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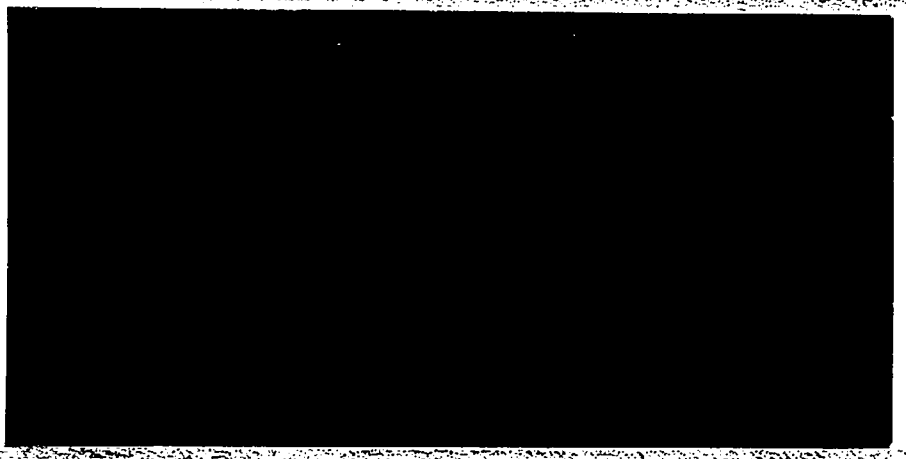

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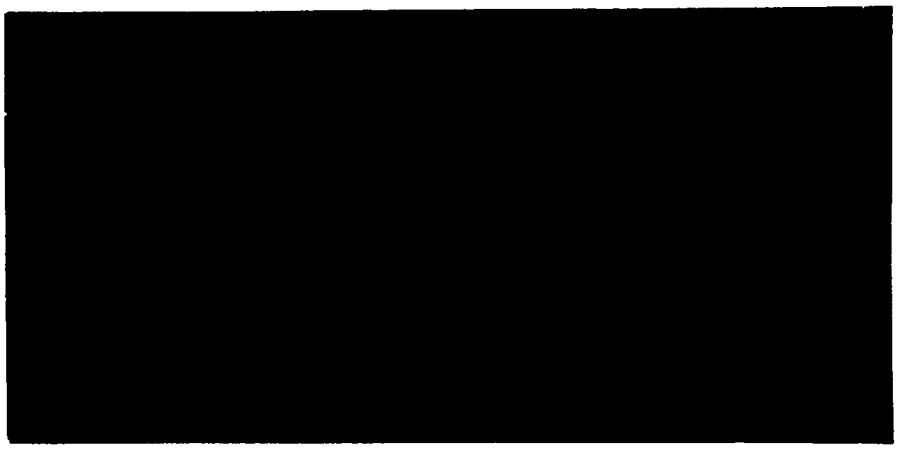

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BIOLOGICAL EFFECTS OF INHALATION OF HIGH CONCENTRATIONS OF TRITIUM GAS

by

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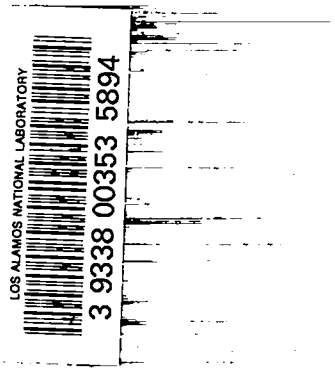

ABSTRACT

The hazard resulting from inhalation of unoxidized tritium (T_2 or HT) in relatively high concentration was investigated. Mice were made to inhale an atmosphere containing 1 per cent tritium gas (20 curies per liter) and the rate of fixation of tritium as HTO in the body fluids was determined.

The animals fixed an LD_{50}^{30} dose of tritium as tritium water (1.5 mc./cc. of body fluids, equivalent to ~800 rep total body exposure) in about 45 minutes, during which time the calculated total dose to the lung surfaces was ~135,000 rep. No histological evidence of acute lung changes from the very high doses of beta radiation was observed. The 30 day survival curve of the mice inhaling 1 per cent tritium gas was compared with that of animals given tritium water (HTO) by intraperitoneal injection. The results showed no significant difference in the LD_{50}^{30} of mice exposed by inhalation of tritium gas and mice that received equivalent amounts of HTO by injection. From these results it was

concluded that the additional large beta radiation exposure of the alveolar surfaces resulted in no additional acute stress to the animal. It appears, therefore, that the fixation of tritium as HTO in the body fluids is the limiting factor with regard to acute inhalation exposure to tritium gas.

ACKNOWLEDGMENTS

The authors wish to acknowledge the help of A. J. Hodges of W-Division, LASL, who helped develop and calibrate the exposure equipment, and to express our appreciation to Dean Meyer and Leo Chelius of H-Division, who originally suggested the problem.

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Introduction

The hazards associated with the inhalation of very low concentrations of tritium gas were investigated experimentally by Pinson.[1,2] He reported that tritium in the form of water (T_2O or HTO) is much more dangerous than tritium in the form of hydrogen gas (T_2 or HT) because of the quantitative fixation of the former by exchange with water vapor in the lungs. HT, on the other hand, is fixed (as HTO) only to the extent of 4 per cent per hour by the rat and 0.08 per cent per hour by man. This oxidation was shown by investigators[3,4] at Hanford to be due largely to bacterial action in the gastrointestinal tract. An additional oxidation mechanism for the conversion of the less harmful HT or T_2 into HTO is "auto-oxidation": the reaction resulting from the radical chains initiated by the beta radiation of tritium. This rate has been measured by Dorfman and Hemmer.[5] From past investigations in animals and humans much has been learned about the absorption, distribution, and excretion of tritium when in the form of HTO.[2,6-14]

Studies of the metabolic turnover of tritium and its exchange with organically-bound hydrogen in the tissues have been reported by Thompson[15-18] and the effect of non-uniform distribution and tritium exchange on the health hazard of exposure to tritium water has been carefully considered by the same investigator.[19] Furchner and Storer[20] found the relative biological effectiveness of tritium beta radiation to be 1.6 compared to the gamma rays of Co^{60} for production of depression of Fe^{59} uptake in the red blood cells of rats. Worman,[21] using splenic and thymic weight loss in mice, obtained an RBE of 1.4 when tritium beta radiation was compared with radium gamma rays.

