IAEA-SM-146/13
CONF-700810--6

EVALUATION OF POTENTIAL HAZARDS FROM TRITIUM WATER

MASTER

V. P. Bond

IAEA Symposium

Environmental Aspects of Nuclear Power Stations

New York City

10-14 August 1970

Brookhaven National Laboratory
Upton, L. I., N. Y.

LEGAL NOTICE

This report was prepared as an account of work sponsored by the United States Government. Neither the United States nor the United States Atomic Energy Commission, nor any of their employees, nor any of their contractors, subcontractors, or their employees, makes any warranty, express or implied, or assumes any legal liability or responsibility for the accuracy, completeness or usefulness of any information, apparatus, product or process disclosed, or represents that its use would not infringe privately owned rights.

July 1970

DISTRIBUTION OF THIS DOCUMENT IS UNLIMITED

Fig

Evaluation of Potential Hazards from Tritium Water*

V. P. Bond

Tritium is produced by reactors, as a ternary fission product and as a result of neutron interaction with coolant additives, deuterium and other reactor components. Some of the tritium is released to the environment in the form of tritiated water (HTO), from the reactor itself and from fuel reprocessing plants. Although tritium has a long half life, and is dispersed in the biosphere with relative ease, the calculated dose (dose equivalent) to the public from reactor-produced HTO is relatively quite small for the present and the next several decades (see below). Thus, although HTO and its possible effects must receive continuing detailed attention, it would appear to present no significant hazard to the public unless the calculated dose does not adequately reflect its toxicity.

The purpose of the present paper is to review the various factors that must be considered in evaluating the toxicity of tritium in the form of tritiated water (HTO). The conclusion is reached that values of dose equivalent for tritium, as calculated by NCRP-ICRP formulae (1, 2) give a conservative and adequate assessment of the potential hazard from this isotope. A given dose of radiation to an organ from the beta rays of tritium has the same radiobiological meaning as the given dose from x-rays or gamma rays (same dose rate pattern), and no significant added hazard is to be attached by virtue of the fact that the dose may have been derived from tritium.

The potential hazards of the radiation from tritium are estimated basically, as in the case of any ionizing radiation, in terms of the ab-

* IAEA Symposium, "Evaluation of Potential Hazards from Tritium Water", New York City, August 1970.

sorbed dose in rads. Maximum permissible "doses", or MPD's (more precisely, maximum permissible dose equivalents) are expressed in terms of rem, i.e., the dose in rads multiplied by a quality factor, (QF). The maximum permissible body burden (MPBB) of an isotope is the amount of activity, e.g., microcuries, that, having gained access to the body, will deliver an MPD to a given organ or organs. Maximum permissible concentrations (MPC's) of radioactive isotopes, e.g., in water or air, are expressed in units of activity per unit volume, e.g., microcuries per cubic centimeter, and are based on the amount of activity that, with a stated rate of exposure, will lead to an MPBB or MPD to a given organ or organs (1, 2).

The several factors that must be considered in determining the toxicity of HTO are discussed separately below, following which an overall evaluation is given. Estimates of dose to the public that may result from HTO released from nuclear power and reprocessing plants are also given.

The Absorbed Dose in Rads. Tritium decays by beta emission only, with a physical half life of 12.26 years. The maximum energy of the beta ray is 18 kev and its average energy is approximately 6 kev. Tritium in the environment is encountered almost entirely in the form of tritiated water. With a single exposure resulting from the breathing or ingestion of HTO, the isotope distributes rapidly and fairly uniformly throughout the body.

The absorbed dose from tritium is calculated in the same manner as the dose from any isotope, and the calculation involves basically the following formula:

$$D = kAE \quad (1)$$

Where

D = absorbed dose rate in rads per unit time

A = activity, in terms of disintegrations per minute, microcuries or other suitable units

E = average energy of the tritium beta ray, and

k = a constant.

Since the energy of the beta from tritium is among the lowest of any isotope encountered practically, the dose in rads, for a given total number of disintegrations per unit mass of tissue, is less than that from any other isotope.

The Dose Equivalent in rem; RBE and QF. For radiation protection purposes, the "dose" (technically, the "dose equivalent") of radiation is expressed in terms of rem, arrived at as follows:

$$DE = (D) (QF) \quad (2)$$

Where

DE = dose equivalent in rem

D = absorbed dose in rads

QF = quality factor, equivalent in concept to the RBE, or the relative biological effectiveness.

