Summary
This report examines the properties of tritium, including its rapid transport and uptake, and its propensities to exchange, bind with organic molecules and concentrate in DNA's hydration shell. The long-running debate over tritium's $W_R$ is outlined. Current biokinetic models for HTO and OBT are described together with their limitations. Tritium’s dosimetry, transmutation effects, ionisation density effects, distribution effects and DNA doses are described. Epidemiological evidence of ill health is discussed. Recommendations are made for the wider recognition of tritium’s hazards, in particular a 15-fold increase in HTO’s dose coefficient, with OBT’s dose coefficient 5-fold greater than that for HTO; and for research to be commissioned to investigate possible teratogenic risks from high transient HTO exposures.

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INTRODUCTION

1. Tritium (³H) 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 12.3 years. Tritium is formed naturally through cosmic ray interaction with H in the upper atmosphere and transfers to the troposphere: emissions from civil and military nuclear facilities considerably exceed natural sources. Tritium is created in nuclear fuel by activation of ¹H and ²H, 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 review will concentrate on the former two forms.

2. Tritium is ubiquitous in all biological systems through mixing, transfer, chemical reaction and dilution of the water cycle. It is exceedingly mobile due to its ease of exchange among chemical species containing hydrogen, including all biota. Consequently tritium may be considered a very efficient distributor of radioactivity in the environment and in humans. Ingested tritiated water has a biological half-life of about 10 days and is assumed to be homogeneously distributed in tissue. Tritiated foodstuffs contain organically bound forms of tritium with longer biological half-lives. These half-lives are variable and may extend to several years. In addition, OBT is heterogeneously distributed in organs. Dose coefficients for OBT are therefore larger than those for HTO. The short track length in tissue of tritium’s beta particle means that tritium’s effectiveness is highly dependent on its distribution in cells.

3. The high mobility and cycling of tritiated water in the biosphere; multiple pathways to man; ease of exchange with stable H atoms in biomolecules; ability to bind with cell constituents to form OBT with higher dose coefficients, and OBT’s heterogeneous distribution in cells suggest that careful attention should be paid to tritium’s properties, dosimetry and radiotoxicity (Fairlie, 1992). Unfortunately, some radiation protection agencies appear ill-informed of tritium’s properties, as tritium is widely regarded to be among the "weakest" of common nuclides. These views are often justified by reference to tritium’s dose coefficients which remain, by some margin, the lowest among common nuclides. The ingestion dose coefficient for H-3 is 660 times lower than that for Cs-137, for example.

4. As a result, official radiotoxicity schemes (IAEA, ILO) continue to list tritium in the lowest radiotoxicity categories. 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 four categories, as set out in Table 1.

Table 1. IAEA Radiotoxicity Scheme

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1. ICRP’s DAC for tritium gas is considerably lower (x 25,000) than that for HTO.
2. In some departments of some UK radiation protection agencies, a new awareness of tritium’s toxicity is noticeably beginning to emerge. But these views are still in the minority in the agencies concerned.
<table>
<thead>
<tr>
<th>Group</th>
<th>Radiotoxicity</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Very High</td>
<td>Ra-226, Pu-239, Am-241, Cf-252</td>
</tr>
<tr>
<td>Group 2</td>
<td>High</td>
<td>Co-60, Th-232, Sr-90</td>
</tr>
<tr>
<td>Group 3</td>
<td>Moderate</td>
<td>low beta yield radionuclides, such as Be-7, Co-57</td>
</tr>
<tr>
<td>Group 4</td>
<td>Low</td>
<td>H-3, Tc-99m, U-238</td>
</tr>
</tbody>
</table>

5. The difficulty is that these guides are based on one factor, i.e., simple ICRP radiotoxicity based on disintegration energies. No comprehensive hazard index exists for radionuclides, contrary to the position with chemicals, for example. Kirchner (1990) has stated that, in addition to mere radiotoxicity, a proper hazard system for nuclides should also take into account-

(i) rapid nuclide transport and cycling in the biosphere,
(ii) global distribution,
(iii) rapid nuclide pathways to humans,
(iv) large nuclide fractions ingested or inhaled,
(v) the property of organic binding, and
(vi) nuclide decay chains and their products.