The effects of the various aspects of tritium metabolism (discussed in the references cited above) on the maximum permissible levels of tritium in air, water, and the human body have been reviewed and summarized in a report by Thompson and Kornberg.[22]

The hazard of inhalation of unoxidized tritium in relatively high concentration, however, had not been investigated experimentally. Calculation of the hazard of inhalation of T_2 on the basis of the HTO uptake is fairly straightforward, and Anderson and Langham have considered the general problem on a theoretical basis.[23] A theoretical estimate of the radiation dose to exposed lung and skin surfaces gives

3000 rep per minute as the dose in an atmosphere containing only 1 per cent T_2. What such a dose rate indicates in terms of actual tissue damage is very difficult to guess, because of the extremely short range of the tritium betas (of the order of 1 micron) in tissue. It seems fairly certain that the horny layer of the skin will afford complete protection, but there remains the possibility of damage to the lungs. The present study was undertaken to determine if any pronounced acute lung damage resulted from the inhalation of unoxidized tritium gas in relatively high concentration.

Experimental Methods and Results

Adult female mice (CF_1 strain) were used throughout this study. Approximately one half of the animals were exposed to the inhalation of T_2 and one half received HTO by intraperitoneal injection. The survival curve of the former group was then compared with the analogous curve obtained from the group receiving HTO by injection. Any pronounced acute lung damage from the very high doses of beta radiation from the unoxidized T_2 should be evidenced by an apparent reduction of the LD_{50}^{30} for the animals exposed by inhalation.

Fourteen groups of 12 mice (24 to 26 g. body weight) each were exposed to the inhalation of an atmosphere containing 1 per cent T_2 (20 curies per liter). The exposure periods ranged from 10 to 200 minutes. Exposures were carried out in a closed system of approximately 1350 ml. volume. Figure 1 shows the complete apparatus used in this study. The exposure chamber was a glass cylinder containing two perforated Lucite shelves to support the mice, 6 mice per shelf, and two trays with NaOH and P_2O_5 to remove CO_2 and water, respectively. A Lucite fan at the bottom of the cage insured rapid air circulation during the exposure. A cold finger condenser in the center of the cage that was connected to a chilled water system (12°C.) provided cooling. Oxygen gas was supplied through a one-way demand valve at about 1 mm. Hg above atmospheric pressure. No metal was used in the cage in order to avoid the possibility of catalytic oxidation of the T_2. The mice could be left in this closed system for several hours without observable ill effects.

The tritium gas came from an uranium tritide (UT_3) source attached to the system. This source when heated to 400 to 450°C. released free T_2 into an evacuated, calibrated gas burette. By means of a manometer the desired volume of T_2 was measured. A nine-fold excess of ordinary hydrogen

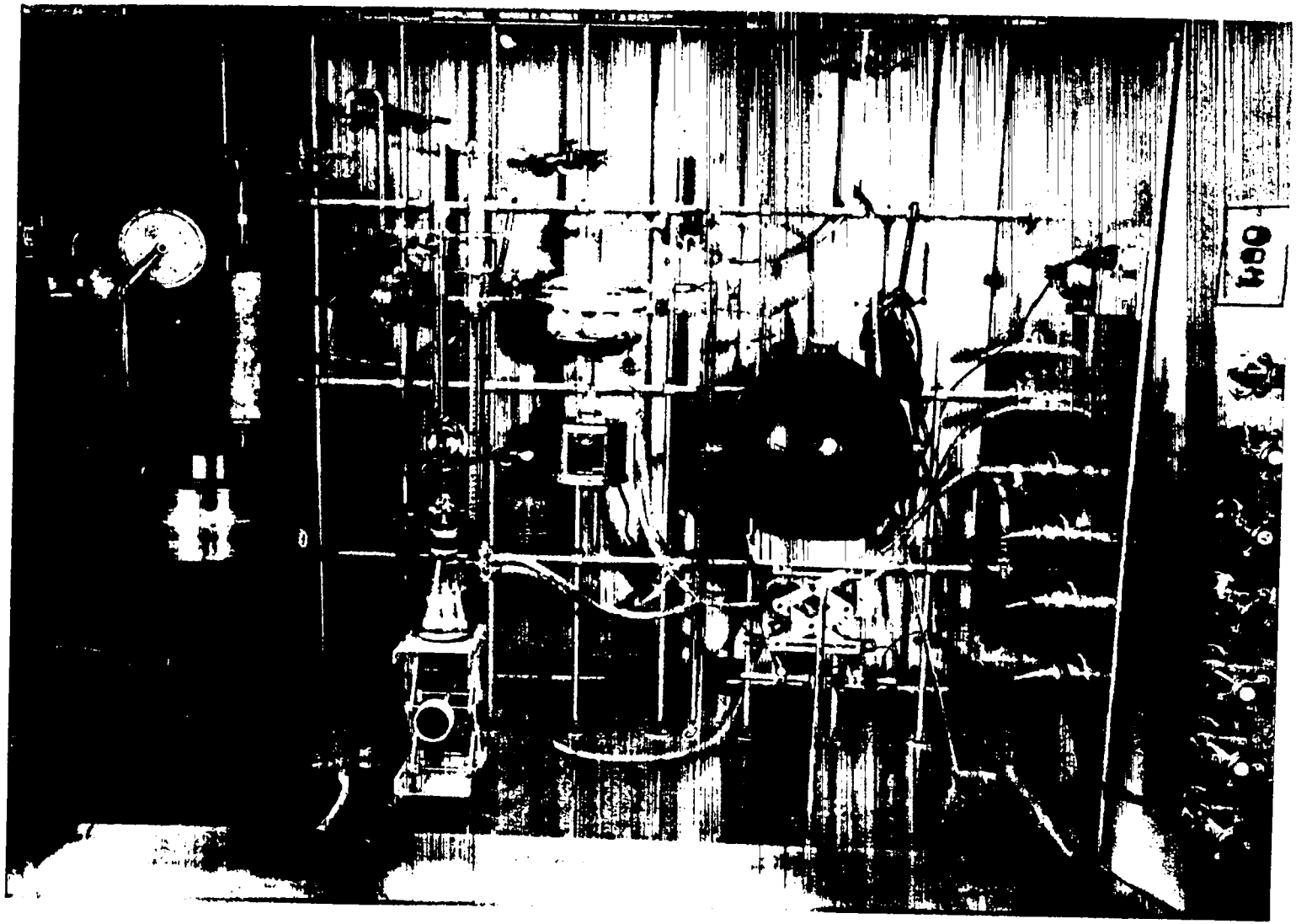

Fig. 1 Apparatus used for exposure of mice to inhalation of an atmosphere containing tritium gas.

