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Internal Dosimetry of Tritiated Hydrogen Gas

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CFFTP will sponsor research, development and studies to extend existing experience and capability gained in handling tritium as part of the CANDU fission program. It is planned that this work will be in full collaboration and serve the needs of international fusion programs.

INTERNAL DOSIMETRY OF

TRITIATED HYDROGEN GAS

Report No. CFFTP-G-84034

Cross Reference Report AECL 8651

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PREFACE

This document is a report on experiments to quantify the doses that may occur from the tritium gas that is converted *in vivo* to tritiated water following the exposure to tritium hydrogen gas contaminated air.

This report also includes theoretical evaluation of the radiological hazards from the uptake through skin of tritium from tritiated hydrogen adsorbed on surfaces.

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EXECUTIVE SUMMARY

This is the final report to the Canadian Fusion Fuel Technology Project (CFFTP) and reports results of work jointly funded by AECL and CFFTP under purchase order 13-30276-11. This work was done in two parts.

Internal Dosimetry of Tritiated Hydrogen Gas

This first part of this project was a measurement of the fraction of tritiated hydrogen gas (HT) inhaled by volunteers that is converted to water (HTO) inside the body. Since HTO is retained in the body for a much longer time than HT dissolved in body fluids, a good estimate of this fraction is required to set radiation protection standards for inhaled HT gas.

There was some question of whether tritiated organic compounds could be formed directly from HT gas, and rats were exposed to HT gas, and injected with HTO to see if there was a significant difference between the amount of organic fractions produced. No evidence of the direct formation of organic compounds from HT was found, leading to the conclusion that if organic compounds were formed, their contribution to dose would not be significant compared to the contribution from HTO.

Six volunteers were exposed to HT gas and the fraction oxidized in vivo estimated by measuring the concentration of HTO in urine. In

particular, the ratio of urine tritium concentration to the integrated exposure to HT gas (Bq L^{-1} per Bq m^{-3} min) was measured, as this ratio gives an estimate of the importance of the HTO contributions to dose. This ratio was measured to be $(2.4 \pm 0.4) \times 10^{-8}$ Bq L^{-1} per Bq m^{-3} min, indicating that the effective dose equivalent from HTO, produced in vivo from HT is approximatly equal to that from HT itself.

Dosimetric Models of Tritium from Skin Absorption Following Contact with T_2 Contaminated Surfaces

The second part of this project was a theoretical study of the importance of uptakes of tritium resulting from contact between surfaces that had been exposed to HT gas and intact skin, and is based on reported studies in rats and humans. Using models that gave results consistent with the reported results, it was found that the dose to skin from tritium bound in skin could be an important consideration in these situations, and that the organic tritium component found in urine adds considerable uncertainty to doses estimated from tritium concentrations in urine. The result of this work was a paper accepted for publication in Health Physics. Experiments to improve our understanding of skin uptake from HT contaminated surfaces are being considered at CRNL.

REPORT TO CFFTP ON WORK PERFORMED
UNDER PURCHASE ORDER 13-30276-11

INTERNAL DOSIMETRY OF TRITIATED HYDROGEN GAS

by

B.F. PETERMAN, J.R. JOHNSON, R.G.C. McELROY AND D.W. DUNFORD

1. INTRODUCTION

The main radiological hazard from tritium in the current Canadian nuclear fuel cycle is associated with releases of tritiated water from heavy water reactors to the workplace or to the environment. The hazard from intakes of tritiated water by inhalation, ingestion or skin absorption have been the subject of many studies in the past, both at CRNL (for example, 1, 2, 3, 4, 5) and elsewhere and is understood well enough for radiation protection purposes. The hazard of ingested tritium that has been incorporated into biological molecules in food is less well understood but it can be readily shown (6) that this hazard is not greatly different than that from the tritiated water in the food provided the biologically incorporated tritium resulted from releases of tritiated water.

The planned tritium extraction plants at CRNL and at Ontario Hydro sites (7, 8), and fusion reactor facilities with planned tritium use, raise the possibility of accidental releases of large amounts of HT (tritiated H_2 gas) to the workplace or the environment. The metabolism of tritium following intakes of HT gas by mammals is not well understood, and since this information is required to set safety standards and procedures for facilities handling large amounts of HT, it was decided to carry out a short term experiment (jointly funded by AECL and CFFTP) specifically designed to supply the necessary information.

With reference to Figure 1, HT entering the lungs will result in a known radiation dose to the lungs from the tritium in the air in the lungs, a known (and small) radiation dose to the whole body from HT dissolved in body fluids, an unknown radiation dose from any organic compounds that may be formed, and an unknown radiation dose from any HT that is oxidized to HTO in vivo. It should be noted that once HTO is formed in body fluids, it will have the same radiation hazard associated with it as an equal concentration of tritium in body fluids arising directly from an HTO exposure.

The questions that need to be answered are therefore:

1. Are any tritiated organic compounds formed directly from HT? There are of course tritiated organic compounds formed from any tritium present as HTO, but the significance of these in terms of radiation hazards can be readily estimated.

2. What fraction of the inhaled HT is oxidized to HTO? Reasonable estimates of this fraction range from 0 to 5×10^{-4}. This latter value is based on a conservative interpretation of the only reported measurements, done at Los Alamos previous to 1957 (9). This range results in a factor of about 10 in the dose resulting from inhaled HT activity. (Or compared to inhaled/absorbed HTO, HT is from 1.7×10^{4} to 1.7×10^{3} times less hazardous.)

The work carried out to provide answers to these questions was:

- constructing and calibrating appropriate tritium-in-air monitors,
- exposing rats to known HT concentrations, injecting controls with HTO and looking for organically bound tritium in tissue and urine,
- following excretion rates in rats to compare retention of tritium following HT and HTO exposures,
- when no organic bound tritium was found in rats, exposing a group of volunteers to find the average fraction of inhaled HT that is converted to HTO, and its variability.

Modes of intake other than inhalation may be important in facilities handling large quantities of HT gas. For example, experiments with rats (10) and humans (11) have shown that tritium will be fixed in skin and transmitted to body fluids when intact skin is brought into contact with surfaces that have been exposed to tritiated hydrogen gas (HT or T_2). Those reports (10, 11) indicated that the hazard associated with this route of tritium uptake was probably small in laboratories handling small quantities of T_2 but could be significant when large quantities are being processed, particularly during repair or maintenance of process equipment. Therefore, it was decided to conduct a preliminary theoretical investigation of the significance of T_2 contaminated surfaces in terms of dose to skin and to body tissues. The results of this investigation are also discussed in this report.

2. EXPERIMENTAL

2.1 HT Gas Supply

Tritiated hydrogen gas (93% HT) was prepared in a gas cylinder at the Tritium Handling Facility at CRNL by a one thousand to one dilution with N_2. The calculated specific activity of this gas at STP was 4.8 x 10^{14} Bq m^{-3}. This gas was further diluted with air from a compressed air cylinder prepared by the CRNL Fire Department to achieve the desired HT concentration, measured as described below.

2.2 HT Exposure Apparatus

Figure 2 shows the HT inhalation exposure system which is located in a fume hood in Building 464. The HT gas from the cylinder is passed through a (25 cm long and 1.2 cm in diameter) zeolite column, two gas washing bottles and a silica gel column (30 cm long and 1.6 cm in diameter) to remove traces of HTO. The HT gas is then mixed and diluted with air and fed into a rubber breathing bag. The bag used in the 1 minute exposures had a 13 L capacity and that for the 10 minute exposures was 100 L. When the concentrations were being adjusted, and during rat exposures, the bag was bypassed. From the rubber breathing bag to the exhaust all the connections were made by plastic spiral tubing of 1 1/8" inside diameter attached with molded rubber ends. The concentration of HT was measured at two points - before and after the point of inhalation or rat exposure - by ionization chambers of coaxial design with an effective volume of 0.8 L. HT gas was supplied to the subject's mouth through an inspiratory/expiratory valve. A nose clip was used to close the nostrils and allow only mouth breathing. Before the inhalation exposure took place the rubber breathing bag was

filled with HT/air mixture and during the exposure the HT/air mixture was fed into the bag at 5 L/min^{-1}. The concentrations of HT during the exposure (before, during and after inhalation) were recorded by chart recorders.

2.3 Animal Exposures

When rats were exposed to HT, a system similar to the one in Figure 2 was used except that the rubber breathing bag was bypassed and the inspiratory/expiratory valve was replaced by a lucite exposure chamber where the rat was placed during the exposure. Rats (Sprague-Dawley, albino CD strain, 8-10 weeks of age, purchased from Charles River, St. Constant, Quebec, Canada) were kept in metabolic cages bought from Canadian Laboratory Supply, Ottawa, Ontario. The metabolic cages were kept, and the animals were cared for, by the Animal Facility at CRNL.