6. Tritium stands out among common nuclides as having properties in 5 out of 6 of these additional fields. Other nuclides including S-35, P-32 and C-14 also have the property of organic binding, but H-3 is much more commonly emitted and in larger amounts. In addition, 60% of the body's atoms are H atoms, of which about 5% are involved in metabolism reactions each day. In addition, H atoms are much more promiscuous in exchange reactions than S, P or N atoms.

7. Concerns have also been expressed at the conjunction of two facts about tritium: its very large emissions and its very low dose coefficient. If tritium's dose coefficients were found to contain significant errors or uncertainties, the consequences could be large.

8. Many researchers have expressed concern about tritium's dosimetry and radiotoxicity: others have pressed for increases in tritium's dose coefficient. A small selection of such views from the past two decades is included in Appendix I. Official studies which have examined tritium's contribution to doses in specific circumstances have usually dismissed a major contribution from tritium (see, for example, COMARE[^3], 1996). However these studies depend on tritium's existing dose coefficient being substantially correct. 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.

9. This report first discusses the phenomenon of tritium labelling of organic molecules which is perhaps the most striking of tritium's properties. It then discusses various problems with tritium's dosimetry including its RBE, distribution, and dose coefficients. It concludes with a number of recommendations and proposals for research.

**ORGANICALLY BOUND TRITIUM**

[^3]: COMARE’s 4^th^ Report 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 trivial.
10. Tritium is bound to organic compounds either by exchange reactions or by enzymatically-catalysed reactions. In exchange reactions, tritium is bonded to oxygen, sulphur, phosphorus or nitrogen atoms (ie as hydroxides, thiols, phosphides and amines). Conventionally, this is termed exchangeable OBT. In enzymatically-catalysed reactions, tritium is bonded to the carbon chain of an organic molecule: this is usually termed non-exchangeable OBT. Tritium so bound is more strongly attached than exchangeable tritium and has longer retention times: such bonds are only dissolved during catabolic reactions.

(a) Exchange Reactions

11. 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 (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 promiscuous of all atoms as regards exchange reactions. The consequence is that all organic molecules downwind of a tritium (HTO) discharge plume which come into contact with HTO will become tritiated very quickly. This includes all plant and animal species, all water-bearing material, and soil. It would include vegetables and fruit in exposed market places, for example.

12. 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 HTO, by definition, is a form of exchangeable OBT. However, historically-speaking, a clear distinction has always been made between HTO and OBT. For this reason, Diabaté and Strack (1993) stated that OBT should mean only non-exchangeable OBT. On the other hand, the Environment Agency (2000) (para 6.7) has more recently stated that “...recognising tritium's ability to exchange is inconsistent with distinguishing tritium as being either tritiated water or organically bound.” Consequently the EA has stated (Environment Agency (2001))

“...it currently takes the view that the more cautious, wider definition of OBT to include any organic matter which contains tritium either exchangeably bound or fixed more firmly to the carbon chain should be used. This will ensure that any dose assessments will take account of both forms of OBT whilst uncertainty remains. Indeed this definition more closely describes the OBT fraction which is measured by current laboratory methods”.

13. This report adopts the same view but will continue 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.

(b) Bikinetic Models for HTO

14. An acute HTO administration in humans results in OBT levels rising to about 6% (range 3-9%) of body HTO levels (Trivedi et al, 1997). Chronic HTO administration results in OBT concentrations increasing to higher levels, depending on how long the administration lasts. Osborne (1972) has stated there is 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 for 56 days to establish a steady state of T turnover: OBT levels rose to 22% of body HTO
levels. However it is unclear what would have happened had the experiment continued. Considerable evidence (see Appendix II) suggests that OBT levels slowly continue to increase and eventually equilibrate with body HTO levels. Most important is the evidence of OBT/HTO ratios of unity from background HTO exposures. Essentially, there is a disjunction between the evidence from animal experiments and the evidence from background levels which has yet to be explained.