The QF and RBE depend upon the quality of radiation, commonly expressed in terms of linear energy transfer, or LET. Because the beta rays from tritium are of relatively low energy, they might be thought of as roughly equivalent to the "end of the tracks" of beta rays of higher energy, and the possibility exists that the RBE for the tritium beta rays might thus be greater than one (3, 4). Some experimental evidence has appeared to be consistent with an RBE (and QF) in excess of 1.0 and a AF of 1.7 was used for many years for radiation protection purposes (1, 2). Precise RBE values for tritium beta radiation are difficult to determine, particularly

in the mammal. Recently the pertinent biological material has been reviewed (5-10), and there appears to be little convincing evidence that the RBE is significantly different from unity. The ICRP has recently revised its evaluation, and is in the process of recommending that a QF of 1.0, rather than 1.7, be used (11). Thus there appears to be little or no basis for ascribing higher toxicity to tritium on the basis of radiation quality and RBE.

Body Distribution and Excretion of HTO. Following the exposure to HTO through either breathing or ingestion, the isotope distributes rapidly, essentially as body water, i.e., almost uniformly throughout the body. It is excreted rapidly, and the biological or effective half time for excretion in man of approximately 12 days (1, 2) is a major determining factor with respect to the amount of radiation exposure received.

Tritium in vivo is exchangeable with hydrogen, and can in principle replace hydrogen on any biochemical compound (5, 12, 13). Also, in metabolic processes, tritium from tritiated water can be incorporated into nonexchangeable position in biological molecules including carbohydrates, proteins, RNA and DNA (5-8, 12-18). These facts were well known when current exposure limits for H^3 were established in 1959 (1, 2), and pertinent references dating back to 1951 were listed (1). With a single exposure the amounts metabolically incorporated into organic compounds may be less than 2% (5, 12). With long-term exposure under extreme conditions in which the organism receives essentially all of its water intake from sources with a given concentration of tritium, organically bound tritium in tissue solids will range from 1% to 2% up to as much as 35% of the concentration maintained in the intake water (14, 16).

Thus the rate of excretion of tritium will depend upon the duration of exposure. The excretion pattern may be regarded as the sum of exponentials (5, 14, 19, 20). Data in man indicate that the excretion rate may be represented as three exponentials, with half times of disappearance of approximately 6, 23 and 345 days (19). Thus the half time of disappearance of 12 days used by the ICRP and NCRP (1, 2) may represent an "effective" rate of excretion.

Possible Concentration of Tritium in Organs. Certain isotopes are known to be selectively retained and thus become relatively concentrated in certain body tissues, e.g., the thyroid has an affinity for iodine. By this process, the concentration of iodine in the thyroid can be many times greater than the concentration of iodine in other organs. Other examples of such selective retention and concentration are well-known, and are taken into account in the calculation of absorbed dose to body organs, in the calculation of maximum permissible concentration (MPC's) of radioactive species in air and water (1, 2), and in the calculation of dose to individuals in specific situations.

Such a concentration phenomenon for tritium, if it occurs at all in the mammal, would be expected to be seen with single or repeated exposures to tritium. Animal studies show the reverse (5, 6, 14-18). In one experiment (5, 15), in mice given tritiated water over a five month period, the concentration of tritium in tissues ranged from approximately 0.1 to 10% (eleven tissues) of that in the body water.

Apparent exceptions have occurred in which the concentration of tritium in some organs in mammals in the wild state have appeared to be higher than that in the plasma (21, 22). However, the previous history of these animals could not be documented, and the animals did have access

to sources of tritium at higher concentrations than those in their plasma water. Under these circumstances the data do not permit one to conclude that a true concentration factor is involved. The apparent tissue concentration is most probably derived from the fact that animals had been ingesting tritium from high concentration sources, and then had changed to lower concentration sources in the environment before being sampled. At any rate, the apparent concentration factor did not exceed approximately 1.4.

The possibility of some concentration of tritium under unusual circumstances cannot be dismissed, however. Although most chemical reactions discriminate against the incorporation of deuterium and tritium in favor of hydrogen (5), it is conceivable that, for some biochemical reactions, the discrimination against tritium "leaving" a biochemical compound once it is incorporated might be greater than that for its "entering" the compound. This could in theory lead to a trapping or concentration factor. There are data indicating a concentration of deuterium in stable biochemical compounds such as honey and beeswax (23,25).

Even if such examples do represent a true trapping phenomenon, it is of no significance practically for the following reasons: Animal experiments involving chronic exposure to tritium under controlled conditions, such as those referred to above (5-7, 14-16, 18), show a marked reverse phenomenon -- the concentration of tritium in body tissues is clearly much lower than that in the source of water. Also, man obviously does not receive all of his food and drink from sources having a uniformly high tritium concentration, and thus a large dilution factor results.

Thus at present, under practical conditions with respect to man, a positive concentration factor does not appear to exist, and a sizable fractional concentration factor applies.