Seven rats were exposed to HT at a concentration of about 3 x 10^{11} Bq m^{-3}, for times ranging from 5 to 10 minutes. Eight rats were injected with HTO as controls. Two rats exposed to HT (HT rats) and two rats injected with HTO (HTO rats) were sacrificed at the end of day 1 and day 7, and tissues analysed for bound tritium after extracting unbound tritium from homogenized muscle and liver samples with $HClO_4$. The urine of two HT rats and two HTO rats at 4 hours post exposure were analysed for organically bound tritium using the distillation technique described by Eakins et al. (11). This technique was used to show that the organic compounds found in urine by Eakins et al. (11) following exposure of intact skin to HT contaminated surfaces did not occur for inhalation exposures. This technique was tested at CRNL using tritiated thymidine as a tritiated organic compound (see Appendix A). Concentrations of tritium in samples were measured with a liquid scintillation counter using identical samples spiked with an NBS traceable standard for calibration.

2.4 Human Exposures

The AECL Clinical Radioisotope C[illegible]mittee reviewed the purpose, aims and benefits of the experiment, especially [illegible]he necessity of using human subjects and the magnitude of the inhalation exposures. Having established that it fully satisfied their Ethical Guidelines, which stress the need to ensure that all volunteers fully understand the possible risks involved, the Committee approved the experiment.

Six volunteers were exposed to HT gas for a total of 16 times (11 one minute exposures, and 5 ten minute exposures). Urine was collected before each exposure, at approximately 1, 2 and 4 hours after exposures, and on the following morning. Volunteers were asked to void their bladder each time a sample was given. Tritium concentrations in urine were measured by liquid scintillation counting, using samples spiked with an NBS traceable standard to calibrate each measurement.

3. RESULTS

3.1 Rat Exposures

3.1.1 Measurement for Organic Tritium in Urine

Table 1 gives the results obtained for the ratio of the tritium concentration in the condensate from the urine of HT and HTO rats to that in urine. The urine samples used were those collected during the day of the exposure and over the first night following the exposure. No evidence for an organic component in urine was observed.

3.1.2 Amount Converted to HTO

Table 2 gives the integrated HT exposure and the resulting HTO measured in urine for the seven rats exposed to HT. Since there is a possibility that some of the HT gas will be oxidized by the exposure system itself, an equal amount of HT gas as used for exposures was passed through the system and the trace of HTO generated in the system was collected by water in a gas washing bottle (bubbler) connected to the exhaust. The concentrations of HTO measured this way were negligible (less than 5%) compared to the amount of HTO found in urine samples of the rats (see Appendix B).

The ratio of urinary concentration to the integrated air concentration predicted from the Pinson and Langham experiment (9) was

$$8.3 \times 10^{-7} \text{ Bq L}^{-1} \text{ per Bq min m}^{-3}$$

The mean value found in this experiment was

$$(3.6 \pm 0.2) \times 10^{-7} \text{ Bq L}^{-1} \text{ per Bq min m}^{-3}$$

for exposures to about 3×10^{11} Bq m^{-3} for times varying between 5 and 10 minutes. The quoted uncertainty is one standard error of the mean of the seven exposures.

The agreement between the predicted and obtained value is good considering that the prediction (9) was based on a 30 hour exposure to rats maintained in a metabolic cage (used to collect urine and feces) where the likelihood of *in vitro* oxidation in excreted and other materials existed. As noted above, a minute amount of *in vitro* oxidization was measured in this experiment, and this was not enough to affect the result significantly.

3.1.3 Bound Tritium in Tissue

Table 3 gives the results of measurements of bound and unbound tritium in muscle and liver on day 1 and day 7 following the exposure. Owing to the difficulty in this type of analysis, there is considerable uncertainty in the measurement of the bound fractions; however, even with these uncertainties, it is clear that even if the fraction of tritium bound in tissue is different for HTO and HT exposures, the difference will not result in significantly different doses because the total contribution to dose from the bound tritium is small.

3.1.4 Retention of Tritium

Table 4 gives the results of fitting the urinary tritium concentration to a single exponential to obtain a half-life of turnover of HTO in the HT rats as compared to the HTO rats. It can be seen that there was considerable variability in the retention between rats. In particular it was noted that rats maintained in metabolic cages during the day, and standard cages at night, had a shorter retention of tritium than those maintained in standard cages during the day and metabolic cages at night, presumably because of physical activity levels. However, there was no observable difference between HT and HTO rats.

3.2 Human Exposures

Table 5 shows the results (HTO activity in urine, HT exposure and HTO/HT ratios) of 11 one minute exposures and 5 ten minute exposures carried out on a group of six volunteers. For ten minute exposures the concentration was approximately one tenth of that prepared for one minute exposures. Figures 3 and 4 show chart-recorded tracings during a one minute and a ten minute exposure. The first HT monitor monitors the concentration of HT in inhaled air and the second HT monitor measures the concentration of HT in exhaled air. As the subject starts inhaling, the HT gas entering his lungs is diluted which is indicated by the drop in the HT concentration in exhaled air. Figure 4 shows that it takes about 7 to 8 minutes to completely saturate the subject's lungs and blood. As discussed before, the amount of HTO generated in the system was estimated by passing the same volume of HT/air mixture as inhaled by the volunteer through the exposure system and trapping HTO by a gas washing bottle connected at the exhaust. The concentration of HTO in the HT/air mixture generated in the system (converted for the dilution in the human body) was then subtracted from the concentration of HTO in subject's urine, as was the background HTO activity measured by the control sample taken before the exposure. The concentration generated by the system (Appendix B) contributed at most 10 to 20% to the total HTO concentration found in urine, the exact amount varying with the individual and exposure. There was no significant difference in the HT/HTO ratio observed between the one minute and ten minute exposures. The average value of the HT conversion for one minute exposures was found to be $(2.5 \pm 0.6) \times 10^{-8}$ Bq L^{-1} per Bq m^{-3} min which agrees well with $(2.1 \pm 0.2) \times 10^{-8}$ Bq L^{-1} per Bq m^{-3} min obtained for ten minute exposures. The overall mean value was $(2.4 \pm 0.4) \times 10^{-8}$ Bq L^{-1} per Bq m^{-3} min.

This value is in good agreement with the only previously reported value (9) of 1.4×10^{-8} Bq L^{-1} per Bq m^{-3} min.

4. DOSE ESTIMATES

The dose due to the inhaled HT gas consists of two major contributions: the dose due to the presence of HT in the air in the lungs and dose due to HTO in soft tissues. The doses to the lungs and soft tissues were estimated for all six volunteers (Table 6). The dose to the lungs was calculated by multiplying the HT exposure by 9.9×10^{-15} Sv/Bq m^{-3} h (12). In this calculation the volume and mass for the lungs of 3×10^{-3} m^3 and 1000 g, respectively, were assumed. The dose to the soft tissue was calculated by multiplying the urine concentrations in µCi L^{-1} by a factor of 32.5 µSv per µCiL^{-1} (3). In this calculation a 10 day half-life was assumed. The average effective dose equivalent from HTO of 12 µSv is essentially the same as the average effective dose equivalent from air in the lung of 11.4 µSv. Thus in calculating the effective committed dose due to inhalation of HT, the dose to the soft tissue should not be neglected.

5. DISCUSSION ON HT INHALATION EXPOSURES

5.1 Product and Site of HT Oxidation

The experiments on rats did not indicate that there were products of HT metabolism other than HTO. Thus, we assume that the only product of HT oxidation in mammals is HTO. Since no mammalian cell can catalyze such a reaction it is of interest to speculate where the oxidation takes place. In the early 1950's Smith et al. (13) assayed the homogenates of various organs of a rat for the rate of tritium gas conversion. The results indicated that the large intestine contains most (about 90%) of the HT oxidation activity. Further work (14) indicated that the presence of various bacteria in the large intestine were responsible for the catalysis of HT to HTO. (The reaction is actually catalyzed by the enzyme hydrogenase which uses HT as a substrate.)

There is no good evidence as to where the oxidation of HT occurs in humans. It is known that some strains of E. coli and Ps. aeroginosa containing enzyme hydrogenase are present in the human gut, and the G.I. tract could represent a potential site of HT oxidation. Thus, the gut flora of a human could strongly influence the rate of oxidation. The bacterial gut flora in turn is influenced by diet. Table 5 indicates that the range of observed fractions of HT converted to HTO was about a factor of 10. Although the average values of the HT conversion should be used for the predicted values, one should bear in mind that the deviation from the mean values can be large.