15. Following HTO intake, the current model (ICRP, 1989) assumes 100% is absorbed and enters the blood. It assumes a turnover half-life of 10 days for HTO. It further assumes that 3% of HTO administered is bound, and that OBT doses from HTO administration can be safely neglected.

(c) Biokinetic Models for OBT

16. OBT has longer retention times than tritiated water as it is incorporated in a variety of biochemical compounds, such as amino acids, sugars, protein, starches, lipids and cell structural materials. Some biomolecules are well-preserved and long-lived, e.g. phospholipids in nerve cells, and, of course, DNA and RNA macromolecules. These longer retention times result in a greater radiotoxicity than tritiated water. OBT ingestion is widely recognised as being more hazardous than HTO ingestion. The ICRP (1989) 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".

17. 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).

18. Serious problems exist with this OBT model. The evidence for a carbon half-life of 40 days is fragmentary and limited: it does not take into account direct experimental evidence of considerably longer half-lives for the long-lived component of OBT. Table 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. The 1st component is conventionally assumed to be exchangeable OBT (ie bound to S, P and N atoms) and the 2nd component to be non-exchangeable OBT.

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

<table>
<thead>
<tr>
<th>Reference</th>
<th>No of cases</th>
<th>Biological Half-life (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>HTO</td>
</tr>
<tr>
<td>Pinson and Langham 1957</td>
<td>9</td>
<td>11.3</td>
</tr>
<tr>
<td>Butler and Leroy 1965</td>
<td>310</td>
<td>9.5</td>
</tr>
<tr>
<td>Osborne 1966</td>
<td>30</td>
<td>10.5</td>
</tr>
<tr>
<td>Snyders et al 1968</td>
<td>1</td>
<td>8.7</td>
</tr>
<tr>
<td>Sanders and Reing 1968</td>
<td>1</td>
<td>6.1</td>
</tr>
<tr>
<td>Minder 1969</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Lambert et al 1971</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td>Moghissi et al 1971</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Moghissi et al 1972</td>
<td>1</td>
<td>9.0</td>
</tr>
<tr>
<td>Balonov et al 1974</td>
<td>-</td>
<td>12.0</td>
</tr>
<tr>
<td>Rudran et al 1988</td>
<td>8</td>
<td>6.0</td>
</tr>
</tbody>
</table>
19. This evidence indicates the current ICRP metabolic model may be incorrect not only for OBT, but for carbon, ie organically bound C-14, as well.

20. Although little human data is available on the percentages of HTO retained as OBT1 and OBT2 after chronic intakes, a reasonable (but not necessarily conservative) assumption would be about 30% and 3% respectively. Using half-lives of 40 days and 550 days for OBT1 and OBT2 would likely result in a ~ 3 fold increase in the HTO dose coefficient and the OBT dose coefficient being 4 to 5 times greater than the HTO dose coefficient. Animal studies are informative here. Commerford, Carsten and Cronkite (1977) found most tritium doses came from OBT, 2 to 3 days after the cessation of chronic HTO administration to mice. Commerford et al (1982) after a transient HTO exposure to mice found all tritium remaining 8 weeks post-exposure was bound to DNA and histone. Although the amounts were small compared to HTO, cell nucleoproteins were much longer-lived: the authors concluded doses from them would exceed HTO doses.

21. If OBT/HTO equilibrium were to be insisted upon, OBT dose coefficients would be greater, but it is unclear whether such equilibria occur in the timespans of most occupational and public exposures to current anthropogenic HTO. Clearly, long-term exposures even to low levels of HTO are contraindicated, due to the build-up of OBT levels.

22. The current ICRP metabolic model for OBT results in ICRP’s OBT dose coefficient being 2.3 times greater than that for HTO. 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 component were included, the OBT dose coefficient would be 4 to 5 times higher than the HTO dose coefficient.

