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Derivation of Dose Conversion Factors for Tritium

G. G. Killough

Prepared for the U.S. Nuclear Regulatory Commission
Division of Health, Siting, and Waste Management
in the Office of Nuclear Regulatory Research
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DERIVATION OF DOSE CONVERSION FACTORS FOR TRITIUM

G. G. Killough

ABSTRACT

For a given intake mode (ingestion, inhalation, absorption through the skin), a *dose conversion factor* (DCF) is the committed dose equivalent to a specified organ of an individual per unit intake of a radionuclide. One also may consider the *effective dose commitment* per unit intake, which is a weighted average of organ-specific DCFs, with weights proportional to risks associated with stochastic radiation-induced fatal health effects, as defined by Publication 26 of the International Commission on Radiological Protection (ICRP). This report derives and tabulates organ-specific dose conversion factors and the effective dose commitment per unit intake of tritium. These factors are based on a steady-state model of hydrogen in the tissues of ICRP's Reference Man (ICRP Publication 23) and equilibrium of specific activities between body water and other tissues. The results differ by 27-33% from the estimate on which ICRP Publication 30 recommendations are based. The report also examines a dynamic model of tritium retention in body water, mineral bone, and two compartments representing organically-bound hydrogen. This model is compared with data from human subjects who were observed for extended periods.

The manner of combining the dose conversion factors with measured or model-predicted levels of contamination in man's exposure media (air, drinking water, soil moisture) to estimate dose rate to an individual is briefly discussed.

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1. INTRODUCTION

In making environmental radiological assessments, it is frequently convenient to think in terms of *dose conversion factors* for the radionuclides released to the environment. For a given intake mode (ingestion, inhalation, absorption through the skin), a *dose conversion factor* (DCF) for a particular radionuclide is the dose equivalent commitment to a specified organ per unit intake of the radionuclide. In lieu of an organ-specific dose conversion factor, one also may consider the *effective dose commitment*, which is a weighted average of organ-specific DCFs, with weights proportional to risks associated with stochastic fatal health effects, as defined by Publication 26 of the International Commission on Radiological Protection (ICRP, 1977). The effective dose commitment is defined in Sect. 2 of this report.

For tritium in the form of tritiated water (HTO), there are two approaches to deriving DCFs. The first is based on fitting decay curves to bioassay data obtained from acutely exposed subjects. The curves are combined with other physiological information to construct dynamic compartment models which simulate the retention of tritium over time following an initial uptake. The second approach assumes a steady-state equilibrium of tritium specific activity in a hypothetical subject's tissues with that in his exposure environment (e.g., food or atmospheric moisture). Knowledge of fractional hydrogen content of specific tissues can then be used to estimate dose-equivalent rate per unit rate of intake of tritium. In Sects. 2, 2.1, 2.2, and 2.3 of this report, we examine both approaches and use the second to estimate organ-specific DCFs for HTO and organically-bound dietary tritium.

In Sect. 3, we indicate briefly the manner of combining DCFs with measured or model-predicted specific activities of tritium in environmental exposure media to estimate annual dose equivalent rates to individuals from such exposure.

It is not possible to include in this report a review of the extensive literature that bears on the estimation of radiation dose due to exposure of individuals to tritium. Our aim is to complement earlier work by Killough et al. (1978) and to clarify the difference between the DCFs given there and other estimates, such as those of ICRP Publication 30 (ICRP, 1979; Eckerman et al., 1981; Dunning and Killough, 1981).

In all calculations, we assume for tritium a radiological half-life of 12.28 yr and average β^- energy of 5.685 keV per transformation (Kocher, 1981).

2. DOSE EQUIVALENT AND METABOLIC MODELS

We base our estimates of dose equivalent due to internal deposition of tritium on the following equation:

$$D_{j \leftarrow i} = S_{j \leftarrow i} q_i \qquad (1)$$

where

$D_{j \leftarrow i}$ = average dose equivalent rate (Sv d^{-1}) to target organ j due to tritium activity in source organ i

q_i = tritium activity (TBq) in source organ i.

The quantity $S_{j \leftarrow i}$ (the "S-factor") in Eq. (1) transforms terabecquerels of tritium activity in source organ i into dose equivalent rate in target organ j.

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For most organs and tissues, the average emitted β^- energy of 5.685 keV is treated as if it were completely absorbed within the source region, and in such cases the only target that need be considered is the source organ itself. Exceptions are transfers of energy among skeletal tissues that are treated as discrete targets (endosteal cells, red marrow) and from contents to walls of the gastrointestinal tract.

The S-factor, for our purposes, may be expressed as follows:

$$S_{j \leftarrow i} = \frac{k(QF)\phi_{j \leftarrow i} E}{M_j} \quad \text{Sv d}^{-1} \text{ to organ } j \text{ per TBq in organ } i \tag{2}$$

(cf. Snyder et al., 1974; Snyder et al., 1975), where

k = 1.38×10^7 g Gy $(\text{MeV})^{-1}\,\text{d}^{-1}\,\text{TBq}^{-1}$

(QF) = quality factor for conversion of absorbed dose (Gy) to dose equivalent (Sv)

$\phi_{j \leftarrow i}$ = fraction of energy emitted by tritium activity in source organ i that is absorbed by target organ j

E = average emitted energy (MeV) per transformation

M_j = mass (g) of target organ j.

In 1966, the International Commission on Radiological Protection recommended continued use of 1.7 as the quality factor for β^-, β^+, and e^- radiation with maximum energies $\leq$0.03 MeV (ICRP, 1966). In a subsequent amendment, the Commission recommended reduction of the quality factor to 1.0 for all β^-, β^+, e^-, γ, and x rays (ICRP, 1969). In all dose estimates given in this report, we have used $(QF) = 1.0$.

Use of Eq. (1) to estimate the dose equivalent rate requires that the levels q_i (TBq) of tritium activity in those source organs and tissues that contribute to dose equivalent in target organs of interest be known in relation to tritium intake. For a constant rate of intake, steady-state tritium-to-hydrogen ratios may be expected to be the same in tissue as in the source (food, water, or atmospheric moisture), apart from radioactive decay and isotopic fractionation. This assumption permits us to estimate dose equivalent rate per unit intake without making specific assumptions about metabolic dynamics (tissues with hydrogen pools that turn over slowly, such as mineral bone, are exceptions to this statement; the turnover rate must be estimated to permit correction for radioactive decay). On the other hand, dynamic metabolic models make it possible to consider dose rate as it varies with time. The responses of such models may be compared with data from human or animal subjects to provide a check on their validity, or from another perspective, the models may be calibrated with such data.

