Suggested Reduction of Permissible Exposure to Plutonium and Other Transuranium Elements

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The historical development of the value of maximum permissible body burden of ^{239}Pu is presented and present considerations for the revision of this standard are given. Some evidence is presented that the linear hypothesis may not be sufficiently conservative at low dose rates and especially for the actinide elements. Until certain questions are answered about the particle problem, it will not be possible to set a satisfactory maximum permissible body burden for ^{239}Pu based on lung as the critical organ, but in the meantime some studies suggest that the present maximum permissible body burden based on bone should be reduced at least by a factor of 200.

Introduction

PERHAPS THERE HAS NEVER BEfore been an enterprise that was planned so carefully for its safety and never before a risk that has been so thoroughly studied and guarded against as has been the case with the nuclear energy industry and its concern to avoid unnecessary exposure to ionizing radiation. It is ironical that in part because of this concern and in spite of the fact that we now probably know far more about the effects of this radiation on man than about any of the other common hazards, exposure to the radiations associated with nuclear energy seem to frighten and engender fear that is all out of proportion in comparison with the everyday risks from such things as medical x-ray, food additives, and environmental pollutants from the burning of fossil fuels. However, on second thought this public concern for radiation exposure probably should not be surprising because, except for unusual precautionary measures and constant vigilance, there likely some day will be a major accident with very serious consequences. Even though most of the public may be convinced of a very low probability of such a serious accident, we are reminded frequently in our newspapers of what could happen from accidental release into the public domain of large quantities of radioactive material from nuclear power plants, from spent fuel operations, or from shipping accidents.

A considerable portion of the credit for the remarkable safety record of the nuclear energy industry as one of the safest of all modern industries must be given to the untiring efforts of members of the health physics profession with whom I have been associated for over 30 years, and which profession I have seen grow from a group of 5 health physicists at the University of Chicago in 1943 to a worldwide organization today of over 10,000 professionals. Our lot as a growing profession of health physicists has been a most interesting and challenging one but it has not always been easy, because there were times when some of my associates were demoted or lost their jobs because they refused to yield to pressures to lower our standards or compromise for unsafe conditions.

We were constantly resisting pressures of engineers and production supervisors to relax what they called our ridiculous conservatism. Sometimes we were forced to set exposure limits that were lower than our management wanted and perforce they were often little better than guesses because in some areas we had almost no experience or supporting experimental data. For example, one of the earliest papers[1] showing how to

calculate dose from internally deposited radionuclides and giving values of permissible body burden and permissible concentration of some 20 radionuclides was delayed for almost a year when I presented it for publication in 1945 because some of the permissible occupational exposure values I calculated were much lower than those in use in weapons production operations. I had at that time almost no metabolic data for some of these radionuclides. For the most part I had to rely on a series of publication by J. G. Hamilton et al.[2] on the metabolism of fission products, plutonium, and other actinide elements in mice and rats and in a few cases data on only 3 or 4 rats were available. The maximum permissible internal dose rates for occupational exposure that I used in making these early calculations were 36 R/y for β and γ radiation and 3.6 rep/y ($\sim$3 rad/y) for α radiation. On this basis and using available metabolic data the value I obtained for ^{239}Pu for maximum permissible lung burden of the occupational worker was 0.035 μCi and for bone burden was 0.42 μCi. The standard man data I used were based on typical human values collected and summarized for me by M. J .Cook.[3]

The first semiofficial values for body burden of the radionuclides were developed at the Chalk River Canada Conference[4] in 1949. These values were later reviewed at the Harwell, England Conference in 1950. From about 1950 to 1973, I was chairman of the Internal Dose Committees of both the International Commission on Radiological Protection (ICRP) and of the National Council on Radiation Protection (NCRP) and so must assume some of the blame for shortcomings of our Handbooks on Internal Dose. During this period there were four principal publications of our Internal Dose Handbooks giving values of organ burden (qf_2) and body burden (q) and maximum permissible concentrations in air $(MPC)_a$

