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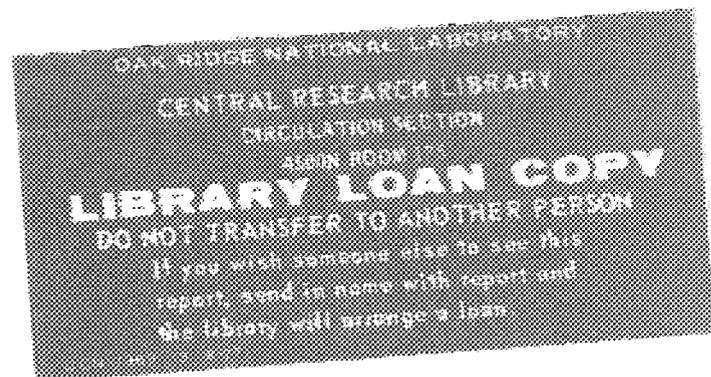
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MARTIN MARIETTA

**Report of Current Work of the
Metabolism and Dosimetry
Research Group
January 1, 1984 - June 30, 1985**

K. F. Eckerman



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Health and Safety Research Division

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THE METABOLISM AND DOSIMETRY RESEARCH GROUP
January 1, 1984 - June 30, 1985

K. F. Eckerman, Group Leader

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INTRODUCTION

This report describes current research efforts within the Metabolism and Dosimetry Research (MDR) Group of the Oak Ridge National Laboratory (ORNL). The MDR group has been funded continuously for over two decades by the Office of Health and Environmental Research (OHER) of the Department of Energy (DOE) to develop dosimetric methods and supporting data for the establishment of radiation protection guidelines. The purposes of this report are to inform OHER of our recent progress and to provide other researchers with summaries of many of our recent results that will appear in the literature only after the considerable lag time that has become standard for journal articles and proceedings.

This report is intended as the first in a new series of annual reports on our work. The work described here is in the form of brief abstracts of publications that appeared during the calendar year 1984 and extended summaries of work 'in progress', including any material that has been accepted for publication or has gone to press since January 1, 1985.

The main task of the Metabolism and Dosimetry Research Group is the development of radiation exposure-dose relationships. This is accomplished through the modeling of the metabolism of radionuclides taken into the body and the modeling of the deposition of ionizing energy within the radiosensitive tissues from these intakes or from exposures to radiation external to the body. Such relationships provide a cornerstone for development of radiation protection guidelines for the numerous radionuclides encountered in the workplace and also serve an important role in evaluation of diagnostic procedures involving radiopharmaceuticals and diagnostic x-ray machines.

Prior to Publication 30 of the International Commission on Radiological Protection (ICRP 30), most of our work had been aimed toward the estimation of organ doses to Reference Man, a model of the human body that has generally been considered adequate for evaluation of occupational exposures. In recent years emphasis in radiation protection has shifted somewhat from occupational exposures to exposures of the general public. In response, this group is now involved in the

development of metabolic and dosimetric models that allow consideration of special characteristics of individuals or of segments of the population, as well as in the continued improvement of models for the reference adult.

The development of models for 'non-reference' humans has required a departure from the curve-fitting approach commonly used in the construction of metabolic models. This is because there usually is insufficient element-specific information to develop these models for special subgroups of the population by typical empirical methods. As far as possible, metabolic models currently being developed by this group are mechanistic rather than empirical in nature. That is, whenever feasible, these models are based strictly on components that correspond to physically identifiable structures, processes, or quantities associated with the human body. We have found that this approach enables the modeler to use the basic physiological literature as an important source of information to supplement any available element-specific data. In addition to the advantages that a physiologically-based modeling approach provides in the consideration of biological variability among humans, it appears to allow more meaningful extrapolation of data from animals to humans, permit more realistic treatment of daughter products of some nuclides, yield better bioassay models of some nuclides by improving estimates of activity in excreta, and lead to improved estimates of doses to heterogeneously distributed radiosensitive tissues.

In closely related work, members of the group are involved in an ICRP task to revise the data for Reference Man given in Publication 23 of the ICRP. This revision is intended to give greater emphasis than did the original publication to the normal variations among humans. In particular, increased attention will be given to variations in anatomical and physiological characteristics with age or sex, or of natural differences that may occur for persons of the same age and sex, for example.

In addition to our primary task of developing exposure-dose relationships for radiation exposures, the group aids in the development of dose-response relationships through the evaluation of radiation doses incurred by subjects of epidemiological studies. Currently, one of the

main tasks of this group is a re-evaluation of the gamma and neutron doses received by the Japanese A-bomb survivors. This study was needed because of the increasingly apparent uncertainties in the total yield of the blast at Hiroshima as well as the relative gamma and neutron yields from the blast. It appears that there may be important modifications of earlier dose estimates and subsequently of the dose-to-risk conversion factors based on epidemiological studies of the A-bomb survivors.

The re-evaluation of the doses received by the A-bomb survivors, the shift in emphasis in radiation protection from occupational to environmental exposures, and the increasing application of organ dose estimates in such areas as risk assessment and probability of causation have required improvements and refinements in our methods for estimating doses to radiosensitive tissues due to irradiation from both external and internal sources. For example, the mathematical phantom developed several years ago for evaluation of energy deposition in the standard Western adult male has now been modified to yield representations of children of various ages, of the Western adult female, and of the Japanese adult male.

To improve our understanding of the factors influencing the effects of radiation on biological systems, we must consider not only the average energy deposited per unit mass of the organ (absorbed dose) but also its magnitude and spatial distribution with respect to the various biological structures within the organ. Prudent application of human experience with radiation in one exposure situation (e.g., the A-bomb survivors) to another situation (e.g., internal emitters) requires careful consideration of the physical and biological description of the irradiation. In this regard, considerable effort has been made to improve estimates of dose and subsequent risk due to irradiation of the skeleton. Particular attention has been given to improving estimates of absorbed energy in the heterogeneously distributed radiosensitive cells of the bone marrow and bone surfaces.

In recent years our funding from OHER has been supplemented through short-term contracts with other agencies, including the Environmental Protection Agency (EPA), the Nuclear Regulatory Commission (NRC), the Food and Drug Administration (FDA), and the Department of Transportation (DOT). This additional support has been critical to the development of

our research program and has provided the opportunity to help standardize the dosimetric methods used by the various federal agencies. In the present fiscal year, approximately 40% of our total funding is from OHER, with most of the remainder about equally split between NRC and EPA.

Work conducted for agencies other than OHER is documented in a manner to meet the specific information needs of those agencies. In general, our efforts for all of the funding agencies contribute to and benefit from a common pool of experience, information, and analytical capability. With few exceptions, each summary of work given in this report represents a combination and distillation of work from several projects supported by several agencies, and no attempt is made here to assign percentages of support to specific summaries.

PART I. Summaries of work in press or in progress after January 1, 1985

REVISION OF ICRP PUBLICATION 23 ON REFERENCE MAN

M. Cristy and G. D. Kerr

INTRODUCTION

At its 1984 meeting in Stockholm, Committee 2 of the International Commission on Radiological Protection (ICRP) formed a task group to revise ICRP Publication 23 on Reference Man, and the Main Commission approved this plan at its meeting later in 1984. Members of the task group are C. R. Richmond, Chairman, M. Cristy, G. D. Kerr, and D. Michelson at Oak Ridge National Laboratory (U.S.), M. C. Thorne of the ICRP (U.K.), L. Karhausen of the Commission of European Communities (Luxembourg), and G. V. Iyengar, now at the U. S. National Bureau of Standards. Cristy and Kerr are members of the Metabolism and Dosimetry Research Group.

The charge to the task group is as follows : "General review and upgrading of ICRP Publication 23 (Reference Man) giving more emphasis to the normal variations of persons and providing more information on young members of the population including age-dependent modelling." The work is expected to take about three years.

HISTORY

The Report of the Task Group on Reference Man, ICRP Publication 23, was published ten years ago, in 1975. However, the references cited end about 1970, so that the report is about 15 years out of date. It was in December 1963 when Committee 2 of the ICRP requested that the Main Commission establish a task group for the revision and extension of the Standard Man concept. Following a suggestion from the Main Commission, the name was later changed from Standard Man to Reference Man. The charge to the original Task Group on Reference Man was as follows:

Estimates of dose equivalent resulting from human exposure to radionuclides that may enter the body depend upon many characteristics which must be indicated clearly if the commission's recommendations on dose commitment, body burden, and MPCs are to be applied correctly. For this reason the

commission's recommendations relating to dose equivalent from internally deposited emitters have been specified in terms of a Reference Man whose characteristics, so far as relevant to the commission's recommendations, are carefully specified. Because of the increased emphasis on exposure of the population, it is desirable that the specifications of the previous Standard Man be reviewed and revised to take account of present needs for evaluation of exposure to radiation.

The task group should review those characteristics of man that relate directly or indirectly to intake, metabolism, distribution in the body, and retention of the various isotopes of concern. In particular, it is desired to define Reference Man, in the first instance, as a typical occupational individual, and it is important that some indication of variability of the occupational group about this norm be indicated. In addition, differences due to age, sex, or habits should be indicated where possible with particular emphasis on fetuses, infants, and children.

It is expected that a separate task group will consider the problem of devising a lung model, and thus the Task Group on Reference Man need not consider the mechanisms of inhalation, deposition, or lung retention. It is expected also that some independent assistance on the gastrointestinal tract will be available but that this will be offered to the task group directly.

It was clear from the outset that the original Task Group on Reference Man needed to produce information in two major areas: (1) a revision and extension of a Reference Man that represented a typical radiation worker and (2) an extension of the concept of Reference Man to give an indication of the extent of individual variation among grossly normal individuals including parameters that depend on age, sex, and other factors that must be considered in estimating dose to individuals of a population.