The *dose equivalent commitment* to organ j, D_j (Sv), is defined as

$$D_j = \sum_i \int_{t_0}^{t_{max}} D_{j \leftarrow i}(t)\,dt \tag{3}$$

where the index i ranges over all source organs whose activity contributes to absorption of energy in target organ j. The time at which intake begins is t_0, and t_{max} may be considered as the end of life of the

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hypothetical individual (common practices are to assign $t_{max} - t_0 = 50$ yr to span a working lifetime in occupational settings, or $= 70$ yr to represent an average lifetime for members of the public). In this report, we apply Eq. (3) only in the case of a unit acute exposure, i.e., instantaneous intake of 1 TBq of tritium at time t_0.

Table 1 shows dose equivalent factors that convert a single intake of tritium (TBq) to dose equivalent commitment (Sv) for each target organ of interest [or equivalently, a constant intake rate (TBq d^{-1}) is converted to dose equivalent rate (Sv d^{-1}) once the body has come into equilibrium with the intake]. The factors corresponding to ingestion may be assumed to apply to tritium that substitutes for ^1H in dietary constituents. The remaining factors are appropriate for intake of tritium as HTO by inhalation or absorption through the skin. Table 1 was computed from steady-state assumptions for which details are given in Sect. 2.2.

The quantity labeled *effective dose commitment* in Table 1 may be defined as that uniform whole-body dose equivalent that corresponds to the same expected number of fatal stochastic health effects as the particular (possibly nonuniformly distributed) dose equivalent commitment under consideration. If H_E denotes the effective dose commitment, then

$$H_E = \sum_j w_j D_j \quad \text{Sv TBq}^{-1} \tag{4}$$

(ICRP, 1977), where index j ranges over the target organs specified in Table 1, w_j = weighting factor that gives the fraction of the total risk of fatal stochastic effects resulting from organ j under uniform whole-

Table 1

Dose equivalent, organ-specific risk factors, and proportional weights

Organ	Dose equivalent commitment (Sv TBq^{-1})	Risk[a] (Sv^{-1})	Proportional weight w_j
Gonads	22	.004	.25
Breast	21	.0025	.15
Red marrow	22	.002	.12
Lung	22	.002	.12
Thyroid	22	.0005	.03
Bone surfaces	17	.0005	.03
Remainder[b]	27[c] (22)[d]	.005	.30
		.0165	1.00
Effective dose commitment	23[c] (22)[d]		

[a]Interpreted as the number of fatal stochastic health effects per sievert of uniform whole-body radiation.

[b]Consists of the 5 organs, other than those listed explicitly, that receive the highest dose-equivalent commitment. The dose-equivalent commitments shown are averages of the estimates for the five organs.

[c]Intake by ingestion. The higher value is due to the tritium in the migrating contents of the gastrointestinal tract.

[d]Intake by inhalation or absorption through the skin.

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body irradiation (Table 1), and D_j = dose equivalent commitment (Sv) to organ j due to intake of 1 TBq of the radionuclide [Eq. (3)]. Thus the product $0.0165H_E$ (of which the first factor is the total stochastic risk of fatal health effects from Table 1) is the expected number of fatal health effects, associated with the effective dose commitment H_E, that would be estimated from the ICRP organ risk factors.

We note that the recommended limits of exposure to tritium in ICRP Publication 30 (1979) are based on an estimate of dose equivalent to soft tissue (6.3 kg) of 17.3 Sv TBq^{-1} (Eckerman et al., 1981). This estimate is computed from exponential removal from a single compartment with a biological half-time of 10 days and differs from the effective dose equivalent estimates given in Table 1 for ingestion and for other intake modes by 33 and 27%, respectively.

2.1 Metabolism

An intake of HTO or T_2O either by ingestion or inhalation is generally assumed to be completely absorbed and to mix rapidly with the water content of the body. Exposure to a contaminated atmosphere results in complete uptake of inhaled HTO and its absorption through intact skin at a comparable rate. Pinson and Langham (1957) estimated that the rates were equal, and results of a study reported by Osborne (1966) suggest that absorption through the skin accounts for about 60% of the total uptake rate.

Numerous studies of excretion data from human subjects after accidental or voluntary exposure to tritium oxide have indicated that the concentration in body water is well represented by a single exponential term for the first two months or more (e.g., Butler and LeRoy, 1965). Some studies have detected one or two additional exponential components with longer decay half-times, i.e., slower removal rates (Snyder et al., 1968; Sanders and Reinig, 1968; Moghissi et al., 1973). Bennett (1973) reviewed 12 studies and concluded that half-times of $T_1 = 9$, $T_2 = 30$, and $T_3 = 450$ days are reasonable average values when the retention of tritium in body water is represented by a function of the form

$$R(t) = c_1\exp(-0.693t/T_1) + c_2\exp(-0.693t/T_2) + c_3\exp(-0.693t/T_3) \quad (5)$$

where $c_1 + c_2 + c_3 = 1$ and $R(t)$ denotes the fraction of an initial acute uptake of tritium by body water, with no subsequent contamination, that remains t days later.

The second and third exponential terms are assumed to be associated with organically bound hydrogen pools in the body, while the first is closely related to the turnover of body water. The value of T_1 has been shown to vary with season, climate, and age; observed values range from 4 to 28 days (Butler and LeRoy, 1965; Wylie et al., 1963; Moghissi and Carter, 1971; Moghissi et al., 1972).

Sanders and Reinig (1968) proposed a three-compartment model for retention of tritium in the body, with a central compartment representing body water and exchanging tritium with two satellite compartments by linear donor-controlled fluxes; in their model, tritium entered and left the body only through the central compartment. They estimated transfer coefficients by mathematical fitting of the tritium retention function to data collected from an accidentally exposed worker, from whom urinary samples had been taken over a period of 415 days. Snyder et al. (1968) fitted a two-compartment model to data from an accidentally exposed subject who was observed for 255 days. The discrete points plotted in Fig. 1 are based on the data from the studies of Sanders and Reinig (1968) and Snyder et al. (1968). In both of the models proposed by these investigators, the additional one or two compartments are assumed to represent an organically bound hydrogen pool. Bennett (1973) assumed that these two compartments accounted for hydrogen fixed in non-fat, non-mineral tissue solids, which he estimated as 120 and 600 gH, and he assigned to them removal half-times of $T_2 = 30$ and $T_3 = 450$ days, respectively. To the 42,000-ml body water compartment, he assigned $T_1 = 9$ days for excretion. Report No. 62 of the National Council on Radiation Protection and Measurements (NCRP, 1979) adopts this model and indicates that it gives a good fit to the data of Sanders and Reinig (1968) and Snyder et al. (1968).