TABLE I
Maximum Permissible Body Burdens for ^{239}PU

Source of Value	Occupational qf_2 (μc)	Occupational q (μc)	For Population at Large qf_2 (μc)	For Population at Large q (μc)
Early Oak Ridge Nat. Lab. (KZM-1947)[1]	0.42 B 0.035 L	0.70 B 0.12 L	—— ——	—— ——
Chalk River Conference 1949[4]	—— ——	0.006 B ——	—— ——	0.00006 B ——
Early Los Alamos Nat. Lab. (WHL-1938)[4]	—— ——	0.063**B ——	—— ——	—— ——
NCRP—Handbook 52 (1953)[5]	0.03 B 0.008 L	0.04 B 0.008 L	(0.003) B (0.0008)*L	(0.004) *B (0.0008)*L
ICRP—Br. J. Radiol. Supp. 6 (1954)[6]	0.03 B 0.02 L	0.04 B 0.02 L	—— ——	—— ——
NCRP—Handbook 69 (1959)[7]	—— ——	0.04 B ——	—— ——	(0.004) *B ——
ICRP—Handbook 2 (1959)[8]	0.036 B ——	0.04 B ——	—— ——	—— ——

B—value based on dose to bone; L—value based on dose to lung; *—values in parentheses are based on suggested safety factor of 10; q—μc in total body based on indicated organ; qf_2—μc in indicated organ (bone or lung); **—W. H. Langham gave 0.032 μCi as a proposed LNL value in 1950.

and water $(MPC)_w$ for a large number of radionuclides including values for ^{239}Pu and some of the other actinide elements. Table I summarizes these values of q and qf_2 for ^{239}Pu. Similar values to those in Table I have been given in these same publications for the other actinide radionuclides and for the most part there have been few changes since 1953. In most cases the ICRP and NCRP recommended dose limits are identical. In 1964. ICRP[9] made a few revisions for the actinide elements but the values for ^{239}Pu remained unchanged.

Changes Being Considered for Revised ICRP Internal Dose Handbook

There are many changes being considered for the ICRP Internal Dose Handbook which has been under revision for over 12 years. Only a few of these changes which relate to the permissible exposure levels for the transuranium radionuclides will be mentioned here. Two rather obvious improvements are: (1) Where possible doses to the bone will be calculated for specific critical tissue of this organ rather than average the dose over the entire bone and (2) The dose to a critical organ (or tissue) will be the sum of the doses to that organ originating from deposits of the radionuclide in all body organs including that from deposits in the critical organ.

The present ICRP and NCRP values[7-9] of q, qf_2, $(MPC)_a$, and $(MPC)_w$ were calculated on the basis of uniform distribution of the radionuclides in the critical body organ (e.g. uniform deposition in the skeleton) and irradiation only from the deposits of the radionuclide within this organ. These assumptions were made because of a lack of biological information. The assumption of uniform distribution of a radionuclide may have given rather reliable results in some cases for gamma and high energy β-emitting radionuclides that are fairly uniformly deposited in an organ but the risk (of bone cancer) from ^{239}Pu could have been seriously underestimated because most of the α-emitting ^{239}Pu is deposited on bone surfaces of the trabecular matrices adjacent to the thin layer of endosteal tissue which happens to be the most critical tissue in this case. Obviously, the inclusion in the calculation of dose only from the radionuclide deposited within the critical tissue itself could lead to underestimates of the risk except for α and low energy β-emitting radionuclides that are highly localized in the critical organ so that cross irradiation from other organs is insignificant. The decision of the ICRP has been to consider the critical tissues of the skeleton the endosteal tissue (as it relates to bone cancer) with an average thickness of 10 μm and the active (red) bone marrow (as it relates to leukemia), and to limit the maximum permissible annual occupational dose (MPAD) to these tissues to no more than 15 rem/y (a limit of 1.5 rem/y for members of the general public). Unfortunately our knowledge of the microdeposition of ^{239}Pu in the bone probably is too limited at the present time to apply these refinements and so it is likely the present practice will be continued; namely, calculate the dose from ^{239}Pu to the entire skeleton, as is done with some justification for ^{226}Ra, and apply an N-factor ($= 5$) to the absorbed dose (rad) as well as the usual Q factor ($= 10$) for α-radiation in obtaining estimates of the dose equivalent (rem).