Information about exposed individuals is required for the estimation of radiation dose to the human body whether from external or internal sources. For external sources, the situation is relatively simple as one is primarily concerned with geometric location, mass, size, and elemental composition of various organs and tissues. In order to calculate secondary limits for radioactive materials taken into the body, however, additional information on physiological processes that might influence the uptake, translocation, residence times, recycling, and excretion of radionuclides is necessary.

The first Standard Man data were presented and formalized at the Chalk River Conference on Permissible Dose, September 29-30, 1949. Three main aspects of Standard Man were considered at that meeting: figures on the mass of the body organs; chemical composition of the total body and the various tissues; and patterns of intake and excretion plus duration of occupational exposure.

At the Chalk River Conference it was decided that these should be averages for normal activities in the temperate zone. Data on water balance, respiration, duration of occupational exposure, and retention of particulate matter in lungs were included in these categories.

Standard Man values were modified at the Sixth International Congress of Radiology in 1950, at the Tripartite Conference on Permissible Dose held at Harriman, New York, in 1953, and at the Seventh International Congress of Radiology, also in 1953. The 1959 report of ICRP Committee 2 (ICRP Publication 2, 1960) contained data on 46 naturally occurring radionuclides in the adult human body. This represented an increase of 31 elements as compared with the previous report. Also included was a table listing the concentrations of 44 naturally occurring elements in 36 tissues. Deposition parameters and elimination halftimes were also compiled in ICRP Publication 2.

For purposes of radiation protection of workers, Reference Man was defined as being between 20-30 years of age, weighing 70 kg, measuring 170 cm in height, and living in a climate characterized by an average temperature of 10-20° C. He is Caucasian and is a Western European or North American in habits and customs. The task group realized that once defined it remained for local or national authorities to determine what modifications of Reference Man might be appropriate for the specific population at risk.

In the work of the present task group, more attention will be given to members of the population who are not young male radiation workers.

PROGRESS TO DATE BY ORNL MEMBERS OF TASK GROUP

(1) To prepare for the first meeting of the Task Group, we have familiarized ourselves thoroughly with ICRP Publication 23 and, as far as possible, with the files of the original Task Group on Reference Man.

These files include photocopies of about 75% of the publications cited in Publication 23. We have solicited opinions from selected experts on shortcomings, errors, or inconsistencies in ICRP Publication 23, and a letter has been sent to journals and newsletters asking for information from users of this document.

(2) A dedicated personal computer (IBM PC) has been acquired by the ORNL members of the Task Group to help in this project. It may be necessary to acquire fixed memory and a small printer.

(3) We have begun searches on journal citations to ICRP Publication 23 to see in what ways the data in that report have been used. To date 266 documents, published from 1978 through 1983, have been identified, and photocopies of 252 of these are in hand. A few of these report errors in Publication 23. Approximately 30% of the citing documents were not related to radiation or radiation protection, which indicates that the Reference Man report is of use to a wide audience.

(4) A proposed Table of Contents has been developed and circulated to all members of the Task Group. A Table of Contents revised according to comments from these members will be presented to ICRP Committee 2 for approval at its June, 1985, meeting in Leningrad.

THE DISTRIBUTION OF INTRACELLULAR ALKALI METALS IN REFERENCE MAN

L. R. Williams and R. W. Leggett

INTRODUCTION

It has been pointed out recently by Mole (1984) that the ICRP Reference Man (ICRP 1975) is a misleading guide to the content and distribution of sodium in the human body, particularly with regard to the skeleton. A review of the recent literature and a reanalysis of the data used in ICRP 23 (1975) have led us to the conclusion that Reference Man is also a misleading guide to the distribution of the predominantly intracellular alkali metals potassium, rubidium, and cesium, and that estimates of the skeletal content of these metals and of the total body content of rubidium may involve especially large errors. The object of this study is to improve estimates of the content and distribution of the intracellular alkali metals in Reference Man. To obtain an internally consistent model of a standard adult, some changes also are suggested in the masses of some of the components of Reference Man.

SUGGESTED MODIFICATIONS TO ICRP REFERENCE MAN

We consider a reference adult male of age 35 years and total mass 70 kg. Suggested changes in the masses of organs from those of the ICRP Reference Man are not large: (1) muscle is assigned a mass of 29 kg instead of 28 kg; (2) adipose tissue is assigned a mass of 14 kg instead of 15 kg; (3) the skeleton is assigned a mass of 10.5 kg instead of 10 kg; (4) the mass of miscellaneous tissue is decreased by 0.5 kg.

Proposed changes in K, Rb, and Cs contents in the organs and tissues of ICRP Reference Man are listed in Table 1. (These are preliminary estimates and may be altered slightly.) The concentration of K, Rb, or Cs in each tissue was estimated as a weighted average of published values (for example, see the compilation by Iyengar, Killmer, and Bowen 1978). Weighting factors were based on the sample size represented by each reported value. As nearly as possible, 20% of the highest published values and 20% of the lowest published values for each

compartment were eliminated as potential outliers before the weighted average was computed. For a few tissues with no available experimental data on natural K, Rb, or Cs, data on radioactive tracers were extrapolated to equilibrium conditions.

Potassium

In Publication 23 of the ICRP (1975), Reference Man is assigned a total body mass of 70 kg and a K content of 140 g. This K content is based primarily on external measurements of the 1.46 MeV gamma ray of K-40 and appears from our review to be a reasonable estimate for a 70-kg male of age 30-40 years. Thus, we have retained this value in our suggested version of Reference Man. There appear to be problems, however, with the estimated K content of some individual organs and tissues of ICRP Reference Man. This becomes evident when one attempts to reproduce total-body K as the sum of the parts, which cannot be done unless an unrealistically high concentration is assigned to tissues not explicitly listed. The estimate of total K in skeleton of ICRP Reference Man appears to us to be based on non-representative skeletal parts and to be too high by 40-50%. On the other hand, there appears to be a substantial underestimate of the K content of skeletal muscle.

Rubidium

The ICRP Reference Man has a total rubidium content of 0.68 g, of which 0.21 g is assigned to the skeleton. Both of these estimates were based on very limited data, and the skeletal content, in particular, appears to have been based on nonrepresentative parts. We estimate a total body content of about 0.25 g Rb, a factor of nearly 3 below the estimate in ICRP Publication 23. The skeletal content of Rb is estimated to be a factor of about 15 below the ICRP estimate. Our estimate of Rb in muscle, which contains most of the body's Rb in our model and in animal experiments with radiorubidium, is in close agreement with the ICRP estimate.

Cesium

Modifications of the distribution of cesium are not as large as those for rubidium. The ICRP Reference Man is assigned a total body content of 0.0015 g Cs, of which about 10% is assigned to the skeleton.

We estimate a total-body content of about 0.0012 g Cs, with approximately 7% of this amount being in the skeleton.

IMPLICATION FOR RADIATION DOSIMETRY

With regard to radiation protection, the most significant modification of ICRP Reference Man discussed here concerns the propensity of Rb for the skeleton. In the metabolic model for Rb given in ICRP Publication 30 (1979), 25% of the body's Rb is assigned to the skeleton on the basis of the distribution of Rb described in ICRP Publication 23. We would assign only about one-fifth as much Rb to the skeleton, given a constant chronic intake. This would lead to a large reduction in the estimated skeletal dose from most isotopes of Rb and some increase in the doses to other tissues.

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- International Commission on Radiological Protection (ICRP), 1975, Report of the Task Group on Reference Man, Publication 23, Pergamon Press: Oxford.
- Iyengar, G. V., Kollmer, W. E., and Bowen, H. J. M., 1978, The Elemental Composition of Human Tissues and Body Fluids, Verlag Chemie, New York.
- Mole, R. H., 1984, "Sodium in Man and the Assessment of Radiation Dose after Criticality Accidents," Phys. Med. Biol. 29, 1307-1327.

Table 1. Preliminary suggestions for changes in masses and contents of major tissues of ICRP Reference Man, given as multiple of value listed in ICRP 23.

Tissue	Mass	K content	Rb content	Cs content
Adipose tissue	0.93	0.75	a,b	b
Brain	1.0	0.9	0.15	0.65
Erythrocytes	1.0	0.95	0.9	b
GI tract	1.0	1.2	1.0	1.1
Heart	1.0	1.1	2.8	1.6
Kidneys	1.0	1.0	0.6	1.5
Liver	1.0	1.1	0.2	1.3
Lungs	1.0	0.9	0.4	2.0
Muscle (skeletal)	1.04	1.1	1.0	1.5
Skeleton	1.05	0.7	0.06	0.6
Skin	1.0	1.7	b	b
Spleen	1.0	1.0	0.3	1.7
Total body	1.0	1.0	0.35	0.8

^aNot estimated by Williams and Leggett.

^bNot estimated in ICRP Publication 23.

REVIEW OF GASTROINTESTINAL ABSORPTION OF RADIONUCLIDES BY YOUNG ANIMALS
AND THE DEVELOPMENT OF THE G.I. TRACT AS IT RELATES TO THIS PROBLEM

M. Cristy

Absorption of several radionuclides present in high-level waste (for example, plutonium, americium, neptunium, and strontium) has been shown to be higher in neonates than in adults of several species by as much as three orders of magnitude. Less information is available on absorption in juvenile animals after weaning and before sexual maturity, especially in animals that have a protracted juvenile period similar to that of humans. Except for strontium, no direct information on absorption by human infants and children of radionuclides present in high-level waste is available. Most of the information on absorption by neonates is from one animal model, the rat, but increased absorption by neonates does seem to be a general phenomenon -- other rodents (hamster, guinea pig) and mammals of other orders (swine, sheep, cow, dog) show increased absorption of plutonium, for example. Human infants and children absorb strontium more readily than do adults (Kahn et al. 1969).