Possible Concentration of Tritium in the Food Chain. Although of little relevance to a short-term (months to years) exposure of man to tritium in the environment, the possibility of concentration should be considered under long-term (many decades) conditions in which added tritium might begin to approach equilibrium in the entire biosphere. Since a trapping or concentration phenomenon in body organs was shown above to be of no practical significance in the mammal, concentration via the food chain would likewise be expected to be of no significance. The worst situation that can be visualized would be if all of the "building blocks" for biochemical molecules were obtained via the diet. Then the newly-formed molecules such as protein, RNA and DNA could have tritium specific activity approaching that in the dietary material. However, higher organisms, including man, also synthesize their own building blocks, e.g., purine and pyrimidine nucleotides, and are not dependent on preformed nucleotides derived from food. A large percentage of nucleic acid precursors given orally are broken down before absorption (8, 26). From these considerations the tritium specific activity in the biochemical molecules in people would approach that in the food they ingest under extreme condition only, and the expectation is that it would be far less under any practical conditions.

Some data indicate that transfer of HTO may be more efficient via one food chain route than another (27). Additional studies on the rates of movement of HTO in food chains are necessary.

HTO in DNA; Possible Additional Effect From. A possible additional effect of tritium disintegration, by virtue of the fact that the tritium may have been incorporated into organic molecules such as DNA, must be considered (28,30). When tritium is disintegrated it becomes helium-3,

which leaves an organic molecule that is positively charged. Some of these reactive organic ions may cause molecular rearrangement to occur, similar to the ions and radicals formed by ionizing radiation. Quantitatively, however, any biological effect is expected to be small in comparison to that caused by the track of the beta ray which yields several hundred ions and radicals. Thus the effect, if it occurs, would be expected to be small.

Possible additional effects of H^3 in DNA cannot be measured in biological systems using tritiated water, because of the small amount of incorporation of tritium into organic molecules (most is in water). However, the possibility of such an effect can be studied under artificial laboratory conditions, which would not be encountered practically in the environment. It is possible to place tritium selectively in various positions in a number of organic molecules, which are incorporated selectively and in a known manner into macromolecules such as DNA and RNA. Examples are tritiated thymidine, which goes exclusively to DNA in the cell nucleus, and tritiated cytidine which is incorporated into both DNA and RNA. A number of other examples could be cited. Because the compounds go selectively into macromolecules, there is a concentration of tritium in the cell nucleus. However, this of course bears no relationship to the situation in which tritium is ingested as tritiated water, as is the case with tritium in the environment.

By means of such selective localization of tritium in specific macromolecules, experiments can be done to determine if there are conditions in which an effect over and above that expected from the absorbed dose from the tritium beta rays can be detected. Such an effect has been looked for extensively with H^3 TDR in mammalian biological systems

(6-8), and over fifty pertinent references were analyzed in detail.

No additional effect was found.

To illustrate the problem more definitively, an example of the type of experiment that is critical is outlined: tritium as tritiated water is introduced in a single application into a biological system. It is thus distributed essentially uniformly throughout the cell and its nucleus and delivers a uniform radiation dose from the beta rays of the tritium. Very rarely are tritium atoms incorporated into DNA or RNA under these circumstances. In a parallel experiment, tritium is given as tritiated thymidine that is incorporated only into DNA in the cell nucleus. Conditions are adjusted so that the cell nucleus receives the same radiation dose in rads in both cases, the only difference being that in the one case the tritium is incorporated into DNA and not in the other. All experiments of this type to date, on somatic and genetic effects in mammals, showed approximately the same degree of biological effect whether or not the tritium was incorporated into DNA, i.e., the tritium showed no significant additional effect by virtue of its having been incorporated into DNA (5-8). This might be expected, since the range of the tritium-beta track is very large compared to the width of a DNA molecule, and most of the energy would be expended outside of the molecule.

Some experiments, however, have indicated that tritium on one specific location in the pyrimidine ring may produce an effect in lower organisms that is in excess of that anticipated from the radiation dose from tritium. These experiments were done with bacteria and with drosophila (31-33). Tritium deliberately placed in the five position in the

pyrimidine ring did show an effect in excess of that expected from the radiation dose alone. A similar effect was looked for with various other tritiated compounds, in which the tritium was located either in a different position on the pyrimidine ring, or in other precursors of DNA and RNA (31-33). No such effect has been found, i.e., the degree of effect was entirely that expected from the beta radiation of the tritium.

It is difficult to estimate the degree to which such an effect might increase the mutation rate over that expected from the beta radiation. One calculation indicates that the maximum increase might be of the order of 25 percent (34). Further work must be done, and it must be determined if an additional effect of H^3 in the five position in the pyrimidine ring can be detected in the mammal.