The new ICRP Internal Dose Handbook probably will not give values of q, qf_2, or $(MPC)_w$ but these quantities can be calculated from values of A (μCi days of residence time in the critical tissue of reference or standard man), B (dose commitment in rem to this critical tissue for the next 50 years per μCi intake), and MPAD (maximum permissible annual dose, e.g. occupational limits of 5 rem/y to total body and gonads; 30 rem/y to total bone, thyroid, and skin; 75 rem/y to hands, feet, arms, and ankles; and 15 rem/y to all other body organs or tissues). Two equations[10] as

follows can be used in making these calculations:

$$q = \frac{5.4 \times 10^{-5}\ m\ (MPAD)}{f_2 \epsilon} \qquad (1)$$

$$q = \frac{(MPAD)A}{365\ f_2 B} \qquad (2)$$

in which A,B and (MPAD) are defined above, f_2 is the fraction of the radionuclide in the critical tissue of that in the total body, ϵ(MeV × Q × N) is the total effective energy deposited in the critical tissue of mass m(g) per disintegration of the radionuclide in the entire body.

The Linear Hypothesis May Not Be Sufficiently Conservative

Frequently in the literature it is stated that the linear hypothesis is a very conservative assumption. During the past few years, however, many studies have indicated that this probably is not true in general and that at low doses and dose rates somatic damage per rad (and especially that from α-irradiation) probably is usually greater than would be assumed on the linear hypothesis. There are many reasons for this, some of which are:

1. The linear hypothesis is based on extrapolations to zero dose of effects of radiation on animals or humans at intermediate to high doses. The points used on the curves at high doses may be on the descending part of the curve, i.e. from portions of the curve where there was overkill or where a large fraction of the highly exposed died of other types of radiation damage and did not survive to die of the radiation effect under study.
2. Extrapolations are made on human data which in general relate human damage such as bone cancer from ^{239}Pu for observation periods of no more than about 20 years. Many of the conclusions are based on studies of animals of life spans less than 10 years. Since man lives for more than 70 years, the slopes of these curves can only increase as more human data are accumulated over his entire life span.
3. The linear hypothesis assumes that man is a uniform and more or less homogeneous population. It applies to the average man and may not be sufficiently conservative for the fetus and for old people. It never takes into consideration special groups such as those studied by Bross[11] where he found that children of age 1-4 had 3.7 times the risk of developing leukemia if they have allergic disease such as asthma and 24.6 times the risk of the children of this age group if they had both allergic disease and had received intrauterine x-ray exposure.
4. There may be cell sterilization at intermediate and high doses. By this we mean there may be many cells in the body which are likely targets to become precursors of a clone of cells which are malignant but they are killed by the higher doses. In other words, these cells may already have two of the "series cancer switches" closed and a low dose of radiation would likely close the final switch in the step toward cancer production. A high dose such as that from which extrapolations usually are made, however, might kill most such cells as it does in radiation therapy which is used to destroy a cancer.
5. For many types of radiation damage the best fit curve is a plot of equation $E = CD^n$ in which E = effect, C = constant, D = radiation dose, and n = constant. For the linear hypothesis n = 1. In some cases $n > 1$ indicating lesser damage per rad at low doses but in many cases the best fit to experimental data is obtained when $n < 1$. Baum[12] recently showed a best

fit for cancer induction when n = ½. In such case the linear hypothesis would be non-conservative.