(H_2) was then added to the T_2. Since the auto-oxidation rate is proportional to specific activity, the ordinary hydrogen competes with tritium for the radiation-produced radicals and lowers by a factor of about 10 the total amount of tritium converted to HTO by the auto-oxidation process. This reduction of the auto-oxidation rate permitted considerably longer exposures before the fixation of a lethal dose of tritium water in the body fluids and provided a larger ratio of lung dose to whole body dose. The gases were allowed to mix for 30 minutes. After the animals were placed in the cage, the pressure was reduced proportionately to allow the admittance of the HT gas. At the end of the exposure period, the remaining HT was recovered by flushing the gas mixture in the cage into a large evacuated flask where the HT was oxidized to HTO by means of an electrical spark. The mice were placed in metabolism cages (4 animals per cage), and urine specimens were collected at specified intervals for 6 days, after which the animals were placed in regular cages and observed for 30 days to determine the LD_{50}^{30} dose of tritium beta radiation.

The urine samples were assayed for tritium activity (HTO) with the liquid scintillation counter (Fig. 2).[24,25] The maximum tritium activity in the body fluids (in mc./ml.)

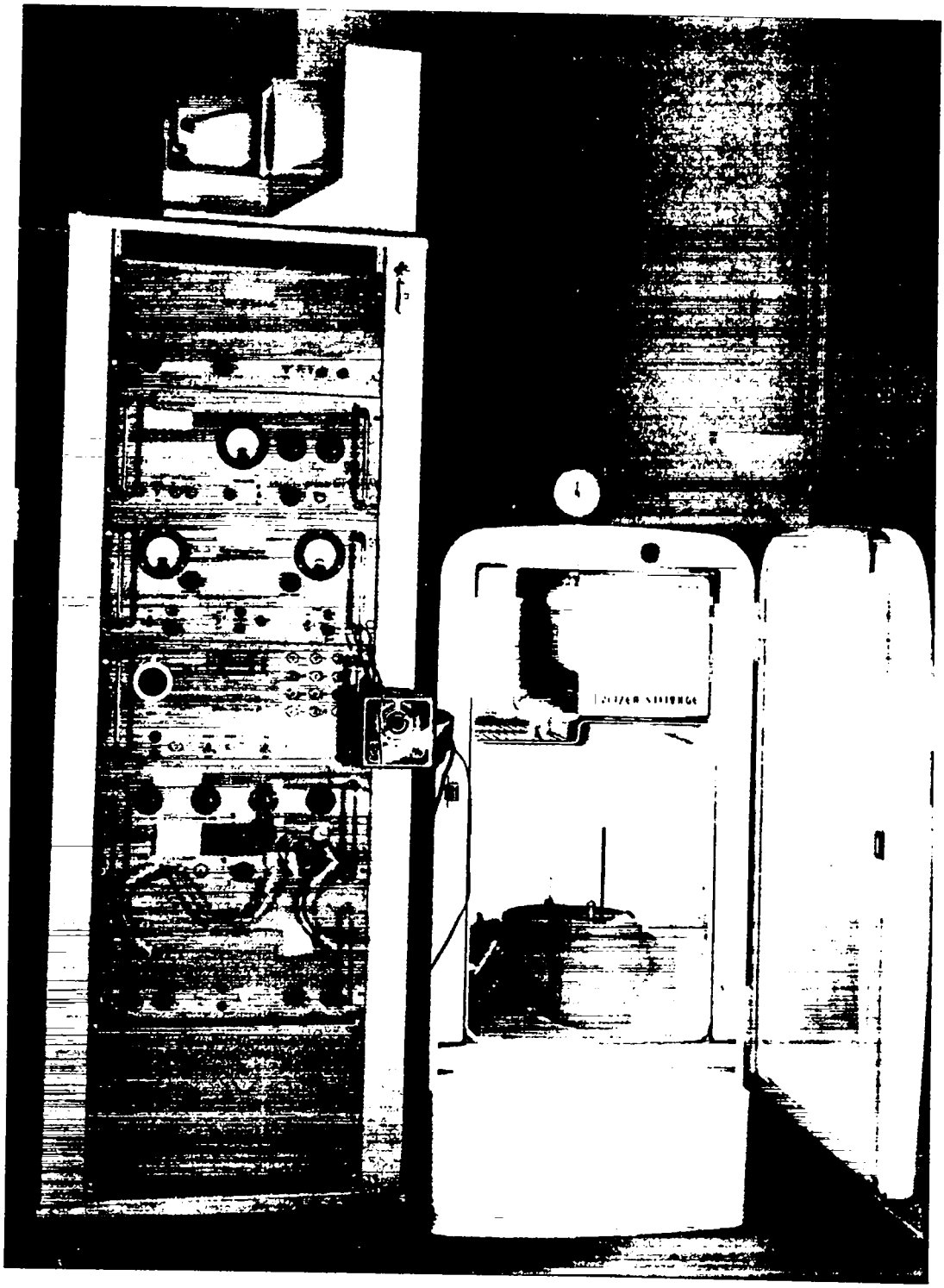

Fig. 2 Fast coincidence liquid scintillation counter used for tritium measurements.

was determined for each group of mice by plotting the HTO concentration in the urine versus time after exposure and extrapolating back to the origin. Figure 3 shows the curves for the HTO turnover of three such groups. The times plotted in this figure represent the midpoint of each collection period.