23. It is concluded here that the biokinetic models currently used for tritium may be incorrect: their use may result in unconservative dose estimates. From this analysis, it is likely that the dose coefficient for HTO may need to 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.

24. Animal studies lend support to this conclusion. As shown later, Commerford et al (1983) estimated that, following an intake of the US-recommended minimum daily amount of 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 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. The authors calculated that OBT doses to DNA in liver cells were 4.6 times higher than after HTO ingestion, not dissimilar to Commerford et al’s estimation of a 4 times increase in concentration.

**ISOTOPE EFFECTS IN DNA’S HYDRATION SHELL**

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4 Assuming the chronic exposure is limited in duration
5 ICRP dose coefficients for adults for tritiated water and OBT are $1.8 \times 10^{-11}$ Sv/Bq and $4.2 \times 10^{-11}$ Sv/Bq. Equivalent dose coefficients for infants are $4.8 \times 10^{-11}$ Sv/Bq and $1.2 \times 10^{-10}$ Sv/Bq respectively.
25. 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 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.

26. Mathur-De Vre and Binet (1982) again using magnetic resonance (MR) techniques, observed that tritiated water was concentrated in the hydration water of DNA due to isotopic discrimination. The isotopic difference between H and T is a factor of three: - the largest difference between any isotopes. Hydrogen-bonded interactions between HTO and macromolecules are stronger than those with ordinary water, because intermolecular bonds between hydrogen and \(^3\)H are energetically more stable than those between hydrogen and hydrogen.

27. 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 \(\beta\)-particles. This points out the importance of microlocalisation of initial energy deposition from hydration tritiated water in defining the RBE of HTO." Commerford (1982) is one of the few scientists to have cited the results of these MR studies. Baumgartner (2002) recently supported Mathur-De Vre et al's findings from both theoretical considerations and experimental results.

INTERNAL DOSIMETRY OF TRITIUM

28. This review focuses on tritium’s internal dosimetry: external tritium is not normally regarded as hazardous. Six aspects will be examined as follows:-

(a) RBE \(w_R\)
(b) transmutation effects
(c) ionisation-density (LET) effects
(d) distribution effects
(c) incorporation in DNA
(f) doses to DNA

(a) Tritium’s RBE \(w_R\)

29. In the past, considerable controversy has existed over tritium’s RBE. Unfortunately, in the past, non-scientific considerations may have played a part in the setting of tritium’s RBE. In 1959, ICRP Report 2 (1959) stated that the RBE for tritium’s beta-radiation was 1.7 and the ICRP (1963) then assigned a Q factor for tritium of 1.7. However, with little explanation, in 1969 the ICRP reduced it to 1.0 (Dunster, 1969) despite the preponderance of evidence to the contrary (Brues et al 1952), (Oliver and Lajtha, 1960), (Dewey et al, 1965)
(Hall et al, 1967) and (Lambert,1969).

30. Professor Karl Morgan, previously chair of a main committee of the ICRP, stated (1990) that in the 1950s and 1960s, during which he was a full member of the ICRP,

"...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."

31. After 1969, many observers (Johnson 1973), (Cronkite, Robertson and Feinendegen, 1973), (Moskalev et al, 1973), (Berry et al, 1973) and (Carsten, 1979) queried the choice of 1 for tritium's RBE. The NCRP (1979a, 1979b) lengthily reviewed the matter and concluded that an RBE of 1 could be ascribed to tritium where the reference radiation was 60-80 kVp X-rays. In the body (ie not the conclusion) of the report, it stated that using the normal referent radiation of 200 kVp X-rays, the RBE was closer to 2. In 1980, Till et al (1980a, 1980b) from the US Oak Ridge National Laboratory mounted a more rigorous and comprehensive examination 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.