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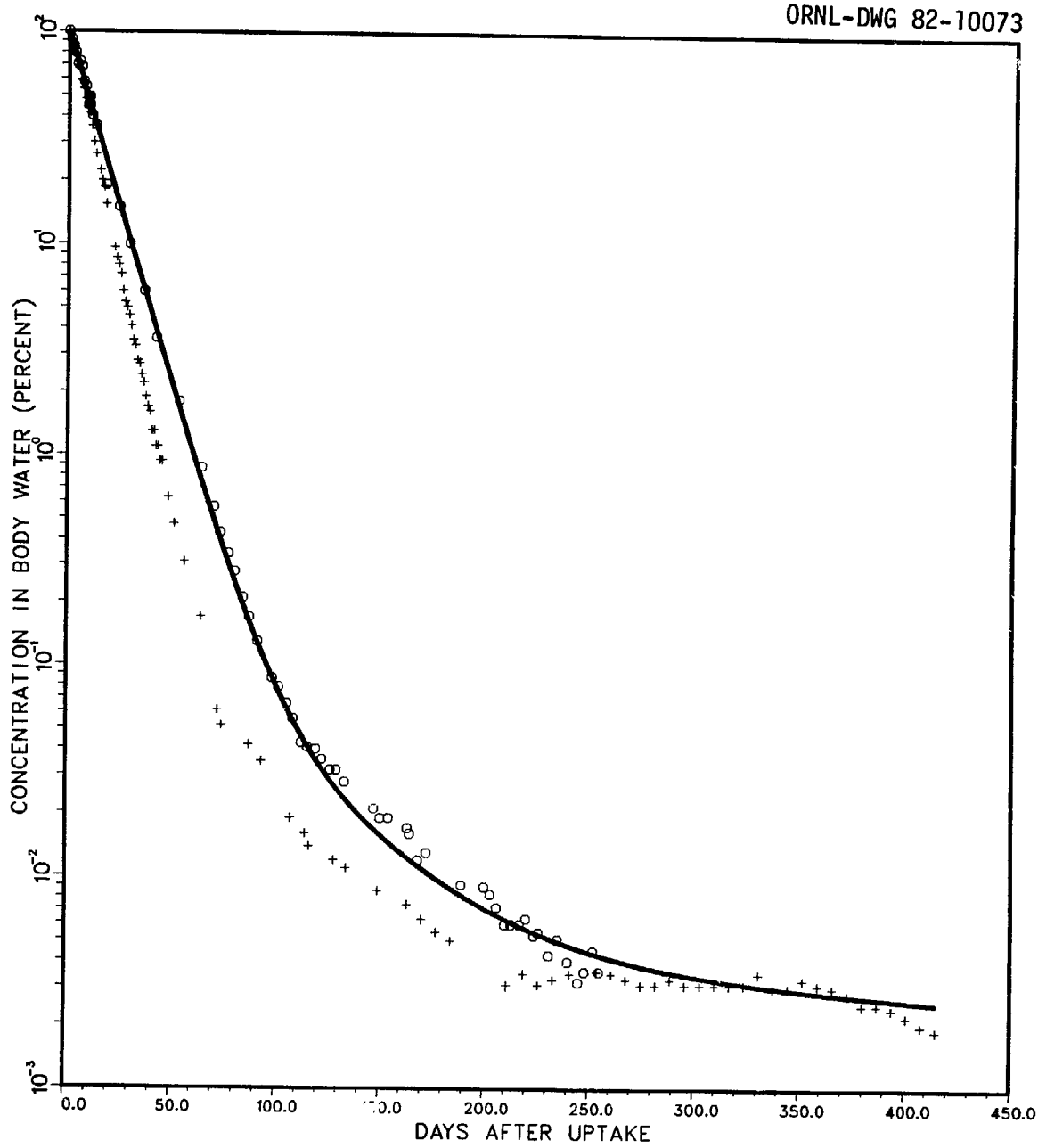

Fig. 1. Dynamic model for retention of tritium in body water (curve). Data are from Snyder et al. (1968; o) and Sanders and Reinig (1968; +).

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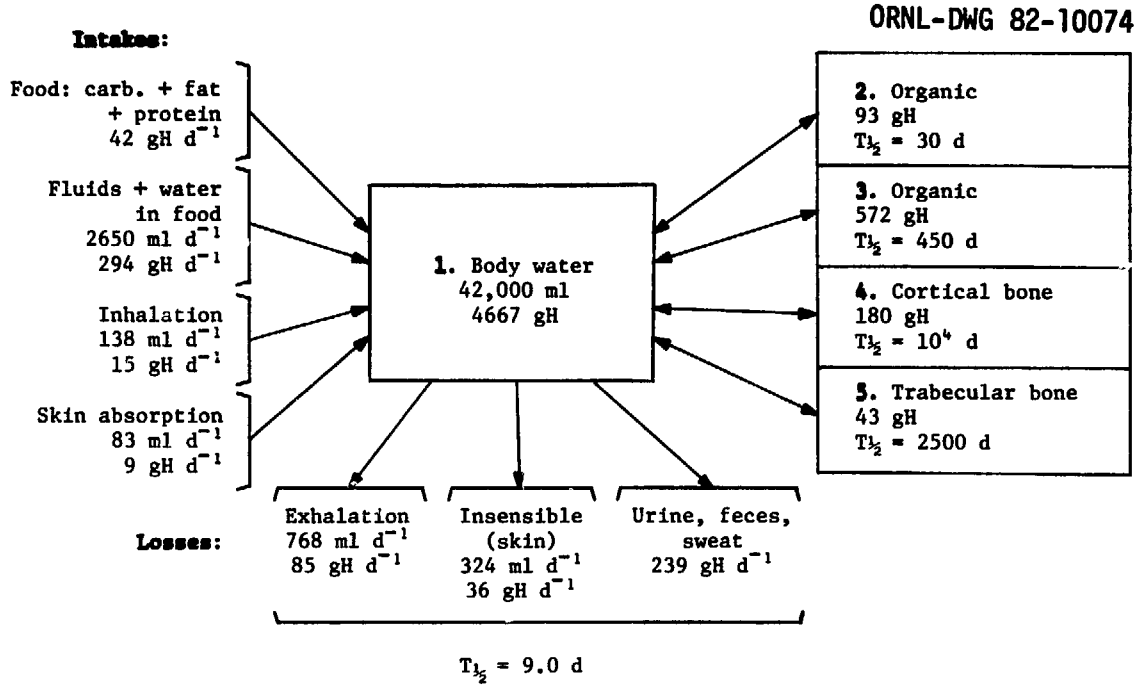

Fig. 2. Compartment diagram for the dynamic and steady-state models of hydrogen uptake, retention, and removal.

We adopt an approach similar to those of the preceding paragraph for constructing a model of the gross metabolism of tritium that is absorbed as HTO or that substitutes for ^1H in dietary protein, carbohydrate, or fat. We impose the added requirement that the physiological assumptions be consistent, insofar as is practicable, with the properties of the ICRP's Reference Man (ICRP, 1975). Moreover, to the structure of the model of Sanders and Reinig (1968), we add compartments for cortical and trabecular bone and impose an explicit breakdown of the modes of hydrogen uptake and loss by the body (Fig. 2).