The data in Fig. 4 show the fixation rate of HTO by mice that inspired 1 per cent T_2 mixed with 9 per cent H_2. The rate of fixation for short periods of exposure was quite unexpected. On the basis of measurements of the biological oxidation rate in rats, it was expected that the mouse, when inhaling 1 per cent tritium gas, would build up HTO activity in the body fluids at a rate of about 0.9 mc./ml./hr. The calculated auto-oxidation rate for a mixture of 1 per cent T_2 is 0.08 per cent per hour. Assuming that the mice exchanged their body water quantitatively with all the tritium water produced in the 1.35-liter cage by auto-oxidation, the expected rate of uptake from this source was 0.15 mc./ml./hr. The sum of these two rates (1.05 mc./ml./hr.) should give the total rate of tritium water uptake by the mouse breathing a mixture of 1 per cent T_2 and 9 per cent H_2. For long exposure times the slope of the concentration versus time of exposure curve gave a value of 1.15 mc./ml./hr., which did agree very well with the calculated value. However, the

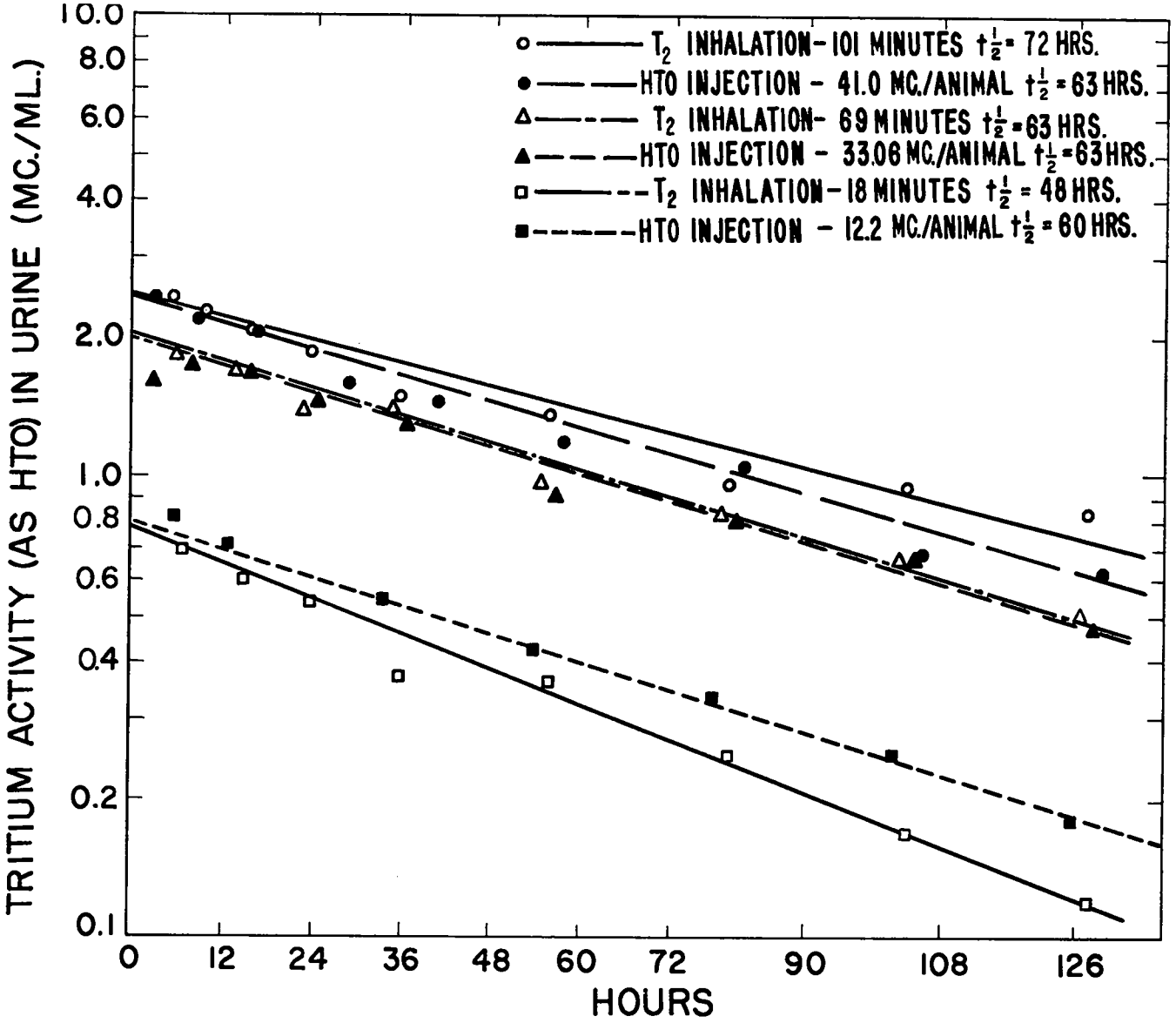

Fig. 3 Rate of HTO turnover in mice that inhaled 1 per cent tritium gas and mice injected intraperitoneally with HTO.