The demonstration of an excess effect in lower organisms by virtue of the location of the tritium on the five position in the pyrimidine ring is of considerable scientific interest, but is of little practical import with respect to the toxicity of tritium encountered in the environment (5-8, 34). First, it is emphasized that the effect is seen at all only under highly specialized laboratory conditions in which great care is taken to insure that the tritium is incorporated, in large concentrations, in a specific location on a specific ring in a specific compound. Under conditions in which tritium is encountered in the environment, only a small percentage is incorporated specifically into such organic compounds under the "worst" conditions of equilibrium of the organism with the source of tritium in the environment. Of the small percent incorporated into all organic compounds, only a small

percent of this can be incorporated into the five position on the pyrimidine ring. The overall conclusion is that under practical conditions in which tritium is ingested or inhaled as tritium water, the effect noted above would not add measurably to the effect of the tritium beta rays. That is to say, the degree of effect is to be predicted on the basis of the absorbed dose in rads (or the dose equivalent in rem), and there appears to be no reason for expecting significant additional effects on the basis that some of the tritium may be incorporated into DNA or other biochemical molecules.

Mass Isotope Effect: Possible Toxicity From. Because tritium is heavier than ordinary hydrogen, the speed of reactions involving the exchange of tritium on a molecule will be different than for hydrogen. If large amounts of tritium relative to hydrogen are involved, biological consequences conceivably could result. It is known that deuterium, another heavy isotope of hydrogen, will lead to toxicity on this basis. However, very high concentrations of deuterium must be obtained (a large fraction of all hydrogen atoms must be replaced by deuterium) before such an effect is seen (12).

In the case of tritium, such a possible effect is of no practical importance. Not only is the number of tritium molecules available in practice much too small, but in addition biological effects from radiation toxicity are encountered long before the necessary high concentrations are reached. Thus this potential factor can be dismissed as having no importance in biological systems.

Effects of Long-Turnover Compartments on Dose Calculations. As noted above, tritium may enter a number of body compartments with different

times of excretion. These compartments in man may have half times of disappearance of approximately 6, 23 and 345 days (19). The question arises as to whether the half disappearance time of 12 days, adopted by the NCRP and ICRP (1, 2) is adequate, and whether the presence of compartments with very long turnover times should be taken into account additionally.

Only the long-term chronic ingestion situation in man is pertinent to the present discussion. As shown above, with long-term exposure to tritiated water, the concentration of tritium in body water or in body tissues will not exceed that maintained in the intake food or water. Further, it was shown above that the biological effect depends principally on the total concentration of tritium in the cells and cell nuclei, and the degree of effect is not dependent on whether the tritium is bound to water, or to other biochemical compounds including DNA. On this basis the presence of tritium in body compartments with a long-turnover time would have no significant influence on the calculated dose or on the degree of postulated biological effect.

The above question has been looked at in some detail (35), and it was asked if the assumption of the distribution of tritium as body water seriously affects the estimate of absorbed radiation dose from this isotope. The conclusion was reached that the assumption of distribution with body water is conservative for acute cases in which the time of tritium exposure is known, and is also conservative for chronic assimilations of tritium.

Conservative Nature of ICRP Limits for Tritium. The NCRP-ICRP have

established 1000 μCi as the maximum permissible body burden of tritium for occupational exposure with body tissue as the critical organ (1, 2). A value of 2000 μCi is also listed with the whole body as the critical organ. Since these figures have been widely misinterpreted, it may be well to explain how they were obtained.

If tritium is uniformly distributed throughout the body so that the energy released is absorbed by the entire 70 kg mass of a "standard man", 1960 μCi (about 2000 μCi) of tritium will give a dose rate of 100 mrem/week corresponding to the maximum permissible occupational exposure of 5000 mrem/year. Since water and body tissues are intimately related this situation will apply approximately in practice either to the equilibrium case where all tissues have become labeled with tritium or to an acute exposure situation where most of the tritium is in the aqueous fraction of the body (body water). In the equilibrium case the dose will vary slightly from tissue to tissue depending on the hydrogen content, but this variation is small. When the tritium is confined to the body water, dose will vary with the water content of tissue and allowance has been made for this as explained in the next paragraph.