In fact, neonates seem to absorb metals in general more than do adults -- this is true for nutritional metals such as iron and calcium as well as toxic, non-nutritional metals such as lead and cadmium. The reason(s) for this general increased absorption is not completely clear. Many workers have suggested that the high rate of pinocytosis by the epithelial cells of the villi in the small intestine that occurs in some species in the immediate postnatal period (and is associated with absorption of immunoglobulins and other macromolecules) is the mechanism or one of the mechanisms involved. However, the evidence is better for some metals than others, and there may be species differences (Henning and Leeper 1984). Neonatal guinea pigs do not absorb immunoglobulins postnatally, although they do take up macromolecules into the intestinal cells by pinocytosis for the first 1-2 days of life (Clarke and Hardy 1970; Lecce and Broughton 1973); Sullivan (1980) reported that guinea pigs 0.5-1 days old show increased absorption of plutonium, and Harrison (1985) recently found that neonatal guinea pigs older than 2 days, where

pinocytotic uptake has ceased, also show increased absorption. Lead is one of the best characterized metals. In neonatal mice, lead is absorbed primarily in the ileum (distal small intestine), by pinocytosis, but lead is also absorbed in the jejunum (just proximal to the ileum) by a different, but unknown, mechanism (Keller and Doherty 1980). In neonatal rats, lead is absorbed primarily in the duodenum (extreme proximal small intestine), where pinocytosis is unimportant. In the ileum, lead is taken up by the intestinal wall, probably by pinocytosis, but little of this is absorbed into the general circulation. Most of this lead is eventually returned to the contents of the intestine, by sloughing of senescent epithelial cells, and eliminated with the feces (Henning and Leeper 1984).

The increased retention of metals in the wall of the neonatal small intestine may be important for radiation protection, since metal radionuclides may irradiate cells in the intestinal wall, including the radiosensitive crypt cells, for longer periods. Several actinides have been shown to be retained tenaciously in the intestinal wall of neonatal rats and swine (Sullivan 1980; Sullivan and Gorham 1982).

Absorption of metals by neonates appears to be a process with at least two steps: (1) uptake of the metal by the epithelial cell and (2) transfer from the epithelial cell to the general circulation. When uptake is by pinocytosis, differences among metals are probably smaller than when by other mechanisms. Greater differences among metals are seen with transfer to blood or lymph; and for radiation protection, these differences are important both for irradiation of tissues distant from the intestine and for irradiation of cells in the intestinal wall. In addition, some metals may be absorbed in part by routes other than through the epithelial cells, but these routes are not well characterized. Damage to the intestinal epithelium, as occurs in diseases such as coeliac sprue, may lead to increased absorption. The mechanisms of absorption of heavy metals by adult animals, either via the epithelial cells or other routes, are also not well known.

How this information on rats and other mammals may be extrapolated to the human infant and child, for the purpose of estimating gut absorption factors for radiation protection, is problematical. How important pinocytosis is for absorption by human infants is not clear,

although it appears that infants do absorb macromolecules to a greater extent than do adults (Walker and Isselbacher 1974). The time period over which the greatly increased absorption seen in neonates of other species may occur in infants is also not clear -- it may occur for only a few days or weeks, or it may occur until weaning from a milk diet. For protection of the public from environmental contamination leading to contamination of food and water, Harrison (1982; 1983a,b) has proposed increased gut absorption factors for plutonium, americium, curium, and neptunium that apply to the first year of life. For all chemical forms of plutonium other than the insoluble oxides and hydroxide, he recommends an average value of 1% be applied to the first three months, when the infant is assumed to be on a milk diet; and absorption is then assumed to decline linearly during the weaning period to 0.05% (his recommended adult value) at nine months of age and remain at 0.05% thereafter. (The ICRP [1979] recommends 0.01% for workers.) For an average value to apply to the entire first year of life, he recommends 0.5%. For the insoluble oxides and hydroxide of plutonium, he recommends that average values of 0.1% and 0.05% absorption be used for the first three months and first year, respectively, and the ICRP (1979) value of 0.001% be used after nine months of age. For all chemical forms of americium and curium, he recommends that average values of 1% and 0.5% absorption be used for the first three months and first year, respectively, and the ICRP (1979) value of 0.05% be used after nine months of age. For all chemical forms of neptunium, he recommends that average values of 1% and 0.5% absorption be used for the first three months and first year, respectively, and 0.1% be used after nine months of age -- note that this latter value, which is 1/10 that recommended by the ICRP (1980) for workers, is based largely on data published after the ICRP made its recommendations. Harrison (1983a, p. 30) states that in 'evaluating the enhancement in the absorption of the actinides in newborn animals, extrapolation of the available animal data to absorption in the human is particularly tenuous.' We agree with this statement and agree that his proposed values are reasonable estimates, given current knowledge. The National Radiological Protection Board of the U.K. has adopted these recommended values for protection of the public (NRPB 1984).

These estimates will need to be reviewed periodically as the mechanisms of absorption of actinides in particular and in heavy metals in general become better understood. These estimates are probably conservative, except perhaps for children older than nine months. The question whether children older than nine months absorb actinides and other radionuclides more readily than do adults has not been adequately addressed in radiation protection. Children appear to absorb several times more iron and lead than do adults (Gorten et al. 1963; Ziegler et al. 1978), but whether this holds true for actinides and other metals is unknown. We suggest that assuming absorption of actinides by children older than nine months is 2-3 times that in adults would be prudent until this question has been tested in an animal with a protracted juvenile period similar to that in humans (it may be necessary to use a primate model).

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AN ESTIMATE OF THE GASTROINTESTINAL ABSORPTION
FRACTION FOR ENVIRONMENTAL PLUTONIUM IN ADULT HUMANS

R. W. Leggett

INTRODUCTION

For evaluation of occupational exposures, the International Commission on Radiological Protection (ICRP) currently recommends the value 10^{-4} for the fraction f_1 of soluble plutonium absorbed from the GI tract to blood (ICRP, 1979). This value is based on data derived in laboratory experiments often conducted under conditions much different from those expected in environmental exposures to plutonium. Recent results and reviews by several authors (Bair 1979; Harrison 1982; Johansson 1983; Kocher and Ryan 1983; Larsen et al. 1981; Sullivan et al. 1979; Sullivan 1980; Sullivan et al. 1980) indicate that chronic intake of low levels of environmental forms of Pu by laboratory animals may lead to observed values of f_1 substantially larger than 10^{-4} . In recent reviews, Bair (1979) and Kocher and Ryan (1983) recommended a value of 10^{-3} , and Harrison (1982) and Johansson (1983) recommended a value of 5×10^{-4} for GI uptake of environmental plutonium. The value recommended by Harrison and Johansson appears to be intended as a 'most likely' value based on the animal data, while the recommendation of Bair and that of Kocher and Ryan may be intended as slightly conservative for considerations of radiation protection. In the present report an upper bound of 0.002 for f_1 is estimated from data for a group of adult humans who had ingested elevated levels of Pu in their normal diet. The value 10^{-3} may be a reasonable choice for f_1 for use in assessments of environmental exposures to plutonium, since it is in close agreement with values recommended in recent reviews, and it is only a factor of 2 below the upper bound estimated here for the group of adult humans.

DERIVATION OF AN UPPER BOUND OF THE ABSORPTION FRACTION FOR ADULT HUMANS

The diet of the Finnish Lapp population is known to contain abnormally high levels of plutonium because of the lichen-reindeer-man food chain peculiar to the region. By estimating the quantities of

plutonium ingested and inhaled by Lapps and their whole-body contents and comparing these with analogous quantities in Southern Finns, Mussalo-Rauhamaa and coworkers (1984) attempted to determine the fraction f_1 of ingested plutonium absorbed to blood. Their approach was to solve the simultaneous equations

$$a_1 X + b_1 Y = c_1 \quad (1)$$

$$a_2 X + b_2 Y = c_2 \quad (2)$$

where Eq. (1) is for Southern Finns and Eq. (2) is for Finnish Lapps, a_i is the lifetime inhalation intake of Pu, b_i is the lifetime dietary intake, c_i is the body burden at death (in 1977-79), and the unknowns X and Y are the fractional absorptions from the airways and from the gastrointestinal tract (f_1), respectively. Their estimates of a_i , b_i , and c_i were 18, 36, and 1.37 pCi, respectively, for Southern Finns and 13, 535, and 1.40 pCi, respectively, for Lapps. An estimate of 9.0×10^{-4} was obtained for Y (f_1) after a slight adjustment for the fraction of activity assumed to be excreted. The authors noted some problems with this approach, but it does nonetheless yield a valuable preliminary result since it is the only available estimate of f_1 for plutonium that is based on data for humans.

The main difficulty with this approach is that the solution for Y will vary substantially as estimates of the body burden and the inhalation intake vary within their ranges of uncertainty. In fact, since autopsy data do not establish a clear difference between the whole-body burdens of the two groups or between their lung burdens, one cannot use this method to rule out the value $f_1=Y=0$, obtained if $a_1=a_2$ and $c_1=c_2$. Although the estimate of 9×10^{-4} agrees well with current estimates based on data for non-humans, one must question whether the agreement is fortuitous in view of the uncertainties in the coefficients in Eqs. (1 and 2). In the following it is shown that, despite the uncertainties involved, the data and general approach of Mussalo-Rauhamaa and coworkers may be used to obtain a reasonably reliable estimate for an upper bound of the GI absorption fraction for environmental plutonium. This upper bound is only a factor of 2 higher than their estimate for f_1 .

The largest error associated with an estimate of the whole-body burden of Pu based on autopsy data generally arises from the non-uniformity of Pu in the skeleton. Mussalo-Rauhamaa and coworkers used rib samples to estimate skeletal burdens. Various results indicate that the concentration of Pu in rib may be higher than average for the skeleton, but it is probably not lower than average (Mussalo-Rauhamaa et al. 1979). Whatever the direction of error, this should be considered a systematic error and thus should apply equally to Lapps and Southern Finns. We are seeking a smallest reasonable upper bound for f_1 , and, as can be shown, the estimate of this value obtained from the approach used here will change in proportion to systematic adjustments in estimated whole-body burdens. Thus, skeletal burdens estimated by Mussalo-Rauhamaa and coworkers should not be adjusted since their error, if any, would probably be on the high side.