The organic compartments of our model are based on the following considerations. Except for fat (13.5 kg), body water (42 liters), and mineral bone (5 kg), the body of Reference Man is 9.5 kg of tissue solids that are assumed to be 7% hydrogen, or 655 gH, which are to be partitioned into two components. With these components we associate the longer-term removal half-times suggested by Bennett (1973), namely T_2 = 30 and T_3 = 450 days, and determine the partition fraction (14% to compartment 2) by requiring the total daily exchange of hydrogen between these compartments and body water to be 3 gH. This value is approximately the difference of Reference Man's dietary intake and food oxidation (42 - 39 gH d^{-1}; see Table 2).

In Reference Man, cortical and trabecular bone have turnover rates of 2.5% and 10% per year, respectively (ICRP, 1975); the respective hydrogen contents are 180 and 43 g. On the basis of these numbers, we have calculated transfer parameters for the bone compartments of the model. The long-term exponential components introduced by these compartments, however, are not observable in existing experimental data, corresponding as they do to removal half-times of 10^4 and 2500 days, respectively.

Body fat is about 12% hydrogen, and while it is known to be actively metabolized, we know of no estimates of its turnover rate in man, though in rats and mice a replacement half-time of about a week has been observed (Bell et al., 1968). We make no attempt to quantify fat in the dynamic model corresponding

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to Fig. 2. In the dose calculations for Table 1, however, which are based on steady-state assumptions, the essential fat incorporated into specific organs and tissues is considered.

Biological removal of tritium from the body occurs by way of urination, fecal excretion, sweat, exhalation, and insensible loss through the skin. Our interpretation of the apportionment of the total hydrogen loss among these pathways is based on a total hydrogen balance for Reference Man (ICRP, 1975), augmented by other data (Table 2). Intake of hydrogen in food and fluids is calculated from Reference Man's daily intake of cabohydrates, fat, and protein, and water balance. For inhalation, we assumed Reference Man's ventilation rate of 23 $m^3\ d^{-1}$ and an absolute humidity of 6 $gH_2O\ m^{-3}$; the latter number is an average value that is typical of the mid-latitudes of the Northern Hemisphere (NCRP, 1979). Diffusion inward through the skin was calculated from the 9.6 liters gas min^{-1} estimated by Osborne (1966) from experiments with volunteer subjects in controlled environments of tritiated water vapor. Loss through the lungs is calculated from the ideal gas law on the assumption that exhaled air is at 32°C and saturated with water vapor; physiological measurements have shown that these assumptions are very nearly satisfied under a wide variety of external conditions (Fenn and Rahn, 1964). The resulting distribution of daily hydrogen intakes and losses is shown in Fig. 2.

We remark that Fig. 2 is not perfectly consistent with Table 2. To force the balance, we chose to reduce the insensible water loss to the value shown in Fig. 2 on the assumption that some part of the number given in Table 2 for Reference Man (850 ml d^{-1}) must be counting exhalation of vapor, and in fact, one reads on p. 360 of ICRP Publication 23 (1975) that ¼ to ⅓ of insensible loss of water is through the lungs, but this information is not included explicitly. Our value seems somewhat high, and indeed, our calculations assign a greater total vapor flux to the lungs, whereas experimental evidence favors the skin.

The curve in Fig. 1 gives the body water response of the dynamic model based on Fig. 2, together with curves and data from the two studies with human subjects described previously (Snyder et al., 1968;

Table 2

Hydrogen intakes and losses for ICRP's Reference Man[a]

	Intakes			Losses	
	ml $H_2O\ d^{-1}$	$gH\ d^{-1}$		ml $H_2O\ d^{-1}$	$gH\ d^{-1}$
Water					
Tap water	150	17	Urine	1,400	156
Milk	300	33	Feces	100	11
Other fluids	1,500	167	Insensible	850	94
In food	700	78	Sweat	650	72
Oxidation	350	39			
Total H_2O	3,000	333		3,000	333
Organic					
Carbohydrate (6.2% H)		14→20[b]	Fecal loss		13
Fat (12% H)		10→15[b]			
Protein (7% H)		5→ 7[b]			
Total		29→42			13

[a]ICRP (1975).

[b]Range based on Figs. 68-70 for carbohydrate, fat, and protein, respectively, ages 20-80, from ICRP (1975).

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Sanders and Reinig, 1968). Explicit time-dependent solutions for the activity in each compartment of the model are given in Table 3. For each of the five compartments, the solution shown represents activity remaining in the compartment t days after the introduction of 1 TBq into body water. Table 4 shows the time integrals of these activity histories. In Sect. 2.3, we illustrate the use of the dynamic compartment model in estimating dose conversion factors.

2.2 Calculation of Dose Conversion Factors

We assume an intake of tritium into the body at a constant rate (1 TBq d^{-1}) and calculate the steady-state level that will occur in each body compartment after an initial period of equilibration. These levels of activity, multiplied by the appropriate S-factors [Eqs. (1) and (2)], give dose equivalent factors with units Sv TBq^{-1}. The following paragraphs give some details of the calculation. The dose conversion factors are displayed in Table 1.

Table 3

Coefficients and removal rate constants for compartment model solutions[a]

(i) Compartment	c_{i1}	c_{i2}	c_{i3}	c_{i4}	c_{i5}
(1) Body water	9.96(-1)	3.51(-3)	5.06(-5)	3.11(-8)	1.20(-7)
(2) Fast organic	-8.35(-3)	8.35(-3)	1.08(-6)	6.23(-10)	2.42(-9)
(3) Slow organic	-2.46(-3)	-3.10(-5)	2.49(-3)	4.00(-9)	1.79(-8)
(4) Cortical bone	-3.42(-5)	-4.11(-7)	-9.21(-8)	3.47(-5)	-1.54(-9)
(5) Trabecular bone	-3.27(-5)	-3.96(-7)	-1.03(-7)	3.83(-10)	3.32(-5)
Total of all compartments	9.86(-1)	1.18(-2)	2.54(-3)	3.47(-5)	3.34(-5)
Removal rate constants λ_j^b (d^{-1})	7.81(-2)	2.29(-2)	1.54(-3)	6.93(-5)	2.77(-4)

[a]Interpretation: with 1 TBq in body water at time zero, the activity in compartment i at subsequent time t (d) is

$$q_i(t) = \sum_{j=1}^{5} c_{ij}\exp[-(\lambda_j^b + \lambda^r)t] \text{ TBq}$$

where $\lambda^r = 1.546\times10^{-4}\,d^{-1}$.