In setting the 1000 μCi limit when body tissue is the critical organ, the ICRP has taken note of the fact that the water content of tissues varies, with some tissues containing up to 80% water. This makes no significant difference for the equilibrium case since the hydrogen content of tissue and water are similar; but for the acute case where tritium is mostly in the body water, an organ with more water will receive a higher dose. To be conservative a hypothetical organ consisting entirely of water was considered. Such an organ would receive the same dose rate as the 43 kg of

body water undiluted by tissue. Computation shows that 1200 μCi will result in 100 mrem/week for this limiting case. This was rounded off conservatively to 1000 μCi and used as the maximum permissible body burden. The 2000 μCi figure is published for use when considering the equilibrium situation or when combining dosage components to the whole body from more than one radionuclide. It should be noted that choice of 1000 μCi as the maximum permissible body burden does not imply a belief that tritium is confined to body water. Rather it results from a conservative approach aimed at keeping the dose rate to any tissue, even for a limited period of time, below 100 mrem/week.

The 1000 μCi maximum permissible body burden recommended by the ICRP and used by AEC facilities is quite conservative. For long-term exposures the exposure rate to the whole body will be only 50 mrem/week, i.e., one half of the maximum permissible value. Since an RBE of 1.7 has previously been used in the tritium computations, the most recent ICRP conclusion that an RBE of 1.0 is correct means that the dose rate is more properly deduced as 29 mrem/week or only 29% of maximum permissible. For short-term exposure, if one uses 80% instead of 100% as the water content of the critical tissue and an RBE of 1, one obtains a dose rate to this tissue of 39 mrem/week or 39% of maximum permissible. Here too, 1000 μCi is conservative.

It should be noted that the ICRP has used the combined intake of water in fluids (1200 ml) and in food (1000 ml), and a maximum permissible body burden of 1000 μCi , in computing the maximum permissible value for water. Thus if intake is entirely in water, the equilibrium dose rate to the body will be $50 \times 12/22 = 27$ mrem/week (15 mrem/week for $\text{RBE} = 1$) or

only about 1/4 of maximum permissible. For the acute case, for a tissue with 80% water, and using an RBE = 1 the rate will be $39 \times 12/22 = 21$ mrem/week. Stated differently, the water limits in addition, to being based on conservative values of maximum permissible body burden, include an allowance for tritium in food. Under conditions in which one receives tritium essentially entirely from chronic ingestion of tritiated water, NCRP-ICRP calculations of the dose equivalent from MPC values for water lead to values that are conservative by a factor of between 5 and 8.

It is a general principal of internal dosimetry that all pathways contributing to the body burden must be counted. Thus the combined effect of tritium activity in air, water and non-aqueous components of food must be evaluated. Considering the conservative nature of the water limits and the substantial allowance for tritium in the water content of food, a separate limit for tritium in food is required only under very special circumstances.

Although the above analysis has been directed towards occupational exposure, the same conclusions apply to population exposure since the latter limits are established as specific fractions of the occupational values.

HTO Turnover in the Embryo; Possible Effects on the Fetus. Possible effects of tritiated water on the embryo have been studied under conditions in which very large amounts of tritiated water were administered to female rats during the 22 day gestation period, and the new born were examined with respect to tritium concentrations and biological effects (18). Concentrations of 1, 10, 50 and 100 $\mu\text{C}/\text{ml}$ of water resulted in total doses

to the embryos of approximately 8, 60, 300 and 600 rads, respectively. No significant effects were observed at the 1 $\mu\text{C}/\text{ml}$ level. At the 10 $\mu\text{C}/\text{ml}$ level significant differences were reported for the weight of the male testes, the kidney and the brain. Results were expressed in terms of percent body weight. If results are expressed in terms of absolute weights of organs, differences in any organ weight except that of the testes at doses below approximately 50 $\mu\text{C}/\text{ml}$ (300 rads) are of questionable significance.

The above results are consistent with those obtained by others with external x- or gamma radiation, namely that doses in the range of tens of rads and above to the fetus can produce observable changes of this type in the new born (37). Thus radiation from tritium appears to act as expected on the basis of absorbed dose, and there is no reason to postulate any unusual effect by virtue of the fact that radiation was derived from tritium. Also, it should be pointed out that effects such as differences in brain weight undoubtedly result from damage to a large number of cells. On this basis the dose-effect curve for such a result would be expected to be markedly curvilinear, i.e. the effect per rad would fall off very rapidly as the dose is reduced. On this basis, one would not expect to find changes of this nature even in very large populations exposed to doses of the order of millirads or a few rads.

Additional laboratory studies have been done (38), in which tritiated thymidine was given to female rats throughout pregnancy. Under these conditions the tritium is located essentially entirely in DNA, and the nucleus of every cell of the newborn is labeled as determined by autoradiography. Large doses of tritiated thymine were used; extensive

studies were done at the level of 1.6 μ c of tritiated thymidine per gram of body weight. Even under these extreme conditions, i.e., high dose, essentially all tritium in DNA, there was no increase in the incidence of abnormal off-spring over that in controls. It was concluded that while some effect could not be excluded, it was small indeed if present. No increase in the number of chromosome abnormalities in the cells of the newborn was found.