32. In 1986, a joint Committee from the ICRP and the ICRU (the International Commission on Radiation Units and Measurements, sister organisation to the ICRP) recommended (ICRU, 1986) an increase in the quality factor for tritium from 1 to 2, for microdosimetric reasons. This recommendation was not implemented by the ICRP. Instead ICRP 60 continued to recommended a Radiation Weighting Factor (wR) of 1 (ICRP 1990) for all electrons (except Auger electrons). For Auger emitters, it recommended microdosimetric techniques be used, although such techniques and their theoretical underpinnings were not discussed.

33. Straume (1991) and Straume and Carsten (1993) comprehensively reviewed tritium data in 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 exposure was to tritiated nucleotides. Table 3 sets out the RBEs in vivo experiments from Straume’s tables.

**Table 3 RBE values for HTO (in vivo studies from Straume, 1991)**

<table>
<thead>
<tr>
<th>Mammary tumours in S-D rats</th>
<th>HTO/chronic X-rays</th>
<th>1.2 ± 0.3</th>
<th>Gragtmans 1984</th>
</tr>
</thead>
<tbody>
<tr>
<td>AML CBA/H mice</td>
<td>HTO/X-rays</td>
<td>1.2 ± 0.3</td>
<td>Johnson 1995</td>
</tr>
<tr>
<td>Various tumours in C57Bl/6NxC3H/He Mice</td>
<td>HTO/acute X-rays</td>
<td>~1</td>
<td>Yokoro 1989</td>
</tr>
</tbody>
</table>

**Chromosome Aberrations**

<table>
<thead>
<tr>
<th>Human lymphocytes</th>
<th>HTO/acute X-rays</th>
<th>1.91 ±0.65</th>
<th>Prosser et al 1983</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human lymphocytes</td>
<td>HTO/sub-acute γ</td>
<td>1.49 ±0.21</td>
<td>Morimoto et al 1989</td>
</tr>
<tr>
<td>Human lymphocytes</td>
<td>HTO/acute X-rays</td>
<td>1.13 ±0.18</td>
<td>Prosser et al 1983</td>
</tr>
<tr>
<td>Human lymphocytes</td>
<td>HTO/acute γ</td>
<td>3.4 ±0.64</td>
<td>Prosser et al 1983, Lloyd</td>
</tr>
<tr>
<td></td>
<td>Method</td>
<td>Value</td>
<td>Reference</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-------------------------------</td>
<td>-------</td>
<td>------------------------------------</td>
</tr>
<tr>
<td>Human lymphocytes</td>
<td>HTO/250 kVp X-rays</td>
<td>2.6</td>
<td>Vulpis 1984</td>
</tr>
<tr>
<td>Human lymphocytes</td>
<td>HTO/250 kVp X-rays</td>
<td>2.0</td>
<td>Prosser et al 1983, Lloyd et al 1988</td>
</tr>
</tbody>
</table>

**Reproductive Effects**

<table>
<thead>
<tr>
<th></th>
<th>Method</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouse spermatogonia</td>
<td>HTO/chronic X-rays</td>
<td>2.4</td>
<td>Lambert 1969</td>
</tr>
<tr>
<td>Mouse spermatogonia</td>
<td>$^3$HTdR/chronic X-rays</td>
<td>1.6</td>
<td>Lambert 1969</td>
</tr>
<tr>
<td>Mouse oocytes</td>
<td>HTO/chronic X-rays</td>
<td>2.9</td>
<td>Dobson and Kwan 1976</td>
</tr>
<tr>
<td>Fish germ cells</td>
<td>HTO/chronic $\gamma$</td>
<td>2.2</td>
<td>Etoh and Hyodo-Taguchi 1982</td>
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<tr>
<td>Fish fertility</td>
<td>HTO/chronic $\gamma$</td>
<td>~2</td>
<td>Hyodo-Taguchi and Hyodo 1985</td>
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<tr>
<td>Mouse testis weight loss</td>
<td>HTO/chronic $\gamma$</td>
<td>1.4</td>
<td>Carr and Nolan 1979</td>
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<tr>
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<td>$^3$HTdR/chronic $\gamma$</td>
<td>2.1</td>
<td>Carr and Nolan 1979</td>
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<td>Matsuda et al 1985</td>
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<td>Ueno et al 1982</td>
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<tr>
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<td>2.7</td>
<td>Kashima et al 1985</td>
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**Genetic Endpoints**

