tritium most commonly occurs as tritiated water (HTO), organically bound tritium (OBT) and elemental tritium gas. This report will concentrate on the former two forms[19].

2. Tritium is ubiquitous in all biological systems through mixing, cycle, transfer, and dilution of the hydrosphere. It is exceedingly mobile, due to its existence as water and to its ease of exchange among all chemical species containing hydrogen, including biota. Consequently, tritium can be viewed as a very efficient distributor of radioactivity in the environment and humans. Ingested tritiated water has a biological half-life of about 10 days in humans: this is assumed to be homogeneously distributed in tissue. Tritiated foodstuffs contain organically bound forms of tritium with longer biological half-lives, which are variable and may extend to several years. OBT dose coefficients are therefore larger than those for HTO.

3. As the radioactive isotope of hydrogen, tritium has a number of unusual properties, including high mobility; ubiquity in the environment; cycling in the biosphere; multiple pathways to man; ease of exchange with stable H atoms in biomolecules; binding with biomolecules to form OBT with higher dose coefficients; and the heterogeneous distribution OBT in cells/tissues. The short range of tritium's beta particle in tissue means that tritium's microdosimetric effects and distribution in tissues and cells must be considered. For these reasons, many scientific concerns have been expressed about tritium (see Appendix I; Fairlie, 1992; Taylor et al, 1990).

[19] ICRP's derived air concentration for HT gas is 25,000 times lower than that for HTO.

Chapter 12 Tritium's Dose Coefficients

4. Paradoxically, tritium's dose coefficients are, by some margin, the lowest among common nuclides. (The dose coefficient of a nuclide is the estimated radiation dose imparted to a cell, tissue or organism by the radioactive decay of one atom of that nuclide.) The ingestion dose coefficient for tritiated water (1.8×10^{-11} Sv per Bq) is ~700 times lower than that for Cs-137, for example. As a result, tritium is widely regarded as a "weak" radionuclide and official radiotoxicity schemes continue to list tritium in the lowest radiotoxicity category. For example, Table 11.1.1.1 of the IAEA's "Health Physics and Radiological Health Handbook" (1990) provides a Toxicity Classification of Radionuclides. The table is similar to the ILO "Guidelines for the Radiation Protection of Workers in Industry—Ionizing Radiations" (1989). This lists nuclides in descending order of radiotoxicity, as shown in Table 12-1. (See also the USEPA website, http://www.epa.gov/radiation/radionuclides/tritium.htm#inthebody.)

Table 1 IAEA Radiotoxicity Scheme

Group	Radiotoxicity	Examples
Group 1	Very High	Ra-226, Pu-239, Am-241, Cf-252
Group 2	High	Co-60, Th-232, Sr-90
Group 3	Moderate	low beta yield radionuclides, eg Be-7, Co-57
Group 4	Low	H-3, Tc-99m, U-238

5. But there is a major misconception here. In radiobiology, so-called "weak" particles in fact have higher radiobiological effectiveness, and the lower their energy, the more effective they become. Tritium beta particles, as we shall see later, are actually 2 to 3 times more damaging[20] than gamma rays, for example. Therefore to call such particles "weak" is very misleading: it is better to call them "low-range".

6. Concern arises at the conjunction of two facts about tritium: its low dose coefficient and the large emissions from various facilities. If tritium's dose coefficient were found to contain significant errors or uncertainties, the health consequences could be large. Studies which have examined tritium's contribution to doses in specific circumstances have usually dismissed a major contribution from tritium (see, for example, in the UK: COMARE[21], 1996). However, these studies depend on tritium's existing dose coefficient's being substantially correct. Many researchers have expressed concern about tritium's dosimetry and have pressed for increases in tritium's dose coefficient. Tritium's dose coefficients were recently reviewed (Harrison et al, 2002) in a report which stated that an ICRP Task Force would be considering recommendations to double tritium's dose coefficients for tritiated water and organically bound tritium, mostly due to uncertainties in tritium's RBE. However, the ICRP has decided not to increase tritium's dose coefficient.

7. For these reasons, it is necessary to review how the ingestion dose coefficient for tritium

[20] The reason has to do with the track structure on ionising radiations. So-called "strong" radiations such as Co-60 gamma rays have very long tracks in tissue, but most of their energy is frittered away over the long track. Damaging amounts of energy are deposited only at the ends of their tracks. Low-range beta emitters such as H-3 consist ONLY of such track ends, and therefore can be more damaging per disintegration than higher energy emitters.

[21] For its 4th Report, the UK Government's COMARE Committee carried out a sensitivity analysis, multiplying tritium doses by 16 (see para 3.105) to account for the possibility of all tritium being bound to DNA. The resulting tritium doses were small.

(HTO) is calculated. Essentially, the dose coefficient is derived from the first principles of radiation physics and from the biokinetic assumptions of (a) homogenous distribution in soft tissue, (b) 100% instantaneous distribution among soft tissues, (c) 63 kg of soft tissues in man, (d) retention described by a single exponential with a half-life of 10 days, and (e) OBT doses neglected. These assumptions are considered next.

Chapter 13 Organically Bound Tritium (OBT)

8. Tritium is attached to organic compounds either by exchange reactions or by enzymatically-catalysed reactions. In exchange reactions, tritium swaps with H atoms bonded to oxygen, sulphur, phosphorus or nitrogen atoms in hydroxides, thiols, phosphides and amines, respectively. Conventionally this is termed “exchangeable” OBT. In enzymatically-catalysed reactions, tritium is bonded to a carbon atom of an organic molecule: this is usually termed “non-exchangeable” OBT. Tritium thus bound is more strongly attached and has longer retention times than exchangeable tritium: such bonds are only dissolved by catabolic reactions. Other nuclides (including S-35, P-32 and C-14) also have the property of organic binding, but H is much more common. Sixty percent of the body’s atoms are H atoms, of which about 5% are involved in metabolism reactions each day. In exchange reactions, H atoms are also much more active than S, P or N atoms.

Exchangeable and non-exchangeable tritium

9. In all elements, atoms engage in exchange reactions with other atoms of the same element to varying degrees. Thus, stable H atoms in organic molecules (i.e., bound to O, N, P or S atoms) can swap positions with tritium atoms in HTO. H, the smallest atom, is by far the most prominent as regards exchange reactions. The practical consequence is that all organic molecules downwind of a tritium (HTO) discharge plume which encounter HTO will quickly become tritiated, albeit at low concentrations. This includes all plant and animal species, all water-bearing material, and soil. It would include vegetables and fruit in exposed market stalls and shops, for example. (See Inoue Y et al, 1993. Uptake of atmospheric tritium by market foods. Fusion Technology 21, pp 494–499.)

10. Differing views exist on whether the definition of OBT should be restricted to non-exchangeable tritium or should include exchangeable tritium. The nub of the problem is that tritiated water, i.e., HTO, by definition, is a form of exchangeable OBT. This review adopts the more common latter view, but continues to make a distinction between the N, P and S forms and the HTO form of exchangeable OBT, because of their different biological half-lives and because of the ubiquity and high concentrations of water in biota.

Biokinetic models for HTO

11. Following HTO intake, the current ICRP model (1989) assumes 100% is absorbed and enters the blood. It assumes a turnover half-life of 10 days for HTO. It also assumes that 3% of HTO administered is bound as OBT, and that OBT doses from HTO administration may be safely neglected. Animal studies are informative and reveal that doses from OBT must be considered. Commerford et al (1982) found that, after a transient HTO exposure to mice, all the remaining tritium was bound to DNA and histone 8 weeks after exposure. Although the amounts were small compared to HTO, cell nucleoproteins were much longer-lived: the authors concluded that doses from them would exceed HTO doses. In addition, Trivedi et al (1997) estimated that an acute HTO administration in humans results in the range 3%–9% being bound as OBT, not the 3% currently assumed by the ICRP.

12. The problem is that the ICRP biokinetic model ignores chronic exposures to tritium. These are important in the case of those living downwind from facilities which discharge tritium 24 hours a day. The ICRP considers chronic exposures to be merely repeats of one acute exposure. That is, in each case, the main dose from HTO and none from OBT, and the HTO

and OBT being excreted before the next acute exposure. However, this is incorrect: animal studies reveal that after chronic exposures to HTO most of the dose comes from OBT. For example, Commerford, Carsten and Cronkite (1977) found most tritium doses came from the OBT component, 2 to 3 days after the cessation of a lengthy chronic HTO administration to mice. Rogers (1992) concluded that because OBT was cleared much more slowly than tissue water tritium, OBT was the principal determinant in estimated radiation doses to mice, following chronic HTO exposure.

13. In fact, chronic HTO administrations result in OBT concentrations increasing to higher levels, depending on how long the administration lasts. In situations of chronic exposures over a few months or so, Osborne (1972) stated there was a theoretical maximum to T-labelling of organic molecules by HTO, due to the fixed percentage (~30%) of exchangeable H bonds in the body. There is some evidence to support this: Rodgers (1992) fed mice tritiated water to establish a steady state of T turnover: OBT levels rose to 22% of body HTO levels after 56 days. However, it is unclear what would have happened had the experiment continued. Much evidence suggests that OBT levels would slowly continue to increase as tritium continues to be taken up by metabolic reactions. Eventually OBT levels, because of their very long half-lives, would equilibrate with body HTO levels. The evidence for this is discussed in Appendix III. Most important are the studies of exposures from naturally-occurring tritium, which indicate OBT/HTO ratios of 1. Essentially, there is a disjunction between the evidence from animal experiments and the evidence from background tritium levels/environmental studies. This may be due to the limited time lengths of existing biokinetic experiments. These experiments may need to continue for years, i.e., for the half-life of OBT in humans, which could be many years.