Table 4

Integrated activities (TBq d) *in the 5 compartments of the model*[a,b]

(1) Body water	12.9
(2) Fast organic	0.256
(3) Slow organic	1.44
(4) Cortical bone	0.152
(5) Trabecular bone	0.0764

[a]Corresponding to an initial uptake of 1 TBq in body water.

[b]An alternate interpretation: the compartment activity levels (TBq) at steady state corresponding to a constant uptake rate of 1 TBq d^{-1} to body water.

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The organically bound hydrogen and mineral bone compartments of our model are corrected for radiological decay as follows:

$$A_j = \frac{A_w \alpha_{j \leftarrow w} \lambda_j^b}{\alpha_{w \leftarrow j} \lambda_j^b + \lambda^r} \tag{6}$$

where A_j denotes the tritium specific activity [TBq $(gH)^{-1}$] in the source compartment in question, A_w is specific activity in body water, λ_j^b is the biological removal rate constant for the compartment, and λ^r is the radiological decay rate constant. The factors $\alpha_{j \leftarrow w}$ and $\alpha_{w \leftarrow j}$ are included to acknowledge the possible presence of an isotope effect in the exchange, but the calculations discussed here have been carried out with $\alpha_{j \leftarrow w} = \alpha_{w \leftarrow j} = 1$.

When source and target organ are the same, the dose equivalent rate is computed as $kE(q_j/M_j)$ Sv d^{-1} [Eqs. (1) and (2)]. But $q_j/M_j = A_j F_j^H$, where F_j^H is the fraction of the mass of organ j that is hydrogen. We have

$$A_j F_j^H = (0.111) F_j^w A_w + (0.122) F_j^{fat} A_{fat} + (0.921)(0.07)(1 - F_j^w - F_j^{fat}) A_w \tag{7}$$

where F_j^w and F_j^{fat} are mass fractions of water and fat, respectively, in source organ j, and 0.111, 0.122, and 0.07 are hydrogen fractions of water, fat, and nonfat, non-mineral tissue solids, respectively. The value 0.921 is the average ratio A_j/A_w for the organically bound hydrogen compartments obtained from Eq. (6). Estimates of F_j^w and F_j^{fat} for various organs of Reference Man are presented in Table 5. With the assumption $A_{fat} = A_w$, equation (7) has been used for computation of most of the dose equivalent estimates on which numbers in Table 1 depend. Exceptions are the skeleton and gastrointestinal tract, as discussed in the following paragraphs.

Skeleton. Source tissues in the skeleton are red and yellow marrow and cortical and trabecular bone; target tissues are red marrow and endosteal cells. S-factors for tritium are given in Table 6 for these source-target combinations (Snyder et al., 1974; Killough et al., 1978). To compute steady-state activity in the source tissue, we use $F_{cort}^H = 0.045$ and $F_{trab}^H = 0.043$ (ICRP, 1975) and the respective ratios A_j/A_w from Eq. (6). For red and yellow marrow, $A_j F_j^H$ are computed from Eq. (7).

Gastrointestinal tract. The target tissues of the gastrointestinal (GI) tract are stomach wall, small intestine wall, upper large intestine wall, and lower large intestine wall. For tritium incorporated in these tissues, we use the formula $kEA_jF_j^H$ Sv d^{-1} per TBq $(gH)^{-1}$ in body water, with $A_jF_j^H$ being obtained from Eq. (6). When the contents of the GI tract are also contaminated with tritium (intake by ingestion), we proceed as follows. Mean transit times for material in each of these four GI segments have been estimated by Eve (1966) to be 1/24, 1/6, 13/24, and 1 day for the stomach, small intestine, upper large intestine, and lower large intestine, respectively. Masses of the contents of these segments are 250, 400, 220, and 135 g, respectively. Absorption is considered to occur in the small intestine at a rate that removes 95% of the tritium from the contents during transit. The dynamic model of the GI tract is catenary, with absorption and movement from one segment to the next by first-order kinetics (Bernard, 1968).

The levels of activity in the contents of the model GI tract are calculated for an intake rate of 1 TBq d^{-1} as follows:

Stomach (S):

$$q_{S\,cont} = \frac{1}{\lambda_S + \lambda^r} = 4.17 \times 10^{-2} \quad \text{TBq}$$

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Table 5

Mass fractions of various organs of Reference Man[a] *that are water and fat* (%)

Organ	Water	Fat
Kidneys	77	5
Liver	72	7
Lungs	78	10
Thyroid	75	10
Pancreas	71	8
Testes	80	3
Red marrow	40	40
Yellow marrow	15	80
Stomach wall	73	6
Small intestine wall	80	6
Upper large intestine wall	81	6
Lower large intestine wall	81	6
Female breast	75	3
Cortical bone[b]	15	1
Trabecular bone[c]	23	1

[a]ICRP (1975).

[b]Protein content: 25%.

[c]Protein content: 24%.

Table 6

S-factors for tritium in skeletal tissues (Sv d^{-1} *to target tissue per* TBq *in source tissue*)[a]

	Source			
Target	Cortical bone	Trabecular bone	Red marrow	Yellow marrow
Red marrow	0.00419	0.537	52.1	0
Total endosteal cells	0.891	1.86	25.0	13.2

[a]Snyder et al. (1974).

Small Intestine (*SI*):

$$q_{SI\,cont} = \frac{\lambda_S q_{S\,cont}}{\lambda_{SI}^{ab} + \lambda_{SI} + \lambda^r} = 8.33\times10^{-3} \quad \text{TBq}, \qquad \lambda_{SI}^{ab} = \frac{0.95\lambda_{SI}}{1-0.95}$$

Upper Large Intestine (*ULI*):

$$q_{ULI\,cont} = \frac{\lambda_{SI} q_{SI\,cont}}{\lambda_{ULI} + \lambda^r} = 2.71\times10^{-2} \quad \text{TBq}$$

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Lower Large Intestine (*LLI*):

$$q_{LLI\,cont} = \frac{\lambda_{ULI}\, q_{ULI\,cont}}{\lambda_{LLI} + \lambda^r} = 5.00\times10^{-2} \quad \text{TBq}$$

The dose rate to the walls from activity in the contents is taken as one-half the dose rate to the contents = $\frac{1}{2}kEq_j/M_j$, in view of Eqs. (1) and (2) and the foregoing data. The estimates of dose (Sv TBq^{-1}) to the segments from the contents are stomach—6.6; small intestine—8.2; upper large intestine—4.8; and lower large intestine—14.5. A weakness of this model is the arbitrary nature of the 95% figure that is applied to absorption of tritium from the small intestine (100% would require an infinite rate coefficient): the complementary fraction determines the estimate of dose to the lower intestinal tract. Moreover, the model does not attempt to account for the loss of tritium by absorption of water from the large intestine. Note that in Table 1, two dose equivalent factors are given for "remainder" and for the effective dose commitment. The larger corresponds to the ingestion of tritium, and its larger value is the result of considering the dose to the walls of the GI tract due to tritium in the contents. The other factor is for other modes of intake—inhalation and absorption through the skin.