HTO in Embryo; Possible Genetic Effects. It has been pointed out that tritium incorporated into the female fetus might be somewhat more hazardous in terms of possible genetic effects in the germ cells of the adult developing from the fetus exposed in utero (29,30). All tissues in the growing embryo are developing, and all cells within these tissues undergo division and synthesize DNA. In many tissues such as the bone marrow, the skin and the epithelium of the bowel, the cells continue to divide after the fetus is born, and throughout adult life. In some tissues however, such as the brain, muscle, the liver, and the ovary, the cells cease to divide at birth or during early development. It is thus possible that in these tissues some tritium might become "fixed" in compounds such as DNA during embryogenesis, and would remain there essentially throughout adult life. That this probably has little significance beyond that expected from the dose or dose equivalent to the oocytes received from tritium or any other radiation is shown below. Both the male and female fetus are considered.

With respect to the male fetus, the germ cells of the testes divide during embryogenesis and continue to divide throughout the adult life of the individual. In the adult an approximate steady state cellular equilibrium

exists, i.e., for every new germ cell produced in the gonads, on the average one cell (and any tritium in that cell) is eliminated from the body. New tritium would be incorporated into newly-formed cells only under conditions of new or continuous exposure to tritium. The most important point, however, is that, as shown above, the potential effect depends on the total dose of radiation from tritium in the cells and cell nuclei, and that it makes little difference whether the tritium be in the form of water or in organic compounds including DNA.

With respect to the oocytes in the female, the situation is similar in many respects. Of the total amount of tritium that might be present in the oocytes or in any other cell, only a very small fraction would be in DNA. A very large percentage of any dose that might be received from tritium would come from tritium in water and in compounds other than DNA. Again, as shown above, the effects are dependent on the total amount of tritium in the cell and cell nucleus, and essentially independent of the relative amounts that may be in any particular biochemical compartment, including DNA. In addition, it has been shown (39) that the apparent degree of recovery from genetic damage from ionizing radiation in the female mouse is enormous and that recovery may be essentially complete, i.e., a threshold situation may pertain for all practical purposes. On these bases, there would appear to be no reason at present to regard tritium in the female embryo as posing an unusual hazard. In other words, if a given dose or dose equivalent is received by the oocytes, either from the beta rays of tritium, from external x-rays or from the gamma rays from any isotope, the degree of potential effect is the same and there appears

to be no reason at present for postulating a significantly greater degree of effect by virtue of the fact that the radiation dose may have been received from tritium,

Dose to the Population from Reactor-produced Tritium. Since it has been shown above that the degree of potential hazard from tritium radiation is adequately and conservatively represented by the calculated dose in millirem, it is appropriate to consider estimates of the dose of radiation that the population may receive from tritium released by power reactors. Since presumably large populations, or the entire population of a country or the world may be exposed, possible genetic effects in the population would appear to be the potential effect of most importance. With respect to genetic effects, it is the average dose to the population that is of most significance, i.e., it makes little difference with respect to genetic effects whether one individual receives 100 units of radiation exposure, or 100 individuals receive 1 unit of radiation exposure.

The probable dose to the population from tritium has been calculated for the year 2000 (40, 41) assuming that power reactors will continue to be installed and operated at the current rate. The average dose to the population from tritium is estimated to be a small fraction of a millirem per year, 0.001 millirems to be precise (41). This value compares with: current exposure to naturally and weapons-testing produced tritium of the order of 0.1 mrem/year (42,43); an estimated 1-2 millirem that may be received in crossing the United States in a jet; the several millirem additional that may be received in going from the shore to spend a few days at a skiing retreat in the mountains; the approximately 100 or more millirem that one may receive per year by virtue of living in a concrete

versus a wooden structure; and the average of some 120 millirem from natural background radiation that the population of the United States receives on the average per year. Thus exposure of the population to tritium from power reactors does not represent a significant additional exposure to the population, additionally when it is kept in mind that the estimates for tritium exposure (41) were made on the basis of formulae provided by the ICRP which were shown above to be conservative by a sizeable factor.

With respect to possible exposure to tritium of local populations near a factor located on a body of water, a specific situation has been considered (44). The dose rate to the local population, if the sole source of drinking water were derived from this source, is estimated to be 0.003 millirads per year. As indicated above, dose rates of this order of magnitude do not represent a significant additional exposure to even very large populations.