5.2 Implications for Radiation Protection and Dosimetry

These experiments indicate that HTO will reach equilibrium in body fluids within 3 to 4 hours after exposure to HT. Thus, if a subject is suspected to have been exposed to HT atmosphere, his/her urine should be monitored for HTO activity 3 or more hours after the exposure to insure equilibrium has been reached, as is done for HTO exposures.

One can estimate the upper limit of the lung dose equivalent (H_L) due to HT in the air in the lungs (15,16) from the measurement of HTO in urine. For short exposures (compared to the effective half-life of HTO of about 10 days, i.e. acute exposures), the lung dose equivalent and the concentration of HTO in urine (measured 3 to 8 hours after the exposure) are linearly related; that is,

$$H_L = k\ C_{HTO}$$

where k is a proportionality constant in Sv/Bq L^{-1} and C_{HTO} is the concentration of HTO in urine (Bq L^{-1})(measured 3 to 8 hours after the exposure). The value for k estimated by this work was found to be $(0.8 \pm 0.33) \times 10^{-8}$ Sv/(Bq L^{-1}) which is slightly lower than the value reported before of 1.2×10^{-8} Sv/(Bq L^{-1}) (15,16). The quoted uncertainty is that arising from the sample standard deviation. It must be emphasized that this is the upper limit on the dose equivalent to the lungs due to inhalation of HT since a large portion of HTO in urine could come from inhaled HTO which is completely absorbed by the body fluids upon inhalation.

A monitoring program for dosimetry purposes in a HT handling facility therefore must have two considerations. The first of these is the monitoring of urine for HTO concentration. This concentration can be used to estimate the effective (whole body) dose from HTO (3,16) and to estimate upper limits on lung dose. This simple program will be adequate provided that urine concentrations

are low enough that the total effective dose equivalent is small compared to the annual dose limit. For example, if the annual average urine concentration is 10^4 Bq L^{-1} (i.e. assuming chronic exposure conditions), the annual dose equivalent to the soft tissue from HTO (which could result from HT and/or HTO exposures) would be 0.2 mSv (3). If we assume that the tritium in urine resulted from the HT exposure only (i.e. no HTO was inhaled), the effective dose equivalent is about 0.4 mSv, made up in approximately equal parts from the dose equivalent to the lung (with a weighting factor of 0.12) and the effective dose equivalent from the oxidized HT. This effective dose is small compared to the annual dose limit of 50 mSv. However, there is a potential for tritium urine concentration to be much higher than 10^4 Bq L^{-1} and this leads to a second consideration. If high urine concentration occurs, there will be a need to assign doses to individuals (16), rather than upper limits on doses. When this occurs, the assignment of a lung dose using the assumption that all of the tritium in urine resulted from HT exposures will be overly conservative, as it is likely that a large portion of it results from exposures to HTO. It would therefore be useful to have a measure of the average ratio of HT to HTO in workplace air. This ratio could be applied to the urine monitoring results to replace the conservation in the assigned lung dose.

6. DOSIMETRIC MODELS OF TRITIUM FROM SKIN ABSORPTION FOLLOWING CONTACT WITH T_2 CONTAMINATED SURFACES

The urinary excretion rate given in Fig. 5 represents our summary of the results reported for volunteers by Eakins et al. (11) following skin contact with T_2 contaminated surfaces and is meant to represent the excretion of tritium for Reference Man (17). The organically bound tritium (OBT) portion is characterized by a rapid increase ($t_{1/2} \sim 0.2$ d), a rapid decrease ($t_{1/2} \sim 0.2$ d) and a slower decrease ($t_{1/2} \sim 1.6$ d). The tritiated water (HTO) portion is characterized by a slower than normal (for HTO exposures) (11) increase, and a slower than normal ($\sim$14 d compared to 10 d) (11) decrease. The models shown in Fig. 6 were developed to describe these observations in terms of the amount of tritium retained in skin, and the amount and rate of OBT and HTO being transferred to body fluids and hence tissues for the purposes of calculating doses. It should be noted that the models are not unique and other models could be developed to describe the observed results.

Model 1 was developed to give the maximum retention in skin consistent with the observed results (Fig. 5). Compartment 1 represents direct oxidization of HT in/on skin (or on the surface or surface/skin interface). The HTO formed is transferred rapidly to body fluids where it is retained with a half-life of 10 d (18). Organically bound tritium formed from incorporation of this tritiated water (3, 18) has been ignored in the model as it will not affect the urinary excretion appreciably over the time post exposure being considered here. Compartment 2 represents OBT in skin

where the surface/skin contact was made. In this model it is assumed that this compartment is the cause of the decrease in urinary concentration of HTO to be slower than expected (11). This assumption is supported by the observation in rats (10) that tritium is retained in skin for a long time at the point of contact, and that the decrease in tritium in skin roughly corresponded to the increase of tritium in body fluids. Compartments 3 and 4 are OBT compartments that result in the tritium being transferred to body fluids and subsequently excreted, giving rise to the observed two component decrease in urine OBT concentration. Compartments 5 and 6 represent HTO and OBT in body fluids, respectively. Only a fraction (0.467) of the HTO excreted is assumed to be in urine (17), the rest being excreted in breath, sweat, etc. In contrast, all OBT is assumed to be excreted in urine, and this assumption would lead to an underestimated dose from this compartment if other routes of excretion existed. It should also be noted that there could easily be a large quantity of OBT in another chemical form that is not excreted, or excreted very slowly, and hence was not seen in the experiment by Eakins et al. (11).

Model 2 was developed to give the minimum retention in skin consistent with the observed results (Fig. 5). Compartment 1 is identical to that in Model 1. Compartment 4 is a short retention skin compartment from which OBT is transferred to body fluids for subsequent redistribution to tissue storage compartments (compartments 2 and 3) or excreted. Compartment 2 is responsible for the HTO urine concentration decrease being slower than expected for an HTO exposure, and compartment 3 is necessary to give the observed two component decrease in OBT urine concentration (11). Compartments 5 and 6 are as described for Model 1.

Differential equations for the models in Fig. 6 were written and solved numerically with the differential equation solution package FORSIM (19) for unit uptake in skin at time t = 0. The rate constants (λ_i) and initial amounts in each compartment were varied until good agreement between the calculated excretion rates and those in Fig. 5 were obtained. In order to achieve this agreement, the values of λ_1 and λ_5 were fixed at 24 d^{-1} and 0.0693 d^{-1}, respectively. The value of λ_1 has little effect on retention or dose provided it is large, and the value of λ_5 is that used for Reference Man (18). Then for Model 1, λ_2 was varied until the HTO excretion agreed with Fig. 5, λ_4 and λ_6 adjusted to achieve agreement with the short-term OBT component of Fig. 1 and λ_3 adjusted to achieve agreement with the long-term OBT component. Once reasonable agreement with the individual components was obtained the relative sizes of the components were adjusted by varying the initial amounts in each compartment and the rates readjusted to improve the fit. This interactive process was continued until the fit was very good. A similar approach was used to obtain good agreement between Model 2 results and Fig. 5. With the rate constants and initial fractions given in Table 7, the calculated results and those derived from the work of Eakins et al. (11) given in Fig. 5 were essentially identical. Table 2 gives the integrated activity per unit uptake in the compartments calculated by FORSIM (19) for both models with the rate constants and initial fractions given in Table 7.

The dose in Sv to various organs or tissues per unit uptake can be calculated by multiplying the integrated activity in the compartments by 1.38×10^{-8} ε/m (3) where ε is the effective energy per tritium decay (0.0057 MeV) and m is the assumed mass of the target organ in kg. If we assume that the area of contaminated skin was 40 cm^2 (11) and that the tritium was in a layer 0.1 cm thick in the dermis, the mass for a density of 1 g cm^{-3} would be 0.004 kg. The sum of the integrated activities

(Table 8) in the skin compartments is 2.3 Bqd and 0.25 Bqd for models 1 and 2, respectively, resulting in a skin dose equivalent per Bq uptake of 4.5×10^{-8} Sv, and 4.9×10^{-9} Sv per Bq uptake, respectively. Similarly, if all the body fluids and tissue compartments are assumed to be equally distributed throughout the soft tissue mass of 63 kg (17) the effective dose equivalent (18) per Bq uptake for model 1 and model 2 is 8.7×10^{-12} Sv and 9.7×10^{-12} Sv, respectively.