2.3 Dynamic Retention Functions

The dynamic compartment model represented by the box diagram of Fig. 2 can be expressed as a system of ordinary differential equations whose solution gives the activity level, $q_i(t)$ (TBq), in compartment i, i = 1,...,5. We have solved this system with an initial level of 1 TBq in body water and zero in all other compartments. The coefficients and decay-rate constants of the solutions are shown in Table 3.

Table 4 gives the time integrals, $\tilde{q}_i$ (TBq d), of the $q_i(t)$, where the time t ranges from 0 to 18,250 d (= 50 yr; for tritium, there is no practical difference in the results for longer times, such as 70 or 100 yr). Given $\tilde{q}_i$, we may apply Eqs. (1)–(3) to compute dose equivalent to compartment i from its time-varying burden of tritium. For 42 kg of body water, the result is

$$kE\tilde{q}_i/M = (1.38\times10^{7})(5.685\times10^{-3})(12.9)/4.2\times10^{4}$$

$$= 24 \text{ Sv TBq}^{-1}$$

The 9.5 kg reservoir of non-fat, non-mineral tissue solids is of particular interest. The estimates of $\tilde{q}_i$ for the two compartments that partition this tissue are mass-averaged:

$$(0.256\times93 + 1.44\times572)/(93 + 572) = 1.27 \text{ TBq d}$$

and the result is used to compute dose equivalent to the tissue:

$$kE\tilde{q}_i/M = (1.38\times10^{7})(5.685\times10^{-3})(1.27)/(9.5\times10^{3})$$

$$= 10.5 \text{ Sv TBq}^{-1}$$

As previously noted, we have not included an explicit representation for fat in the dynamic model. To estimate dose equivalent to the fat compartment from tritium incorporated in it, we assume rapid turnover so

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that the specific activity ratio $A_{fat}/A_w \approx 1$. From Table 4, we see that a sustained uptake of 1 TBq d^{-1} to body water results in a steady-state value of 12.9 TBq in body water, from which we calculate

$$A_w = (12.9 \text{ TBq})/[(4.2\times10^4 \text{ g } H_2O)/9]$$

$$= 2.76\times10^{-3} \text{ TBq (gH)}^{-1}$$

$$A_{fat} \approx A_w$$

Fat is 12.2% H. Therefore we compute

$$kEA_{fat}F^H_{fat} = (1.38\times10^7)(5.685\times10^{-3})(2.76\times10^{-3})(0.122)$$

$$= 26.4 \text{ Sv TBq}^{-1} \text{ to fat.}$$

Dose equivalent to body water, lean tissue solids, and fat computed above may be combined to estimate dose in soft tissue, given the mass fraction of each constituent. The thyroid, for example, is 75% water, 10% fat, and 15% lean tissue solids. We have

$$0.75\times24 + 0.15\times10.5 + 0.10\times26.4 = 22.2 \text{ Sv TBq}^{-1}.$$

Depending as it does on the assumption $A_{fat} \approx A_w$, this value possibly overestimates the contribution to the dose by tritium bound in fat molecules. The opposite extreme is $A_{fat} = 0$, and the estimate corresponding to this case is 19.6 Sv TBq^{-1}. Omitting both fat and the organic hydrogen compartments results in an estimate of 18 Sv TBq^{-1} due to tritium in body water. With the latter figure as a baseline value, the components associated with fat and other organically bound tritium contribute an increment of 23%. Bennett (1973) similarly estimated a 20% increase resulting from the addition of the organic hydrogen compartments in lean tissue.

We have already referred to recommended limits in ICRP Publication 30 (ICRP, 1979) that are based on the estimate 17.3 Sv TBq^{-1} to 63 kg of soft tissue (Eckerman et al., 1981). This value was derived from a 10-day half-time for biological removal, without consideration of differential uptake and retention of tritium by different tissue components; in particular, it fails to take into account the diminishing fractional removal rate that is evident in the data of Fig. 2 and represented by the model of Fig. 1 and Tables 3 and 4.

3. ENVIRONMENTAL DOSIMETRY OF TRITIUM

The dose conversion factors derived in the foregoing sections must be combined with concentrations or specific activities of tritium in man's various exposure media to yield estimates of annual dose equivalent for use in radiological assessments. Levels of contamination in the exposure media (air, water, soil moisture) are estimated by means of direct measurements or environmental modeling with assumed rates of release. An extended treatment of these techniques is beyond the scope of this report. We give only some basic indications of ways in which the esimates of dose equivalent rate to an individual may be obtained on the basis of an assumed steady state of contamination levels.

The model of hydrogen balance assumed for Reference Man (Table 2 and Fig. 5) provides a conversion of tritium specific activities in food and fluids and the subject's ambient air humidity to daily intake rates (TBq d^{-1}) for steady-state conditions. We have

$$I_{air} = (15 + 9)A_{air} \text{ TBq d}^{-1} \qquad (8)$$

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$$I_{ingestion} = 183A_{water} + 33A_{milk} + 120A_{food} \text{ TBq d}^{-1} \quad (9)$$

where the specific activities A_{air}, A_{water}, etc., are in units of TBq $(gH)^{-1}$ and the coefficients are gH d^{-1} derived from the respective sources. Note that water in food is included in the ccefficient 120 associated with A_{food} in Eq. (9). We point out that the coefficients 15 and 9 gH d^{-1} in Eq. (8) (inhalation and absorption through the skin, respectively) are sensitive to the ambient absolute humidity, and the values given correspond to 6 g H_2O m^{-3}.

The specific activities A_{water}, A_{milk}, and A_{food} depend in turn on the environmental sources of the water, milk, and food ingested by the reference individual. In this connection, we illustrate with assumptions similar to those taken in NCRP Report No. 62 (NCRP, 1979) and assume that 20% of drinking water is taken from deep wells (ground water). The remaining 80% is assumed to come from freshwater lakes and streams. The specific activities of milk and the water content and organically bound hydrogen in all other foods (A_{milk} and A_{food}) are taken as the arithmetic mean of A_{air} and the specific activity of surface soil water. In exception to the foregoing rule, however, we assume that 2 g of the daily dietary hydrogen intake derives from seafoods. When the appropriate substitutions are made in Eq. (9), the expression for $I_{ingestion}$ becomes

$$I_{ingestion} = 36.6A_{gw} + 146.4A_{fw} + 75.5(A_{air} + A_{sw}) + 2A_{os} \text{ TBq d}^{-1} \quad (10)$$

where the meanings of the subscripts are gw = ground water, fw = freshwater lakes and streams, sw = surface soil water, and os = ocean surface water. Equation (10) is applied to measured or model-predicted tritium specific activities in these environmental exposure media.