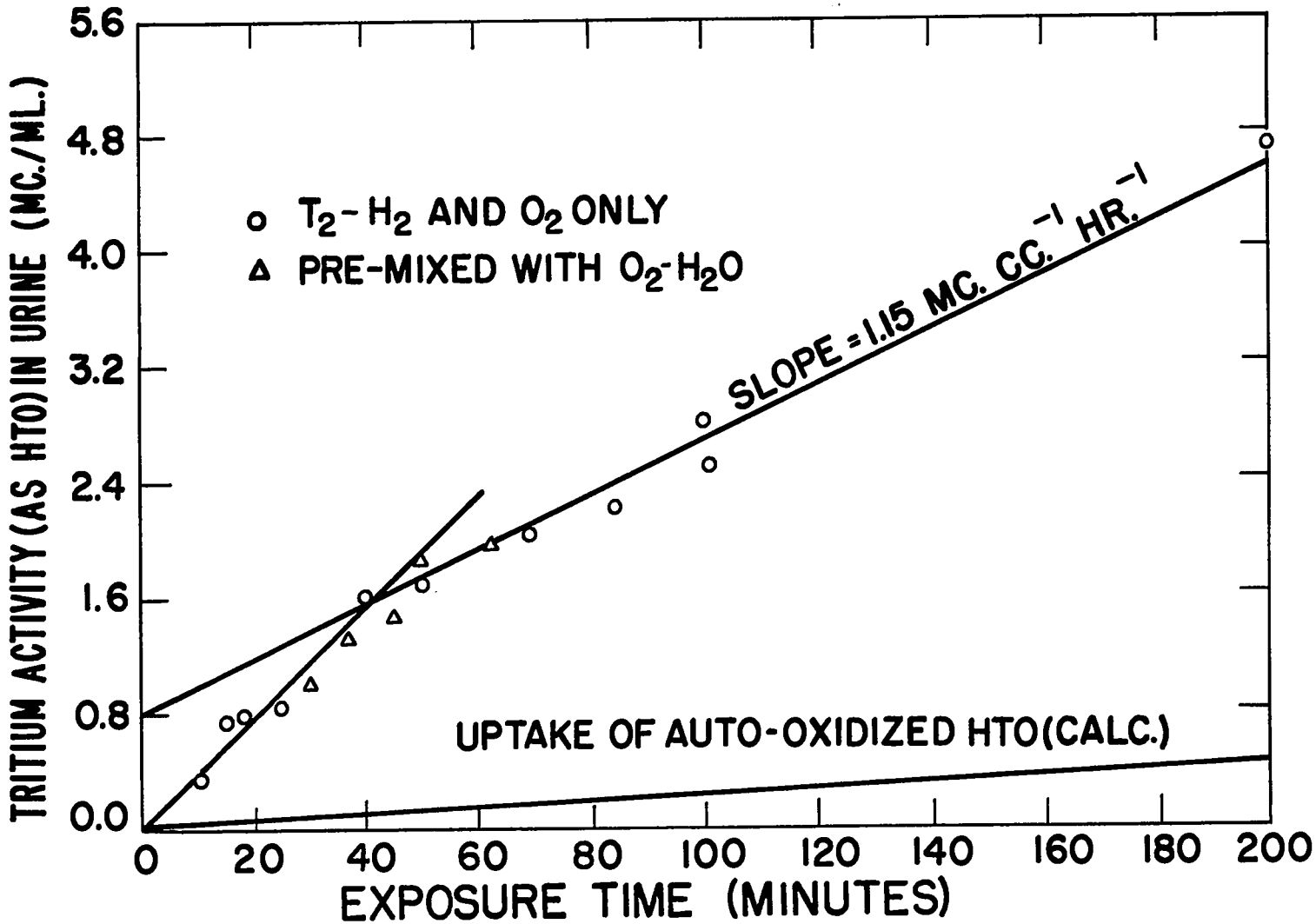

Fig. 4 Rate of fixation of HTO by mice inhaling 1 per cent T_2 and 9 per cent H_2.

experimental curve did not extrapolate back to the origin. The observed results indicated a rapid initial uptake of a total of 0.8 mc./ml., an uptake with a half-time of 22 minutes which was completed in about 1 hour, after which the expected rate was observed.

An analysis of the initial portion of the curve (Fig.5) indicated that an exponential absorption equation of the form

$$C = C_0(1 - e^{-ut})$$

was compatible with the data. The above rate equation would follow if the observed concentration was a result of the uptake by the mice of a small amount of T_2O originally present in the T_2. However, 0.5 per cent of the total amount of T_2 in the cage would have to be in the form of HTO to provide for the observed results. Also, the uptake would be quantitative and the rate of uptake would be determined by the rate at which the mice turned over the volume of air in the cage. Assuming a respiratory volume of 25 ml./min. per mouse, the calculated time required for 12 mice to turn over the 1350 ml. volume of the cage is 3 minutes. If the local mixing of the exhaled air with the rest of the cage was poor, this half-time could be lengthened considerably, but 22 minutes is rather extreme. Furthermore, attempts to demonstrate

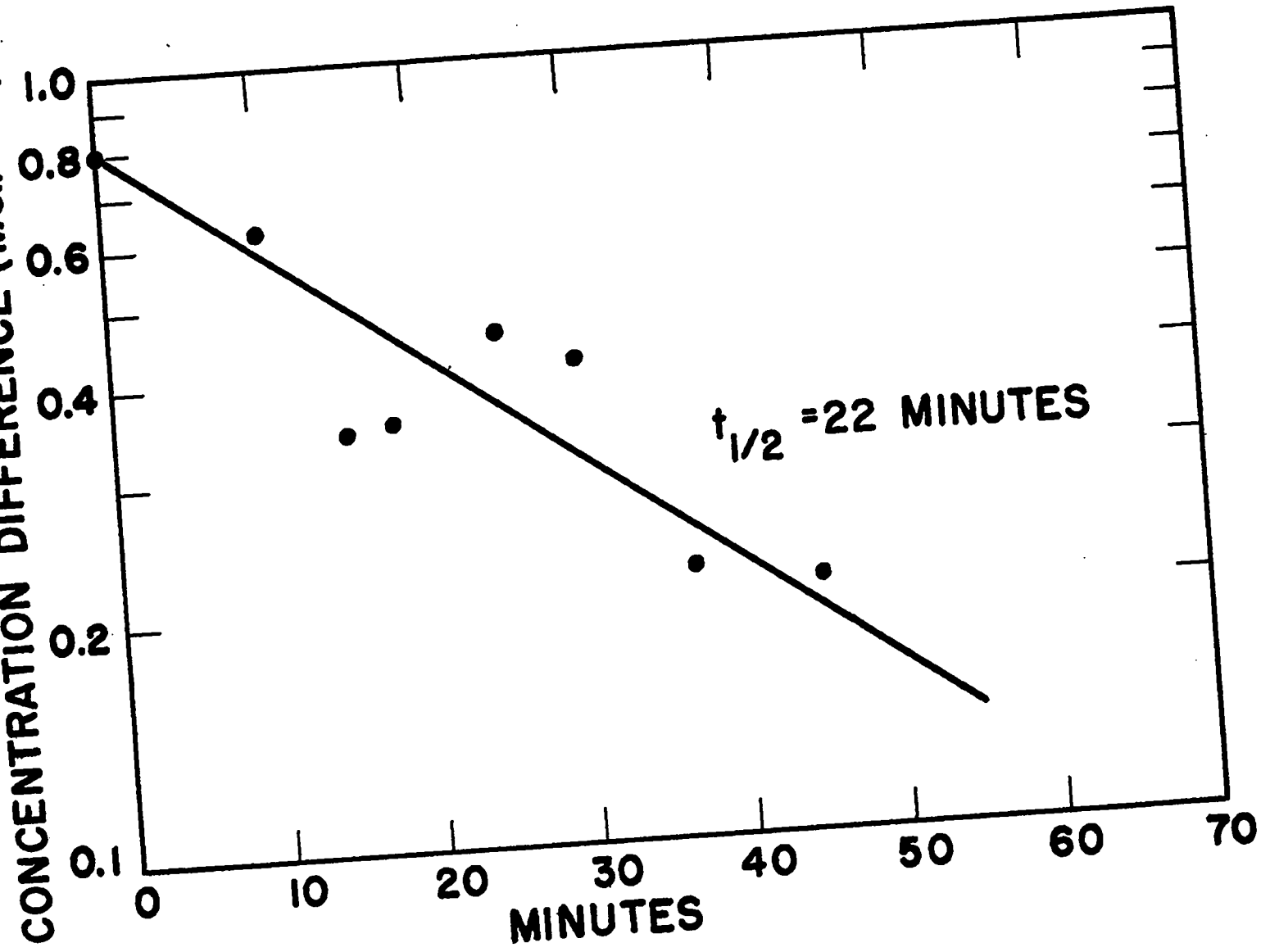

Fig. 5 Initial portion of the fixation curve showing a fixation half-time of 22 minutes.

the identity of the "impurity" with water or with any chemical species of tritium exchangeable with water failed. Passing the T_2 gas over P_2O_5, extracting it with a large volume of liquid water, and premixing the T_2-H_2 gas with excess air or O_2, followed by exposing the mixture to liquid water, all failed to affect the initial uptake rate.