Biokinetic models for OBT

14. OBT has longer retention times than tritiated water, as it is incorporated in a variety of biochemical compounds, including amino acids, sugars, starches, lipids and cell structural materials. Some biomolecules are well-preserved and long-lived, e.g., phospholipids in nerve cells, and DNA and RNA macromolecules. These longer retention times result in a greater radiotoxicity than that of tritiated water. The ICRP (1989) has stated that "the major part of tritium uptake of the public will occur by ingestion of food into which tritium has been incorporated into both plant and animal components", and has recommended an OBT ingestion dose coefficient 2.3 times greater than that for HTO[22].

15. Following OBT intake, the current ICRP (1989) model assumes 50% reaches the blood as OBT and the rest is catabolised to HTO, which also reaches the blood. OBT is assumed to be excreted with a turnover half-life of 40 days. This figure comes from the assumption that all OBT is bound to C and the assumption of a 40 day half-life of C in the body (ICRP, 1975).

16. Serious problems exist with this model. The evidence for a carbon half-life of 40 days is derivative: it does not take into account experimental evidence in humans of considerably longer half-lives for the long-lived component of OBT. Table 13.2 below shows the 1st OBT component has a half-life of about 30 to 40 days, and the 2nd OBT component has a half-life of over 500 days in humans. The 1st component is conventionally assumed to be exchangeable OBT (i.e., bound to S, P and N atoms) and the 2nd component to be non-

[22] ICRP dose coefficients for adults are 1.8 x 10^{-11} Sv/Bq for tritiated water and 4.2 x 10^{-11} Sv/Bq for OBT.

exchangeable OBT.

Table 2 Tritium retention half-lives in humans after ingestion of HTO

Reference	No. of cases	Biological half-life (days)		
		HTO	OBT1	OBT2
Pinson and Langham 1957	9	11.3	-	-
Butler and Leroy 1965	310	9.5	-	-
Osborne 1966	30	10.5	-	-
Snyders et al 1968	1	8.7	34	-
Sanders and Reinig 1968	1	6.1	23	344
Minder 1969	1	-	1-30	139-230
Lambert et al 1971	1	9.1	36	-
Moghissi et al 1971	-	-	21-26	280-550
Moghissi et al 1972	1	9.0	30	450
Balonov et al 1974	-	12.0	39-76	-
Rudran et al 1988	8	6.0	30	226

17. Although little human data is available on the percentages of HTO retained as OBT1 and OBT2 after chronic intake, a reasonable (but not necessarily conservative) assumption would be about 30% and 6% respectively, assuming a continuous exposure limited to a few months' duration. Using half-lives of 30 days for OBT1 and 550 days for OBT2 would result in a ~ 3-fold increase in the HTO dose coefficient. If a more precautionary approach were adopted, i.e., if OBT/HTO equilibria were created, then OBT dose coefficients would be greater. However, it is unclear whether such equilibria occur in the timespans of most occupational and public exposures from current HTO releases. Clearly, long-term exposures from even low levels of HTO are contra-indicated, due to the build-up of OBT levels.

18. The current ICRP metabolic model for OBT results in ICRP's OBT dose coefficient being 2.3 times greater than the HTO dose coefficient. More accurate models of tritium metabolism (Etnier et al, 1984) and (Saito et al, 1989) indicate OBT doses are 1.7 to 6.4 times higher than calculated HTO doses, depending on OBT/HTO ratios in consumed food. Rudran (1988) estimated the OBT/HTO dose ratio was 3.4 for workers. If the longer-lived OBT components were included, the OBT dose coefficient would be 4 to 5 times higher than the HTO dose coefficient. This estimate is suggested by the findings of Komatsu et al (1990), who fed rats tritiated OBT. After 22 days' ingestion, 37% of the total tritium dose came from DNA and was due to the long half-lives of DNA. The authors calculated that OBT doses were 4.6 times higher than after HTO ingestion.

19. It is concluded here that the ICRP's biokinetic models for both HTO and OBT are significantly in error in a number of respects: their use may result in unconservative dose estimates. From this analysis, the dose coefficient for HTO should be increased by a factor of 3, and that OBT's dose coefficient should be increased to be 4 to 5 times greater than that for HTO.

Chapter 14 Internal Dosimetry of Tritium

20. This review focuses on tritium's internal dosimetry: external tritium is not normally regarded as hazardous. Five aspects are examined:

(a) tritium's RBE
(b) tritium's w_R value
(c) ionisation-density (LET) effects
(d) distribution effects
(e) incorporation in DNA

(a) RBE values for tritium

21. Tritium's RBE values have been the subject of much controversy in the past. (See Appendix IV for a short history of the matter.) Some may reflect that this controversy continues to this day. Straume (1991) and Straume and Carsten (1993) comprehensively reviewed tritium RBE experimental data for a range of species and endpoints. They concluded that a radiation-weighting factor of 3 was appropriate for tritiated water (HTO). Higher RBEs were found when the comparison radiation was gamma rays. Table 3 sets out the RBE values from the radiobiological experiments cited in Straume's tables. Since Straume's review, an RBE for HTO of 1.2 ± 0.3 for the induction of myeloid leukaemia in mice (Johnson et al, 1995) has been reported and is included in Table 3.

Table 3 Tritium RBE values

Cancer induction			
Mammary tumours in S-D rats	HTO/chronic x-rays	1.2 ± 0.3	Gragtmans et al 1984
AML CBA/H mice	HTO/x-rays	1.2 ±0.3	Johnson et al 1995
Various tumours in C57Bl/6NxC3H/He mice	HTO/acute x-rays	~1	Yokoro 1989
Chromosome aberrations			
Human lymphocytes	HTO/acute x-rays	1.9 ±0.7	Prosser et al 1983
Human lymphocytes	HTO/sub-acute γ	1.49 ±0.21	Morimoto et al 1989
Human lymphocytes	HTO/acute x-rays	1.13 ±0.18	Prosser et al 1983
Human lymphocytes	HTO/acute γ	3.4 ±0.64	Prosser et al 1983, Lloyd et al 1988
Human lymphocytes	HTO/250 kVp x-rays	2.6	Vulpis 1984
Human lymphocytes	HTO/250 kVp x-rays	2.0	Prosser et al 1983, Lloyd et al 1988
Reproductive effects			
Mouse spermatogonia	HTO/chronic x-rays	2.4	Lambert 1969
Mouse spermatogonia	^{3}HTdR/chronic x-rays	1.6	Lambert 1969
Mouse oocytes	HTO/chronic x-rays	2.9	Dobson and Kwan 1976
Fish germ cells	HTO/chronic γ	2.2	Etoh, Hyodo-Taguchi 1982
Fish fertility	HTO/chronic γ	~2	Hyodo-Taguchi, Hyodo 1985
Mouse testis weight loss	HTO/chronic γ	1.4	Carr and Nolan 1979
Mouse testis weight loss	^{3}HTdR/chronic γ	2.1	Carr and Nolan 1979
Mouse zygotes	HTO/chronic γ	1.8	Matsuda et al 1985
Micronuclei in mammalian cells	HTO/chronic γ	2.0	Ueno et al 1982
Micronuclei in mammalian cells	HTO/chronic γ	2.7	Kashima et al 1985

Genetic endpoints			
Drosophila mutations	HTO/γ	2.7	Byrne and Lee 1989
Mouse mutations	HTO/chronic γ	2.7	Nomura and Yamamoto 1989
Mouse dominant lethals	HTO/chronic γ	2.5	Searle 1974
Mouse dominant lethals	HTO/chronic γ	1.5	Carsten, Commerford 1976
Mouse dominant lethals	HTO/chronic γ	2.5	Xiang-yan et al 1986
Mouse specific locus mutations	HTO/chronic γ	2	UNSCEAR 1982
6-Thioguanine resist. in mouse cells *in vitro*	HTO/chronic γ	2.9	Ueno et al. (1989)
6-Thioguanine resist. in mouse cells in vitro	3H-amino acid/chronic γ	2.6	Ueno et al. (1989)
6-Thioguanine resist. in mouse cells in vitro	tritiated thymidine (3H-Tdr) and chronic γ	5.9	Ueno et al. (1989)
6-Thioguanine resist. in mouse cells in vitro	HTO/ γ at 10^{-5} mutant frequency acute	1.5	Nakamura et al. (1985)
6-Thioguanine resist. in mouse cells in vitro	HTO/γ at 10^{-5} mutant frequency	2.4	Nakamura et al. (1985)
Chromosome aberrations in human sperm in vitro	HTO/chronic X rays	3	Kamiguchi et al. (1990)
Chromosome aberrations in fish lymphocytes in vitro	HTO/chronic γ	1.9	Suyama and Etoh (1985)
Chromosome aberrations in mouse zygotes	HTO/chronic γ	1.8	Matsuda et al. (1985)
Chromosome aberrations in CBA/H mice lymphocytes	HTO/X rays	1.1	Chopra and Heddle (1988)
Chromosome aberrations in CBA/H mice spermatogonia	HTO/X rays	1.2	Chopra and Heddle (1988)
Developmental effects			
Mouse embryo	HTO/chronic γ	1.7	Yamada et al 1982
Rat embryo	HTO/chronic γ	2.6	Satow et al 1989
Cell killing in vitro	HTO/chronic γ	1.3	Ueno et al 1989
Cell killing in vitro	^{3}H-amino acids/chronic γ	1.7	Ueno et al 1989
Cell killing in vitro	^{3}H thymidine/chronic γ	3.5	Ueno et al 1989

Source: Straume (1991).