With the above assumptions, the ratio of skin dose equivalent to effective dose equivalent is 5.2×10^{3} and 5.1×10^{2}, respectively. It is possible therefore that significant skin doses could result from this route of uptake without urinary excretion exceeding a significant concentration if only HTO intakes are assumed. As an example, if only HTO in body fluids is assumed, a urinary tritium concentration of 10^{4} Bq L^{-1} in urine would result in a committed effective dose equivalent to Reference Man of about 10 μSv. However, if the uptake resulted from skin contact with T_2 contaminated surfaces, and the above models adequately represent the range of metabolism of tritium in this circumstance, the committed effective dose equivalent to Reference Man from a 10^{4} Bq L^{-1} urinary concentration could range from about 0.4 μSv to 10 μSv, depending on the time post exposure, and the skin dose equivalent could range from about 250 mSv to 100 mSv, depending on the time post exposure and the model used.

Actual skin doses could be considerably higher than those calculated above if some of the tritium initially bound in skin was removed by diffusion to the atmosphere (11). Skin doses are also directly proportional to the mass that is assumed to contain the OBT bound in skin (Compartments 2, 3 and 4 for Model 1 and Compartment 4 for Model 2) and there is considerable uncertainty in the correct value to use. The thickness of the skin was assumed to be 0.1 cm in the above calculations. However, there is no reason to believe that the OBT is confined to the dermis, with an average thickness of about 0.1 cm (17) and other assumptions could be made. For example, the T_2 could be somehow converted to OBT and stored in sebaceous glands, and the mass involved could therefore be considerably different than 0.004 kg. In addition, the cells in these glands have been implicated in the production of skin cancers in rats by alpha rays (21) and doses to these cells might be of primary concern in humans also.

The effective dose equivalent could also be considerably higher if the OBT in body fluids and tissues was concentrated in a small organ, or a component of OBT is not excreted in urine, and hence not considered in this work. In addition, some of the OBT could be in DNA precursers (20) and the hazard from this tritium could be higher than that calculated from the average dose to tissue.

There is considerable uncertainty about the validity of the excretion curve (Fig. 5) and of the models (Fig. 6) used to describe tritium metabolism in this report. However, it is clear that significant skin doses could occur, and that significant uptakes of HTO and OBT could also occur from skin contact with T_2 contaminated surfaces if the T_2 concentrations

are large enough. The tritium air concentrations, the time between surface exposure to T_2 gas and skin contact ($\sim$24 h), size of surface (10 to 100 cm^2) and contact time (1 to 10 min) used by Eakins et al. (11) are all well within those expected during maintenance and repair of tritium processing equipment. With the above conditions Eakins et al. (11) reported a peak urine tritium concentration greater than 10^5 Bq L^{-1}. Hence significant doses from this route of uptake appear likely in T_2 processing facilities unless care is taken to prevent them.

Experiments to develop methods of prevention, and to improve our understanding of the metabolism of tritium in this situation, are being considered.

7. CONCLUSIONS AND RECOMMENDATIONS

ICRP (12), based only on the dose to the lungs due to HT in the air in the lungs, recommended a value for the Derived Air Concentration for HT of 2×10^{10} Bq m^{-3}. As a result of this work and the work done by Pinson and Langham the Derived Air Concentration should be lowered to 1×10^{10} Bq m^{-3}.

This work has also given a relationship between the concentration of tritium in urine and the upper limit on lung dose. This upper limit on lung dose could be very conservative, and it is recommended that a monitor to give average HT to HTO ratios in air be developed to reduce this conservatism.

Estimated doses to skin and to whole body following uptake of tritium through intact skin in contact HT contaminated surfaces cannot be estimated with confidence. It is recommended that experiments to reduce the uncertainty in dose estimates following this mode of intake be initiated.

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TABLE 1

RATIO OF TRITIUM IN CONDENSATE TO TRITIUM IN URINE

ANIMAL	SAMPLE	RATIO
HT3	1	1.02
	2	0.98
HT4	1	0.94
HTO3	1	1.03
	2	0.98
HTO4	1	1.02
	2	1.04

Average of HT rat samples = 0.98 ± 0.02

Average of HTO rat samples = 1.02 ± 0.01
(Uncertainty given is the standard error of the mean)

TABLE 2

RESULTS FOR HT RAT EXPOSURES

RAT NO.	HT EXPOSURE (Bq m^{-3} min)	HTO IN URINE (Bq L^{-1})	HTO/HT (Bq L^{-1}/Bq m^{-3} min)
HT1	5.6×10^{5}	1.6×10^{12}	3.5×10^{-7}
HT2	6.7×10^{5}	1.8×10^{12}	3.8×10^{-7}
HT3	4.4×10^{5}	1.3×10^{12}	3.4×10^{-7}
HT4	1.4×10^{6}	2.9×10^{12}	4.7×10^{-7}
HT5	5.4×10^{5}	1.5×10^{12}	3.6×10^{-7}
HT6	7.0×10^{5}	2.3×10^{12}	3.1×10^{-7}
HT7	5.1×10^{5}	1.9×10^{12}	2.7×10^{-7}
MEAN			3.5×10^{-7}
SAMPLE STANDARD DEVIATION			0.6×10^{-7}
STANDARD ERROR OF MEAN			0.2×10^{-7}

TABLE 3

COMPARISON OF BOUND TRITIUM IN RAT MUSCLE AND LIVER FOR HT AND HTO EXPOSED RATS

RAT NO.	MUSCLE			LIVER		
	UNBOUND (dpm g^{-1})	BOUND (dpm g^{-1})	RATIO	UNBOUND (dpm g^{-1})	BOUND (dpm g^{-1})	RATIO
Sacrificed on Day 1						
HT1	39300	620	0.016	24800	1200	0.048
HT2	36600	2370	0.065	34100	1960	0.057
HTO1	84000	607	0.007	82200	4910	0.060
HTO2	86300	788	0.009	78400	2220	0.028
Sacrificed on Day 8						
HT3	3740	736	0.20	3040	220	0.072
HT4	14100	586	0.042	13800	1100	0.080
HTO3	16133	2540	0.16	14300	1120	0.078
HTO4	30600	2720	0.089	29500	2820	0.096

TABLE 4

RETENTION HALFTIMES IN HT AND HTO RATS

RAT NO.	DURATION (days)	$T_{1/2}$ (days)	CONDITIONS
HT3	8	2.9	A
HT4	8	3.0	A
HTO3	8	3.0	A
HTO4	8	2.9	A
HT5	14	4.7	B
HT6	14	5.5	B
HTO5	14	4.4	B
HTO6	14	3.9	B
HT7	6	3.1	C
HTO7	6	2.7	C
HTO8	6	4.1	C

A = Metabolic cages during night, standard cage during day.

B = Metabolic cage during day, standard cage during night for 8 days, then metabolic continually.

C = Metabolic cage continually.

TABLE 5

SUMMARY OF RESULTS FOR ONE AND TEN MINUTE EXPOSURES

SUBJECT	TRIAL	HTO IN URINE ($\mu Ci\ L^{-1}$)	HT EXPOSURE ($Bq\ m^{-3}\ min$)	HTO/HT ($Bq\ L^{-1}/Bq\ m^{-3}\ min$)
1 (a)	0	0.29	5.6×10^{11}	1.9×10^{-8}
1 (a)	1	0.23	5.3×10^{11}	1.6×10^{-8}
1 (a)	2	0.27	5.0×10^{11}	2.0×10^{-8}
1 (b)	3	0.28	5.8×10^{11}	1.8×10^{-8}
3 (a)	1	0.56	5.9×10^{11}	3.5×10^{-8}
3 (a)	2	1.1	5.2×10^{11}	7.8×10^{-8}
4 (a)	1	0.37	6.6×10^{11}	2.1×10^{-8}
4 (a)	2	0.31	4.9×10^{11}	2.3×10^{-8}
4 (b)	3	0.31	6.42×10^{11}	1.8×10^{-8}
5 (a)	2	0.44	5.0×10^{11}	3.3×10^{-8}
5 (b)	3	0.42	5.8×10^{11}	2.7×10^{-8}
6 (a)	1	0.10	5.2×10^{11}	0.7×10^{-8}
6 (b)	3	0.49	7.1×10^{11}	2.6×10^{-8}
7 (a)	1	0.20	6.3×10^{11}	1.2×10^{-8}
7 (a)	2	0.18	5.3×10^{11}	1.3×10^{-8}
7 (b)	3	0.27	6.8×10^{11}	1.5×10^{-8}
		Average		2.4×10^{-8}
		Sample Standard Error		1.6×10^{-8}
		Standard Error of Mean		0.4×10^{-8}

(a) One minute exposures

(b) Ten minute exposures

TABLE 6

SUMMARY OF DOSES RECEIVED BY VOLUNTEERS

VOLUNTEER	NUMBER OF EXPOSURES	LUNG DOSE FROM HT IN LUNG (μSv)	EFFECTIVE DOSE FROM HTO IN TISSUE (μSv)	TOTAL EFFECTIVE DOSE (μSv)
1	4	358	35	78
3	2	183	54	76
4	3	295	32	67
5	2	178	28	49
6	2	203	19	43
7	3	303	21	57
TOTALS	16	1520	189	370
AVERAGE		95	12	23

TABLE 7

Rate constants (d^{-1}) used with the models of Fig. 6 to give the urinary tritium excretion in Fig. 5. Initial activity for Model 1 is in Compartments 1, 2, 3 and 4 are 0.33, 0.15, 0.17 and 0.35 Bq, respectively. Initial activities for Model 2 for Compartments 1 and 4 are 0.3 and 0.7 Bq, respectively. Initial activities in all other compartments are zero.

