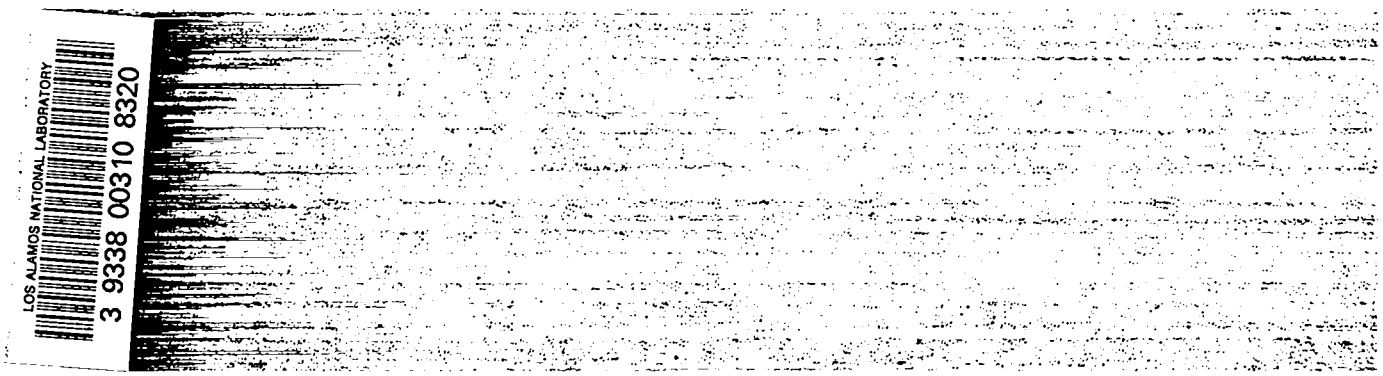
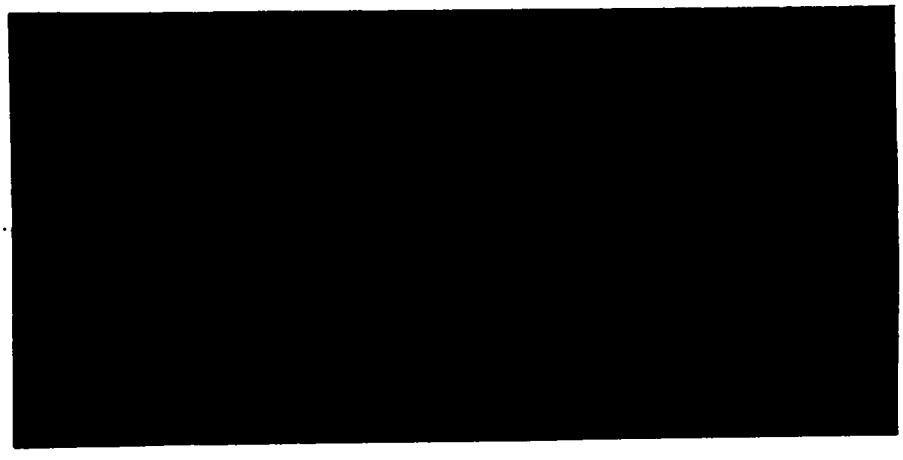
LOS ALAMOS SCIENTIFIC LABORATORY

OF THE UNIVERSITY OF CALIFORNIA

LOS ALAMOS, NEW MEXICO

CONTRACT W-7405-ENG.36 WITH THE
U.S. ATOMIC ENERGY COMMISSION





LOS ALAMOS SCIENTIFIC LABORATORY
of the
UNIVERSITY OF CALIFORNIA

Report written:
February, 1954

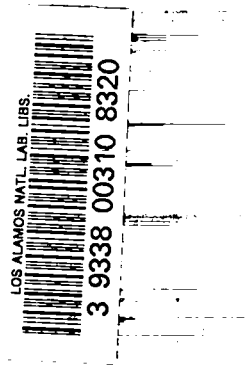
Report distributed: APR 2 9 1954

LA-1646

A THEORETICAL CONSIDERATION OF THE HAZARDS ASSOCIATED WITH ACUTE EXPOSURE TO HIGH CONCENTRATIONS OF TRITIUM GAS

Report written by:

E. C. Anderson
Wright Langham



HEALTH AND BIOLOGY

ABSTRACT

Theoretical consideration of the hazards associated with acute exposure to high concentrations of T_2 gas indicates that a lethal radiation dose would result only from explosive oxidation of very rich T_2-air mixtures. If the gas remained as T_2, the dose rate to a 1 μ layer of the respiratory surfaces would be very high (thousands of rep/sec) but the effect of doses of this magnitude on a very thin layer of these surfaces is completely unknown. Other hazards considered are whole body radiation from dissolved T_2 in body fluids, biological oxidation of T_2 and subsequent absorption of the tritium water produced, auto-oxidation of the T_2 by the tritium beta rays and absorption of the tritium water, and Bremsstrahlung radiation of tissue surfaces. The orders of magnitude of these hazards are a few tenths rep/sec of exposure.

ACKNOWLEDGMENT

The authors are grateful to Dean Meyer and Dr. T. L. Shipman of the Health Division who suggested the problem considered in this report.

CONTENTS

TABLES

1. Introduction

Various aspects of the hazards associated with chronic exposure to tritium gas and tritium water have been carefully considered in previous reports from this laboratory by Anderson,[1] Pinson,[2,3,4,5,6,7] Furchner and Storer,[8] and by the very competent group at Hanford.[9] The results reported in the studies listed indicated that total body radiation from absorption of tritium as HTO was the principal hazard associated with chronic exposure to tritium. They found that the hazard from exposure to HTO was at least a factor of 10^3 greater than the hazard from exposure to tritium gas.

There seems to be only one report[10] in which any consideration is given to the hazards associated with more acute exposure to tritium water and no considerations have been given to very acute exposures to tritium gas. The problems involved are complex, due to the variety of possible mechanisms of entry and damage and to the great uncertainties as to the effects of very weak beta rays in producing radiation damage to the lung surfaces.

In the present report an attempt is made to estimate theoretically the relative orders of magnitude of the several hazards and to point out the critical areas in which experimental information is needed to clarify the situation. Many of the numbers quoted are rough approximations for preliminary estimation only and should not be used as wholly reliable figures for the hazard involved.

2. Potential Hazards to be Considered

In the case of very acute exposures in accidents involving the escape of large quantities of T_2 gas, there are at least four potential mechanisms of radiation damage that must be considered.

2.1 Direct Irradiation of Tissue Surfaces by the Tritium Beta Rays

Any direct effect of tritium beta rays on tissue surfaces must by very superficial because of the extremely short range of the tritium beta particles (maximum range in tissue $\sim 7\ \mu$, average range $\sim 1\ \mu$). Even a very thin layer of dead cells or of water will offer a very large protection factor to the tissue. It seems certain that the skin will be immune to damage from this source and the surfaces of the respiratory tract may be the sensitive area. It is completely unknown to what extent such a soft beta particle is capable of producing damage to the respiratory tract surfaces. Tolerance may well be measured in thousands of rep.

2.2 Bremsstrahlung

Bremsstrahlung (very soft X rays) produced by the stopping of the tritium betas in

tissue will carry a much smaller radiation dose much deeper. The penetration will be of the order of millimeters, but the dose rate will be of the order of 10^{-5} of the beta dose at the surface.

2.3 Solution of Tritium Gas in Body Fluids

Some tritium gas will dissolve in the body fluids as T_2 and hence rapidly permeate the total body according to the equilibration rate of the total body water. The result will be a whole body dose exactly as if tritium water were inhaled but with two outstanding differences:

a. The elimination time will be much shorter than the body water turnover time. On removal from the T_2-containing atmosphere, the concentration of T_2 in the body fluids will fall rapidly.

b. The saturation level is much lower. Instead of 100% retention of the material inhaled, as is the case with water, the body fluids will take up only as much T_2 as will produce a concentration per unit volume which is about 1.6% of the concentration per unit volume of the inhaled air. As discussed in Section 3.2, the dose will not be truly whole body for short exposure times.

2.4 Oxidation of Tritium Gas to Tritium Water and Subsequent Absorption

The tritium gas may be oxidized to water and taken up as such. Several oxidation mechanisms must be considered. Since the exchange of activity between T_2 and H_2O has been shown not to occur spontaneously extraneous factors are necessary, such as:

a. Biological oxidation, observed and measured by Pinson.[2]

b. Catalytic oxidation by the beta radiation of the T_2 itself, measured by Dorfman and Hemmer.[15]

c. Ignition of an explosive T_2-air mixture by spark or flames, followed by inhalation and skin absorption of the T_2O produced.