6. As pointed out above ^{239}Pu is an α-emitting, bone seeking, radionuclide like ^{224}Ra, but unlike ^{226}Ra, it is deposited on the bone surfaces adjacent to the radiosensitive endosteal and perisoteal tissues. The use of the N-factor equal to 5 for all α-emitting radionuclides in bone except ^{226}Ra somewhat compensated for this increased risk from surface deposition but has always left some questions to be answered when we determined all q and qf_2 values for bone as given in Table I by comparison with ^{226}Ra burdens in man. Our 50 year human experience with ^{226}Ra has been of extreme importance in setting these values for bone but one was not completely satisfied in using the University of Utah[13] data on ^{239}Pu and ^{226}Ra in dogs to provide guidance in making these extrapolations in humans where there are very little ^{239}Pu data. Fortunately, a recent finding may be of great assistance in relating ^{239}Pu exposure to ^{226}Ra which has been studied intensively for many years in some humans who have varying quantitatively determined body burdens of ^{226}Ra in their skeletons. Here I refer to the important studies of Mays et al.[14] of over 1000 patients in Germany who were injected with known amounts of the short lived (3.64 day), α-emitting radionuclide, ^{224}Ra as a treatment for extra-pulmonary tuberculosis. Because of its short radioactive half life ^{224}Ra, unlike ^{226}Ra, does not have time to be deeply imbedded in bone and thus may simulate to a considerable degree the deposition of ^{239}Pu in man. Mays[14] et al. have made an interesting observation regarding human exposure to ^{224}Ra which may have important bearing on chronic exposure of large populations to α-emitting, bone surface seeking radionuclides; namely, there is a greater incidence of bone sarcoma from a given total dose of radiation when the span of ^{224}Ra injections was increased. This increased risk with increased protraction of α-radiation exposure is opposite from what has been observed generally with exposure to x-rays where protracted dose allows time for more repair of radiation damage. Mays has suggested that maybe this may be attributable to (a) increased number of cells irradiated, (b) less kill of pre-malignant cells (i.e. cell sterilization), (c) prolonged stimulus of cell division, and (d) greater difficulty for cell repair of local α-damage.

Since ^{239}Pu when dispersed into the environment in very low concentration (except in the unlikely accident) delivers a protracted rather than an acute exposure to man, the risks may be greater than those suggested by animal studies at high acute levels of exposure to ^{239}Pu.

Changes in the Permissible Exposure Level for ^{239}Pu as Suggested by the Author

As noted in Table I, no values of q and qf_2 for occupational exposure are given at the present time in NCRP and ICRP Handbooks on Internal Dose for lung. However, using the data provided in ICRP Handbook 2, the value of 0.015 μCi ^{239}Pu for uniform distribution can be obtained. This of course raises the question of the so-called hot particle problem and adequacy of a value of q or qf_2 based on the assumption that the risk of lung damage (i.e. lung carcinoma) is proportional to the average dose delivered to the entire lung ($m = 10^3$ g).

No one knows the answer to this question at the present time. Certainly we would like to have more information. Tamplin and Cochran[15] suggest that because of the very large dose (thousands of rem/y) in the vi-

cinity of a micron size particle of ^{239}Pu lodged in lung tissue, the present q for lung ($\sim$ 0.015 μCi) and the corresponding values of $(MPC)_a$ for occupational exposure as well as those for members of the public should be lowered by a factor of 10^5. Perhaps they are right, but I believe they have not made a strong case for this factor simply because adequate biological data are not available and much of that which we have seems to give contradictory information. Early experiments of Lisco, Finkel, and Brues[16] have indicated there is a high probability (about 50%) of a malignancy at the site of injections of as little as one μg ($\sim$ 0.06 μCi) of ^{239}Pu in the skin of animals and data of Cember[17] perhaps suggest a higher risk due to localized doses in the lungs. On the other hand, later experiments of Brues[18] have shown when plaques of radioactive materials are placed on the skin of an animal, the risk of skin carcinoma is greater for a uniform distribution of a μCi than for a μCi localized in hot spots. The outstanding research of Bair and Thompson[19] shed much light on the hot particle problem but unfortunately they do not provide us with unequivocal proof that there is or isn't a hot particle problem. They[19] leave the question as one still to be resolved when they state "The mean dose to a tissue may be less important, however, than the dose to localized regions within the tissue." There is no question that epithelial cells of the skin are very radiosensitive and local doses such as are produced by μg quantities of ^{239}Pu in wounds are very carcinogenic. The tissues at risk in the lungs also are epithelial and the most important question remaining is whether or not this large localized dose to the epithelial cells of the lung can likewise result in a high incidence of lung tumors when small dust particles of the highly insoluble $^{239}PuO_2$ are inhaled and find their way to the terminal bronchioles, alveolar epithelial cells, or are translocated to thoracic and abdominal lymph nodes. It certainly is encouraging that there is no clear evidence at the present time that human occupational exposure to plutonium and other transuranium elements has resulted in any form of cancer. We should realize, however, that no extensive epidemiological and autopsy study of the exposed human populations has been completed and with man the average incubation period for tumors of the lung, bone, liver, or lymph nodes may be 40 to 50 years.