<table>
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<th></th>
<th>Method</th>
<th>Value</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Drosophila mutations</td>
<td>HTO/$\gamma$</td>
<td>2.7</td>
<td>Byrne and Lee 1989</td>
</tr>
<tr>
<td>Mouse mutations</td>
<td>HTO/chronic $\gamma$</td>
<td>2.7</td>
<td>Nomura and Yamamoto 1989</td>
</tr>
<tr>
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<td>HTO/chronic $\gamma$</td>
<td>2.5</td>
<td>Searle 1974</td>
</tr>
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<td>HTO/chronic $\gamma$</td>
<td>1.5</td>
<td>Carsten and Commerford 1976</td>
</tr>
<tr>
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<td>2.5</td>
<td>Xiang-yan et al 1986</td>
</tr>
<tr>
<td>Mouse specific locus</td>
<td>HTO/chronic $\gamma$</td>
<td>2</td>
<td>UNSCEAR 1982</td>
</tr>
</tbody>
</table>

**Developmental Effects**

<table>
<thead>
<tr>
<th></th>
<th>Method</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouse embryo</td>
<td>HTO/chronic $\gamma$</td>
<td>1.7</td>
<td>Yamada et al 1982</td>
</tr>
<tr>
<td>Rat embryo</td>
<td>HTO/chronic $\gamma$</td>
<td>2.6</td>
<td>Satow et al 1989</td>
</tr>
</tbody>
</table>

34. Tritium’s $w_R$ presently remains at unity. Harrison et al (2002) recently reviewed this matter (inter alia) and recommended an approximate doubling of tritium’s dose coefficients mainly due to tritium RBE uncertainties. From this analysis, it is also recommended that the $w_R$ for HTO should be increased from unity to at least 2.5. Indeed, as stated above, Straume and Carsten have recommended an increase to 3.

(b) Transmutation effects

35. When a tritium atom disintegrates, it not only emits radiation in the form of a $\beta$-particle but it also transmutes to a helium ion which is chemically and physically different from a hydrogen atom. This helium ion recoils from its emission of a $\beta$-particle 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.

36. NCRP Reports 62, 63 and 87 (1979a, 1979b, 1987) reviewed earlier studies on tritium’s transmutation effects: there are few recent studies. Feinendegen and Bond (1973) concluded tritium’s transmutation effects were small in comparison to the effects of its $\beta$-radiation. Experimentally, it is difficult to distinguish these effects because of the short track length of the $\beta$-particle.
37. Concern about transmutation has focussed primarily on the mutagenic effects of tritium incorporated into protein precursors 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. Tislijar-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.

38. 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 doses from OBT, RBE evidence and distributional effects.

(c) Ionisation-density (LET) effects

39. 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 energy (5.7 keV), or maximum energy (18.6 keV), has a range in tissue of about 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."

40. When beta-particles move through tissue, they lose energy by ionising and exciting molecules along their track, until they (and any secondary electrons they produce) slow down to thermal energies. Towards the end of the electron tracks, the average distance between ionisations is small, so they deposit a relatively large amount of energy in a very short distance. 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."
41. These conclusions were widely supported (e.g. Frankenberg et al\textsuperscript{6} (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-like (nanometre) dimensions, which could be responsible for a major part of the biological effectiveness of low-LET radiations.

42. 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 tritium's short beta particle track.

(d) Distribution

43. Because of tritium's short beta track, tritium's energy deposition closely follows its cellular distribution. Tritium's radiotoxicity (and RBE) is therefore highly dependent on its congruence with radiosensitive sites. Current dosimetric models assume tritium is homogenously distributed. This is a wide generalisation, and in most cases this is not known. In addition, it is difficult to distinguish experimentally whether a given level of biological effect from tritium, relative to that from another radiation, is the result of a heterogeneous concentration of tritium in the cell or cell nucleus (rather than being uniformly distributed) or is a result of an inherent greater effectiveness of the tritium decaying from one particular organic molecule rather than another.