Autopsy data for five adult male Lapps are listed by Mussalo-Rauhamaa and coworkers (1984). The maximum estimated body burden of these subjects (three reindeer herders, a fisherman, and a farmer) is 1.56 pCi. Because of the relatively small sample of Lapps considered, this value is adopted here as a conservatively high estimate of the whole-body burden of a typical adult male Lapp. The average whole-body burden of 1.37 pCi determined for adult male Southern Finns is based on a relatively large number of samples and will be adopted for this analysis.

The estimated dietary intake of Pu for the Lapps during the period 1954-1978 is 535 pCi (Mussalo-Rauhamaa et al. 1984). There is no way to determine whether this is representative of the sample population's intake, but it should be assumed that some error is involved. Since the upper-bound estimate will increase as the assumed Pu intake by the sample population decreases, it will be assumed conservatively that this estimate is 50% higher than the real value, which would then be about 360 pCi. The relatively small dietary intake for Southern Finns has little effect on the final estimate of f_1 when allowed to vary within a reasonable range; the original estimate by Mussalo-Rauhamaa and coworkers (1984) of 36 pCi is used here.

The inhalation intake for the entire lifetime of the Lapps and Southern Finns was based on the estimated air concentration of Pu in northern and southern Finland, respectively, and a breathing rate of 20 m³/day (Mussalo-Rauhamaa et al. 1984). It was noted that the breathing rates of the Lapps may not be the same as those of typical Southern Finns, since the Lapps are generally outdoor workers. The amount of plutonium inhaled by Lapps was estimated as 13 pCi, compared with 18 pCi for Southern Finns, with the difference arising from the fact that the average concentration of Pu in surface air in the Lapp region was estimated to be only 72% of the concentration in southern Finland. It is assumed here that the inhalation intake by Southern Finns is k_1Q and that of Lapps is $k_2(0.72Q)$, where k_1 denotes the appropriate breathing rate in each case and Q is the integrated concentration of plutonium in surface air in southern Finland. The following equations may then be used to obtain an upper bound for f_1 , without having to assign a numerical value to the breathing rate:

$$k_1QX + 36Y = 1.37 \quad (3)$$

$$0.72k_2QX + 360Y = 1.56. \quad (4)$$

From Eq. (4) we know that $Y < 0.0043$. Substituting this into Eq. (3) yields $X > 1.22/k_1Q$, and putting this back into Eq. (4) gives $Y < 0.0043 - 0.0024k_2/k_1$. There are two reasons why one might suspect that $k_2 > k_1$: the Lapps may perform heavier work than Finns, as an average; and the limited autopsy data did not show a lower concentration of Pu in lungs of Lapps than in lungs of Southern Finns, despite the lower air concentrations experienced by the Lapps. An upper bound for Y can be found by assuming $k_2 = k_1$; this gives $Y = f_1 < 0.0019$. An iteration of the procedure described in this paragraph yields $f_1 < 0.0017$, and allowance for the small amount of plutonium lost from the body (ICRP 1979) between 1954 and 1978 would raise this upper bound to about 0.002.

A slightly more detailed description of this work will appear in Health Physics, probably in 1986.

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RELIABILITY OF THE DOSIMETRIC MODELS OF ICRP 30
AND PROSPECTS FOR IMPROVED MODELS

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INTRODUCTION

Publication 30 of the ICRP represents the culmination of an extensive effort to characterize the metabolism and dosimetry of radionuclides in a reference worker. This is generally considered the most reliable comprehensive collection of internal dosimetric models now available and as such is often used in radiation risk analyses for environmental as well as occupational exposures, even though these models were not designed or intended for evaluation of exposures to the public. Thus, it is important to examine the reliability of the ICRP 30 internal dosimetric models for use in radiation risk analyses for arbitrary populations, as well as for the reference adult for which they were originally developed. The objects of this ongoing study are (1) to identify sources of uncertainty or error in the metabolic and dosimetric models of ICRP 30, (2) to quantify the reliability of these models for application to various subgroups of the population, and (3) to suggest improvements in specific models as well as in the general modeling approaches.

In ICRP 30, estimates of absorbed energy per gram of tissue for various radiosensitive organs following internal exposure to a given radionuclide usually are derived by combining three models in series: (1) a model for the retention and translocation to blood of inhaled material by the respiratory or gastrointestinal tract; (2) a "metabolic model" of the allotment of activity among the various organs and retention in those organs; (3) a model of the dose received by each organ from the given distribution of the radionuclide and its radioactive progeny. During the past year much of our effort has been directed toward assessing the reliability of some of the metabolic models of ICRP 30 and considering ways of improving the present modeling approach. Some attention also has been given to improving estimates of

specific absorbed fractions, particularly with regard to the heterogeneously distributed radiosensitive tissues of the skeleton.

METABOLIC MODELS

Derivation of metabolic models was simplified considerably in ICRP 30 by restricting attention to the average adult, considering only integrated doses over relatively long periods, not explicitly considering the recirculation of radionuclides among the organs, and assuming that daughter radionuclides produced from their parent within the body stay with and behave metabolically like their parent, among other assumptions. There are several advantages in imposing these restrictions: one may deal only with an 'average metabolism' in adults and need not describe variation with age, sex, and other factors; it is not necessary to provide a detailed description of retention and excretion over the short term, such as in the first few days after exposure to a radionuclide; retention of nearly all radionuclides can be described using a relatively neat and simple format, namely, as an exponential function or as a simple combination of a few exponential functions, each with constant coefficients and exponents. The retention functions are usually empirically derived as best-fitting curves relative to animal or human retention data. Thus the advantages of this modeling approach are that the models have a simple, common format, they are easy to understand, they are easy to use in the calculation of dose, they need not be particularly accurate over the short term, and they do not require information concerning variation of metabolism among humans.

Several disadvantages and weaknesses in the ICRP 30 modeling approach have also become apparent. For example, although the models have been constructed largely from animal data, they are not constructed in such a way that extrapolation to humans has strong logical support. Also, doses to heterogeneously distributed radiosensitive tissues of an organ (e.g., skeleton) cannot be estimated accurately, since the actual movement of radionuclides in the body is usually not accurately tracked, even in cases where the whole-body retention is estimated fairly well. Another weakness is that some radionuclides are assigned the model of an apparently related nuclide (for example, americium, curium, neptunium

are assigned the model derived for plutonium) although some differences in metabolism are known. A fourth problem is that the growth of radioactive daughters is often not handled realistically, and the format of the models makes it difficult to apply alternate assumptions.

If one considers the models with regard to their common applications for purposes other than estimating dose commitments to radiation workers, then other disadvantages become evident. For example, the models often do not yield accurate estimates of excretion even for the average adult; thus, better models are needed for bioassay programs. Perhaps the major problem with these models is that they are not flexible. Their construction does not allow extension to non-standard man (any person with anatomical or metabolic characteristics different from Reference Man, such as a child). This is because the components of the models usually were derived as fits to experimental data and hence do not correspond to identifiable anatomical or physiological entities. There is generally insufficient data with which to develop new models for special subgroups by the fitting techniques that characterize most of the standard-man models.

We have evaluated uncertainties associated with the ICRP retention models for potassium, rubidium, cesium, strontium, and plutonium and have suggested alternate models based on a mechanistic approach in which the components of the model are defined in terms of actual anatomical and physiological entities and recirculation of activity is considered explicitly. Some of these models are described in other summaries and abstracts in this report.

SPECIFIC ABSORBED FRACTIONS

In ICRP 30, estimates of absorbed dose from photon and neutron radiations are derived using a mathematical analogue of the body with a homogeneous representation of the skeleton. While the assumption of a homogeneous skeleton is adequate for considerations of scatter and total absorption of energy by the skeleton, the assumption of charged particle (electronic) equilibrium which would hold in a homogeneous skeleton does not appear to be satisfied in the vicinity of the real, inhomogeneous skeleton. The transport of energy by secondary electrons must be

considered in deriving realistic estimates of absorbed dose in soft tissues surrounding the bone mineral.

Complexities involved in modeling the geometry of soft-tissue regions within the skeleton have led to a conservatively oversimplified formulation in ICRP 30 of energy deposition in the skeleton. In particular, the total energy deposition in the assumed homogeneous skeleton usually is partitioned among the various skeletal tissues, including active marrow, by skeletal mass fraction. This results in overestimation of the absorbed dose to the active marrow. Also, in ICRP 30 energy deposition in endosteal tissue is equated to that of the skeleton as a whole. This probably leads to underestimates of absorbed dose in the endosteal tissue.

We are developing a computational approach for photon and neutron irradiation that formulates the absorbed dose in the soft tissues of the skeleton in terms of the physical and anatomical factors that are thought to control the location of energy deposition. This approach relies on information from the literature on the microscopic structure of trabeculation in the skeleton. The results of our computations are expressed in terms of response functions or photon fluence-to-dose factors that relate absorbed dose in active marrow to particle fluence in the skeleton. These factors can be applied to fluence estimates derived from Monte Carlo transport calculations in mathematical analogues of the body, with a homogeneous representation of the skeleton, to yield estimates of absorbed dose in active marrow from photon radiation incident upon or emitted within the body.

A MODEL FOR THE KINETICS OF POTASSIUM IN HEALTHY HUMANS

R. W. Leggett and L. R. Williams

INTRODUCTION

The scheme currently recommended by the International Commission on Radiological Protection (ICRP 1979) for evaluation of occupational exposures to radiopotassium depicts the body as a single well-mixed pool from which K is lost with a biological half-time of 30 days. More detailed models of the behavior of K in the body have appeared in the physiological and medical literature, but these models generally consist of hypothetical, mathematically derived compartments that may not correspond to identifiable anatomical compartments. The purpose of this study is to develop a model that describes the normal movement of K through the human body in much greater qualitative and quantitative detail than has been offered previously. This work arose as part of our efforts to develop a small set of general schemes that could be used to improve the empirical curve-fitting approach used by the ICRP for depicting the retention of radionuclides in humans. Potassium was used as a starting point for one of these schemes because of the large body of information on this element and the importance of some radioisotopes that follow the movement of K. In subsequent work the model framework will be applied to the elements rubidium and cesium.