22. From this table it can be seen that two reference radiations are commonly used in RBE studies: hard gamma rays (usually from Co-60) and x-rays (usually 200-250 kVp). Paragraph 53 of ICRP 92 (2004) noted RBE differences of 2 to 3 between these two radiations with the latter being more effective. As a result, these two reference radiations have been the source of much confusion in determining the RBEs of tritium because a factor of 2 to 3 embraces tritium's own RBE values when compared with these radiations.

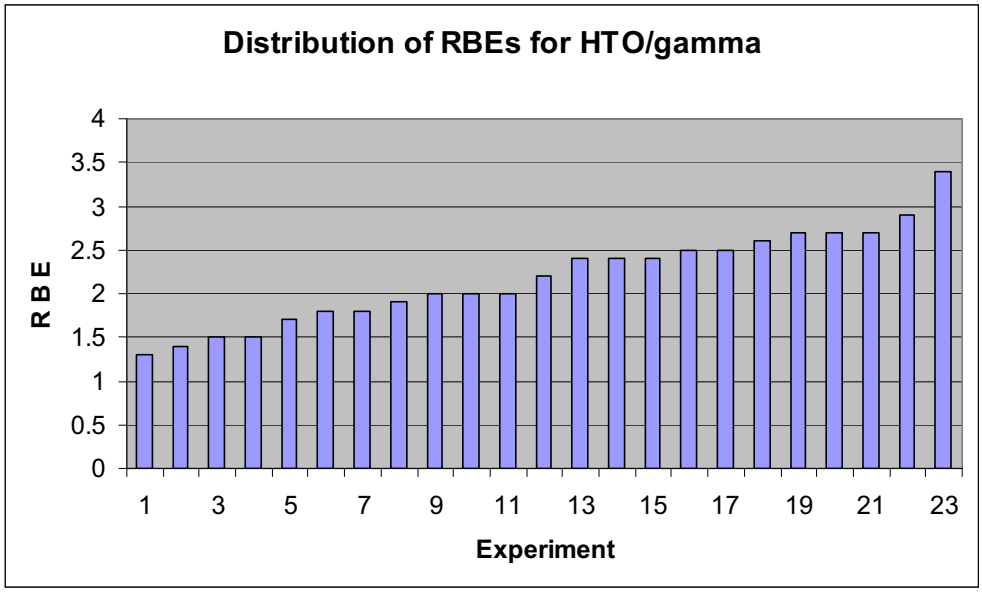
23. Hard gamma radiation (rather than 250 kVp x-rays) is the more appropriate reference radiation to use with tritium for three reasons:

(a) The Compton/photoelectron ratio produced by gamma rays is more similar to that produced by beta particles.

(b) The ICRP in Publication 92 (2004) has recommended that hard gammas should be used as the reference radiation for a number of other technical reasons.
(c) ICRP 92 also stated that x-rays are approximately twice as effective as hard gamma rays, indicating that the RBE values in the X ray experiments in table 3 above are about half the true RBE value.

24. For these reasons, Figure 1 presents a histogram of RBE results for tritiated water (HTO)/ gamma comparisons from Table 3. These range from 1.3 to 3.4, with a median of 2.2.

Figure 1 Distribution of RBE values for HTO/gamma comparisons



25. Harrison et al (2002) reviewed the data on which current ICRP dose coefficients for HTO were based and estimated that the RBE for tritium (HTO) was uniformly distributed between 1 and 2.5, with a median of 1.75. The authors also stated that an ICRP Task Force would be considering recommendations to double tritium's dose coefficients for tritiated water and organically bound tritium mostly due to uncertainties in tritium's RBE. These recommendations were not implemented, as the w_R for tritium (HTO) remains at unity as of June 2005 and tritium's dose coefficients have not been increased. Nevertheless, some authors (see Lambert, 2001) have used increased dose coefficients for tritium in specific dose studies.

(b) w_R value for tritium

26. The latest ICRP Publication to discuss this matter was ICRP 92 (2004). This report stated that it considered all the new RBE evidence, but it did not discuss the above evidence on tritium. The ICRP decided to continue with a w_R of 1 for all photons and electrons including tritium. The report contained a number of arguments for this position which are considered in detail in Appendix VI. The conclusion is that the ICRP's arguments are unconvincing and that the scientific evidence for an increase in tritium's w_R is very strong.

(c) Ionisation-density (LET) effects

The range of the beta-particle (i.e. electron) from the decay of a tritium nucleus is small because of its low energy of emission. A tritium beta particle of average (5.7 keV) or maximum (18.6 keV) energy, has a range in tissue of approx 1 μm or 7 μm, respectively (ICRU 1970). Hence, energies deposited per unit length of track are relatively large. Vennart (1969) stated they are "far greater for tritium than for carbon-14 and other ß-emitting particles."

27. When beta-particles move through tissue, they lose energy by ionising and exciting molecules along their track, until they (and their secondary electrons) slow down to thermal energies. Towards the end of the electron tracks, the average distance between ionisations is small, so they deposit relatively large amounts of energy in very short distances. From experiments with ultrasoft (photon energies 0.28–8.0 keV) x-rays, Goodhead and Nikjoo (1990) concluded that:

- ultrasoft x-rays, which interact in tissue to produce low-energy electrons, were more biologically effective than equal doses of hard x-rays or gamma rays;
- their RBEs increase with decreasing x-ray energy down to very low energies, for which the electron track lengths are very short (~7 nm);
- low-energy electron track ends were a predominant cause of cell inactivation in all low LET radiations, by virtue of the contribution from the low-energy secondary electrons that are always produced within the tissue; and
- the isolated sparse ionisations and excitations along the remainder of the track were "of relatively little biological significance."

28. These conclusions have been widely supported, for example, by Frankenberg et al[23] (1990) and other studies summarized by Hill et al (2001). The particular microdosimetric approach taken by the ICRU (1986), based on lineal energy over micrometre dimensions, also predicted increased effectiveness for tritium beta particles and other low-energy electrons. Nikjoo and Goodhead (1991) further reported that low energy electrons were particularly efficient at producing highly localised clusters of atomic damage over DNA-size (nanometre) dimensions, which could be responsible for a major part of the biological effectiveness of low-LET radiations.

29. These findings indicate that so-called "weak" radiations, which pack almost all of their punch at track ends, are equally as effective as "strong" radiations which dissipate as much as 70% of their energies as sparse ionisations during the higher energy parts of their tracks. ICRP 30 (1979) implicitly recognised this when it stated that the Q factor for internal emitters varied along their disintegration tracks. However, the ICRP assumed that the Q factor was constant for a given radiation. This may be a reasonable assumption for external radiations but it is inadequate to describe microscopic features of the short track of tritium's beta particle.

30. Because of its short track, tritium’s energy deposition closely follows its cellular distribution. Tritium’s RBE is therefore considered highly dependent on its congruence with radiosensitive sites. Current dosimetric models assume tritiated water intakes are homogenously distributed as water. This is an assumption, as in most cases this is not known. In addition, it is difficult to distinguish experimentally whether a biological effect from tritium, relative to that from another radiation, is the result of

(i) the heterogeneity of its concentration, or

[23] Who observed, inter alia, that electron track ends induced double strand breaks in DNA.

(ii) an inherently greater effectiveness of tritium decaying from one particular organic molecule rather than another.

(d) Tritium's incorporation in DNA

31. Perhaps the most important target is DNA. Chromosome diameters for most cells are between 0.3 and 1.0 μm. The average track length of the tritium beta particle in tissue is 0.5 to 0.7 μm (NCRP, 1979a). In this distance, the beta particle from tritium decay creates many ionisations. Because this track length is of the same magnitude as the diameter of a human chromosome, concern has been expressed (Ikushima et al, 1984), (Mathur-De Vre and Binet, 1984) and (Commerford, Carsten and Cronkite, 1977) about tritium doses when incorporated in DNA.

32. Although tritium is not the only nuclide which may be incorporated in DNA, hydrogen is by far the most common element in the DNA molecule. Saito and Ishida (1986) indicated that OBT from tritiated food was clearly incorporated within DNA. Commerford et al (1977) indicated the same from chronic HTO ingestion. Commerford et al (1982) also found, after a transient HTO exposure to mice, all the tritium remaining 8 weeks post-exposure was bound to DNA and histone[24]. Although the amounts were small compared to the HTO in the cell, cell nucleoproteins were much longer-lived. The authors concluded that doses from them would exceed doses from HTO. DNA half-lives were extremely long: 318 days for mice liver DNA and 593 days for mice brain DNA, i.e., about the lifespan of mice. In humans, DNA half-lives would be longer. The authors concluded that the cells at most risk would be those dividing at the time of exposure which afterwards were long-lived, i.e., key cells in embryos, nerve cells and oocytes.

33. Tritium doses to DNA occur because of its incorporation within the nucleus. The constituents of the nucleus are, on average, 70% (by weight) water, about 20% proteins, 3% lipids, and the rest nucleoproteins: DNA constitutes about 3% of nucleus weight[25] (Commerford, 1982). Saito and Ishida (1986) calculated the percentage contribution of various cell components, including DNA, to total cell dose, following the chronic ingestion of tritiated milk by suckling mice. They observed that initially between 1%–3% of the total dose in liver cells came from DNA-incorporated tritium. After 14 weeks' ingestion of tritiated food, this rose to 10% and after 41 weeks' ingestion to 52% of total tritium dose.