	MODEL 1	MODEL 2
λ_1	24	24
λ_2	0.095	0.15
λ_3	0.3	1.1
λ_4	1.9	3.0
λ_5	0.0693	0.0693
λ_6	3.5	5.0
λ_7	-	5.0
λ_8	-	0.8

TABLE 8

Integrated activities (Bq d) in compartments given in Fig. 6 using the rate constants and initial amounts given in Table 7.

MODEL	COMPARTMENT					
	1	2	3	4	5	6
1	0.014	1.5	0.58	0.18	6.9	0.15
2	0.012	0.85	0.72	0.24	6.1	0.21

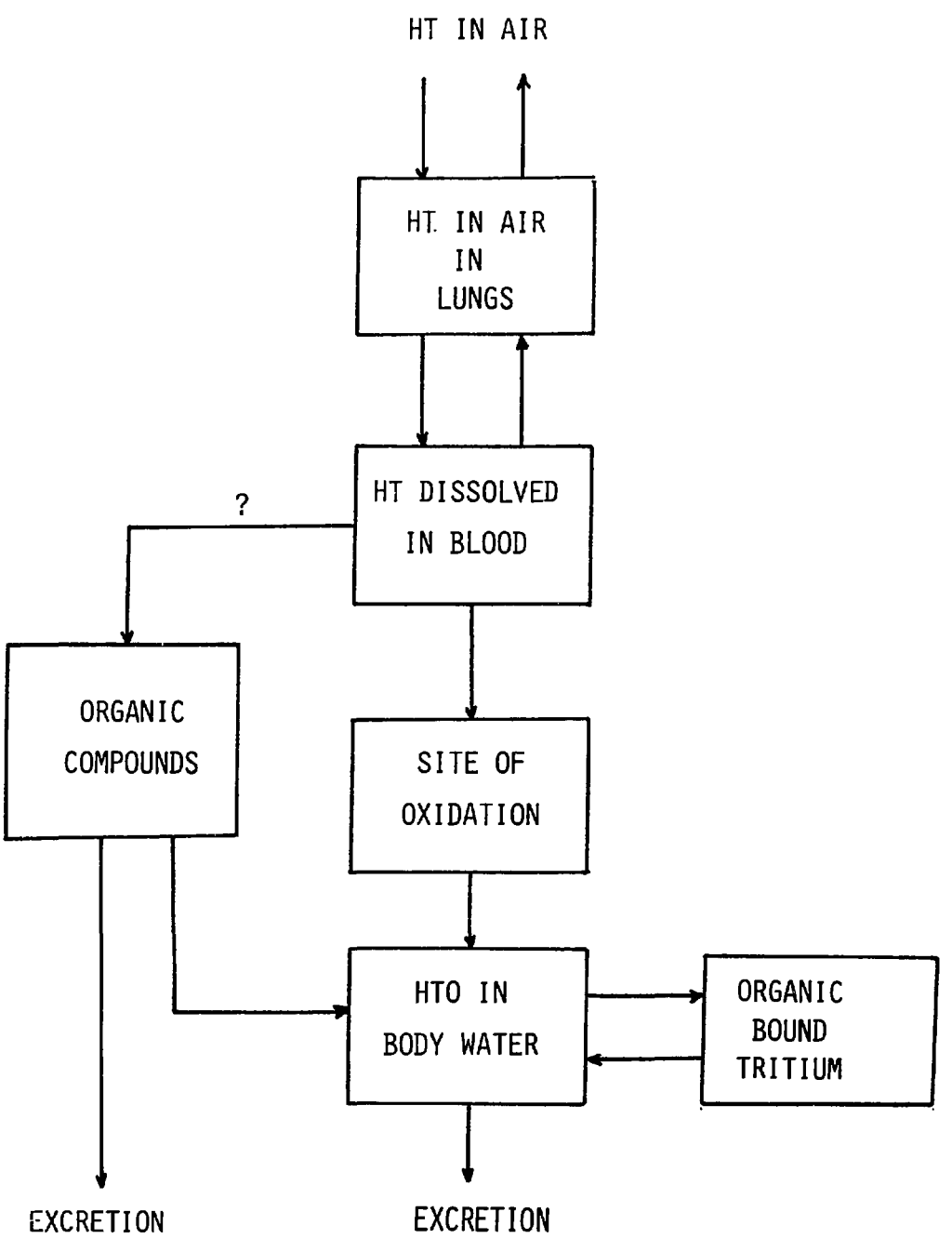