An attempt has been made to estimate the relative importance of the tritium hazards via the various mechanisms and it is possible to arrive at some tentative conclusions as to the orders of magnitude of the hazards produced by each.

3. Methods of Estimation of Specific Hazards

3.1 Direct Irradiation of Tissue Surfaces by Beta Rays

An estimate of the magnitude of the radiation dose to a tissue surface from T_2 gas can be made by the method of Mayneord.[11] In T_2 gas with a specific activity of 2.6 curies/cc and an average disintegration energy of 6 kev (= 10^{-8} ergs), the energy release is 960 erg sec^{-1} cc^{-1}. If on one side of a plane through the gas, all the activity be removed, the

energy release in small volumes lying in the plane decreases by just 1/2 to 380 erg sec^{-1} cc^{-1}. If the density of the material occupying the space from which the activity was removed be increased to unity (e.g., tissue), the energy release per unit volume is increased approximately by the ratios of densities (a more exact calculation is not justified here); that is, by a factor of 1000. Converting the dose to units of rep, we find a surface dose to the tissue of 4000 rep sec^{-1} during exposure to pure T_2 gas.

Because of the extremely short range of the tritium beta particles, the dose will be a very superficial one. The average range of the tritium beta particles in tissue is only $\sim 1\ \mu$, which may be compared with an average cell diameter of $\sim 5\ \mu$.

It is certain that the horny layers of the skin (assumed to be 70 μ thick[12]--the range of a 70 kev beta particle) will afford virtually complete protection but the response of lung tissue is completely unknown. Experiments are planned to investigate this extremely important point.

3.2 Bremsstrahlung

The intensity of Bremsstrahlung (X rays) from the tritium betas can be estimated from the usual X-ray formula[13]

$$\epsilon = 10^{-9} ZV$$

where ϵ is the fraction of the electron energy appearing as X radiation, Z is the atomic number of the target (6 for carbon), and V is the energy of the electrons in volts (6000 for the average tritium beta). Therefore $\epsilon = 4 \times 10^{-5}$ for tritium betas incident on tissue. The production efficiency of X rays in the air adjacent to the tissue surface will be about the same. These X rays will have an average effective energy of a few kilovolts and hence a penetration of a few millimeters at most. This will reduce the dose rate by distributing the energy through a larger volume compared with the beta's themselves. On the other hand these X rays will reach the tissue surface from a larger gas volume, thus tending to cancel the first effect. Since only an order of magnitude approximation is aimed at here, we may assume that to a first approximation the Bremsstrahlung dose will be about 4×10^{-5} of the direct beta ray dose at the surface and that it will be given to the top few millimeters of tissue. (Note that the skin is presumably not immune to a radiation of this penetration.) This rate is then ~ 0.2 rep/sec for an exposure to pure T_2.

This number is negligible compared with the direct beta dose to the lung surface unless the latter should turn out to be completely harmless because of its superficial nature.

3.3 Solution of Tritium Gas in Body Fluids

Some estimates on the rate of uptake of T_2 gas by body fluids can be made on the basis

of results reported by Tobias et al.[14] They studied the rate of uptake of krypton and other rare gases and found that the results are compatible with the theory that transport by the blood is the limiting factor. The gases were inhaled and gamma measurements made at the hand and the knee of the subject to determine the relative concentrations at those points. Both during uptake and during elimination they observed three principal half-lives. These were about 4 min, 30 to 60 min, and a very long period of several hours. The shortest period was about what one might expect from a crude consideration of blood circulation. If the blood volume is 5 liters and the total body fluid is 40 liters, then the blood contains at any time ~12% of the total amount of gas dissolved in the total body fluids. Assuming a cardiac output of 6 liters/min, the entire blood volume is circulated through the lungs in 0.8 min. If the blood were completely deaerated on passage through the lungs, this number would correspond to a half-time of 4 min in agreement with the shortest period observed by Tobias et al.[14]