Inhalation and absorption through the skin. To estimate the dose due to intake by inhalation, we assume that for a steady release rate of HTO to the atmosphere, the time-averaged air concentration at ground level at a given location is χ TBq m^{-3}. Reference Man (ICRP, 1975) breathes 23 m^3 d^{-1}, and we assume that all inhaled HTO is absorbed. Consequently, the effective dose equivalent rate, due to inhalation, is

$$\frac{D_{h,\text{HTO}}}{\chi} = (22 \text{ Sv TBq}^{-1})(23 \text{ m}^3 \text{ d}^{-1})(365 \text{ d yr}^{-1}) \quad (11)$$

$$= 1.85\times10^5 \text{ Sv yr}^{-1} \text{ per TBq m}^{-3}$$

The factor 22 Sv TBq^{-1} is from Table 1.

For absorption of HTO through the skin, recall that our intake model assumes an inward flux of 9.6×10^{-3} liters of gas per minute (Osborne, 1966). The resulting effective dose equivalent rate at the location in question is

$$\frac{D_{s,\text{HTO}}}{\chi} = (22 \text{ Sv TBq}^{-1})(9.6\times10^{-3} \text{ m}^3 \text{ min}^{-1})(5.26\times10^5 \text{ min yr}^{-1}) \quad (12)$$

$$= 1.11\times10^5 \text{ Sv yr}^{-1} \text{ per TBq m}^{-3}$$

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A portion of the release is often HT gas, which behaves quite differently from HTO in the body and in environmental systems. Bush (1972) reports that HT is absorbed only slightly through the lungs, and not at all through the skin. When human subjects are exposed to equal concentrations of HTO and HT in air, the uptake of HT is 1% of that of HTO. Bush (1972) estimates the dose from HT in the atmosphere as 0.01% of the dose that would result from an equal concentration of HTO. Essentially the same conclusions were reported earlier by DeLong et al. (1953). Pinson and Langham (1957) compared the rates of appearance of tritium in the body fluids of man during inhalation of HT and HTO and found them to be equal when the specific activity of HT in air was approximately 15,000 times that of HTO. In Publication 2 of the ICRP (1959), however, the maximum permissible concentrations in air for the 168-hour week differ only by a factor of 200 for the two chemical species, and some investigators (e.g., Jacobs et al., 1979) have based environmental assessments on this figure. For immersion in HT gas at a concentration of χ TBq m^{-3}, we adopt the factor 10^{-4}. Lung tissue is irradiated directly by the HT in inspired air. In ICRP Publication 30 (1979), recommendations for limiting the HT concentration are based on an estimate that is equivalent to 87 Sv yr^{-1} to the lung per TBq m^{-3} concentration of HT in the air. Including this component of dose, we estimate the effective dose equivalent rate due to inhalation of HT as

$$\frac{D_{HT,h}}{\chi} = (10^{-4})(1.85\times10^{5} + 1.11\times10^{5}) + (0.12)(87) \tag{13}$$

$$= 40 \text{ Sv yr}^{-1} \text{ per TBq m}^{-3}$$

where 0.12 is the risk coefficient w_{lung} from Table 1.

Ingestion. The annual effective dose equivalent rate is

$$D_g = (23 \text{ Sv TBq}^{-1})(365 \text{ d yr}^{-1})I_{ingestion} \text{ Sv yr}^{-1} \tag{14}$$

where 23 Sv TBq^{-1} is the effective dose equivalent from Table 6 and $I_{ingestion}$ [Eqs. (8) and (9)] is the intake rate of tritium (TBq d^{-1}) as HTO and organically bound dietary components, between which we make no distinction in uptake or metabolism.

4. SUMMARY AND DISCUSSION

We have presented estimates of committed dose equivalent to various organs of ICRP's Reference Man per unit intake of tritium as HTO or organically bound in dietary materials (Table 1). The basis of these numbers is a 5-compartment model of hydrogen uptake, removal, and exchange among major reservoirs in the body (Fig. 2). The model is calibrated to account for slow-decay terms in the retention function as estimated by Bennett (1973) on the basis of a review of data from several long-term studies of exposed subjects. The dose conversion factors arrived at by this method are combined with weights proportional to risks of stochastic fatal health effects to give the effective dose equivalent, as defined in ICRP Publication 26 (1977). We have also given a brief indication of the manner in which the dose conversion factors may be combined with results of environmental monitoring or modeling to produce estimates of dose equivalent to exposed individuals following releases of tritium as HTO or HT.

The effective dose equivalent estimates shown in Table 1 (22 and 23 Sv TBq^{-1}) are, respectively, 27 and 33% larger than the value assumed in arriving at the recommendations for occupational exposure limitation in ICRP Publication 30 (1979).

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We list several limitations of the models and methods discussed in this report:

- Dynamics of the metabolism of fat in human beings are poorly quantified. Our estimates are based on the assumption of extremely rapid turnover and therefore could be high, but only by about 12% at most for tissue that is 10% fat (Sect. 2.3).
- Our model of the GI tract does not attempt to account for loss of tritium by absorption of water from the large intestine. It is assumed (somewhat arbitrarily) that 95% of the ingested tritium is absorbed from the small intestine, and that the remaining 5% passes through the lower tract without further absorption. This assumption is almost certainly pessimistic, but the extent of its effect on the effective dose commitment is relatively minor (Table 1).
- Isotopic fractionation of ^{3}H with respect to ^{1}H in physical and chemical transformations has been neglected in the calculations reported here. Substantial discrimination against the heavier ^{3}H has been reported in experiments with plants (McFarlane, 1976; Garland and Ameen, 1979), resulting in $^{3}H/^{1}H$ ratios in plant tissue that were significantly lower than those maintained in the controlled environment. Results vary from one species of grain to another, however, and in any event could not be extrapolated to man. Any attempt to derive estimates for man by direct application of the fundamental thermodynamic relations to diffusion and reaction kinetics would require a bio-physico-chemical model of man that greatly exceeds the one discussed here.
- Dose estimates presented in this report do not account for dependence of anatomical and physiological parameters of the models on age.

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REFERENCES

Bell, G. H., Davidson, J. N., and Scarborough, H. 1968. *Textbook of Physiology and Biochemistry.* (Edinburgh and London: E. & S. Livingstone Ltd).

Bennett, B. G. 1973. "Environmental Tritium and the Dose to Man." In: *Proceedings of the Third International Congress of the International Radiation Protection Association, Washington, D. C., Sept. 9-14, 1973*:1047-1053. Rep. No. CONF-730907-P2 (Springfield, Va.: National Technical Information Service).