Summary and Conclusions. The possible biological effects of tritium have been analyzed, and several factors that must go into an evaluation have been considered both from the theoretical and experimental standpoints. Maximum permissible concentrations or maximum permissible doses of tritium are based principally on the absorbed radiation dose delivered by the tritium beta rays. Several possible factors that might possibly increase the degree of toxicity over that expected from the absorbed dose are considered in some detail. These include the possibility of concentration of tritium, and the possibility of concentration of tritium, and the possibility of additional toxicity by virtue of the fact that some tritium may be

incorporated into biological molecules such as DNA. The conclusion is reached that these factors do not significantly increase the dose that might be expected from a given concentration of tritium in the environment, or the effects of that dose. A given dose (e.g., an MPD) of radiation from the beta rays of tritium (from either inhalation or ingestion) has the same radiobiological and radiation protection meaning as the given dose from x-rays or gamma rays (same dose rate pattern), and no added significance or potential hazard is to be attached by virtue of the fact that the dose may have been derived from tritium. Calculated population exposure from reactor-produced HTO is very small, compared to that from natural and weapons-testing produced HTO, and from other sources of radiation to which the population is exposed.

Note: The author is indebted to F. Cowan, A. Hull, and C. Meinhold for helpful discussions during the preparation of this material.

REFERENCES

1. Report of ICRP Committee II on Permissible Dose for Internal Radiation, Health Phys. 3 (1960) 1-380.
2. NCRP report, Handbook 69, Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and in Water for Occupational Exposure, U. S. Department of Commerce, U. S. Gov't. Printing Office (1959).
3. HARDER, D. Spectra of Primary and Secondary Electrons in Material Irradiated by Fast Electrons. In Biophysical Aspects of Radiation Quality, IAEA Publ. Tech. Report Series 58 (1966).
4. ZERMENO, A. and COLE, A. Radiosensitive Structure of Metaphase and Interphase Hamster Cells as Studied by Low-Voltage Electron Beam Irradiation, Rad. Res. 39 (1969) 669-684.
5. FEINENDEGEN, L. E. Tritium-labeled Molecules in Biology and Medicine, Acad. Press, New York and London (1967).
6. BOND, V. P. and FEINENDEGEN, L. E. Intranuclear ^3H Thymidine: Dosimetric Radiological and Radiation Protection Aspects, Hlth. Phys. 12 (1966) 1007-1020.
7. VENMART, J. Radiotoxicity of Tritium and ^{14}C Compounds, Hlth. Phys. 16 (1969) 429-440.
8. ROBISON, W. L. and ANSPAUGH, L. R. Biological Hazards from Project Rulison, Livermore, Calif. Draft Report.
9. SHTUKKENBERG, Y. M. Application of Tritium in Biological Research, U. N. Scientific Committee on Effects of Atomic Radiation, A/AC, 82/G, L. 552 (1960).
10. LAMBERT, B. E. Cytological Damage Produced in the Mouse Testes by Tritiated Thymidine, Tritiated Water and X-rays, Hlth. Phys. 17 (1969) 547.
11. SNYDER, W. S. Personal Communication.
12. SIRI, W. F. and EVER, J. Tritium Exchange in Biological Systems, in Tritium in Biological Systems, Symp. IAEA 2 (1961) 71-84.
13. RITTENBERG, D. The Effect of Isotopic Substitution in the Living Cell, J. of Chem. Phys., 60 (1963) 318-322.
14. THOMPSON, R. C. and BALLOU, J. E. Studies of Metabolic Turnover with Tritium as a Tracer, V., J. of Biology and Chem. 223 (1956) 795-809.
15. PINSON, E. and LANGHAM, W. Physiology and Toxicology of Tritium in Man, J. Appl. Physiol. 10 (1957) 108-126.
16. KHAN, A. A. and WILSON, J. E. Studies of Turnover in Mammalian Subcellular Particles: Brain Nuclei, Mitochondria and Microsomes, Neurochem. 12 (1965) 81-86, Perg. Press, N. Ireland.