For very short exposure times such as would presumably be involved in the sort of accident under consideration, only the shortest half-time need be considered. This in turn means that all the body fluids have not reached equilibrium and the dissolved gas is confined to the major circulation. (The radiation dose received is therefore not a true whole body dose.) Also, for short exposures, the amount of gas dissolved in the body fluids can be assumed to be a linear function of time with a rate constant of 0.003 sec^{-1}. That is, the concentration reached in body fluids as a result of breathing an atmosphere containing T_2 gas for a time t sec is 0.003t times the saturation concentration (provided t is less than, say, 200 sec). The T_2 is then eliminated with a 4 min half-time on termination of the exposure. During elimination the total dose is just D_o/λ where $D_o = D_{sat}t$ is the dose rate in rep/sec due to the highest T_2 concentration reached and λ is the rate corresponding to the 4 min half-time (0.003 sec^{-1}). D_o in turn is given by $D_{sat}\lambda t$, where D_{sat} is the dose rate corresponding to saturation of the body fluids with T_2. Neglecting the comparatively small dose received during the buildup period, the total dose D due to an exposure of duration t is

$$D = \frac{D_{sat}\lambda t}{\lambda} = D_{sat}t$$

If the saturation concentration of T_2 in body fluids is taken to be 1.6% of the concentration of T_2 in the inspired air, then the whole body dose D_{sat} due to saturation of the body fluids by breathing of pure T_2 is ~0.16 rep/sec.

It is interesting that D is independent of the elimination time, because a longer elimination time reduces the amount taken up but lengthens the duration of exposure by an exactly compensating factor. The total dose is just that which would be received from an exposure to complete saturation for a period equal only to the duration of the exposure and not extending

over the elimination time of several minutes. The important effect of the elimination half-time is that it prevents the dissolved T_2 from reaching equilibrium in the body fluids so that the radiation is not truly whole body.

3.4 Oxidation of Tritium Gas to Tritium Water and Subsequent Absorption

The hazards produced by oxidation of the T_2 to T_2O involve absorption and equilibration of the tritium water in body fluids. The radiation dose is delivered as whole body irradiation over the period of time necessary for the elimination of the HTO from the body. Assuming that after a severe exposure, drastic therapeutic measures could be taken to increase the water turnover rate and reduce the half-time to, say, 2 days, for example,* then the total integrated dose delivered to the body is $\sim 10^6$ rep for an initial concentration of 1 curie/gm of body weight. On the basis of this factor, the total dose expected can be calculated in terms of time of exposure in seconds, regardless of the mode of oxidation of the T_2. For slower water turnover rates the dose increases linearly with the half-time.

Biological oxidation -- Pinson has observed and measured an apparent biological oxidation of HT to HTO by rats inhaling tritium as HT. Tritium as HTO was found in the body fluids of the animals despite elaborate drying of the gas prior to inhalation. The mechanism of the fixation is not known, but it is established as an in vivo phenomenon. For concentrations of HT in the inspired air of 9 and 0.6 μc/cc, he found rates of appearance of HTO in body fluids of 0.085 and 0.083% of the inhaled level per hour, respectively. Assuming this rate of fixation to apply to biological oxidation of high concentrations of T_2 gas by man during short exposure times, we find a rate of uptake of 0.34 μc/gm of tissue per second of exposure to a gas with a specific activity equal to that of pure T_2 (2.6 curies/cc). The integrated dose, if the water turnover half-time is 2 days, is then 0.4 rep/sec of exposure. If T_2 gas diffusing through the skin is also fixed at the same rate as that inhaled (no experimental data are available on this point), then the hazard will be proportionally higher.

Auto-oxidation -- By auto-oxidation is meant the oxidation of T_2 to T_2O catalyzed by the radiation field of the T_2 itself. This effect has been recently measured[15] and the ion yield found to be 3.25 molecules of T_2 oxidized per ion pair. This value compares favorably with results previously obtained with alpha rays[16] and with cathode rays.[17] In both the latter cases it was found that ~ 4 molecules of water were formed per ion pair produced in the gas mixtures. The yield appears to be more or less independent of the relative concentrations of H_2 and O_2 and unaffected by the presence of inert gases such as N_2. Using this number, the

*This half-time was achieved by Pinson by the intake of large quantities of water (12 liters per day). Presumably in an extreme emergency a still shorter half-life could be obtained by using sweat-baths, blood transfusions, etc.