(i) Doses from HTO

34. Commerford, Carsten and Cronkite (1977), after chronic administration of HTO to mice, found nearly all the tritium in cell nuclei was tritiated water. Immediately at the end of ingestion, tritiated water gave the largest dose. Two to three days after the cessation of HTO administration, most doses came from OBT. After 15 months, the DNA of the cells of various mice organs was still tritiated at about the same concentration as the originally administered HTO. Ovary DNA had the highest concentration ratio (1.07) of the HTO activity ingested, and spleen DNA the least (0.72). The same authors (1982) later found, after a transient HTO exposure, all the tritium remaining after 8 weeks was bound to DNA and histone.

(ii) Doses from OBT

35. Etnier, Travis and Hetrick (1984) estimated that most protein in food crosses the human gut

[24] Protein bead on which the DNA is helically wound inside the chromosome.
[25] These proportions are illustrative, as ratios vary considerably among cell types.

wall and is available for incorporation, particularly essential amino acids. Commerford, Carsten and Cronkite (1983) estimated that, on average, 55% of dietary protein was transferred to tissue protein. They further estimated that, following an intake of the US recommended minimum daily amount of daily tritiated protein, a standard nucleus would contain 4 times more tritium in the form of nuclear proteins than in the form of water. Komatsu et al (1990) fed rats with shrimps containing tritiated protein. After 22 days' ingestion, 37% of the total tritium dose came from DNA and was due to the long half-lives of DNA. They calculated that OBT doses to DNA in liver cells were 4.6 times higher than after HTO ingestion.

36. Feinendegen et al (1980) found that chronically-administered amino acids were 2 to 4 times more efficiently incorporated than thymidine into "the long-lived components of proliferating tissues", i.e., DNA. They also found the distribution of amino acids among these components was similar to that of nucleic acid precursors (see next paragraph) in some organs, and the same was true for their turnover rates. They concluded that doses from tritiated amino acids in bone marrow stem cells were similar to tritiated thymidine and that tritiated amino acids were therefore radiologically 25–50 times more effective than HTO.

37. Nucleic acid precursors have been studied extensively, particularly thymidine and deoxycytidine, which is incorporated only in DNA. About 20 grams of DNA are synthesized each day in humans: 4 grams of thymidine are required for this production. Other precursors, e.g., uridine and cytidine, are incorporated into RNA as well as DNA. Very small amounts of tritiated thymidine and other precursors can be obtained directly from eating tritiated plants and animals (NCRP, 1979b). Lambert and Clifton (1968) estimated that, after an acute ingestion of tritiated thymidine, more than 90% was broken down in the GI tract, and only 2% was incorporated into DNA. NCRP Report 63 (1979b) estimated that 5% to 10% of tritiated thymidine would be incorporated in humans after eating tritiated foodstuffs. Takeda (1991) observed uptake levels of 7%–20% into OBT (i.e., some into DNA), depending on the organ, after 22 days' chronic ingestion of tritiated thymidine in rats.

38. In the past, differences of view have existed about the dangers of tritiated thymidine, which is a laboratory tracer, not a food. In practice, the greater danger is likely to emanate from tritiated protein in foodstuffs, as tritiated protein is more likely to be ingested (e.g., from foods grown, reared or cultured near tritium-releasing facilities) than tritiated thymidine. However, great care has to be taken with facilities which manufacture (and discharge to the environment) tritiated proteins and nucleic acid precursors.

39. It is concluded that tritium clearly can be incorporated in DNA. The studies by Commerford et al, Komatsu, and Saito and Ishida indicate that tritiated foods (OBT) are quicker and more effective than HTO in delivering radiation doses to the cell nucleus and DNA. Proteins (amino acids) are more efficiently incorporated into nucleoproteins than thymidine, and doses from tritiated proteins to DNA are four times greater than from HTO.

Chapter 15 Conclusions

40. Conclusions and recommendations are made on the following matters:

- tritium's hazardous properties,
- tritium's dose coefficients, and
- possible risks from tritium exposures.

(a) Tritium's hazardous properties

It is concluded that tritium's properties, including its ubiquity, rapid transport, and propensities to exchange, bind with organic molecules, concentrate in DNA's hydration shell, and have ionisation density in small volumes should be more widely recognised and discussed. It is recommended that up-to-date information on tritium's properties should be published and made available to regulatory agencies. In particular, a hazard guide for common radionuclides should be published, taking into account the properties of nuclides as well as their radiotoxicities. Such properties should include (see Kirchner, 1990):

(i) radiotoxicity (i.e., ICRP dose coefficient),
(ii) rapid nuclide transport and cycling in the biosphere,
(iii) global distribution,
(iv) rapid pathways to man,
(v) large nuclide fractions ingested or inhaled,
(vi) organic binding,
(vii) long half-lives, and
(viii) nuclide decay chains and their products.

(b) Dose coefficients

It is recommended that tritium's dose coefficients should be increased as follows. First, tritium's w_R should be increased to 2. Second, its biokinetic model should be improved to recognise tritium's long-lived OBT components and chronic exposures. This would increase HTO's doses by a factor of ~3. Third, tritium's other properties of rapid transport and uptake merit a factor of 2. Multiplying these factors means that HTO's dose coefficients should be increased by 2 x 3 x 2 = 12—or more simply, an order of magnitude. This would still leave tritium's dose coefficient ~70 times lower than that for Cs-137.

41. It is also recommended that OBT's dose coefficient should be ~5 times greater than that estimated for HTO; currently it is 2.3 times greater. The rationale for the factor 5 is as follows. The most hazardous form of OBT in diet appears to be tritiated protein. It is recalled that about 22 of the 40 or so amino acids which constitute proteins are essential amino acids (e.g., leucine and lysine), in that they are not manufactured in the body but taken up directly from diet. From the study by Komatsu et al (1990), OBT doses following tritiated protein intake were 4.6 times greater than from HTO. Feinendegen et al (1980) estimated even greater doses from tritiated proteins. A conservative approach would be to provide a level of protection for tritiated protein which would cover the other constituents of food, i.e., tritiated lipids and tritiated carbohydrates. Therefore a factor of 4 or 5 should be applied to all OBT in diet.

(c) Possible risks from tritium exposures

42. In pregnant women, cell proliferation occurs in the zygote, embryo, and foetus stages. Transient high concentrations of HTO could result in the ingestion/inhalation of HTO by pregnant women and tritium labelling at crucial points of embryo development. This could result in increased rates of untoward pregnancy outcomes, including stillbirths, congenital malformations and neonatal deaths. This concern was first raised by Professor Edward Radford (Provincial Government of Ontario, 1978) in testimony to the Ontario Select Committee on Hydro Matters, which examined possible health effects of large tritium discharges from nuclear facilities near Toronto, Canada. It was also raised by Commerford et al (1982), who stated that cells at most risk would be those dividing at the time of exposure which afterwards were long-lived, i.e., key cells in embryos (including nerve cells and oocytes). Straume (1991, 1993) estimated that tritium's teratogenic risks were 6-fold greater than tritium's carcinogenic risks, although strictly speaking it is difficult to compare these different risks. It is recommended that further epidemiological research should be carried out on possible teratogenic risks of exposure to tritium, which takes due account of the findings of the AECB reports.

Appendix I to Part 2 Previously Expressed Concerns about Tritium

"The present study clearly demonstrated that HTO [tritium] severely injures human stem cells to the same extent as 252 Cf neutrons, especially in the low dose range." Shigeta et al (1989).

"Although classified among the least toxic of the important radioactive atoms, there is considerable hazard with the ingestion of even low doses of tritium." Killen and Carroll (1989).

"The extreme sensitivity of the pre-implanted mammalian embryo to the ß radiation of tritiated compounds of metabolic importance points to the necessity for a re-evaluation of tritium risks for human beings, not only for workers exposed to occupational hazards, but also for those subject to chronic low doses." Clerici et al (1984).

"The question of main practical concern is ... the possibility that significant biological effects may result from protracted exposure to low tritium concentrations in water." Vulpis N (1984).

"For mouse immature oocyte killing, tritium administered chronically as ^{3}HOH is of near ***maximum possible*** radiobiological effectiveness. The implication: tritium in the form most commonly encountered as an environmental pollutant may actually be as effective as the most damaging high-LET radiations in reducing the fertility of certain other species as well... a highly sensitive germ-cell stage exists prenatally in at least some primate species... By implication, such a highly vulnerable stage may also exist in the prenatal human female." Straume T, Kwan TC, Goldstein LS, and Dobson RL (1989) (emphasis in original)

"...through various metabolic pathways [tritiated water] may enter any hydrogen position in organic matter including DNA, the most sensitive target for various radiation effects. The low energy of the ß emission from tritium produces relatively dense radiation tracks and causes localised deposition of dose in tissue. Considering these facts, there is concern about the ability of HTO to produce cytogenetic damage." Ikushima et al (1984)

"...the absorption of tritium energy occurs in the immediate vicinity of the tritiated nucleotide (DNA). Thus, tritiated water represents a very complex internal source of radiation causing concern about the health risks arising from exposure of human beings to it." Mathur-De Vre R and Binet J (1984)

"The tritium content of the chromosome, especially DNA, is particularly significant since such tritium is likely to cause genetic and somatic damage and to persist for very long periods of time." Commerford SL, Carsten AL, Cronkite EP (1977).
"When tritium is incorporated into biologically important molecular sites such as the DNA, its production of genetic damage per unit of energy deposited has been measured to be higher than a similar dose from protracted exposure from most (low-LET) external radiation. This may be related to the increase in LET at the end of the ß-particle range and the short range of the ß-particle from ^{3}H. This would result in much of the energy being deposited in the nucleus with a higher LET and effectiveness." NCRP Report No. 89 (1987).