The nature of this peculiar effect, therefore, remains unknown. It was important for the present experiment only in that it reduced appreciably the permissible duration of the exposure and decreased the ratio of the lung dose to total body dose. The mice fixed a lethal dose of HTO in about 45 minutes; whereas, if the 1.15 mc./ml./hr. rate of fixation had prevailed from the start of the experiment, a time of 75 minutes would have been required and the lung dose associated with the LD_{50}^{30} of HTO would have been 225,000 instead of 135,000 rep.

Fifteen groups of 12 to 21 mice (24 to 26 g.) each were injected intraperitoneally with HTO in amounts sufficient to produce tritium concentrations in the body fluids corresponding to those of the animals exposed by inhalation of HT.

Urine samples were collected from the injected mice and the HTO turnover established in the same manner given above (Fig. 3). In both experiments the integrated total body beta radiation dose to the mice by the HTO was calculated using

the following physical constants and formulae:

5.69 kev = average energy release per tritium disintegration

3.7×10^7 d/sec. = 1 mc.

3.6×10^3 sec. = 1 hr.

1.6×10^{-9} erg = 1 kev

93 ergs/g. = 1 rep

0.67 ml. of water per gram of tissue

$$\text{Dose (rep)} = \frac{5.69 \times 3.7 \times 10^7 \times 3.6 \times 10^3 \times 1.6 \times 10^{-9}}{93}$$

$$= 13 \text{ rep/hr./mc./g.} \times 0.67 \text{ ml./g.}$$

$$= 8.7 \text{ rep/hr./mc./ml.}$$

$$\text{Integrated dose} = 8.7 \times C \times \frac{t_{1/2}}{0.693}$$

In the above expressions the calculated dose delivered per hour by tritium at a concentration of 1 mc./g. is 13 rep, 67 per cent is the observed body fluid content, $t_{1/2}$ is the turnover time of HTO in hours and C is the tritium concentration in millicuries per milliliter of body water.

The data showing the exposure conditions, radiation doses, and 30 day survival of the animals that were exposed by inhalation of HT and by intraperitoneal injection of HTO are summarized in Tables 1 and 2, respectively.

When the per cent survival at 30 days was plotted against total body radiation dose in rep, the survival curves

TABLE 1 SURVIVAL OF CF_1 FEMALE MICE FOLLOWING INHALATION OF AN ATMOSPHERE CONTAINING 1 PER CENT TRITIUM GAS

Exposure (min.)	Tritium conc. as HTO in body fluids (mc./ml.)	Turnover, $t_{1/2}$ (hrs.)	Calc. dose to lungs (rep)	Calc. dose to body (rep)	30 day survival (%)
200	4.70	40	600,000	2370	0
101	2.50	72	300,000	2260	0
84	2.20	63	252,000	1740	0
69	2.05	63	207,000	1630	8.3
65	1.95	46	195,000	1130	8.3
50	1.70	57	150,000	1220	25.0
45	1.45	44	135,000	800	66.6
37	1.30	66	111,000	1080	33.3
35	1.60	54	105,000	1090	25.0
30	1.00	55	90,000	690	41.6
25	0.83	43	75,000	450	100.
18	0.78	48	54,000	470	100.
15	0.74	45	45,000	420	100.
10	0.36	43	30,000	195	100.

Note: 12 mice were used for each exposure.

TABLE 2 SURVIVAL OF CF_1 FEMALE MICE FOLLOWING INTRAPERITONEAL INJECTION OF HTO

HTO injected per mouse (mc.)	Tritium water conc. in body fluids (mc./ml.)	Turnover, $t_{1/2}$ (hrs.)	Calculated body dose (rep)	30 day survival (%)
41.0	2.50	63	1980	0
35.8	2.17	69	1890	0
35.1	2.30	40	1160	5
33.06	2.02	61	1550	0
30.65	1.95	38	930	10
28.5	1.85	51	1190	0
27.9	1.80	66	1500	0
26.5	1.63	43	870	38
24.2	1.42	62	1110	24
21.85	1.22	40	615	90
16.4	0.95	42	500	100
13.4	0.93	59	690	100
12.2	0.80	60	603	100
11.4	0.65	39	320	100
5.6	0.37	45	210	100

Note: 12-21 mice were used per injected dose.

shown in Fig. 6 were obtained. From these curves it appears that the LD_{50}^{30} total body radiation dose for animals exposed by the inhalation of HT was about 900 rep and that for animals receiving tritium beta irradiation by intraperitoneal injection of HTO was about 840 rep. Probit transformations of the two survival curves are shown in Fig. 7. Statistical analysis of the probit regression lines gave a value of about 800 rep for the LD_{50}^{30} tritium beta irradiation dose in both cases and failed to show any significant difference in the values obtained under the two different exposure conditions. The slopes of the two regression lines appeared to be significantly different. It is doubtful, however, that this difference in slope has any interpretable meaning insofar as the experiment was concerned. Unfortunately, the high initial fixation rate of HT (as HTO) by the animals exposed by inhalation resulted in 100 per cent deaths in the majority of the exposed groups of the first experiment. It was, therefore, necessary to repeat the experiment using additional animals that were not randomly selected from the same population as those exposed by intraperitoneal injection of HTO. It is quite likely that the difference in slopes of the two regression lines is an indication of lack of homogeneity between the two animal populations from which the experimental groups were selected.