DESCRIPTION OF THE MODEL

The conceptual framework for this model is indicated in Fig. 1. Inflow and outflow rates chosen for the various compartments are summarized in Table 1. Plasma (solid arrows) serves as a primary feeding compartment, although it cannot be regarded as a central compartment since transport of K among compartments by other materials (dashed arrows) is also considered. The rate of flow of K from plasma into a compartment is viewed as being related to blood flow but not totally controlled by this factor. Other factors, such as the tissue-specific fraction of K extracted by a compartment during a single passage from arterial to venous plasma, are considered whenever possible. At points in the construction of the model where data for

humans are sparse or nonexistent, extrapolation from data for other species is made by appealing as much as possible to the apparent similarity among many species in the behavior of K at the tissue level.

Movement of K among the compartments is viewed as a system of first-order processes. Based on a review of experimental data for non-humans and comparisons of model predictions with data for humans it was concluded that this approach is adequate provided attention is restricted to the net movement of K over periods of at least 2-3 minutes. Thus, no attempt was made to incorporate into the model any delays or other peculiarities associated with the extracellular-membrane-intracellular exchange of K that may occur on the order of seconds. For consideration of net movement of K over periods of 2-3 minutes or longer, it was not necessary to consider cellular and extracellular fluids within an organ or tissue as separate pools of K except in the case of skeletal muscle. Skeletal muscle appears to be an exception due to a combination of two factors: (1) the cells of this compartment equilibrate more slowly with ECF than do cells of most other tissues, so that the assumption of uniform mixing over a short period is less reasonable for skeletal muscle than for most other compartments; and (2) the assumption of uniform mixing in skeletal muscle would lead to an unrealistically rapid increase of K in skeletal muscle, which would lead to large errors for all compartments in the model because most of the body's K is in skeletal muscle.

Although the parameter values described in this paper are for a typical, healthy, resting adult male, the model framework has been designed for consideration of other conditions and subgroups of the population, provided the equilibrium distribution of K, regional blood flow rates, and K extraction fractions can be modified appropriately. For example, to apply the model to a non-resting adult male, we would alter blood flow rates and change the K extraction fraction for muscle to correspond to the greater blood flow. To apply the model to an adult female or to a child, we would reduce the fraction of total-body K in muscle and increase the fractions in other organs, modify the cardiac output and regional blood flow rates to the extent possible, and alter the extraction fraction for muscle if necessary to correspond to the different blood flow rate.

Comparisons of model predictions with observations of human subjects receiving oral or intravenous doses of radiopotassium (Burch, Threefoot, and Ray 1955; Corsa et al. 1950; Hamilton 1938) are made in Figs. 2 and 3. In Fig. 3 the model predictions are given in terms of a 'disequilibrium factor'. For a given tissue, the disequilibrium factor is defined as the fraction of the unexcreted tracer K in the substance at a given time after ingestion or injection of a unit activity of tracer K, divided by the fraction of whole-body K in that compartment at equilibrium. For urine, the disequilibrium factor at any given time is the urinary excretion rate of the administered unit activity of tracer K, divided by the urinary excretion rate of tracer K after equilibration with body K has been attained.

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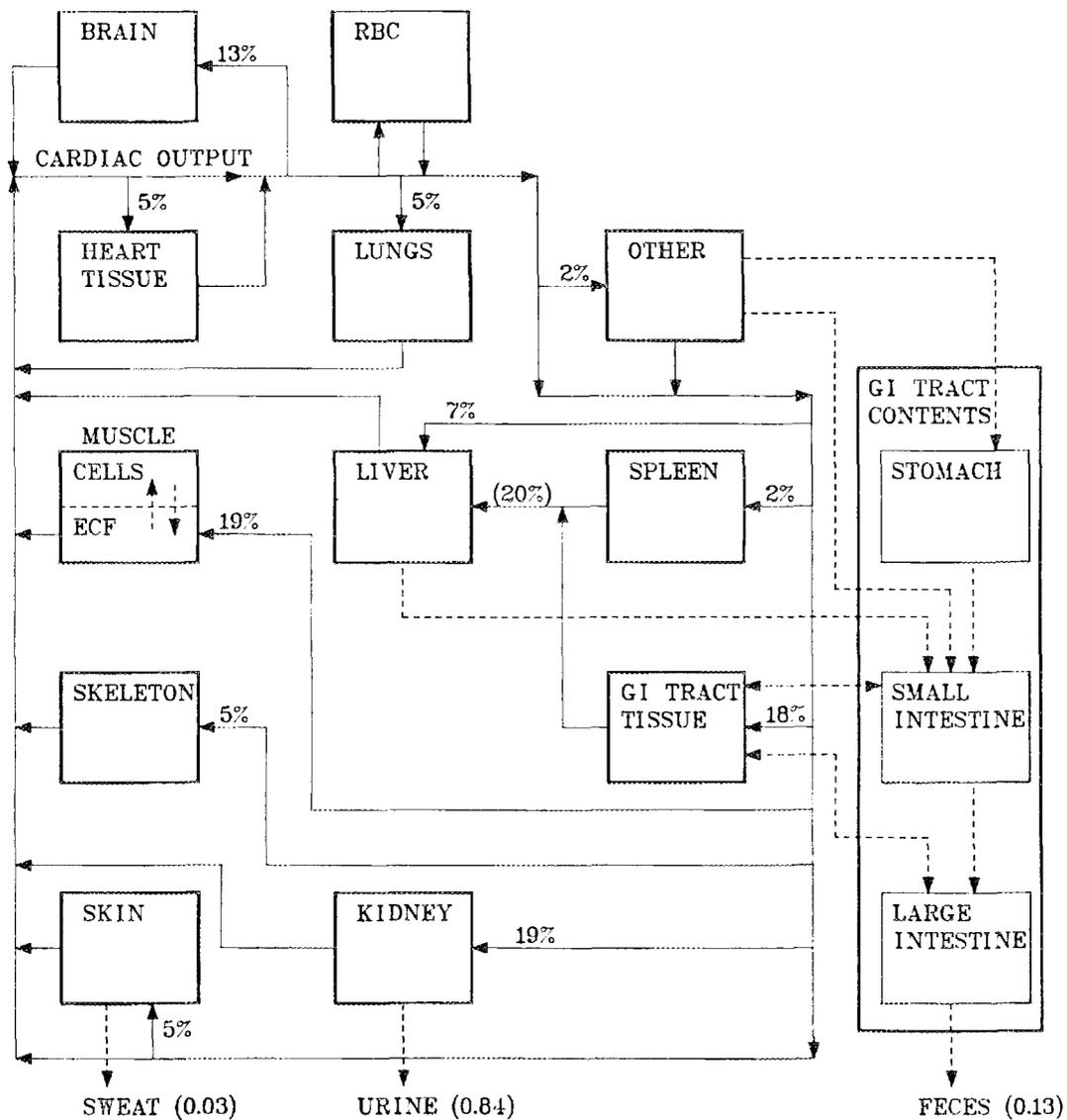


Figure 1. Direction of flow of potassium among compartments of model. Solid arrows indicate plasma flow and dashed arrows indicate flow not involving plasma. Numbers next to compartments refer to percentages of cardiac output passing through the compartments. Numbers to the right of sweat, urine, and feces are typical relative fractions of potassium excreted along these routes.

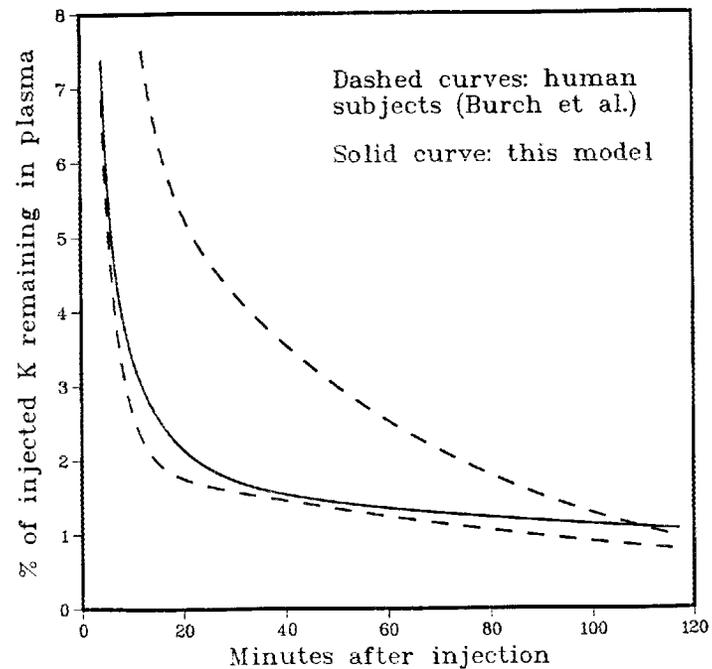
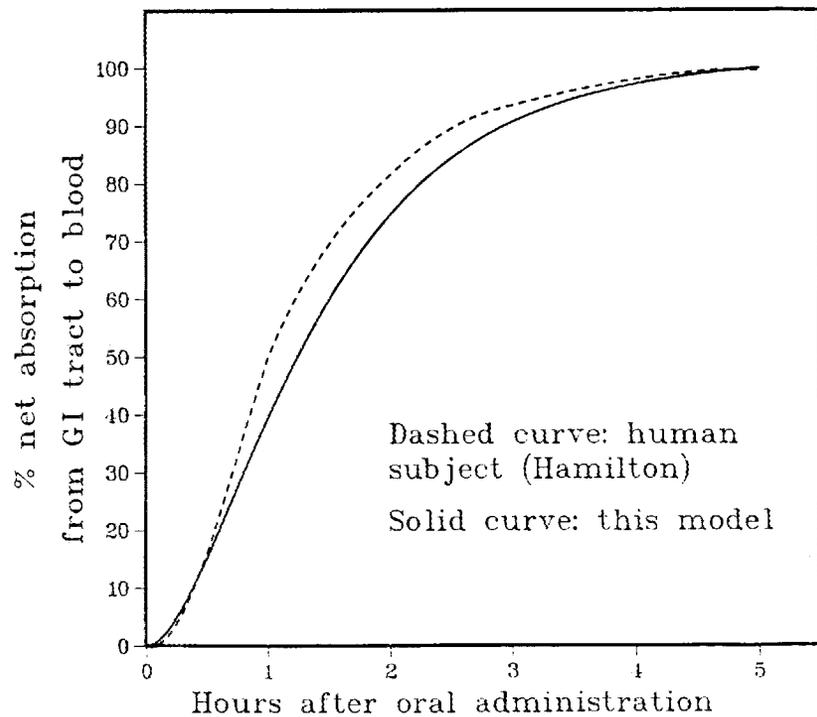