maximum rate of formation of T_2O due to the T_2 beta radiation (assuming that four molecules of water are formed for every ion pair and that no ion pairs are lost through recombination, or by other reactions) is calculated to be 3×10^{13} molecules of water per second per curie or 5×10^{-11} moles of water per second per curie. Assuming no dilution with inert water vapor, the water formed will have the same specific activity per mole as the tritium gas, so that the fixation of tritium activity as water proceeds at the rate of 3×10^{-6} curies (T_2O) per second per curie of T_2. In a gas with the specific activity of pure T_2 the rate of T_2O formation is 7.5×10^{-6} curies/cc per second. If the tidal volume is 1 liter, and if this volume of the air in the lungs is assumed to be in equilibrium with the external air, then the oxidation rate in the lungs is 7.6×10^{-3} curies/sec. This is also the rate of uptake of tritium water by the body if the exchange in the lungs is 100%. As a result of a calculation analogous to that for biological oxidation, this corresponds to a total integrated dose of 0.1 rep/sec of exposure if the water turnover time is 2 days.

Explosive oxidation -- If the conversion of T_2 to T_2O proceeds simultaneously with the escape of the tritium gas (by ignition of escaping gas by sparks, flame, metal catalysis, etc.) then the hazard becomes that of exposure to extremely high concentrations of T_2O. For the extreme case of exposure to T_2O, i.e., steam at 1 atm pressure, the specific activity of the steam is that of T_2 gas (2.6 curies/cc). Inhalation will result in absorption of 2600 curies per breath (3 sec) giving an integrated dose ($t_{1/2}$ of 2 days) of 10^4 rep/sec of exposure. One breath would result in a certainly lethal dose of more than 10,000 rep. (For complete conversion of T_2 to T_2O, the concentration of T_2 in air cannot exceed 30%, so the maximum hazard via this mechanism is 3000 rep.)

Even if the exposed person did not inhale while escaping from the accident, absorption through the skin would occur. The only data bearing on this hazard are those of Pinson,[5] obtained by immersing the arm of a man in HTO or HTO vapor up to the elbow. The rate of HTO appearance in the body fluids indicated a water uptake of 0.05 mg cm^{-2} min^{-1}. Assuming a skin area of 10^4 cm^2 and the same rate of water uptake from T_2O steam, the calculated integrated dose ($t_{1/2}$ = 2 days) is 300 rep/sec of exposure, or 100 rep/sec at the 30% level.

4. Discussion and Conclusions

The results of a theoretical consideration of the hazards associated with acute exposure to high concentrations of tritium gas are summarized in Table 1. The hazard is assumed to be pure T_2--an assumption which may be unrealistic, but which represents the maximum possible danger. Appropriate scaling factors can be easily applied to calculate the hazard for

lower concentrations of tritium. A few of the scaled values are also given in Table 1. For example if 100 liters of pure T_2 were distributed through a room 10' x 15' x 30', the dilution factor would be 10^3. In general an accidental exposure would be likely to involve a very non-uniform distribution of the T_2 around the site of the accident and calculations of exact local concentrations would be difficult if not impossible.

The principal conclusions of this study are:

1. The greatest danger from an acute exposure to a mixture of T_2 gas in air is that it will be converted to water by ignition of an explosive mixture. A single breath from such a concentrated T_2O air mixture could well be lethal in spite of drastic therapeutic measures to increase water turnover. Even if the victim did not breathe while escaping from the contaminated area, skin absorption of the T_2O is sufficiently high to constitute a serious hazard.

2. If the T_2 is not ignited, the situation is difficult to interpret due to lack of sufficient information on the effects of very soft beta particles on tissue surfaces. Leaving open the possibility of damage to respiratory surfaces, the next most important hazards are whole body doses from the solubility of T_2 in body fluids and from the biological oxidation of T_2 to HTO. These are of nearly equal importance. The hazard is not a lethal one, a 10 sec exposure even to pure T_2 would result in a dose of only $\sim$6 rep. If room air were 10% T_2, a 10 min exposure would result in a dose of 40 rep.

3. Bremsstrahlung can probably be neglected because of its low intensity and small penetration.

4. Auto-oxidation of T_2 by its own radiation field appears to be negligible compared to biological oxidation and solubility.