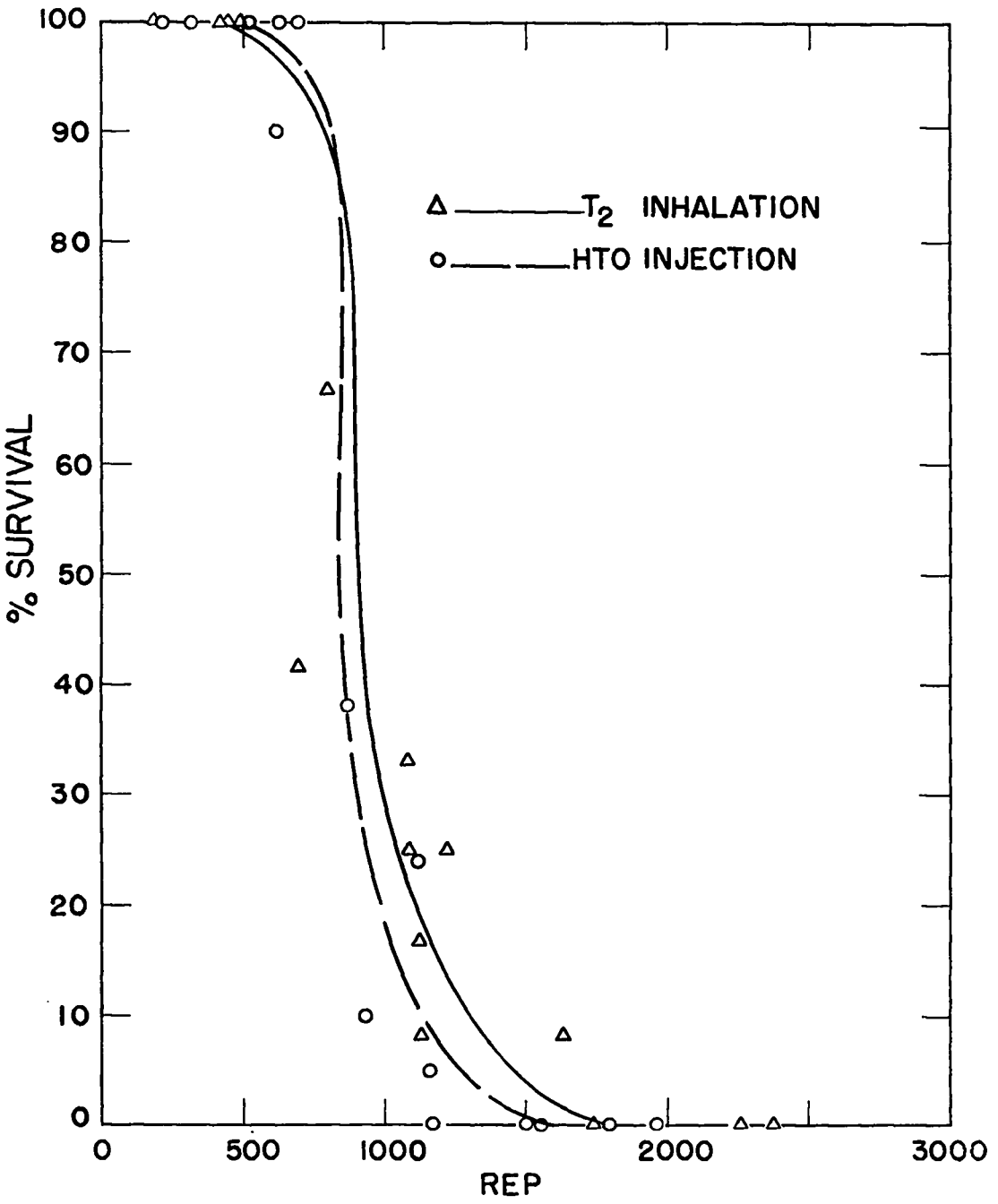

Fig. 6 **The 30 day survival curves of mice following exposure by inhalation of 1 per cent tritium gas and by intraperitoneal injection of HTO as a function of total body beta radiation dose from the HTO in body fluids.**