In theory at least the occupational exposure values of q and qf_2 for α-emitting radionuclides that are bone seekers have not been set by the use of equations 1 and 2 in the past but by direct comparison with the value of q = 0.1 μCi of ^{226}Ra in the human body. It develops, however, that the same values of q and qf_2 as are given by NCRP[7] and ICRP[3] can be obtained by setting (MPAD) in equation 1 equal to 30 rem/y for bone seeking radionuclides. This standard of 0.1 μCi of ^{226}Ra was set by the U. S. Advisory Committee on Safe Handling of Radioactive Luminous Compounds[20] in 1941. The ICRP[3] stated, "At the present time, it would be difficult to say which is more harmful to man (a) the dose rate to the total body of 0.1 rem/wk or (b) the dose rate to the bone resulting from a body burden of 0.1 μCi of ^{226}Ra . . . Although tumors have not been observed in persons with body burdens of radium as low as 0.1 μCi, the factor of safety may not be as large as 10 because tumors have been observed in persons having a body burden less than 1 μCi of radium at the time the tumor was first detected . . . Several workers have described changes in skeletal density and/or histopathological changes in the bone of patients who had 0.1 μCi or less of radium, and more pathological changes may be expected as these individuals become older." In spite of uncertainties regarding the 0.1 μCi standard for ^{226}Ra, it is based on over 50 years of human (not other animal) experience. With proper adjustments to determine the equivalent dose (rem) to the critical body tissue

from α-emitting actinide radionuclides, I believe comparison with ^{226}Ra and ^{224}Ra provides the best method now available for setting suitable radiation protection standards for these radioactive materials.

I believe the most reliable values of q based on bone as the critical tissue can be obtained for ^{239}Pu and some other transuranium radionuclides by making use of the comparative data on bone carcinoma and sarcome incidence in dogs that have been injected with known amounts of ^{226}Ra and ^{239}Pu as well as a number of other α-emitting radionuclides. This outstanding work has been carried out over a period of many years by a team at the University of Utah[13] and as pointed out by Bair and Thompson[19] these data can be used in making comparison of the values of q for ^{239}Pu and the other transuranium α-emitting radionuclides with ^{226}Ra. If one makes these comparisons, the corrections listed below should be made to the value of q = 0.04 μCi of ^{239}Pu which as indicated above is based on the 0.1 μCi ^{226}Ra standard when setting N = 5 or on the average dose rate of 30 rem/y to the adult skeleton:

(a) The value of q = 0.04 makes use of an N-factor of 5 for the α-radiation of ^{239}Pu and other α-emitting radionuclides in the skeleton. As pointed out above, this N is intended to be the relative risk from bone seeking, α-emitting radionuclides (e.g. ^{239}Pu) in comparison with ^{226}Ra on the basis of absorbed dose (i.e. on a per rad basis). Data of Daugherty and Mays[21] have shown that this value of N for dogs is somewhere between 5 and 15. If we accept the value of 15, the appropriate correction factor for ^{239}Pu is 5/15 or 1/3.

(b) The surface to volume ratio for the trabecular bone of the dog (the tissue in which it is believed most of the bone cancers originate) is about twice that for man. Thus the same amount of ^{239}Pu in man would have twice the concentration of ^{239}Pu near the trabecular surfaces as that in the dog. This would be a correction factor for ^{239}Pu of 1/2.

(c) The rate of turnover (burial) by apposition of new bone of the deposits of α-emitting radionuclides on the trabecular surfaces is probably about ten times that in the dog of that in man. This corresponds to a correction factor for ^{239}Pu of 1/10.