44. Perhaps the most important target is DNA. Chromosome diameters for most cells are between 0.3 and 1.0\,\mu m. The track length of the tritium beta particle in tissue is 0.5 to 0.7\,\mu m (NCRP, 1979a). In this distance, the beta particle from tritium decay creates many ionisations. Because this track length is 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. This is explored next.

(e) Tritium's incorporation in DNA

45. 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 tritium from tritiated food clearly enters the DNA molecule. Commerford et al (1977) indicated the same from chronic HTO ingestion. Commerford et al (1982) found, after a transient HTO exposure to mice, all the tritium remaining 8 weeks post-exposure was bound to DNA and histone\textsuperscript{7}. 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 HTO doses. DNA half-lives were extremely long: 318 days for mice.

\textsuperscript{6} who observed that electron track ends induced double stand breaks in DNA

\textsuperscript{7} the protein bead on which the DNA is helically wound inside the chromosome
liver DNA and 593 days for mice brain DNA: ie 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, ie key cells in embryos (including nerve cells and oocytes).

(f) Tritium’s Doses to DNA

46. Tritium doses to DNA originate from its incorporation in the constituents of the nucleus. These are, on average, 70% (by weight) water, about 20% proteins, 3% lipids, and the rest nucleoproteins: DNA constitutes about 3% of the nucleus (Commerford, 1982). These proportions are illustrative, as ratios vary considerably among cell types. 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

47. 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. After 2 to 3 days after the cessation of HTO administration, most dose came from OBT. After 15 months, the DNA of the cells of various mice organs was tritiated to 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

48. Etnier, Travis and Hetrick (1984) estimated that most protein in food crosses the human gut wall and is available for incorporation, particularly the 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, similar to Commerford et al's (1983) estimation of a 4 times greater OBT than HTO concentration in cell nuclei following tritiated protein intake.

49. 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", ie 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 25-50 times radiologically more effective than HTO.
Nucleic acid precursors have been studied extensively, particularly thymidine and deoxycytidine which is incorporated only into 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.

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 discharging facilities) than tritiated thymidine. However great care has to be taken with facilities which manufacture (and discharge) tritiated proteins and nucleic acid precursors, e.g. at Cardiff.

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.

EPIDEMIOLOGICAL EVIDENCE

Few studies directly examine effects which may arise from tritium exposures.

The following Canadian studies are of interest as Canadian heavy water stations emit unusually large amounts of tritium (~2.5 PBq annually). In 1988, Durham Nuclear Awareness, an environment NGO in Canada, published a report by an independent researcher (McArthur, 1988), 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 Mr Hugh Richards who also found a correlation between lagged tritium emissions from a plant near Cardiff with neonatal deaths. The Committee is considering this report separately. The similarity of the findings is interesting. On 2 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 heavy water reactors at Kota in Rajasthan in India which discharge large amounts of tritiated water. However none of these reports has been published in the scientific literature.

AECB studies (1989, 1991a) found childhood leukemia death rates near the Bruce and Pickering reactors in Ontario were 1.4 times higher than expected (Observed = 36, Expected = 25.7). Childhood leukemia deaths were greater after the plant opened than before, with before and after mortality ratios 1.08 and 1.34 respectively at Pickering. More leukemia deaths were counted at the child's place of birth than the child's place of death. These
findings were statistically significant at the p=0.06 level.

56. Another AECB report (1991b) found elevated birth defect rates near the Pickering nuclear station adjacent to Toronto, Ontario. The study found an 80% \((O = 24, E = 12.9)\) increased birth prevalence of Down's syndrome at the nearby town of Pickering, and a 46% \((O=14, E = 9.6)\) increase at Ajax, a town further away. These were correlated with airborne tritium releases in the case of Pickering, and ground monitored tritium levels in the case of Ajax. The report also found an association between high tritium emissions and central nervous system anomalies in births at Pickering.