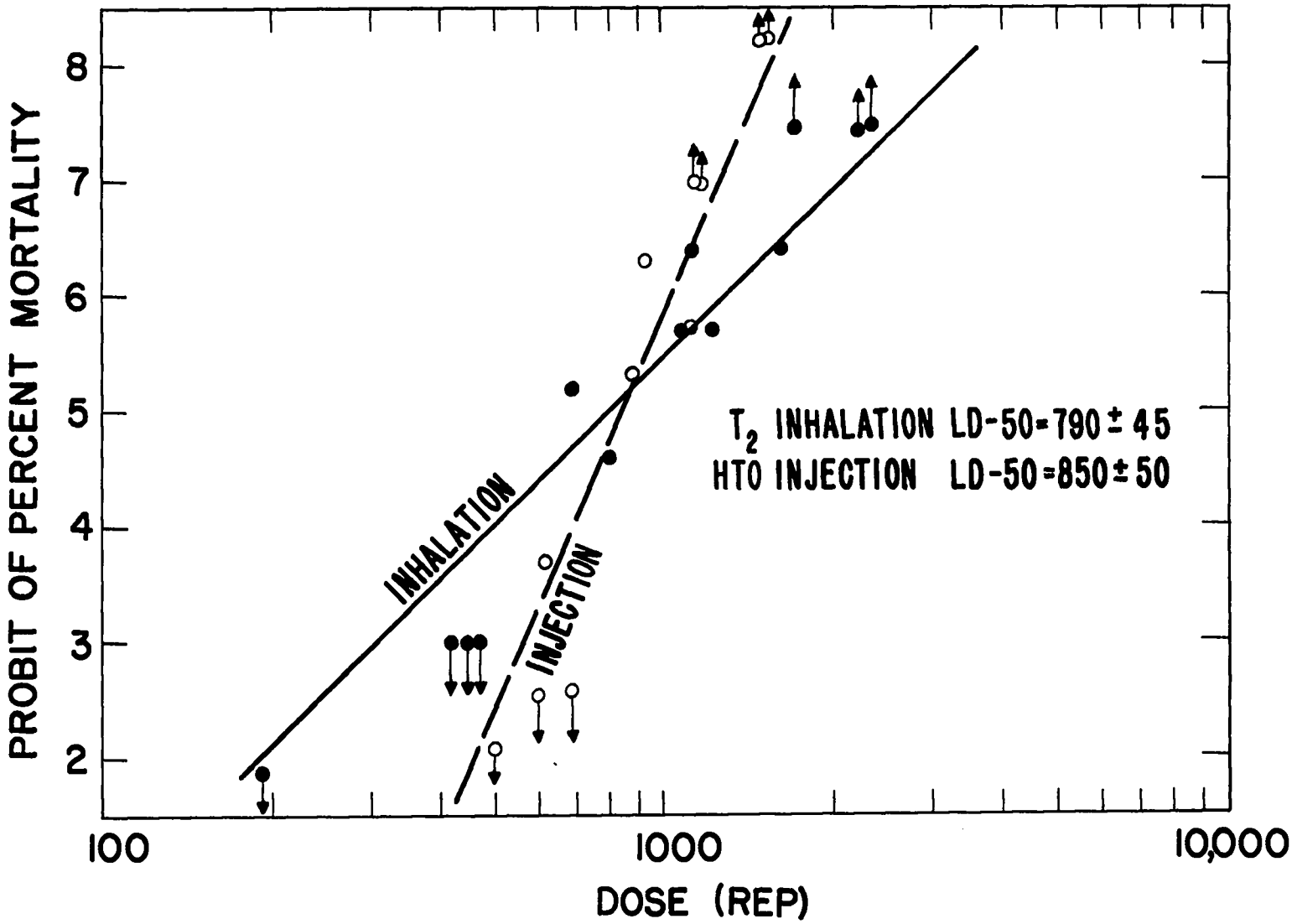

Fig. 7 Probit transformation of 30 day survival curves given in Fig. 6.

The data in Table 3 show the survival times of the mice following the two methods of exposure. These data show no obvious difference in the time sequence of deaths in the two experimental groups, which suggests that the modes of death of animals that received HTO by the inhalation of HT and those receiving HTO by intraperitoneal injection were quite similar, and in both cases due primarily to total body irradiation from the HTO in the body water.

Discussion and Conclusions

Theoretically, the inhalation of unoxidized tritium gas in relatively high concentrations should produce very high beta radiation doses to the lung surfaces. A portion of the inhaled tritium is oxidized to HTO by biological oxidation within the animal and a portion undergoes auto-oxidation by the beta particles of the tritium itself. If the acute beta radiation of the lung surfaces resulting from the inspiration of the unoxidized tritium produces a significant additional stress to the animal, the LD_{50}^{30} dose of HTO taken into the animal via inhalation of HT should be lower than that of HTO administered via intraperitoneal injection by some amount that would be a measure of the contribution of the lung damage to the death of the animal. In the present studies animals were forced to inhale an atmosphere containing

TABLE 3 SURVIVAL TIMES OF MICE EXPOSED TO THE INHALATION OF 1 PER CENT TRITIUM GAS AND MICE INJECTED INTRAPERITONEALLY WITH HTO

No. of mice	Dose (rep)	Days after exposure and number of animals dying at each dose																										Total dead
		4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	
T_2 inhalation exposures																													
12	195																												0
12	420																												0
12	450																												0
12	470																												0
12	690												2	1	1				4										8
12	800									1	1						1							1					4
12	1080									1	1	3		1		1				1									8
12	1090						1			1	1	1	2	1	1			1											9
12	1130						1		1	1	3	1	1	1	2														11
12	1220							1	2	1	2	1	1	1															9
12	1630				1			2	2	1	2	1	2																11
12	1740		1				3		3	3	1	1	X																12
12	2260		1	1		1	5	2		1			1	X															12
HTO injection exposures																													
12	210																												0
21	320																												0
21	500																												0
12	603																												0
21	615													1			1												2
12	690																												0
21	870												2	3	4	3		1											13
21	930								1	2	4	4	3	3	1			1											19
21	1110							4	1	5	2	1	1	1	1														16
21	1160						1	2	5	3	4	2	1	1						1									20
21	1190			1	1	3	1		2	3	3	1	4	2	X														21
12	1500								2	1	4	2	2	1	X														12
12	1550			1	2	2		1	4	1		1	X																12
12	1890							8	3		1	X																	12
12	1980			1	1		4	4	2	X																			12

Note: X indicates death of all animals in the exposure group.

1 per cent of unoxidized tritium for varying periods of time. Under the experimental conditions the mice fixed a lethal dose of tritium (1.5 mc./ml. of body fluids) as HTO in about 45 minutes. During the 45 minute exposure period, the calculated tritium beta radiation dose to the lung surfaces was approximately 135,000 rep. The HTO concentration of 1.5 mc./ml. of body fluids delivers a total body beta radiation dose of approximately 800 rep. The probit regression lines for per cent survival versus tritium beta radiation dose (Fig. 7) indicate that there was no significant difference in the LD_{50}^{30} dose for mice that had received HTO via inhalation of unoxidized tritium and those that received HTO via intraperitoneal injection.

The data on median survival time of animals exposed via inhalation of HT and via intraperitoneal injection of HTO (shown in Table 3) indicate also that there was no essential difference in the mode of death of the animals dying in each exposure group. Although not conclusive, the above observation at least suggests that the very high beta radiation dose delivered to the lung surfaces by the unoxidized tritium did not contribute materially to the death of the animals, and histological examination of the exposed lungs failed to reveal any gross or microscopic signs of damage.

The present studies strongly suggest, therefore, that

animals dying from inhalation of high concentrations of unoxidized tritium die primarily from total body beta radiation resulting from the absorption of HTO formed by biological and auto-oxidation of the tritium gas. The apparent absence of acute damage to the lung surfaces from the high beta radiation doses resulting from inhalation of T_2 or HT may possibly be due to absorption of the beta energy by a thin film of water present on the respiratory surfaces.

From these studies it appears that the acute hazard resulting from the inhalation of tritium gas is confined primarily to the total body beta radiation dose resulting from the tritium activity absorbed and incorporated into the body fluids as HTO.

It is not practical, however, to increase the maximum permissible air tolerance levels on the basis of the lower toxicity of T_2 compared with HTO because monitoring instruments do not differentiate between the two forms. It is necessary, therefore, to assume that in any emergency the more dangerous species is present.

In considering the acute effects of exposure to high levels of tritium known to be in the form of T_2, it may be concluded that direct lung damage can be disregarded. The possibility of delayed effects after sub-lethal exposures has not been eliminated, however, and the animals surviving

the exposures are being followed with this possibility in mind. Experiments to study the effects of chronic sub-lethal exposures would be desirable.

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