FIGURE 1 Schematic of HT metabolism in humans

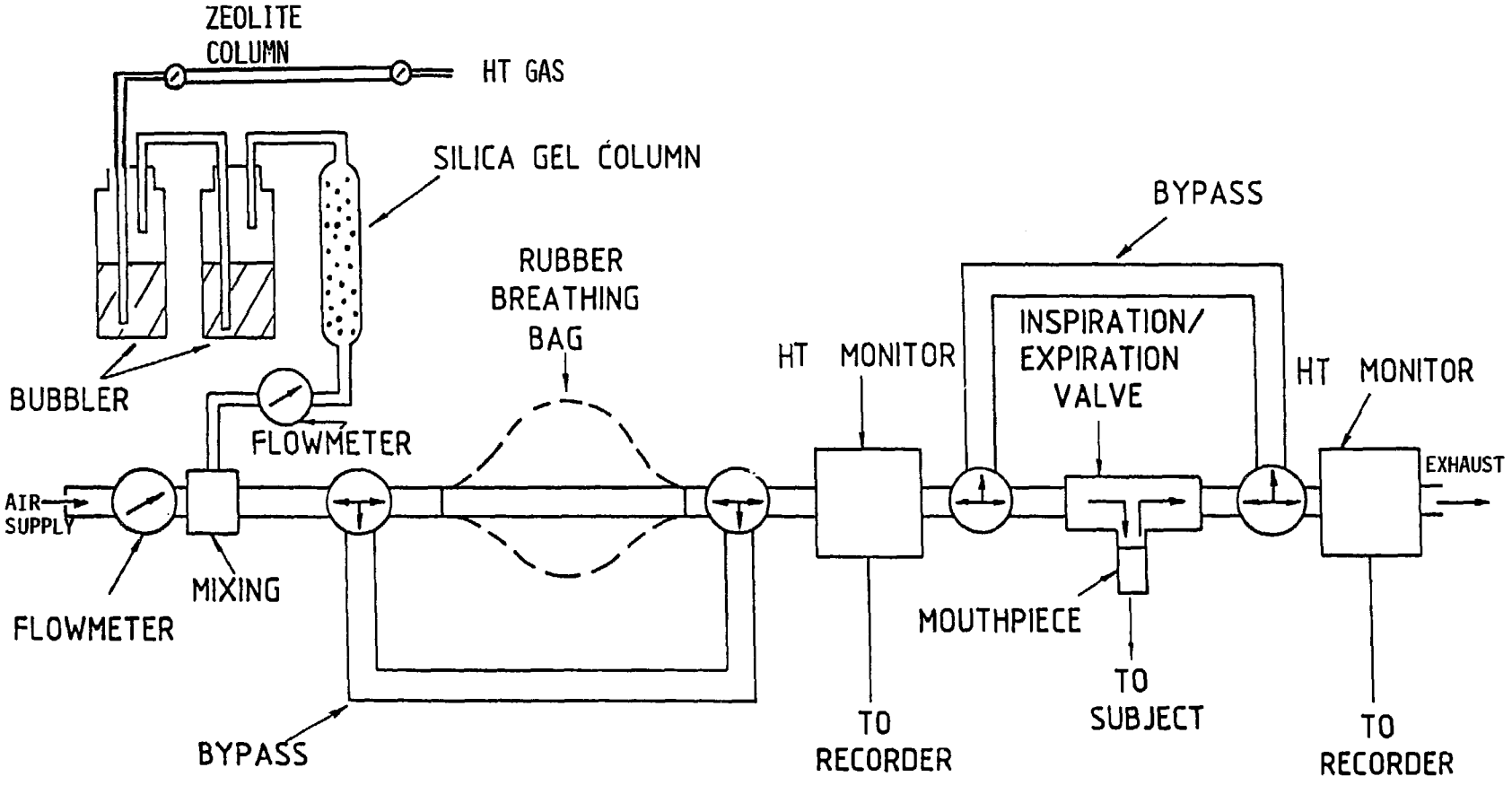

FIGURE 2 Schematic of the HT inhalation exposure system

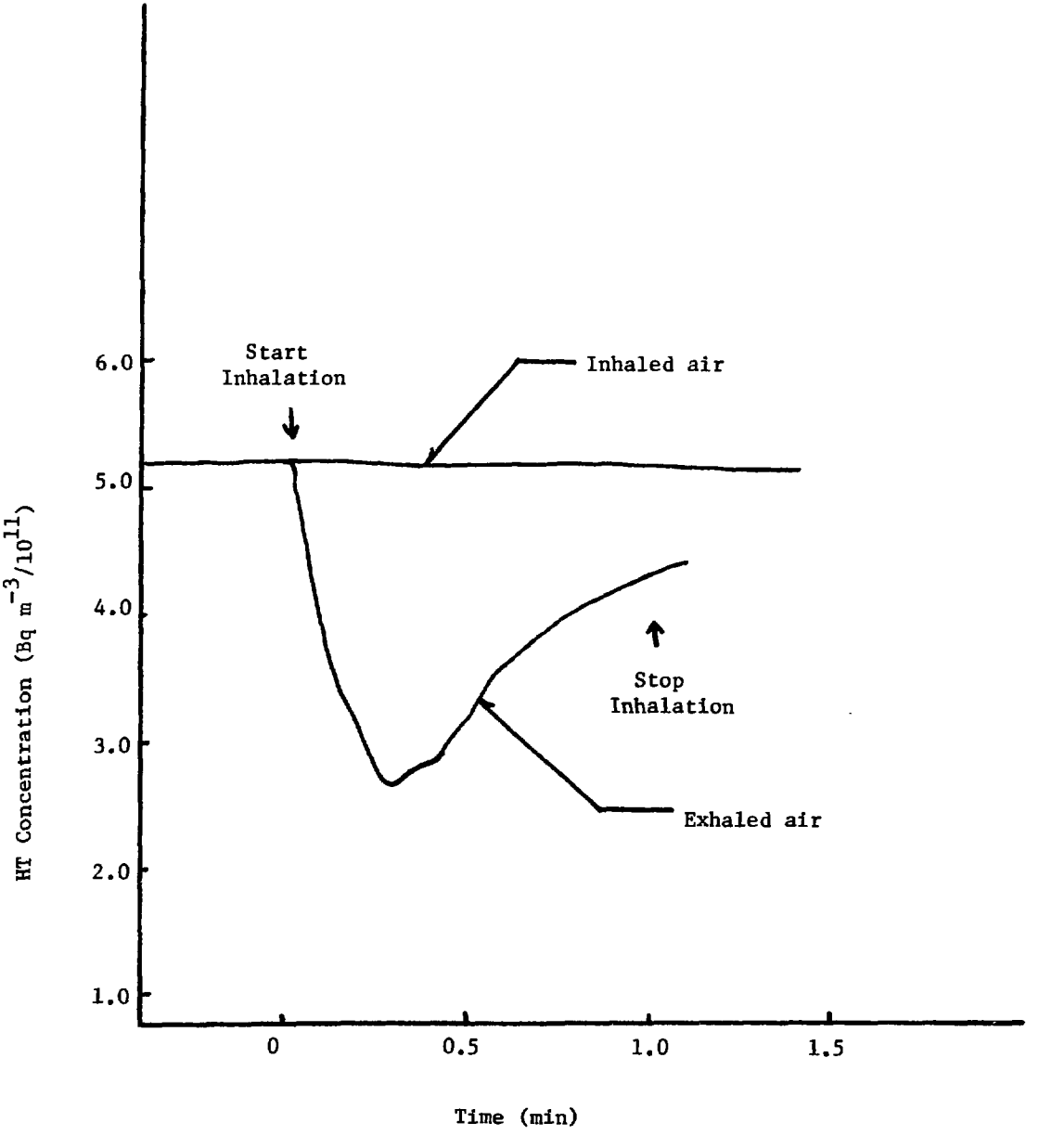

FIGURE 3 Typical air concentration results during a one minute exposure

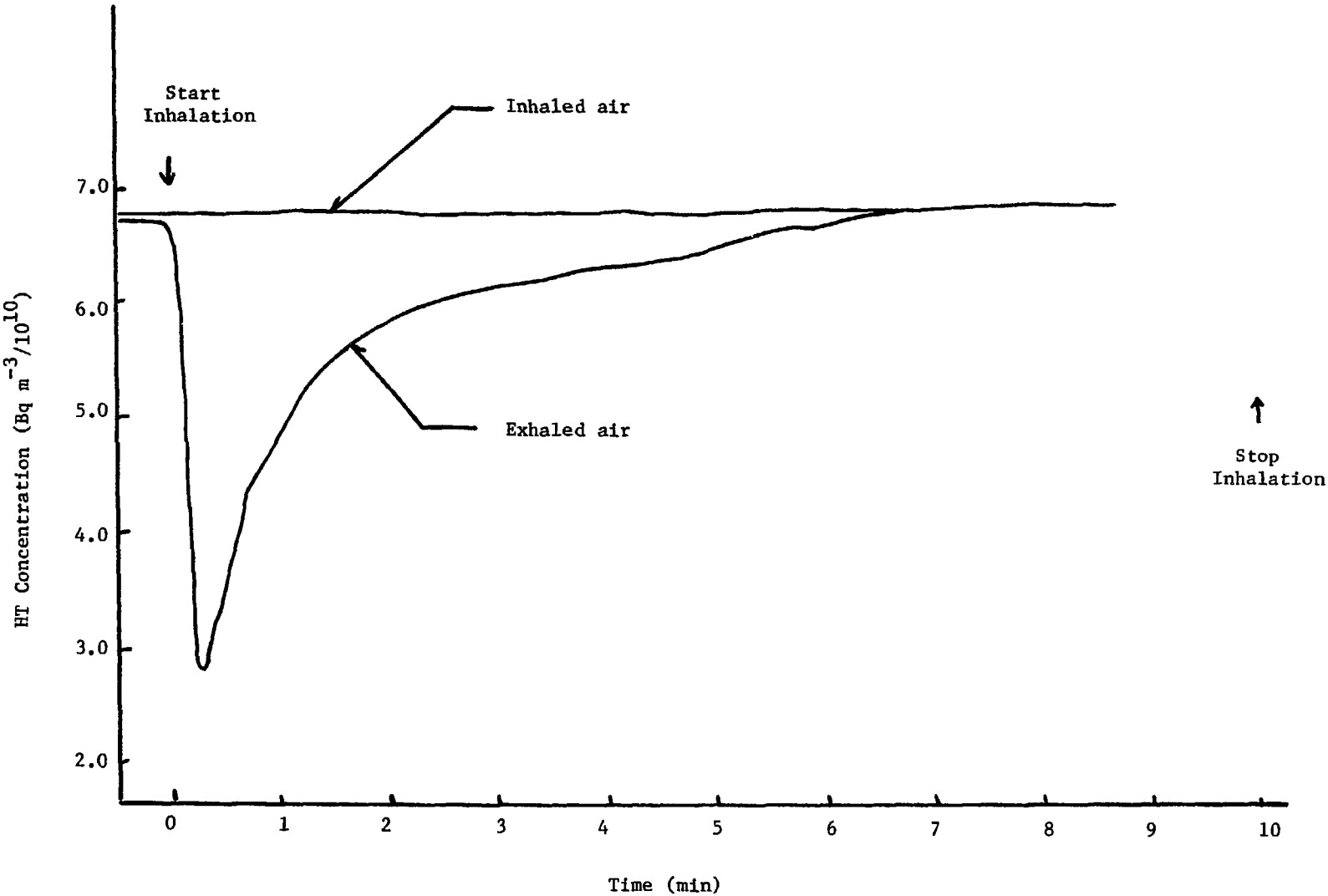

FIGURE 4 Typical air concentration results during a ten minute exposure