"...it is...becoming increasingly important to study the genetic damage that this isotope can produce in man after incorporation, resulting both from irradiation by the tritium ß particles...and from the local effects of transmutation." Pelliccia et al (1988).

“.. there has been considerable apprehension about the incorporation of tritium into genetic material as the result of the very short range of the tritium ß particle and the possible effects of transmutation." Cronkite EP, Robertson JS and Feinendegen LE (1973).

"the radiation dose [of tritium] delivered to specific tissues, for example bone marrow, may be greater following the ingestion of organically bound tritium by almost an order of magnitude as compared to HTO [tritiated water]." Taylor DM, Moroni JP, Snihs O, Richmond CR (1990).

"The major part of the tritium uptake by members of the public will occur by ingestion of food into which tritium has been incorporated into both plant and animal components. Such organically bound tritium (OBT) will be present in many different chemical compounds including proteins, carbohydrates, fats and nucleic acids." ICRP Publication 56, 1989.

"All tritium models currently in use ignore the fact that organically-bound tritium in foodstuffs may be directly assimilated in the bound compartments of body tissue without previous oxidation ... properly accounting for metabolism of organically bound tritium in foodstuffs can increase cumulative dose estimates by as much as a factor of 4 or 5 over doses estimated for free body water [tritium] alone.” Travis CC et al (1984).

"... in view of the relatively large amounts of ^{3}H and ^{14}C which may be produced from expanded fission energy or from future fusion energy programmes, it is desirable to re-evaluate their potential to cause serious damage to human health." Taylor DM, Moroni JP, Snihs O, Richmond CR (1990).

"The hazards of tritium in the environment are becoming of increasing concern as by-products of nuclear power generation and fusion research." Little JB (1988).

Appendix II to Part 2 Equilibria between OBT and HTO Levels after Chronic HTO Exposures

In animals, Commerford et al (1977) showed OBT/HTO ratios between 0.73 and 1.07 in the DNA of cell nuclei of various mice organs after 15 months' HTO ingestion. Other studies (Koranda and Martin 1973) (Hatch et al, 1970) and (Evans, 1969) of animals living in tritium-contaminated sites indicated OBT/HTO ratios between 0.85 and 1.5. However it is possible these animals could have consumed OBT as well as HTO in their natural habitats.
In humans, Pinson and Langham (1957) found tritium concentrations in the organic constituents of fat and hair (i.e., OBT) removed from a man who died after eight months' chronic exposure to tritiated water were higher than that in body water at the time of autopsy. Rudran (1988b) studied the retention of tritium in eight workers chronically exposed to HTO at a heavy water nuclear station. She concluded that the fraction of HTO organically bound was larger than that assumed by the ICRP, and that the committed OBT dose equivalent was 3.4 times higher than that based on HTO alone.

Everyone is chronically exposed to background tritiated water levels (approximately 5–10 Bq/L). A number of older studies have examined naturally-occurring OBT/HTO ratios in humans. Bogen et al (1973), Bogen et al, (1979), Ujeno et al (1989), and Hisamatsu et al (1989) in the US and Japan found these ratios were usually unity or slightly higher than unity. The same is found in older studies of background OBT/HTO ratios in plants (Belot, 1986).

Appendix III to Part 2 Isotope Effects In DNA's Hydration Shell

Mathur-De Vre and Binet (1984) and Mathur-De Vre (1979), using magnetic resonance (MR) techniques, showed that water molecules were bound to DNA and RNA by electrostatic interactions and extensive hydrogen bonding. This water of hydration accounts for 60% (Cooke and Kuntz, 1974) of the weight of the DNA macromolecule. Mathur-De Vre et al (1983) estimated that the biological half-life of the hydration water of human DNA was 30 days. Gregolis et al (1982) found using MR techniques that hydrated DNA was more sensitive to gamma radiation than DNA without its bound water.

Mathur-De Vre and Binet (1982) observed that tritiated water was concentrated in the hydration water of DNA. This was due to the fact that hydrogen bonds between macromolecules and HTO are stronger than those with ordinary water, because intermolecular bonds between H-1 and H-3 are energetically more stable than those between H-1 and H-1. This is an example of an isotopic effect. The isotopic difference between H-1 and H-3 is a factor of three—the largest difference between any isotopes.

Mathur-De Vre and Binet stated that the role of hydration water in mediating initial radiation damage was "of great significance but has received little attention so far." They added that "the hydration water of DNA represents the fraction of cell water that is most effective for inducing radiobiological effects." Mathur-De Vre and Binet concluded "the hydration fraction of HTO constitutes an effective source for inducing initial molecular damage. At least some of these tritiated water molecules are located in very close contact with the macromolecular chains at a distance that is shorter than the maximum range of tritium ß-particles. This points out the importance of micro-localisation of initial energy deposition from hydration tritiated water in defining the RBE of HTO." However, few other scientists have discussed these findings (see Commerford,1982; and Baumgartner, 2002).

Appendix IV to Part 2 Controversy over Tritium's RBE

In the past, considerable controversy existed over tritium's RBE and its Q or w_R value. ICRP Report 2 (1959) established the RBE for tritium's beta-radiation to be 1.7 and the ICRP (1963) assigned a Q factor for tritium of 1.7. However in 1969 the ICRP reduced it to 1.0 (Dunster, 1969) with little explanation, despite the preponderance of evidence to the contrary at the time (Brues et al, 1952; Oliver and Lajtha, 1960; Dewey et al, 1965; Hall et al, 1967; and Lambert,1969).

Unfortunately, in the past, non-scientific considerations may have played a part in the setting of tritium's quality factor, at least in the US. Professor Karl Morgan, former member of the ICRP and former chairman of a main ICRP committee, stated (1990) that during the Cold War in the 1950s and 1960s

> "…there was constant pressure to set the RBE at 1.0 instead of 1.7. One ICRP member even went so far as to lament the difficulties they were having in keeping down to the tritium Maximum Permissible Concentrations (MPCs) in their weapons production plant, and our lowering the RBE to one would be a great help... Thus the way of reducing risk in a weapons production plant has been to get the ICRP and the US National Council on Radiation Protection (NCRP) to relax radiation protection standards, raise the MPC values, and have them adopted by the US Department of Energy and the US Nuclear Regulatory Commission."

After 1969, many observers (Johnson 1973; Cronkite, Robertson and Feinendegen, 1973; Moskalev et al, 1973; Berry et al, 1973; and Carsten, 1979) continued to query the choice of 1 for tritium's RBE. The NCRP (1979a, 1979b) reviewed the matter at length and concluded that an RBE of 1 could be ascribed to tritium, but only where the reference radiation was 60–80 kVp x-rays. In the body (but not the conclusion) of the report, it stated that, using the normal reference radiation of 200 kVp x-rays, its RBE was closer to 2. In 1980, Till et al (1980a, 1980b) from the US Oak Ridge National Laboratory mounted rigorous and comprehensive examinations of tritium's RBE. Their reports criticised the choice of unity and concluded that copious evidence indicated tritium's Q factor should be at least 2.

In 1986, a joint Committee from the ICRP and the ICRU[26] recommended an increase in the quality factor for tritium from 1 to 2, for microdosimetric reasons (ICRU, 1986). This recommendation was not implemented by the ICRP. Instead, ICRP 60 continued to recommend a radiation weighting factor (w_R) of 1 (ICRP 1990) for all (except Auger) electrons. For Auger emitters, it recommended microdosimetric techniques be used, although the likely doses from doing so were not discussed.

[26] International Commission on Radiation Units and Measurements, sister organisation to the ICRP.

Appendix V to Part 2 Transmutation Effects

When a tritium atom disintegrates, it emits radiation in the form of a ß-particle and transmutes to a helium ion, chemically and physically different from a hydrogen atom. This helium ion recoils from its ß-particle emission and has associated excitation energy. These processes rupture the bond to the compound to which the former tritium atom had been attached; this results in the compound acquiring a positive charge and becoming chemically active. The above effects, apart from radiation, are collectively known as transmutation effects. Since the recoiling nucleus is the second smallest of all radionuclides, the associated recoil velocity might be expected to be considerable.

NCRP Reports 62, 63 and 87 (1979a, 1979b and 1987) reviewed earlier studies on tritium's transmutation effects: no recent studies exist. Feinendegen and Bond (1973) concluded tritium's transmutation effects were small in comparison to the effects of its ß-radiation. It is difficult to distinguish these effects experimentally because of the short track length of the ß-particle.