Bernard, S. R. 1968 "A Model for Estimation of Mean Activity Present in Segments of the Gastrointestinal Tract at any Time post Ingestion." In: *Health Physics Division Annual Progress Report for Period Ending July 31, 1968*:283-287. Oak Ridge National Laboratory Rep. ORNL-4316.

Bush, W. R. 1972. *Assessing and Controlling the Hazard from Tritiated Water.* Atomic Energy Control Board (Canada) Rep. AECL-4150.

Butler, H. L., and LeRoy, J. H. 1965. "Observation of Biological Half-Life of Tritium." *Health Phys.* **11**(4):283-285.

DeLong, C. W., Thompson, A. C., and Kornberg, H. C. 1953. "Percutaneous Absorption of Tritium and Tritium Oxide." In: *Biology Research—Annual Report 1952*:52-58. Hanford Atomic Products Operation Rep. HW-28636.

Dunning, D. E., Jr., and Killough, G. G. 1981. "A Comparison of Effective Dose Equivalents from Three Major Internal Dose Compilations." *Rad. Protection Dosimetry* **1**(1):3-9.

Eckerman, K. F., Ford, M. R., and Watson, S. B. 1981. *Internal Dosimetry Data and Methods of ICRP—Part 2, Vol. 1: Committed Dose Equivalent and Secondary Limits.* Oak Ridge National Laboratory Rep. NUREG/CR-1962 Volume 1, ORNL/NUREG/TM-433/VI.

Eve, I. S. 1966. "A Review of the Physiology of the Gastrointestinal Tract in Relation to Radiation Doses from Radioactive Materials." *Health Phys.* **12**(2):131-161.

Fenn, W. O., and Rahn, H. 1964. *Handbook of Physiology. Section 3: Respiration, Volume I* (Washington, D. C.: American Physiological Society).

Garland, J. A., and Ameen, M. 1979. "Incorporation of Tritium in Grain Plants." *Health Phys.* **36**:35-38.

International Commission on Radiological Protection (ICRP). 1959. ICRP Publication 2 (Oxford: Pergamon Press).

International Commission on Radiological Protection (ICRP). 1966. ICRP Publication 9 (Oxford: Pergamon Press).

International Commission on Radiological Protection (ICRP). 1969. Progress report from ICRP. *Health Phys.* **17**(2):389-390.

International Commission on Radiological Protection (ICRP). 1975. *Report of the Task Group on Reference Man,* ICRP Publication 23 (Oxford: Pergamon Press).

International Commission on Radiological Protection (ICRP). 1977. ICRP Publication 26. *Ann. ICRP* **1**(3):2-47.

International Commission on Radiological Protection (ICRP). 1979. *Limits for Intake of Radionuclides by Workers,* ICRP Publication 30, Supplement to Part 1. *Ann. ICRP* **3**(1-4):1-555.

Jacobs, D. G., Easterly, C. E., and Phillips, J. E. 1979. "The Influence of the Rate of Conversion of HT to HTO on Projected Radiation Doses from Release of Molecular Tritium." In: *Behaviour of Tritium in the Environment*: 373-383. (Vienna: IAEA).

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Killough, G. G., Dunning, D. E., Jr., Bernard, S. R., and Pleasant, J. C. 1978. *Estimates of Internal Dose Equivalent to 22 Target Organs for Radionuclides Occurring in Routine Releases from Nuclear Fuel-Cycle Facilities, Vol. 1.* Oak Ridge National Laboratory Rep. NUREG/CR-0150, ORNL/NUREG/TM-190.

Kocher, D. C. 1981. *Radioactive Decay Tables—A Handbook of Decay Data for Applications to Radiation Dosimetry and Radiological Assessments.* Technical Information Center, U. S. Department of Energy DOE/TIC-11026.

McFarlane, J. C. 1976. "Tritium Fractionation in Plants." *Env. and Exp. Botany* **16**:9-14.

Moghissi, A. A., and Carter, M. W. 1971. "Long-Term Evaluation of the Biological Half-Life of Tritium." *Health Phys.* **21**(1):57-60.

Moghissi, A. A., Carter, M. W., and Bretthauer, E. W. 1972. "Further Studies on the Long-Term Evaluation of the Biological Half-Life of Tritium." *Health Phys.* **23**(6):805-806.

Moghissi, A. A., Patzer, R. G., and Carter, M. W. 1973. "Biokinetics of Environmental Tritium." In: *Tritium* (Moghissi, A. A., and Carter, M. W., eds.):314-321. Rep. No. CONF-710809 (Springfield, Va.: National Technical Information Service).

National Council on Radiation Protection and Measurements (NCRP). 1979. *Tritium in the Environment*, NCRP Report No. 62. (NCRP Publications, P. O. Box 30175, Washington, D. C. 20014).

Osborne, R. V. 1966. "Absorption of Tritiated Water Vapour by People." *Health Phys.* **12**(11):1527-1537.

Pinson, E. A., and Langham, W. H. 1957. "Physiology and Toxicology of Tritium in Man." *J. Appl. Physiol.* **10**:108. Reprinted: *Health Phys.* **38**:1087-1110 (1980).

Sanders, S. M., and Reinig, W. C. 1968. "Assessment of Tritium in Man." In: *Proceedings of the Symposium on Diagnosis and Treatment of Deposited Radionuclides*:543. Rep. No. CONF-670521 (Springfield, Va.: National Technical Information Service).

Snyder, W. S., Fish, B. R., Bernard, S. R., Ford, M. R., and Muir, J. R. 1968. "Urinary Excretion of Tritium Following Exposure of Man to HTO—A Two-Exponential Model." *Phys. Med. Biol.* **13**:547-559.

Snyder, W. S., Ford, M. R., Warner, G. G., and Watson, S. B. 1974. *A Tabulation of Dose Equivalent per Microcurie-Day for Source and Target Organs of an Adult for Various Radionuclides.* Oak Ridge National Laboratory Rep. ORNL-5000.

Snyder, W. S., Ford, M. R., Warner, G. G., and Watson, S. B. 1975. *"S," Absorbed Dose per Unit Cumulated Activity for Selected Radionuclides and Organs.* NM/MIRD Pamphlet No. 11, Medical Internal Radiation Dose Committee, Society of Nuclear Medicine.

Till, J. E., Meyer, H. R., Etnier, E. L., Bomar, E. S., Gentry, R. D., Killough, G. G., Rohwer, P. S., Tennery, V. J., and Travis, C. C. 1980. *Tritium—An Analysis of Key Environmental and Dosimetric Questions.* Oak Ridge National Laboratory Rep. ORNL/TM-6990.

Wylie, K. F., Bigler, W. A., and Grove, G. R. 1963. "Biological Half-Life of Tritium." *Health Phys.* **9**(9):911-914.

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