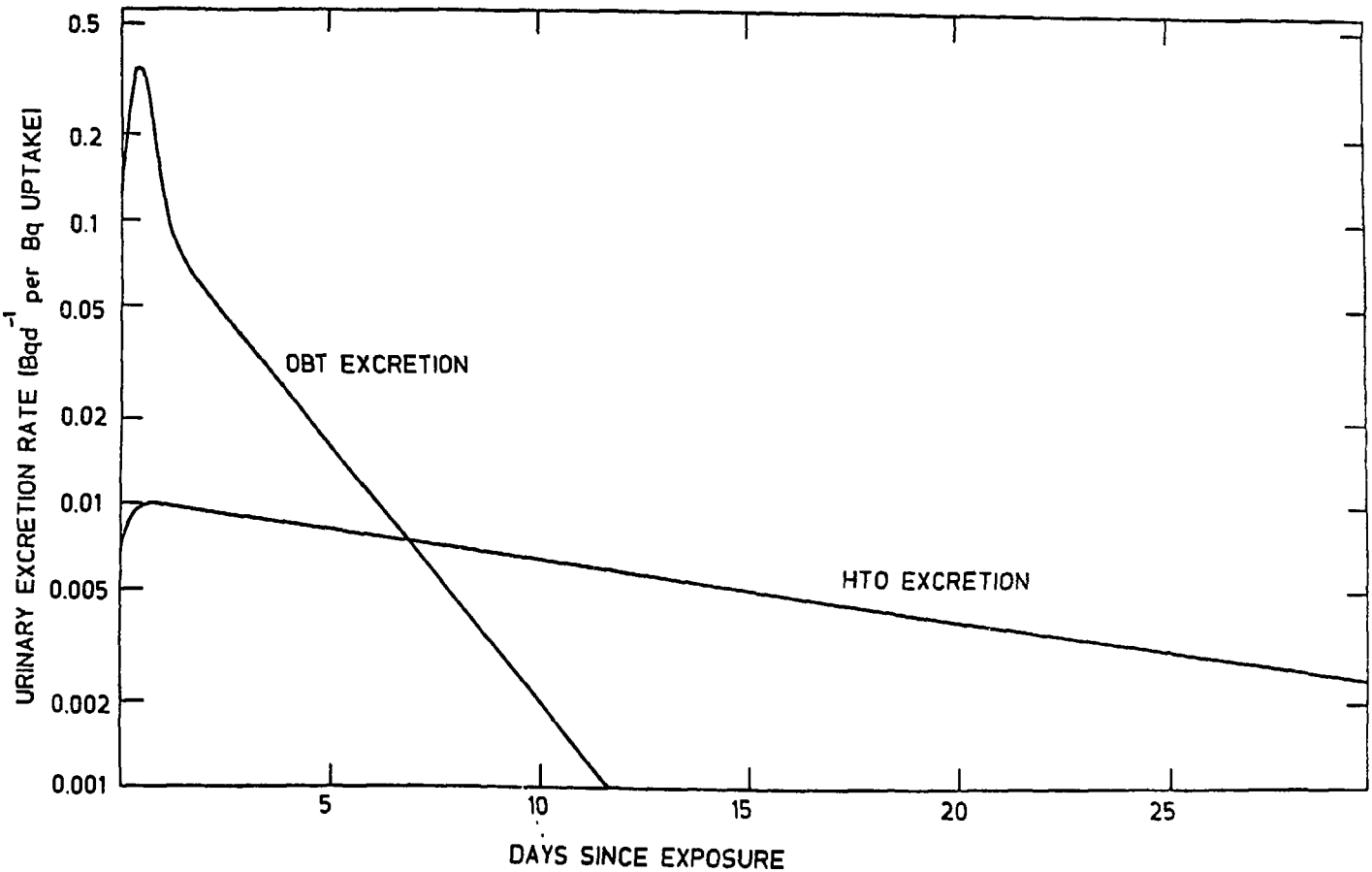

FIGURE 5 The assumed urinary excretion rate following skin contact with T_2 contaminated surfaces used in this preliminary theoretical investigation.

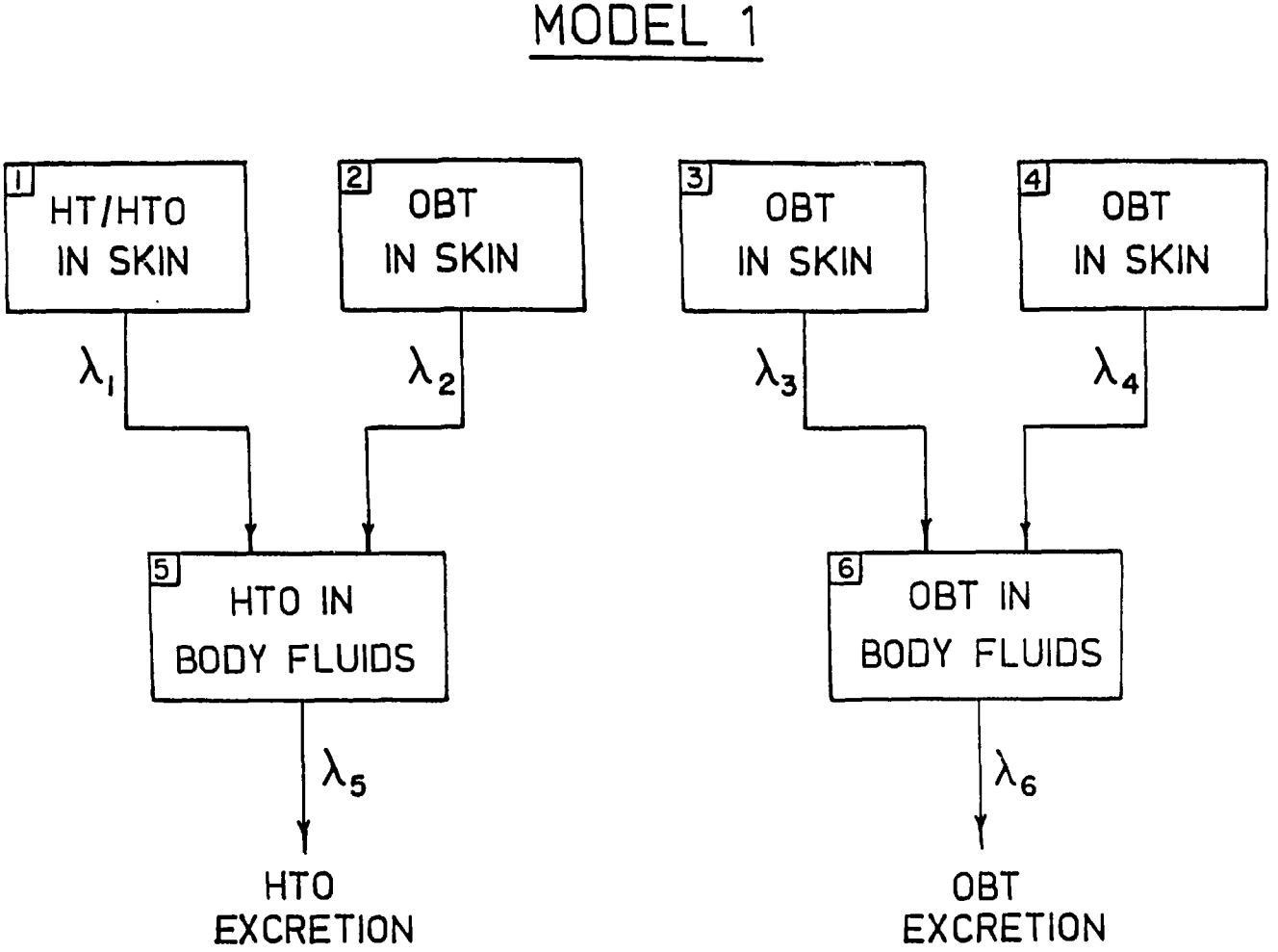

FIGURE 6 (a) Model 1 used to describe tritium metabolism following skin contact with T_2 contaminated surfaces.