Concern about transmutation has focussed primarily on the mutagenic effects of tritium incorporated into protein, including DNA, precursors. For reasons that are not understood, strong transmutational effects occur when tritium is situated at certain carbon positions in their purine and pyrimidine rings. Tritium at the number 5 carbon position in cytosine (incorporated in DNA as uridine or uracil) was found (Person et al, 1964) to be 7 times more mutagenic than tritium at other positions on the ring. This increase was estimated to be due to transmutation alone and not to radiation. Smaller transmutation effects for tritium at 6-thymidine and 2-adenine positions have also been found (Person et al, 1976). Krasin et al (1976) observed that half of tritium decays in 2-adenine in DNA resulted in strand-strand DNA cross-links, due to transmutation alone, over and above radiation effects. Tisljar-Lentulis et al (1983) found about a third of single-strand DNA breaks caused by tritium decay in 6-thymidine were due to transmutation. Ueno et al (1989) found that tritium in 6-thymidine position was 2.7 times more lethal to cells and 2 times more mutagenic than HTO. They postulated this could be due to "mechanisms other than radiation." Ueno et al (1982) and Tano (1986) found similar effects for thymidine tritiated at the methyl position, not previously reported as having transmutational effects.

The percentage of hydrogen atoms at the positions mentioned above in a DNA molecule is small. For example, only about 2% of the hydrogen atoms in a DNA molecule are at the 5-position of cytosine. More research needs to be carried out to elucidate the mechanisms and effects of transmutation. However, for the time being, these do not appear to have as much significance as OBT doses, RBE evidence and distributional effects.

Appendix VI to Part 2 ICRP 92 Arguments for Retaining a wR of 1 for Tritium

ICRP 92 defended its recommendation of a w_R of 1 for all photons and electrons by stating that there was little epidemiological evidence on which to base any differences (preface Editorial). It also stated that uncertainties in risks were large

> "… given the UNSCEAR 2000 judgement of several-fold uncertainty on the judgement of the nominal risk coefficient for cancer, including that for the dose and dose-rate effectiveness factor (DDREF), we do not see the need to ascribe different values of *w*R to different low-linear-energy-transfer (LET) radiations. A *w*R of 1 may therefore be retained for all low-LET radiations" (preface Editorial).

However the same points (scarcity of epidemiological evidence, uncertainty in risks) also apply with greater force to neutrons and protons, but this did not stop recommendations being made for different w_R values in their cases. In fact, the report recommended differences in w_R values for neutrons (which vary by about a factor of 4, depending on energy), and for protons (whose w_R is reduced from 5 to 2, i.e., a factor of 2.5). However the ICRP 92's findings (see paragraph 53) of RBE differences of 2 to 3 between conventional x-rays and hard gamma rays were not accommodated. Similarly, RBE differences between beta particles and gamma rays were also not discussed.

In the extract above, the ICRP states that a w_R of 1 may be applied to "all low-LET radiations," but this leaves unexamined the higher LET radiations from low-energy beta and Auger emitters. This matter was examined in the mid-1980s by a joint ICRU and ICRP Task Group. Its report—ICRU Report 40—stated that RBE values of photons/electrons were inversely energy dependent: as photon/electron energies decreased, their RBEs increased. For photons/electrons with energies below about 10 keV, ICRU Report 40 stated that differences in RBE could amount to 2 at doses below 100 mGy and it therefore recommended a Q value of 2 for low-energy beta particles, including those from tritium (ICRU, 1986). These recommendations were never implemented by the ICRP.

Second, the ICRP is understood to have defended its decision to continue with a w_R of 1 for tritium by referring to RBE values of 1.2 in two RBE studies (by Gragtmans et al, 1984; and Johnson et al, 1995) on cancer induction in mice and rats. Although animal data on carcinogenicity need to be taken into account, the ICRP reply nevertheless indicates a selective view of the large body of RBE evidence available on tritium, as set out in Table 3.

More important, these two animal studies used x-rays as their reference radiation. However, hard gamma would have been the more relevant reference radiation to have used, for two reasons. First, the Compton/photoelectron ratio produced by gamma rays is more similar to that produced by beta particles. Second, the ICRP itself has recommended that hard gammas should be used as the reference radiation for a number of other technical reasons. As stated by ICRP 92, x-rays are approximately twice as effective as hard gamma rays, which means that the correct RBE would be about double the observed value of 1.2, thus fitting in with most other values observed with gamma as the reference radiation.

Third, ICRP 92 stated that w_R and RBE values may be different as they are used for different purposes:

> "…there is no conflict between the fact that all photon radiations are …given the same weight, while risks… of soft x-rays, conventional x-rays, and hard gamma rays are assessed differently. *w*R is designed for the practice of radiological protection, not for specific risk assessment. … it is not meant to be applied in the derivation of quantitative risk estimates under specific conditions" (paragraphs 82 and 83).

It further explained that

> "…w_R is a quantity intended for use in radiological protection and was not developed for use in epidemiological studies or other specific investigations of human exposure. For these other studies, absorbed dose in the organs of interest and specific data relating to the RBE of the radiation type in question are the most relevant quantities to use" (preface Editorial).

These statements are disingenuous as the distinction between RBE and w_R is simply not made in practice: when quantitative risk estimates under specific conditions are being estimated, ICRP w_R values are almost invariably used by practitioners and regulators. The reason is that ICRP guidance on recommended RBE values does not exist at present (except for Auger emitters). Given this absence, w_R values and effective doses will continue to be used routinely in retrospective dose assessments, epidemiological studies or medical exposures, regardless of the ICRP's admonitions to the contrary. If the ICRP really wishes w_R values not to be used in the above circumstances, then it will need to recommend RBE values for use in such situations, i.e., for specific nuclides, ideally in their various chemical forms and for specific organs or tissues. Without such guidance, a danger exists of different regulatory bodies using differing RBE values for the same nuclides in specific dose assessments, etc., which would be a recipe for confusion.

The ICRP policy not to recommend more realistic higher w_R values for low-range photons and electrons is unsatisfactory for a number of reasons. It is not precautionary: that is, it does not err on the safe side for radiation protection purposes. The ICRP's disinclination to allocate higher w_R factors to low-range photons and electrons has direct implications, as regards radiation protection, for low-energy beta emitters are commonly discharged from nuclear and other industrial facilities. It may also have consequences for radiation protection in nuclear medicine, as regards widely-used Auger emitters, and for mammography x-rays.

Appendix VII to Part 2 Epidemiological Evidence

For a number of reasons, epidemiology studies are a blunt tool for discovering whether adverse effects result from radiation exposures. This is because epidemiology studies, generally speaking, have many methodological limitations. These include the following:

- Strict data requirements: Ideally, epidemiology data is required with good case identification, uniform registration, clear diagnostic criteria and uniformity of data collation. These data requirements are often difficult to fulfil and make large demands on time and resources.
- Confounding factors: The true causes of morbidity or mortality can be uncertain, due to confounding factors such as socio-economic status and competing causes of death.
- Bias: Smoking and alcohol cause major increases in overall mortality and morbidity, and in cancer and cardiovascular disease. These require careful handling of the raw data to avoid bias.
- Poor signal to noise: Only large, expensive and lengthy epidemiology studies are able to reveal effects where the signal (added cancers) is weak, and the noise (large numbers of spontaneous cancers) is strong.
- Uncertain doses: Establishing causality often requires estimating doses in order to show a dose-effect relationship. However, large uncertainties often exist in estimating doses—especially from internal radiation, e.g., from tritium.
- Wide confidence intervals: Usually findings (e.g., risks or odds ratios) are expressed with 95% confidence intervals. That is, the range of values within which the true value lies 95% of the time. But often this range can be very wide—simply because of low numbers of cases—which severely limits what we can conclude from the findings.

Many epidemiology studies are first stage “ecologic” studies, that is, they are quick studies which look at health or population data and not at individual data. Their findings should be regarded as perhaps indicative and not conclusive. If their findings are suggestive of an adverse effect, then these should be investigated further by more detailed second-stage epidemiology studies, that is, cohort or case-control studies. These studies match “cases” (i.e., those who have an adverse effect) with randomly-selected similar individuals, in order to minimise bias and confounding influences. However, fewer of these are carried out because of their greater expense and longer timespans. The conclusions are that we need to be more aware of the factors to be taken into account when considering epidemiology studies, and that we need to interpret their findings with great care. Therefore readers are advised to lower their expectations when considering individual epidemiology studies.

Another problem is that the results of epidemiology studies are unfortunately often the subject of controversy. On the one hand, official agencies are often accused of “playing down” the findings of increased incidences of ill health, much to the concern of local groups who may have a different viewpoint. It is often difficult to keep to the scientific findings of epidemiology studies and to accurately assess their worth, especially when they are contested in the media. But that is what is necessary, and is what this report will attempt to do.

Despite these caveats, epidemiology studies are still useful as they are one of only two sources of information (the other being cell or animal studies) about the effects of radiation. They are also the main source of information about their effects in humans.

1. Leukemia in children near Candu nuclear facilities (ecologic study)

Clarke et al. (1989, 1991) studied mortality and incidence of childhood leukemia near nuclear facilities in Ontario. The first report (Clarke et al. 1989) considered leukemia deaths and cases at ages 0–4, and the second (Clarke et al. 1991) considered cases and deaths at ages 0–14. Data for areas "nearby" (<25 km), the 16 reactors at Bruce and Pickering over the period 1971–1987, were pooled together to increase statistical significance. The findings were 36 leukemia deaths at ages 0–14 vs 25.7 expected (SMR = 1.40, 95% CI 0.98–1.9) indicating an excess leukemia mortality with borderline statistical significance, However the confidence intervals were wide: the data were consistent with there being no increase and with there being a 90% increase in leukemia.

However there were indications which warranted further investigation: higher leukemia death rates after the reactors had started than before; more deaths when counted at place of birth than at place of death; and the magnitude of the higher confidence interval. It is notable that different levels of statistical significance were adopted by the two reports. The first was 10%, and the second 5%. If the 10% level had been chosen in the second study as it had been in the first, the leukemia increase would have been considered "statistically significant". Unfortunately there were no estimates of tritium doses in this study. The authors recommended further case-control research.