5. Summary

From a theoretical consideration of the hazards associated with exposure to pure T_2 gas, it is concluded that a lethal hazard would result only from explosive oxidation of the gas. If the gas remains as T_2 the apparent dose rate to a 1 μ layer of the respiratory surfaces would be very high (thousands of rep/sec) but the effect of doses of this magnitude on these surfaces is completely unknown. The problem is being approached experimentally.

The other important hazard is a whole body dose due to the entry of tritium into body fluids by direct solution as T_2 and by biological oxidation of T_2 to HTO. The expected exposure is $\sim$0.6 rep/sec of exposure time to pure T_2 at 1 atm. Bremsstrahlung and oxidation due to the radiation field of the tritium itself appear to be negligible.

References

1. E. C. Anderson, "Preliminary Report on Human Excretion of Tritium," LAMS-1099, April 1950.
2. E. A. Pinson, "The Body Absorption, Distribution, and Excretion of Tritium in Man and Animals," LA-1218, March 1951.
3. E. A. Pinson, "Absorption of Ingested Tritium Water and the Water Dilution Volume of Man," LA-1464, June 1952.
4. E. A. Pinson, "Lung Absorption of HTO by Man Upon Inspiration of HTO Water Vapor," LA-1465, June 1952.
5. E. A. Pinson, "Absorption of Liquid HTO and HTO Vapor Through the Skin of Man," LA-1466, June 1952.
6. E. A. Pinson, "Incorporation of Tritium into the Organic Components of Various Tissues of the Mouse," LA-1467, October 1952.
7. E. A. Pinson, "The Specific Activity of Tritium in the Organic Components of the Skin and Fat of Man Following Eight Months' Chronic Exposure to HTO in Body Fluids," LA-1468, October 1952.
8. J. Furchner and J. Storer, "The Relative Effectiveness of Tritium in Depressing Iron Uptake in Rats," LA-1544, March 1953.
9. R. C. Thompson, HW-20092 (1951).
10. E. A. Pinson, "Theoretical Considerations of Acute Exposure of Man to HTO or T_2O Activity in the Atmospheric Environment," LA-1469, October 1952.
11. W. V. Mayneord, Brit. J. Radiology, Supplement No. 2, 191 (1950).
12. A. A. Maximow and W. Bloom, A Textbook of Histology, p. 311, W. B. Saunders Co., Philadelphia, Sixth Edition (1952).
13. A. H. Compton and S. K. Allison, X Rays in Theory and Experiment, p. 90, D. Van Nostrand Co., New York (1946).
14. C. A. Tobias, H. B. Jones, J. H. Lawrence, J. G. Hamilton, J. Clin. Invest. 28, 1375 (1949).
15. L. M. Dorfman and B. A. Hemmer, Bull. Am. Phys. Soc. 29, No. 1, 17 (1954).
16. S. C. Lind, "The Chemical Effects of Alpha Particles and Electrons," p. 100 A. C. S. Monograph No. 2, Reinhold, New York (1928).
17. S. Glockler and S. C. Lind, "The Electro-Chemistry of Gases and Other Dielectrics," p. 82, John Wiley and Sons, New York (1939).

TABLE 1

HAZARDS ASSOCIATED WITH ACUTE EXPOSURE TO HIGH CONCENTRATIONS OF TRITIUM GAS

T_2 Conc. (Vol. %)	Radiation Dose (rep/sec of Exposure)						
	Acute Dose During Exposure			Integrated Dose Following Exposure*			
	β-Radiation to Tissue Surfaces ~1 μ	Bremsstrahlung to Tissue Surfaces ~1 mm	Total Body β-Radiation from Sol. of T_2	HTO from Biological Oxidation	HTO from Auto-oxidation	HTO from Explosive Oxidation	
						Inhalation	Skin Absorption
100	4000	0.2	0.2	0.4	0.1	--**	--**
30	1300	0.07	0.07	0.13	0.03	3000	100
10	400	0.02	0.02	0.04	0.01	1000	30
1	40	0.002	0.002	0.004	0.001	100	3

*Calculated on basis of biological half-time for HTO = 2 days

**Complete oxidation to T_2O possible only below 30% T_2 concentration in air