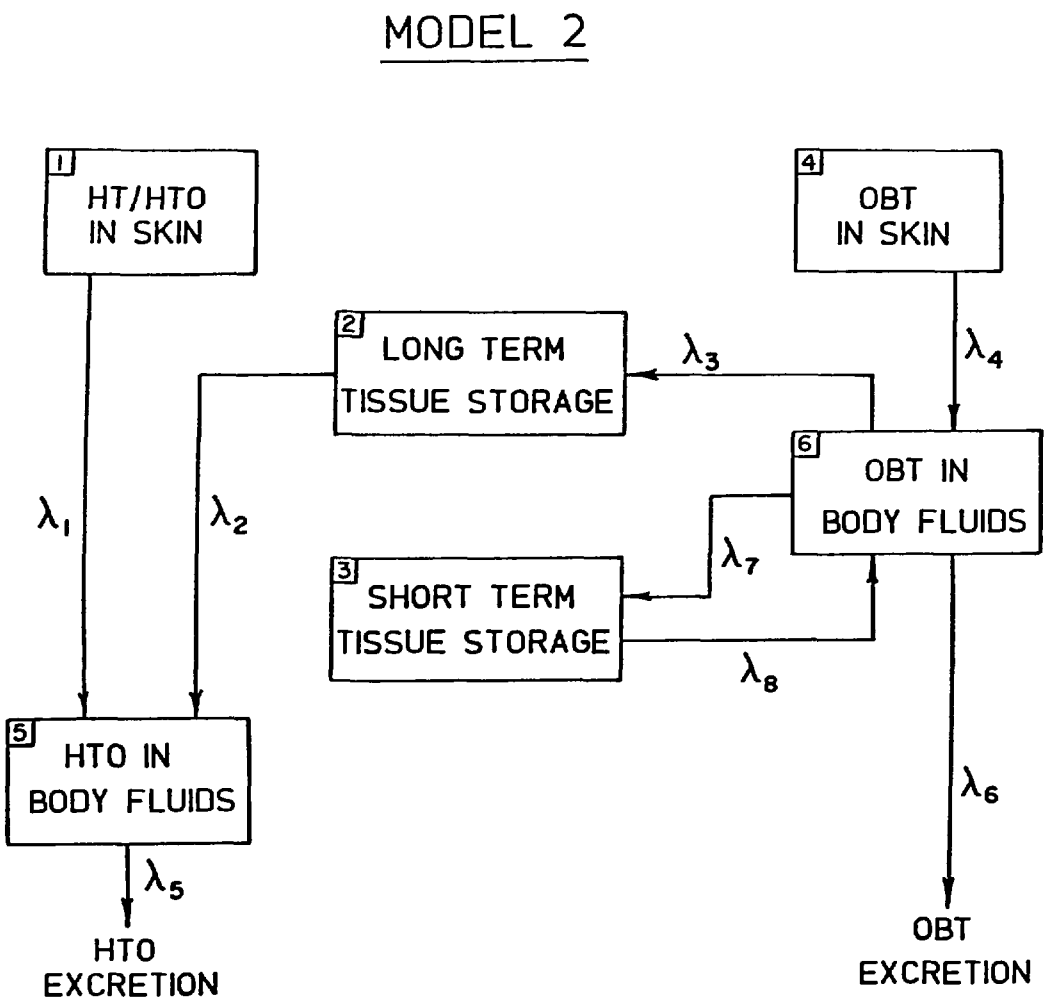

FIGURE 6 (b) Model 2 used to describe tritium metabolism following skin contact with T_2 contaminated surfaces.

APPENDIX A

MEASUREMENT OF ORGANICALLY BOUND TRITIUM IN URINE

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Eakins et al. (A1) found that a large fraction of the tritium in urine excreted following contact between intact skin and HT contaminated surfaces was organically bound. The method they used was "low temperature distillation". In this method, the tritium concentration of the urine sample is first measured. The urine sample is then placed in a covered petrie dish and a beaker of ice water placed on the cover. The urine sample is heated to about 40°C and the water that condenses on the cover collected and analysed for tritium. The difference between the initial urine tritium concentration and the condensate tritium concentration was attributed to organically bound tritium.

This method was tested before being used to demonstrate that organically bound tritium was not excreted following HT gas exposures, and the results of these tests are given in Table A-1. Test 1 used tritiated water, and the measured tritium concentrations in the original solution and the condensate were indistinguishable, as expected. Tests 2 to 5 were done using urine that was known to have a very low tritium concentration spiked with known amounts of tritiated water. Again, the results for the original solution and the condensate were indistinguishable. Tests 6 to 8 were done on an old stock solution of tritiated thymidine that had been stored at a very high specific activity ($\sim$10 mCi/mL) and diluted for this test. Test 6 showed that a considerable fraction of the tritium was no longer bound to thymidine, whereas test 8 (after most of the tritiated water had been "flushed" out) indicated that very little of the organically bound tritium would be transferred to the condensate. Test 9 was done on a urine sample from an employee who has been chronically exposed to tritium oxide for several years. There was no indication of organic tritium in this sample.

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TABLE A-1

TESTS ON TECHNIQUE TO MEASURE THE CONCENTRATION OF TRITIUM IN SOLUTION AND CONDENSATE (μCi/L)

SAMPLE	ORIGINAL SOLUTION	CONDENSATE ON COVER	SOLUTION LEFT IN DISH
Tritiated Water	5.38	5.29	NT*
Tritiated Water in Urine	6.15	5.95	NT
Tritiated Water in Urine	5.65	5.77	NT
Tritiated Water in Urine	5.34	5.48	NT
Tritiated Water in Urine	5.27	5.41	NT
Tritiated Thymidine no evaporation	1693.3	200.7	NT
Tritiated Thymidine one evaporation	535.7	23.2	NT
Tritiated Thymidine 4 evaporations	477.7	1.8	626
Tritiated Thymidine 4 evaporations	311	6.25	876
Urine	6.82	6.89	NT

* NT = not tested

APPENDIX B

HT OXIDATION IN THE HT INHALATION SYSTEM

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It has been observed that T_2 in air will be oxidized more rapidly than H_2 (B1). The more rapid oxidation probably occurs due to the self radiation of T_2 (B1). Beta particles emitted by T_2 can cause formation of OH^- and H^+ (or T^+) radicals which can initiate the formation of HTO. It has also been observed that metals such as steel, brass, aluminum and platinum will catalyze the conversion of HT to HTO (B2). The second order rate constants for the oxidation of HT were estimated to be 1.6×10^{-17} $(Bq\ m^{-3})^{-1}\ hr^{-1}$ for brass, 1.1×10^{-16} $(Bq\ m^{-3})\ hr^{-1}$ for platinum. In the presence of water vapor the rate constants were observed to increase from one to two orders of magnitude (B2). Other materials such as glass were also found to catalyze the oxidation but to a much smaller extent (B3).

The possibility of HT oxidation was recognized before the exposures were undertaken. To remove the HTO that could be found in the gas tank a zeolite and a silica gel columns were installed (see Figure 2). The ratio of HTO/HT of the HT/air mixture after the passage through both columns (but before the first ion chamber) was measured to be 0.5×10^{-6}.

All the components of the HT inhalation/exposure system were plastic or rubber except the HT ionization chambers which were made of stainless steel. Using the above value for the rate constant one can estimate the amount of HTO formed during the passage of HT through one ionization chamber. This was calculated to be 7.4×10^4 Bq/m^3 (2×10^{-3} μCi/L) for standard breathing rates. This would contribute to the subject's HTO concentration in urine of about 5×10^{-4} μCi/L. Since air that passed through the monitor was humid the concentration of HTO in urine could increase 10 times to 5×10^{-3} μCi/L.

The concentrations of tritium oxide in air was estimated by passing the same amount of HT through the system as was inhaled by the subject. This HTO would contribute about 0.02 μCi/L to the concentration of HTO in urine. This amount was deducted from the total HTO concentration found in subject's urine.

The difference between the calculated value and the measured value of HTO measured at the exhaust of the inhalation system could be attributed to:

(1) before HT equilibration is achieved HT/air mixture is passed through the system for 15 to 20 minutes. It could be that during this period some HTO was formed which was trapped and later released into the stream,

(2) the exposure system was used in the same configuration about ten times and although after each experiment it was purged for about two days with air (at the end of the purging period no activity was detected) it is possible that some HT remained adsorbed on the surfaces and was gradually oxidized. The trapped HT could have oxidized and was then released into the HT/air stream, and

(3) the rate constant for the catalysis of HT by steel were obtained with steel foils in a static environment. Different configurations and different surfaces in our experiment could strongly affect the rate of oxidation.

References

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