2. Birth defects and infant mortality in the vicinity of the Pickering nuclear facility, Ontario (ecologic study)

Johnson and Rouleau (1991) studied birth defects, stillbirths, perinatal, neonatal and infant mortality within 25 km of the Pickering nuclear station. They also studied these endpoints in relation to airborne and waterborne discharges of tritium from Pickering, concentrating on the Pickering and Ajax townships closest to the Pickering plant. Again there were no estimates of tritium doses.

The incidence of central nervous system defects was significantly elevated in Pickering township for the highest level of airborne emissions (odds ratio in highest group = 4.01 (95% CI 1.25, 14.04), based on 6 cases), but no statistically significant trends with tritium emissions (p=0.197) or ground-monitoring data (p=0.24) were observed.

There was a statistically significantly raised prevalence of births with Down syndrome in Pickering township (24 observed vs 12.9 expected [relative risk = 1.85, 95% CI 1.19, 2.76]), But 23 other birth defect endpoints did not show such an excess. There was a correlation, although not statistically significant (p=0.468), between Down syndrome prevalence and airborne tritium release, but no correlation with ground tritium concentrations. There were no such excess birth defect risks in Ajax township (slightly further away), although there was a positive correlation, but again not statistically significant (p=0.282) with ground tritium concentrations.

The very mixed results from this study are difficult to interpret, but the raised incidence of Down Syndrome (formerly called mongolism) cases is notable, as many Chernobyl studies (see box below) also indicated excess cases in areas exposed to radioactive fallout, including tritium. However, the authors of the study stated that it was difficult to see why the incidence of Down Syndrome alone should be increased and not the many other forms of congenital malformation, e.g., spina bifida and cleft lip/palate. This is a good question and there is no answer at present. However, just because we do not know the answer does not

provide a reason to discount the observed association and a possible connection between tritium exposures and Down Syndrome.

Studies of increased incidences of Down Syndrome after Chernobyl exposures

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3. Offspring of Canadian nuclear workers (case control study)

Green et al (1997) assessed cases of congenital abnormalities and matched controls in the offspring of Canadian nuclear workers. (763 case-control pairs of fathers, and 165 case-control pairs of mothers.) Tritium doses were assessed for those cases/controls having a recorded tritium dose 60 days before conception versus those with no dose. The study revealed possible evidence of increased chromosomal disorders with tritium exposure, but the number of cases (two) is very small and confidence intervals very wide. Why there should be a tritium-associated increase only in chromosomal disorders but not other disorders is not known. However, as stated above, just because we do not know the answer does not provide a reason to discount the observed association and a possible connection between tritium exposures and chromosomal disorders.

Table A Adjusted odds ratios (and 95% CI) for congenital abnormalities according to tritium exposure (recorded dose 60 days before conception vs none)

Disorder	Odds Ratio (95% CI)
Single gene disorders	-
Chromosomal disorders	1.46 (0.24, 8.80)
Multifactorial disorders	1.13 (0.66, 1.94)
Genetic, unspecified	0.80 (0.27, 2.32)
Unknown disorders	0.84 (0.40, 1.76)
Total	0.99 (0.67, 1.47)

Source: From Table 2 in Green et al (1997).

4. Offspring of Ontario radiation workers

McLaughlin et al (1992, 1993) considered cases of childhood (ages 0–14) leukemia in the offspring of Ontario radiation workers and matched cases. Tritium workers were those employed at the AECL laboratories at Chalk River, and 5 power stations (Rolphton, Pickering (A, B), Bruce (A, B) (112 cases and 896 controls). Preconceptional tritium doses were assessed for this group. There was some evidence of raised risks with internal tritium plus external radiation exposures but with wide confidence intervals (see Table B). It is difficult to be more precise, as apparently there were no leukemia cases in the offspring of parents estimated to be only receiving tritium doses (see Table C).

Table B and Table C

Odds ratios (and 95% CIs) for leukaemia cases among offspring of workers monitored for tritium in Ontario

Table B

	Estimated whole dose (external + tritium) before diagnosis (mSv)	
	0 mSv	**≥0.1 mSv**
Cases (number)	103	9
Controls (number)	826	64
Odds ratio (95% CIs)	1.00	1.19 (0.51, 2.73)

Table C

	Estimated tritium dose before conception (mSv)	
	0 mSv	**≥0.1 mSv**
Cases (number)	112	0
Controls (number)	876	14
Odds ratio (95% CIs)	1.00	0.00 (0, 2.39)*

Source: From table III in McLaughlin et al (1993).

*1-sided p=0.25 (Fisher's exact test).

The odds ratio is defined as the ratio of the odds of an event occurring in a group under study to the odds of it occurring in another (control) group. An odds ratio of 1 indicates that the incidence under study is equally likely in both groups. An odds ratio greater than 1 indicates that the condition is more likely in the study group. And an odds ratio less than 1 indicates that the condition is less likely in the study group.

5. Canadian nuclear workers

Zablotska et al. (2004) studied mortality in more than 45,000 Canadian nuclear workers between 1957 and 1994. Tritium doses were calculated from urinalysis data, and added to external (film-badge) doses. The study did not indicate tritium doses, but states that for some workers these could have been large. Overall, the mean dose was 13.5 mSv, and among those recorded as having "some dose" it was 19.7 mSv. The resulting excess relative risks (ERRs) per Sv were high and were mostly statistically significant, though with wide CIs (see Table D).

Table D

	ERRs per Sv	**95% CI**	**2-sided p test**
All leukemia, excluding CLL	52.5	0.205, 291	0.048*
Rectal cancer	34.1	1.41, 165	0.029*
All solid cancers	2.80	-0.038, 7.13,	0.054+

*Statistically significant at 5% level.
+Borderline significance.

Of interest, is the fact that, with an ERR per Sv of 52.5 for leukemia, the ERR for nuclear workers with the average dose of ~20 mSv would be 1.05, i.e., their leukemia risk was about double the spontaneous risk of leukemia in the population. Similarly the ERR for all solid cancers of 2.8 means that the ERR for nuclear workers with the average dose of ~20 mSv would be 0.056, that is, about a 5% or 6% increase in the spontaneous risk—or a rise from 30% to 35% in their risk of dying from a solid cancer.

Interestingly, the study examined the risks of those exposed only to external radiation (i.e., gamma rays) and compared them with the risks of those exposed to **both** external gamma and internal tritium doses. If the average risks per Sv for two groups were found to be the same this would indicate that the risk from (say) one Sv of tritium's beta radiation was approximately the same as the risk from one Sv of gamma rays. However, this was not found. Instead, those exposed to tritium were subject to higher risks, per sievert of tritium exposed, as indicated in Table E below. Unfortunately it is not possible from the study data to ascertain by how much tritium doses are more effective (risk-wise) than gamma doses. These data suggest that tritium doses were underestimated, or that tritium's dose coefficients are too low, or both of these.

Table E

	ERR Sv^{-1} without tritium doses	**ERR Sv^{-1} with tritium doses**	**% Increase**
All leukaemia	16.3	18.9	16%
All solid cancers	2.67	2.80	5%

Studies of radiation-associated risks among workers chronically exposed to low doses of radiation are important, both to estimate risks directly and to assess the adequacy of extrapolations of risk estimates from high-dose studies. Zablotska et al examined 45,468 nuclear power industry workers from the Canadian National Dose Registry, monitored (for more than one year) for chronic low-dose whole- body ionising radiation exposures between 1957 and 1994 (mean duration of monitoring = 7.4 years, mean cumulative equivalent dose = 13.5 mSv). The excess relative risk for **leukemia** (excluding chronic lymphocytic leukemia)

was 52.5 per sievert (95% confidence interval [CIs]: 0.205, 291) with the association having a p-value close to 0.05 (i.e., the association was statistically significant at the 5% level). Relative risks for leukemia increased as the dose increased.

The excess relative risk for all **solid cancers** was 2.80 per sievert (95% CIs: -0.038, 7.13) with the association having a p-value also close to 0.05. The relationship between solid cancer risk and dose was less consistent than with leukemia risk. Although these point estimates are considerably higher than those found in other studies of whole-body irradiation, the difference could still be due to chance because of the wide confidence intervals. (Recall that readers were warned earlier not to have high expectations of epidemiology studies).

6. Congenital malformations in India

In April 1991, the UK Channel 4 TV programme "The Price of Power" revealed apparently high incidences of congenital malformations in babies born downwind of the two Candu reactors at Kota in Rajasthan in India, which discharge large amounts of tritiated water. Gadekar et al (1994) also found significantly increased incidences in congenital malformations in children, compared with those in an area remote from the reactors. The relative risk (RR) of congenital malformations in children (<18 years) for the area near the reactors was 3.45 (CI 1.67 and 7.59). In younger children (<11 years) the RR was 5.08 (CI 2.14 and 12.06). The authors stated that tritium discharges from the reactors were larger than the published emissions from Canadian reactors and they recommended further studies. These have not been carried out.

7. Unpublished studies

In 1988, Durham Nuclear Awareness, an environment NGO in Canada, published a report by an independent researcher (David McArthur), which found a correlation between fatal birth defects and neonatal deaths with lagged tritium water emissions from the Pickering nuclear plant between 1978 and 1985. This report is similar in its findings to that of another independent researcher (Hugh Richards) who also found a correlation between lagged tritium emissions from a plant near Cardiff with neonatal deaths. It is understood that the 2 authors are unaware of each other’s findings. The similarity of the findings is interesting. However neither of these reports has been published in the scientific literature and they are understood to have been criticised by others for statistical inadequacies.