CONCLUSIONS

57. Conclusions and recommendations are made on the following matters

(a) hazardous properties
(b) dose coefficients
(c) possible risks of tritium exposures
(d) further research

(a) hazardous properties

58. 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 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 drawn up and published and made available to regulatory agencies. In particular, it is recommended that a hazard guide for most common radionuclides should be published taking into account other properties of nuclides as well as their radiotoxities. Such properties should include those listed in paragraph 5 above with the addition of the property of very long radiological half-lives.

(b) dose coefficients

59. It is recommended that tritium’s dose coefficients should be increased as follows. First, tritium’s \(w_R\) should be increased to 2.5. Second, its biokinetic model should be improved to recognise tritium’s long-lived OBT components. This would increase HTO’s doses by a factor of \(\approx 3\). Third, there should be some recognition of tritium’s hazardous properties, ie its ease of uptake, propensity to exchange, and concentration in DNA’s hydration shell. Although there is much uncertainty here, an overall factor of 2 is suggested: this may need to be changed in future. Multiplying these three factors means that HTO’s current small dose coefficients should be increased by an overall factor of \(2.5 \times 3 \times 2 = 15\). This would leave tritium’s dose coefficient \(\approx 50\) times lower than that for Cs-137.

60. It is also recommended that OBT’s dose coefficient should be \(\approx 5\) times greater than that estimated for HTO; currently the OBT coefficient is 2.3 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

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8 However the report concluded only that the findings were “not statistically significant” ie at the 95% confidence level. Although factually correct, the statement was perhaps misleading.
are essential amino acids (e.g., leucine and lysine) as they are not manufactured in the body but taken up directly from diet. From the studies by Commerford et al. (1983) and Komatsu et al. (1990), OBT doses to DNA are 4.6 times greater than from HTO following tritiated protein intake. Other authors have estimated greater effects\(^9\) from OBT. 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 5 is applied to all OBT in diet.

(c) possible risks of tritium exposures

61. In pregnant women, cell proliferation occurs in the zygote, embryo, and foetus. Transient high concentrations of HTO could result in the ingestion/inhalation/absorption of HTO by pregnant women and heavy T labelling of embryos 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 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 (including nerve cells and oocytes). Straume (1991, 1993) estimated that tritium’s teratogenic risks were 6 fold greater than tritium’s carcinogenic risks.

(d) further research

62. 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 McArthur, Richards and AECB reports.

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March 23, 2003

\(^9\) As stated above, Feinendegen et al. (1980) estimated tritiated protein was 25-50 times more effective than HTO, but their reasoning for this is unclear.
APPENDIX I: EXPRESSIONS OF SCIENTIFIC CONCERN RE: TRITIUM

1. Radiological Effectiveness of Tritium

"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)

"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 β 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)

2. Tritium's Effects on DNA

"...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 β 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 of 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)
3. Tritium’s Genetic Effects

“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 β-particle range and the short range of the β-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 β 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 β particle and the possible effects of transmutation.” Cronkite EP, Robertson JS and Feinendegen LE (1973)

4. Organically Bound Tritium

“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)

5. Future Tritium Releases

“... 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: EVIDENCE FOR EQUILIBRIA IN OBT/HTO LEVELS AFTER LONG-TERM HTO EXPOSURES

Long-term mice studies by Commerford et al (1977) showed OBT/HTO ratios between 0.73 and 1.07 in the DNA of cell nuclei of various 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 likely these animals would 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 (ie 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 in India. 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.

All humans are chronically exposed to background tritiated water levels, therefore it is instructive to examine background OBT/HTO ratios in humans. Several studies (Bogen et al, 1973) (Bogen et al, 1979), (Ujeno et al, 1989) and (Hisamatsu et al, 1989) in US and Japan reveal these ratios usually are unity or in some cases, slightly higher than unity. The same is found with studies of background OBT/HTO ratios in plants (Belot, 1986).
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