Figure 2. Net absorption of potassium from GI tract to blood and percent of injected potassium remaining in plasma, as measured in human subjects and predicted by this model.

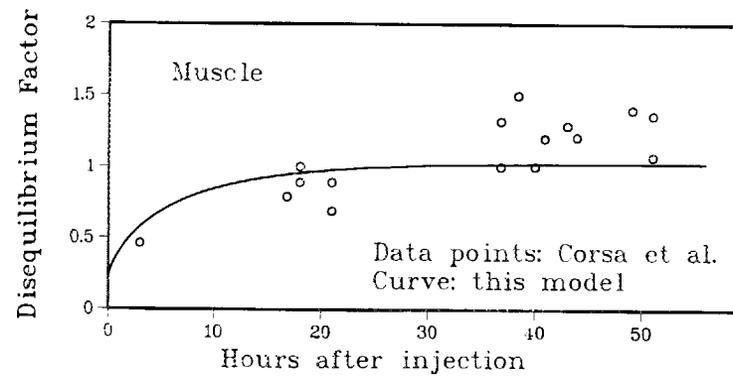
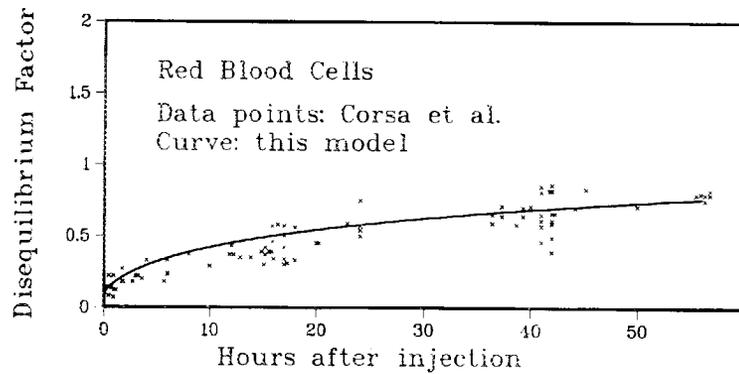
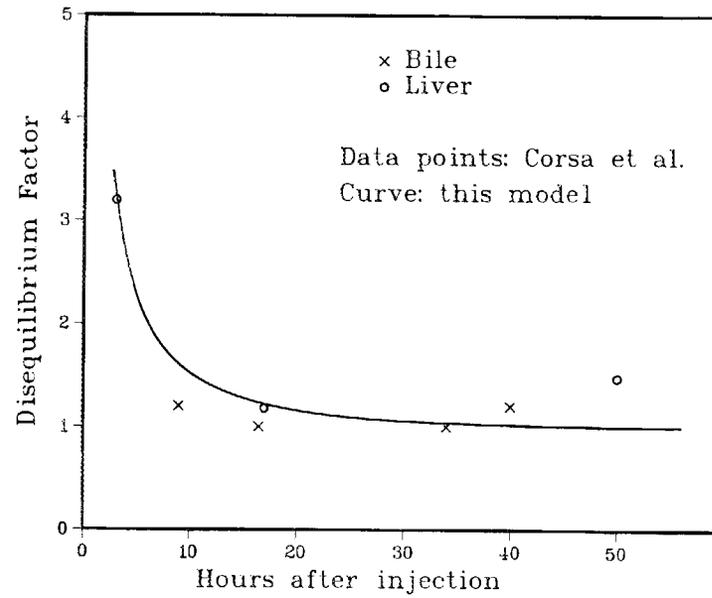
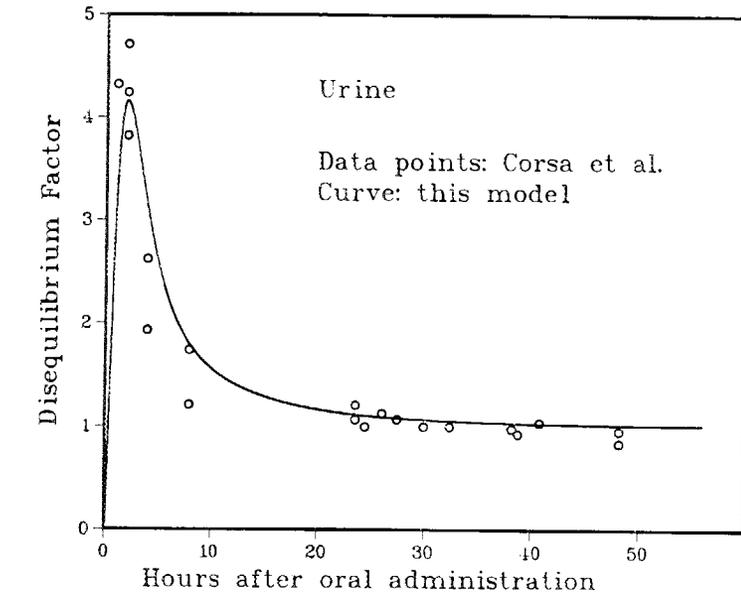


Figure 3. Relative concentration of ingested potassium in urine and injected potassium in liver, bile, red blood cells, and muscle, as measured in human subjects and predicted by this model.

Table 1. Inflow and outflow rates for compartments and subcompartments.

Compartment	Inflow ^a	Source	Outflow ^b	Destination
RBC	6	Plasma	0.38	Plasma
Kidneys	257	Plasma	209.4 4.6	Plasma Urine
Muscle ECF	242	Plasma	242	Plasma
	2.65	Muscle cells	500	Muscle cells
Muscle cells	500	Muscle ECF	2.65	Muscle ECF
Skeleton	56	Plasma	2.9	Plasma
Heart	67.5	Plasma	42.2	Plasma
Lungs	67.5	Plasma	33.8	Plasma
Stomach contents	3.3	Food	40	SI contents
	0.11	Other		
	0.48	GI tract walls		
SI contents	40	Stomach contents	11.35	Plasma
	0.36	GI tract walls	17	Liver
	0.03	Other	1.65	LI contents
	0.05	Liver		
LI contents	1.65	SI contents	0.43	Feces
	0.13	GI tract walls	0.06	Plasma
			0.09	Liver
GI tract walls	216	Plasma	0.48	Stomach contents
			0.36	SI contents
			0.13	LI contents
			23.6	Plasma
			35.4	Liver
Spleen	21	Plasma	7	Plasma
			10.5	Liver
Liver	100.8	Plasma (direct)	0.05	SI contents
	10.5	Spleen ^c	26.35	Plasma
	0.09	LI contents ^c		
	17	SI contents ^c		
	35.4	GI tract walls ^c		
Skin	67.5	Plasma	8.206 0.024	Plasma Sweat
Brain	2.9	Plasma	0.38	Plasma
Other	120	Plasma	7.06 0.11 0.03	Plasma Stomach contents SI contents

^aSource compartment volumes of potassium per day, except food, which is in g/day.

^bCompartment or subcompartment volumes of potassium per day.

^cA brief intermediate residence in plasma is ignored.

PREDICTING THE RETENTION OF CESIUM IN THE INDIVIDUAL

R. W. Leggett

INTRODUCTION

Various factors have been offered to explain the wide variation in the retention of cesium among humans and to predict retention patterns for this element in individuals. For example, the biological half-time of cesium has been expressed as an increasing function of body mass and also as an increasing function of age throughout life (Eberhardt 1967; Cryer and Baverstock 1972; McCraw 1965). Some early investigators attempted to describe accumulation of Cs-137 in the body in terms of discrimination factors between cesium and the chemically similar element potassium, such as the ratio of Cs/K in the body to Cs/K in total diet, milk, total excreta, or urine (Anderson 1957; Booker 1959; McNeill and Trojan 1960), but such factors were found to oversimplify the physiological relationship between these two elements and were soon abandoned. The goal of this study was to construct a predictive model for retention of cesium in the individual. It was found that potassium is a useful index for this purpose, after all.