17. METZGER, H. P. The Effect of Unilateral Visual Stimulation on Synthesis of Cortical Proteins in Each Hemisphere of the Split-Brain Monkey, *J. Neurochem.* 14 (1967) 183-187.
18. CAHILL, D. F. and YUILE, C. L. Some Effects of Tritiated Water on Mammalian Fetal Development, Rochester, New York, Draft Report.
19. REINIG, W. C. and SANDERS, S. M. (Jr.) Assessment of Tritium in Man, Presented at Symp. on Diagnosis and Treatment of Deposited Radionuclides, May 15-17, 1967, Richland, Washington, for Publication in Symp. Proceed. by Excerpta Medica.
20. SNYDER, W. S. Urinary Excretion of Tritium following Exposure of Man to HTO -- a Two Exponential Model, *Phys. Med. Biol.*, Vol. 13, No. 4 (1968) 547-559.
21. SHORE, B. The Fate and Importance of Radionuclides Produced in Plowshare Events (preprint). Prepared for submittal to the U. S. Public Health Symp., Las Vegas, Nevada, April 7-11, 1969.
22. EVANS, A. G. New Dose Estimates from Chronic Tritium Exposures, *Health Phys.* 16 (1969) 57-63, Pergamon Press, N. Ireland.
23. BOWEN, J. J. M. Trace Elements in Biochemistry. Biologic Fractionation of Isotopes, Academic Press, London (1966) 137-139.
24. HELVEY, T. C. The National Concentration of Deuterium in Honey, *Science* 117 (1953) 276-277.
25. KRAICHEVSKY, M. I., FRIEDMAN, I., NEWALL, M. F., and SISLAR, F. D. Deuterium Fractionation during Molecular Hydrogen Formation in a Marine Pseudomonad. *J. Biol. Chem.* 236 (1961) 2520.
26. WADE, L. (Jr.) and SHAW, E. I. Preliminary Estimate of the Hazard from Ingestion of Tritiated Thymidine, Lawrence, Kansas, Draft Report.
27. KIRCHMAN, R. and VAN DEN HOCK, J. Transfer et Incorporation du tritium dans les Constituants de l'Herbe et du lait, en Conditions Naturelles, International Radiation Protection Association meeting, 3-8 May 1970, Brighton, England.
28. HUVER, C. W. Biological Effects of Tritium, Presented to Minnesota Pollution Control Agency, February 19, 1969.
29. RADFORD, E. P. Testimony before the Subcommittee on Air and Water Pollution of the Committee on Public Works, November 19, 1969, Washington, D.C. S3042, 96-183.

30. RADFORD, E. P., MERZ, T., RAY, C., HERRIOT, R. M., KRUSE, C. W., SOUTHWICK, C. H. AND SLADEN, B. K. Statement of Concern, Environment, 11 (1969) 18-27.
31. PERSON, S. Lethal and Mutagenic Effects of Tritium Decay Produced by Tritiated Compounds Incorporated into Bacteria and Bacteriophages. In, Symp. Report, Biological Effects of Transmutation and Decay of Incorporated Radioisotopes, IAEA, Vienna, (1968).
32. FUNK, F., PERSON, S. Cytosine to Thymine Transitions from Decay of Cytosine-5-³H in Bacteriophage S 13, Science 166 (1969) 1629-1631.
33. KIEFT, P. Induction of Recessive Lethals by ³H Uridine and ³H Thymidine in Drosophila. In, Symp. Report, Biological Effects of Transmutation and Decay of Incorporated Radioisotopes, I.A.E.A., Vienna (1968).
34. PERSON, S. Personal Communication.
35. SANDERS, S. M. (Jr.) Dose Estimates from Chronic and Acute Tritium Exposures, Draft Report.
36. CAHILL, D. F. Personal Communication.
37. United Nations Scientific Committee on the Effects of Atomic Radiation, Report of General Assembly, 17th Session, Suppl. 16, A/5216 (1962).
38. FLIEDNER, T. M., HAAS, R. J., STEHLE, H. AND ADAMS, A. Complete Labeling of all Cell Nuclei in Newborn Rats with ³H-Thymidine, Lab. Investigation 18 (1968) 249-259.
39. RUSSELL, W. L. Factors that Affect the Radiation Induction of Mutations in the Mouse, Annals of the Brazilian Acad. of Sciences 39 (1967) 65-75.
40. PETERSON, H. T. (Jr.) Environmental Tritium Contamination from Increasing Utilization of Nuclear Energy Sources, Presented at Seminar on Agricultural and Public Health Aspects of Environmental Contamination by Radioactive Materials, Vienna, Austria March 24-28, 1969.
41. JACOBS, D. G. Sources of Tritium and its Behavior Upon Release to the Environment. In, Hearings of the Joint Committee on Atomic Energy, Part I, October 28 - November 7, 1969, 500.
42. THOMPSON, T. J. Testimony on "Environmental Effects of Producing Electrical Power". Joint Committee on Atomic Energy, October 30, 1969.
43. MOGHISSI, A. A. and LIEBERMAN, R. Tritium Body Burden of Children, 1967-68. Radiol. Health Data and Reports 11 (1970) 227-231.

44. AEC Comments on Tritium Releases from the Proposed Calvert Cliffs Nuclear Power Plant. In, Selected Materials on Environmental Effects of Producing Electric Power, Joint Committee on Atomic Energy, 91st Congress (1969) 507.