Conclusion

As stated in the introduction, epidemiology studies are a blunt tool for finding out about the risks of hazardous substances. The reality is that there is a very large gap between the cancer risks that most individuals will tolerate from industrial activities (e.g., 1 in a million) and the risks that can be detected by epidemiological study of reasonable-sized populations. The above studies, except for Zablotska et al, show either no increases or possible increased incidences with borderline statistical significance in most cases. However it must be remembered that just because these studies were unable to detect increased effects, this does not mean that there are no effects. As Altman and Bland (1995) stated “absence of evidence is not evidence of absence”. The methodological limitations of many epidemiology studies mean that they are simply not able to detect any effect with statistical certainty.

Another matter is that if the detected excesses of childhood leukemias around Pickering and Bruce turn out to be both real and caused by radiation exposures from the nearby Candu stations, then either the estimated radiation doses to people are too small, or the risk coefficients for radiation are too small, or a combination of both. Indeed the real doses and real risks could be higher than our regulators assume, and could even be higher than this report suggests.

With this in mind, our conclusion is that the above studies taken together provide suggestive, albeit limited, evidence for increased health effects from exposure to tritium. However this evidence remains indicative and not conclusive. We conclude that more epidemiology studies should be carried out in areas downwind of the Canadian nuclear reactors.

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Annex A to Part 2 Acronyms and Abbreviations

AECB	former Atomic Energy Control Board (now CNSC qv)
ACRP	former Canadian Advisory Committee on Radiation Protection
Bq	becquerel (unit of radioactivity)
CERRIE	UK Committee Examining the Radiation Risks of Internal Emitters
Ci	curie (unit of radioactivity)
COMARE	UK Committee on the Medical Aspects of Radiation in the Environment
CNSC	Canadian Nuclear Safety Commission
ddref	dose and dose-rate effectiveness factor
DNA	deoxyribose nucleic acid
DoE	US Department of Energy
EC	European Commission
EPA	US Environmental Protection Agency
err	excess relative risk
Gy	gray (unit of absorbed radiation dose)
HPA-RP	Radiation Protection Unit of the UK Health Protection Agency (formerly NRPB qv)
IAEA	International Atomic Energy Agency
IEER	Institute of Environment and Energy Research
ICRP	International Commission on Radiation Protection
LET	linear energy transfer
LNT	linear no-threshold (radiation's dose-effect relationship)
NEA	Nuclear Energy Agency of the OECD
NCI	US National Cancer Institute
NRC	US Nuclear Regulatory Commission
NRPB	former UK National Radiological Protection Board
OECD	Organisation for Economic Cooperation and Development
rad	rad (unit of absorbed radiation dose used in the US)
rem	rem (unit of radiation dose used in the US)
RERF	Radiation Effects Research Foundation
Sv	sievert (unit of equivalent or effective radiation dose)
TRF	tritium reduction facility
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
WHO	World Health Organisation

Annex B to Part 2 Radiation Dose Units

A measure of the effect of radiation is the amount of energy it deposits in unit mass of body tissue. This quantity is called the **absorbed dose**. The unit of absorbed dose is the **gray** (Gy). One gray is equal to the energy deposition of 1 joule in 1 kilogram of tissue.

The biological effects of alpha particles and neutrons (high-LET[27] radiation) are, in general, greater than the effects of beta particles and gamma rays (low-LET radiation) of the same energy. The **radiation weighting factor** (w_R) was introduced to take account of the different biological effectiveness of alpha and beta particles, neutrons, and x- and gamma rays.

The quantity **equivalent dose** is then defined as: equivalent dose = absorbed dose multiplied by the w_R. The unit of equivalent dose is the **sievert** (Sv).

In studies of low-dose radiation, the sievert is too large a unit and doses are usually given in millisieverts (mSv), where 1 Sv = 1,000 mSv (see below).

For low-LET radiation, $w_R = 1$, so grays and sieverts will be numerically equivalent. However, for alpha particles $w_R = 20$, so an absorbed dose of 1 mGy produced by alpha particles will have an equivalent dose of 20 mSv.

International System of Nomenclature (commonly used units)

E = exa	$= 10^{18}$	d = deci (one tenth)	$= 10^{-1}$
P = peta	$= 10^{15}$	c = centi (one hundredth)	$= 10^{-2}$
T = tera (one trillion)	$= 10^{12}$	m = milli (one thousandth)	$= 10^{-3}$
G = giga (one billion)	$= 10^{9}$	μ = micro (one millionth)	$= 10^{-6}$
M = mega (one million)	$= 10^{6}$	n = nano (one billionth)	$= 10^{-9}$
k = kilo (one thousand)	$= 10^{3}$	p = pico (one trillionth)	$= 10^{-12}$

Common examples are:

PBq	= petabecquerel (one million billion becquerels)	$= 10^{15}$ Bq
TBq	= terabecquerel (one trillion becquerels)	$= 10^{12}$ Bq
GBq	= gigabecquerel (one billion becquerels)	$= 10^{9}$ Bq
mSv	= millisievert (one thousandth of a sievert)	$= 10^{-3}$ Sv
μSv	= microsievert (one millionth of a sievert)	$= 10^{-6}$ Sv
nSv	= nanosievert (one billionth of a sievert)	$= 10^{-9}$ Sv

[27] LET= linear energy transfer, i.e., the energy transferred per unit length of the radiation track.

Annex C to Part 2 Conversion between European and US units

a. Radioactivity units

CURIES TO BECQUERELS

1 curie	= 1 Ci	= 37×10^{9} becquerels
1 millicurie	= 1 mCi (10^{-3} Ci)	= 37×10^{6} becquerels
1 microcurie	= 1 µCi (10^{-6} Ci)	= 37×10^{3} becquerels
1 nanocurie	= 1 nCi (10^{-9} Ci)	= 37 x 1 becquerels
1 picocurie	= 1 pCi (10^{-12}Ci)	= 37×10^{-3} becquerels

BECQUERELS TO CURIES

1 petabecquerel	= 1 PBq (10^{15} Bq)	= 27×10^{3}	curies
1 terabecquerel	= 1 TBq (10^{12} Bq)	= 27	curies
1 gigabecquerel	= 1 GBq (10^{9} Bq)	= 27×10^{-3}	curies
1 megabecquerel	= 1 MBq (10^{6} Bq)	= 27×10^{-6}	curies
1 kilobecquerel	= 1 kBq (10^{3} Bq)	= 27×10^{-12}	curies
1 becquerel	= 1 Bq	= 27×10^{-15}	curies

b. Radiation units

REMS TO SIEVERTS

1 rem	= 1 rem	= 10^{0} rem	= 10 millisieverts
1 millirem	= 1 mrem	= 10^{-3} rem	= 10 microsieverts
1 microrem	= 1 µrem	= 10^{-6} rem	= 10 nanosieverts

SIEVERTS TO REMS

1 sievert	= 1 Sv	= 10^{0} Sv	= 100 rem
1 millisievert	= 1 mSv	= 10^{-3} Sv	= 100 millirem
1 microsievert	= 1 µSv	= 10^{-6} Sv	= 100 microrem

Annex D to Part 2 Glossary of Scientific Terms

Absorbed dose — Quantity of energy imparted by ionising radiation to unit mass of matter such as tissue. 1 Gy = 1 joule per kilogram.

Activity — The rate at which radioactive substances decay. Unit: the becquerel; symbol: Bq. One Bq = 1 disintegration per second.

Annual limit of intake (ALI) — The amount of material inhaled or ingested in 1 year that would result in a committed effective dose of 20 mSv.

Becquerel — See activity.

Beta particle — An electron emitted by the nucleus of a radionuclide.

Decay — The process of spontaneous transformation of a radionuclide. The decrease in the activity of a radioactive substance.

Decay product — A nuclide or radionuclide produced by decay. It may be formed directly from a radionuclide or as a result of a series of successive decays through several radionuclides.

Dose — General term for quantity of radiation. See absorbed dose, effective dose, equivalent dose.

Dose factor — The committed effective dose resulting from the inhalation or ingestion of 1 Bq of a given radionuclide. Unit: sievert per Becquerel; symbol: Sv/Bq.

Effective dose — The quantity obtained by multiplying the equivalent doses to various tissues and organs by the tissue weighting factor appropriate to each and summing the products. Unit:
sievert; symbol: Sv.

Equivalent dose — The quantity obtained by multiplying the absorbed dose by the appropriate radiation weighting factor to allow for the different effectiveness of the various ionizing radiations in causing harm to tissue. Unit: sievert; symbol: Sv.

Gamma ray — A discrete quantity of electromagnetic energy, without mass or charge.

Gray — See absorbed dose.

Half-life — The time taken for the activity of a radionuclide to lose half its value by decay.

Ionisation — The process by which a neutral atom or molecule acquires or loses an electric charge. The production of ions.

Ionising radiation — Radiation that produces ionisation in matter.

Nuclear fission — The process in which a nucleus splits into two or more nuclei and energy is released.

Radionuclide — An unstable nuclide that emits ionizing radiation.

Risk factor — The probability of fatal cancer or leukaemia per unit effective dose.

Sievert — See effective dose.

Annex E to Part 2 Recommended Reading

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B. On tritium

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