RESULTS OF THE STUDY

Whole-body retention of cesium in a person usually can be approximated closely by a two-exponential expression (ICRP 1979; NCRP 1977)

$$R(t) = a \exp(-0.693t/T_1) + (1 - a) \exp(-0.693t/T_2), \quad (1)$$

where $R(t)$ is the fraction of activity at reference time zero still retained in the body t days later, a and $1-a$ are fractions of the initial activity associated with two hypothetical compartments that together make up the total body, and T_1 and T_2 are the biological half-times of cesium in those compartments. After a review and analysis of the physiological and radiobiological literature on cesium and biologically similar elements, it was conjectured that the parameters a , T_1 , and T_2 of Eq. (1) could be expressed in terms of K_t , the amount of K in the total body. It was suspected that each of these parameters depends on the fraction F of K_t that is in skeletal muscle, where most

of the body's K resides; in turn, F appears to increase with K_t . It has been found that the skeletal muscles exchange Cs with plasma at a slow rate in comparison with most of the other tissues, particularly the viscera. In relative terms, a smaller value of F should correspond to a smaller slow-exchange pool for Cs and a larger amount of Cs entering plasma and available for excretion over the first few days after exposure. Thus, decreasing values of F should correspond to increasing values of the short-term fraction a in Eq. (1). Also, a smaller value of F means a smaller muscle pool, which should correspond to a smaller value of T_2 in Eq. (1), not only because of a potentially shorter time for a single turnover of that pool but also because of a smaller fraction of material being recycled to that pool. A similar argument applies to the correspondence between decreasing values of F and increasing values of T_1 , although the argument may be weaker in this case because of the more heterogeneous nature of the fast-exchange 'pool'. A positive correlation between F and K_t is suggested by some results. Differences with sex in the relations between K_t and the parameters of Eq. (1) might be expected since there could be differences with sex in the relation between the fraction F defined above and K_t .

The relation between K_t and the parameters of Eq. (1) was investigated using data from a study by Lloyd and coworkers (1973), who measured the retention of Cs-137 and Rb-83 in 38 persons of various ages, some healthy and some with muscle disease. It was found that T_2 increases with K_t in a nearly linear fashion (Fig. 1), and a also increases with K_t but in a nonlinear fashion (Fig. 2). A relation between K_t and T_1 is revealed indirectly by first relating K_t and a and then relating a and T_1 (Fig. 3). The pairs (K_t, T_2) for healthy males are approximated by the line

$$T_2 = -1.22 + 0.72K_t \quad (R=0.91). \quad (2)$$

The pairs (K_t, T_1) for healthy males may be better approximated by an exponentially declining curve:

$$T_1 = 18 \exp(-0.016K_t) \quad (R=0.87). \quad (3)$$

Curves (2) and (3) intersect at about $K_t = 20$ g, which is the value of K_t at approximately one year of age. At $K_t = 20$ g, $T_1 = T_2 = 13$. For

smaller values of K_t it will be assumed that $T_1 = T_2$; based on empirical data, this common value is assumed to decrease linearly with K_t from 22 days at $K_t = 5$ g (birth) to 13 days at $K_t = 15$ g (approximately 6 months of age), and then remain at 13 days through $K_t = 20$ g. For healthy males, the pairs (K_t, a) also may decline more in an exponential pattern than a linear one, with the best-fitting exponential function being

$$a = 0.81 \exp(-0.014K_t) \quad (R=0.92). \quad (4)$$

Because of the small number of data points for females, especially for small values of K_t , the information for males was used as a point of departure for constructing a model for females, by appealing to the apparent lack of difference with sex in K_t and in retention of cesium in young children. The model for females was taken to be the same as that for males through age 6-7 years ($K_t=43$ g), and functions agreeing with the model for males at $K_t=43$ g and representing the data for females for higher ages were found. These are given in Eqs. (5-7).

$$T_2 = -17.1 + 1.09K_t \quad (K_t > 43 \text{ g}); \quad (5)$$

$$T_1 = 14 \exp(-0.01K_t) \quad (K_t > 43 \text{ g}); \quad (6)$$

$$a = 0.89 \exp(-0.016K_t) \quad (K_t > 43 \text{ g}). \quad (7)$$

The data of Lloyd and coworkers also were used to compare the relative values of the indices age, total body mass, and total body potassium in predicting the equivalent biological half-time T_Q of cesium in humans. The equivalent biological half-time for an individual is defined as the average of all the component half-times in his/her retention function, each weighted by the relative component size (coefficient). The coefficient of correlation, R , between T_Q for cesium in Lloyd's subjects and the factor age, weight, or K_t is given in Table 1 for each of three categories: children, all subjects, and adults (age 18 years or greater). In all three categories T_Q is more highly correlated with K_t than with either age or total body mass.

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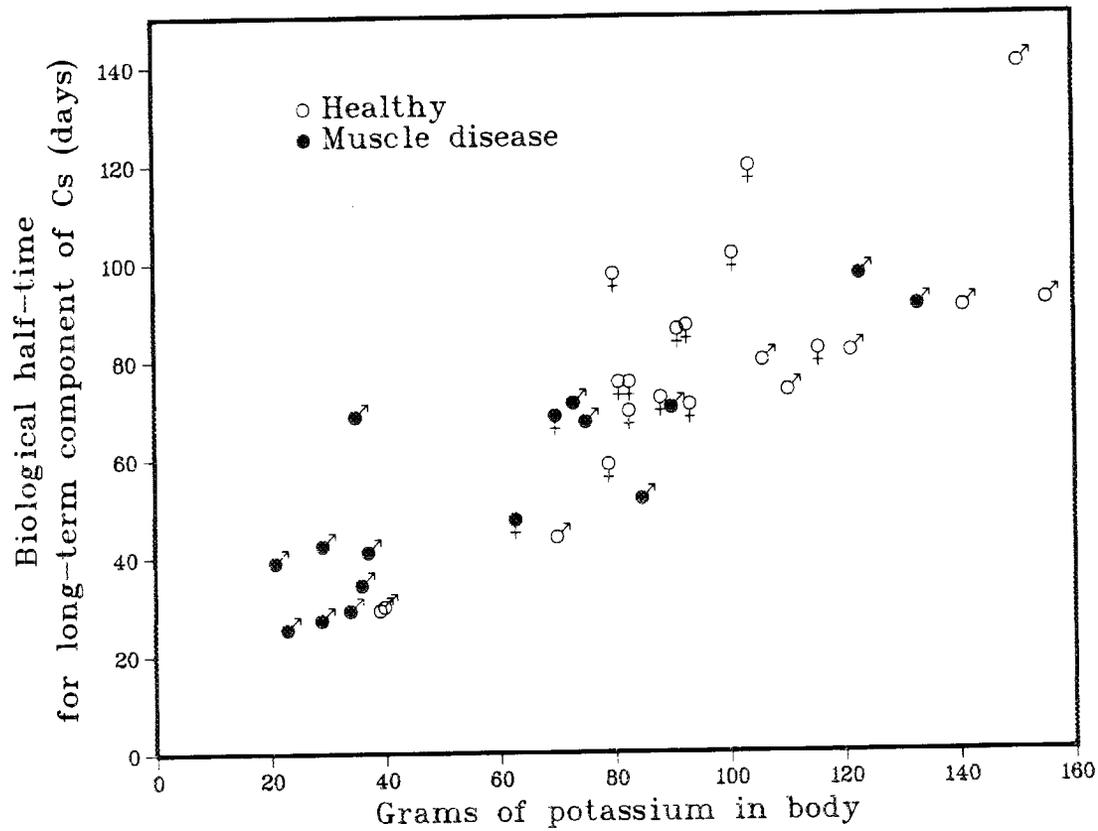


Figure 1. Total-body potassium vs. the biological half-time for the long-term component of cesium retention in the Utah subjects.

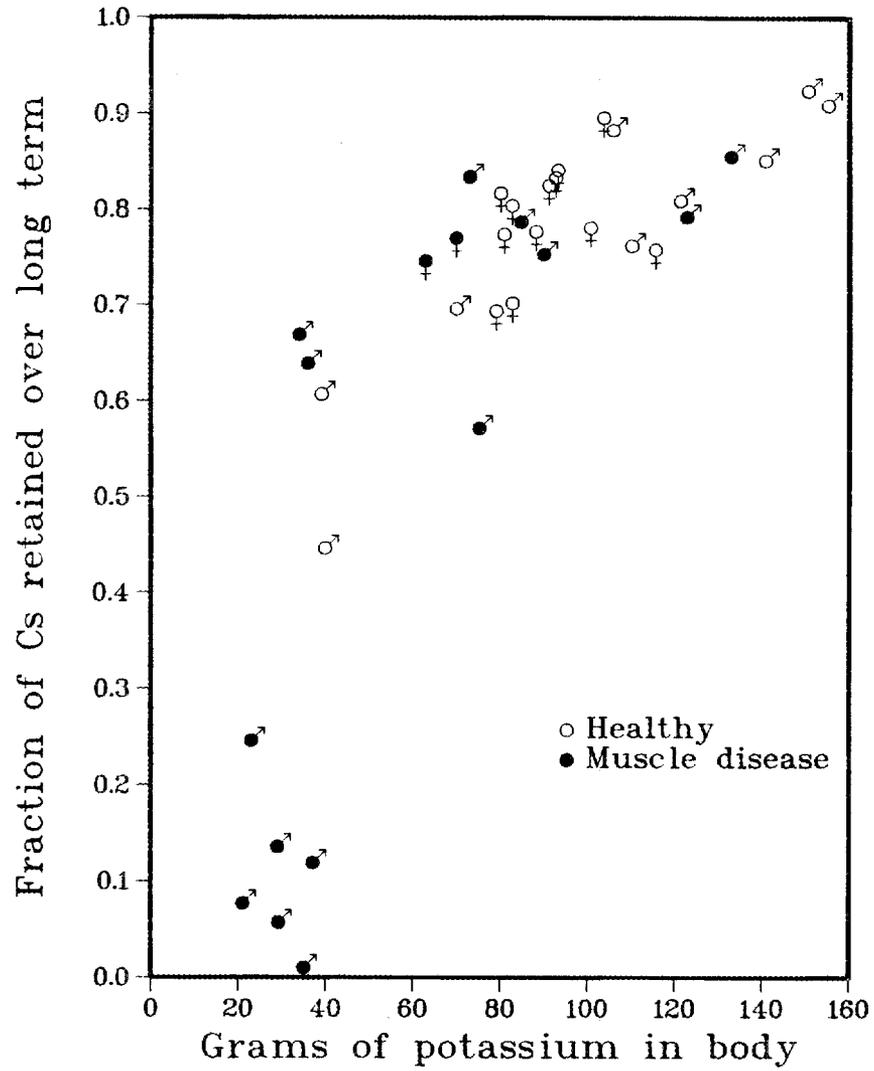


Figure 2. Total-body potassium vs. the fraction of cesium retained over a long term by the Utah subjects.