(d) Studies of Metivier et al.[22] on the survival time of baboons relative to the dog for various concentrations of $^{239}PuO_2$ in the lungs suggest that the baboon is about 4 times as radiosensitive as the dog. Assuming this same ratio would apply for bone burden of ^{239}Pu (perhaps a poor assumption) and that the radiosensitivities of the baboon and man are the same we have a correction factor for ^{239}Pu of 1/4.

The above would correspond to an overall reduction in q for ^{239}Pu of 1/240 (or q = 0.00017 instead of 0.04 μCi) when endosteal tissue of the bone is the critical tissue. Insufficient data are available to attempt any such correction to the value of q for the lungs other than apply correction (d) above. Thus we would have q = 0.015/4 $\cong$ 0.004 μCi when total lung is the critical tissue. This of course does not address the hot particle problem but rather shelves it until we have more data. This unfortunately is what society has done for generations in the case of environmental pollutants from burning of fossil fuels.

A somewhat similar problem, namely the possible use of pulmonary lymph nodes as the critical body organ for $^{239}PuO_2$ has been under discussion for many years by Committee 2 of ICRP. There is no question but that when dogs inhale $^{239}PuO_2$ in finely divided particles a major fraction ends up in the

thoracic lymph nodes. Park et al.[23] for example give the percents of alveolar-deposited $^{239}PuO_2$ 11 years after exposure of about 40% for thoracic lymph nodes, 13% for liver, and 5% for bone. After many years of consideration of this question the ICRP finally decided not to use the lymph nodes as critical body tissue because no animal studies had indicated this to be the critical tissue in terms of carcinogenesis. Perhaps in this case of large doses to the lymph nodes we have a good example of cell sterilization or complete kill of all the radiosensitive cells in the nodes that are within the range of the α-radiation. The picture might be quite different for lesser $^{329}PuO_2$ concentrations in these nodes which might be experienced by members of the public from chronic exposure to low dust levels of $^{239}PuO_2$. Perhaps only time can tell whether or not the present practice of ICRP of averaging the ^{239}Pu dose in the pulmonary lymph nodes and in alveoli and terminal bronchioles with the dose to the total lung mass (1000 g) is non-conservative. Likewise, as many researchers have pointed out, plutonium and the other transuranium elements tend to localize in the liver during chronic environmental exposure or from chronic leakage of Pu from the lymph nodes to the body fluids. Thus in the years ahead we could have some surprises and find that not the bone but the liver or even the lymph nodes after all are the critical tissues for human damage from chronic exposure to low levels of the transuranium elements. Hopefully, in the meantime we will learn more also about other environmental insults because when we do, I believe we will recognize an even greater urgency to keep their exposure to man as low as practicable.

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Vienna, Austria to Host International Atomic Energy Agency Symposium

Development of nuclear-based techniques for the measurement, detection and control of environmental pollutants will be the theme of the symposium, to be held March 15-19, 1976.

Inquiries on participation should be directed promptly to John H. Kane, Special Assistant for Conferences, Office of Public Affairs, MS: A1-5216, United States Energy Research and Development Administration, Washington, DC 20545.

Errata, Changes, Addition . . .

June, 1975 The Market Basket: Food for Thought
by William B. Deichmann, Ph.D., M.D. (hon.)

page 411—In the author's line, *Deichman* should have read *Deichmann.*

page 415—The phrase *"(nine calories per gram)"* is changed to read *"(nine Calories per gram)".*

page 421—The statement "The *diminishing* incidence of metastatic . . ." is changed to read "The *increasing* incidence of metastatic . . ."

June, 1975 Occupational Exposure Limits for Novel Work Schedules
by R. S. Brief and R. A. Scala

page 469—The author requests that preparation of the *"Comments"* portion of this article be credited to Dr. Herbert Stockinger, Chairman of the ACGIH Committee.

First European Plant Engineering Exhibition Opens September 15th

A major five-day conference will accompany this show, to be held at Earls Court, London. Among the subjects to be covered are health and safety law compliance and physical working environments. U.S. and Canadian visitors information from Clapp & Poliak, Inc., 245 Park Ave., New York, New York 10017.