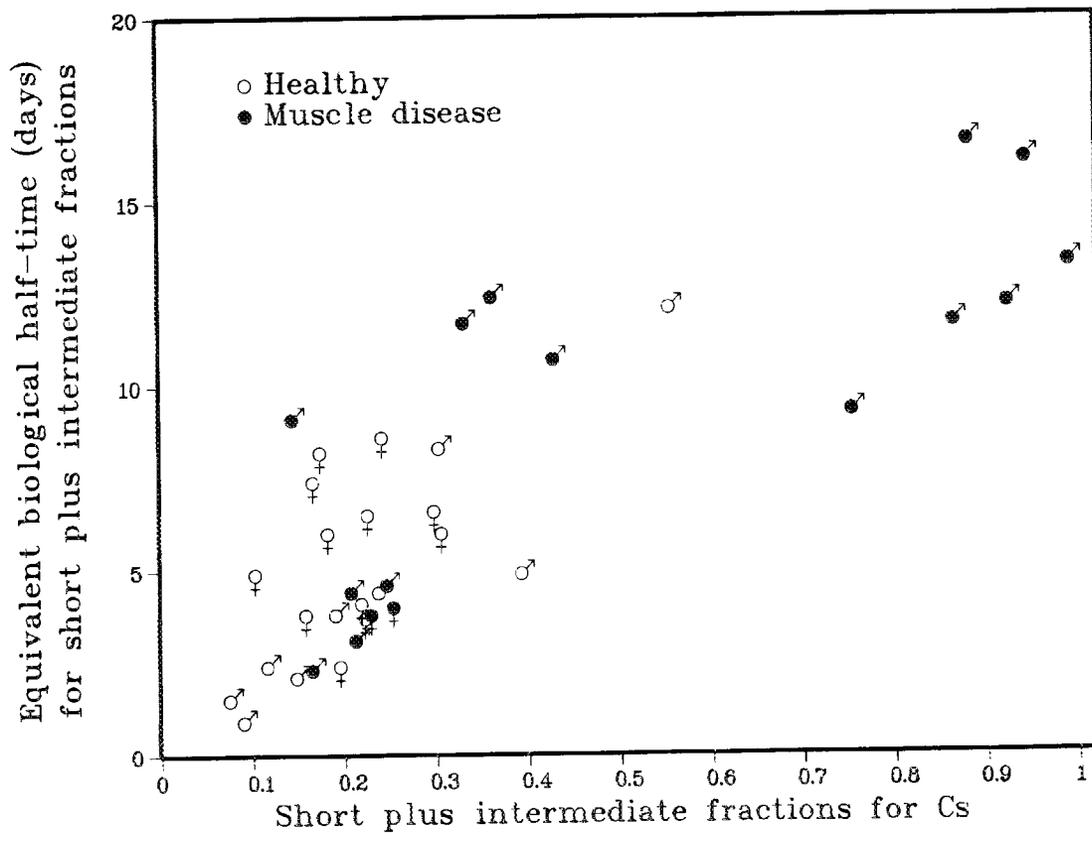


Figure 3. Total-body potassium vs. the equivalent biological half-time for the fraction of cesium in the Utah subjects associated with early excretion.

Table 1. Coefficients of correlation between the equivalent biological half-time T_Q of Cs and age, weight, and K_t based on data of Lloyd et al.

Parameters	Correlation coefficient		
	All subjects	Children	Adults
Age vs T_Q	0.53	0.45	0.05
Weight vs T_Q	0.74	0.69	0.52
K_t vs T_Q	0.89	0.97	0.78

MODELING THE BEHAVIOR OF DAUGHTER NUCLIDES BORN IN THE BODY

R. W. Leggett, D. E. Dunning, Jr., and K. F. Eckerman

INTRODUCTION

For some radionuclides, estimates of dose to radiosensitive tissues depend strongly on assumptions regarding retention of decay products. Examination of experimental data reveals that a variety of complex relationships exists between the retention of parent and daughter nuclides, and that broad assumptions concerning decay products such as those used in Publication 2 (ICRP 1959) or Publication 30 (ICRP 1979) of the ICRP cannot adequately cover all important parent nuclides. The purpose of this study is to examine problems related to modeling retention of decay products and to suggest ways of improving present methods.

DEPENDENCE OF ESTIMATES OF DOSE ON ASSUMPTIONS
CONCERNING RETENTION OF DECAY PRODUCTS

We have examined, for various parent radionuclides, the change in estimates of activity and dose equivalent as assumptions concerning behavior of decay products are altered. The characteristic retention function used for each nuclide (that is, the retention function that would apply to the nuclide if it were injected into the bloodstream) is that given in ICRP 30, although some computationally convenient approximations have been used to describe retention of the alkaline earth elements. To make our analysis manageable we restricted attention to a choice among the following simple assumptions concerning decay products:

Assumption A: Growth of the daughter is viewed as a fresh deposit in the organ from body fluids, and the daughter is assigned its own characteristic retention function for the organ.

Assumption B: The daughter is assumed to follow the metabolic pathways of the parent, and each term of the retention function for the

parent is assumed to represent an actual physical compartment. The daughter is assumed to remain in the compartment where it was born until removed with the same removal rate as the parent.

Assumption C: The daughter is assumed to follow the metabolic pathways of the parent, but the terms of the parent's retention function are not considered as actual physical compartments. In this case the total activity of the parent in the organ is continuously pooled, and each daughter atom is assigned the total retention function of the parent, rather than a single term from that function. (If the retention function has only one term, then this assumption is the same as Assumption B.)

Assumption A was used in ICRP 2 and Assumption B was used in ICRP 30. Because of the uncertainty regarding the physical significance of the multiple terms of the retention functions in ICRP 30, Assumption C may be as reasonable as Assumption B for use with those retention functions. These assumptions are by no means exhaustive; they were chosen for our analysis simply because of their common use and because their dosimetric implications can be determined using the ICRP 30 modeling approach.

The committed effective dose equivalent (that is, the sum of the dose equivalents in individual organs, each weighted by an organ-weighting factor as described in Publication 26 of the ICRP (ICRP 1977) for a fifty-year period following injection of a parent nuclide into blood was calculated for eight parent nuclides using the above Assumptions A, B, and C. Results are listed in Table 1. It is apparent that, depending on the parent nuclide, estimates of dose may be strongly affected, slightly affected, or virtually unaffected by the choice among these three assumptions.

IMPROVING PRESENT DOSIMETRIC METHODS FOR CHAINS OF RADIONUCLIDES

We have concluded that dosimetry for chains of radionuclides could be improved in many cases by avoiding the type of blanket assumptions made in ICRP 2 or ICRP 30 and by considering parent radionuclides on a case by case basis. This could be done most effectively if metabolic models were revised to reflect the actual processes involved in the

retention and translocation of nuclides. For the most part the models recommended in ICRP 30 are not flexible enough to allow incorporation of physiological considerations. This is because these models usually were derived as fits to experimental data, and the terms of their mathematical representations do not correspond to identifiable anatomical or physiological entities. Thus, we have concluded that the problem more accurately describing the behavior of radioactive progeny born in the body cannot be addressed adequately without changing the basic modeling approach used in ICRP 30 for parent nuclides.

Physiological considerations appear to be particularly important in the case of bone-seeking radionuclides. If a nuclide produced on bone surfaces is classified as a 'surface-seeking' nuclide, then it may be reasonable to assume that it remains on bone surfaces until removed by processes of burial and resorption. If the nuclide is not a surface-seeker, then its migration from the parent might be limited by the half-life of the nuclide and the rate of entry of surface material into the bone volume. Except where there is information to the contrary (such as for noble gases), radionuclides produced in bone volume should probably be assumed to remain there until released by bone remodeling processes. In general, the daughter may be less likely to remain with the parent over an extended period in trabecular bone volume than in cortical bone volume, because trabecular bone is completely remodeled in a few years, and the daughter may not be recycled to the same extent as the parent. It appears that metabolic models will have to include (1) cortical and trabecular bone as separate compartments (some ICRP models already do this) and (2) explicit consideration of the processes of bone addition and resorption before the problem of the migration of daughter products from the skeleton can be handled adequately.

Examples indicate that some radionuclides produced in soft tissues may migrate extensively and quickly from the parent, while others remain with the parent to a large extent. If there is insufficient experimental and/or physiological information to guide the development of a model for a particular daughter in soft tissue or in bone, then one conservative approach would be to estimate doses under different plausible assumptions regarding decay products, and apply the highest estimate of dose.

A more complete description of this work will appear in Radiat. Prot. Dosim. 9, 1985, 77-91.

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Table 1. Committed effective dose equivalent and maximum organ dose equivalent from radionuclide deposition in blood (Sv/Bq).

Nuclide	Committed Effective Dose Equivalent				Maximum Committed Dose Equivalent to any Organ				
	A	B	C	Ratio B:A	A	B	C	Ratio B:A	Organ
Sr-90	1.1E-07	1.1E-07	9.8E-08	1.0	1.3E-06	1.3E-06	1.1E-06	1.0	Bone surface
Sr-91	2.1E-10	1.5E-10	1.5E-10	0.71	5.3E-10	3.5E-10	3.5E-10	0.66	Red marrow
Cs-137	1.4E-08	1.4E-08	1.4E-08	1.0	1.4E-08	1.4E-08	1.4E-08	1.0	Whole body
Bi-210	6.2E-08	7.3E-09	4.6E-09	0.12	9.7E-07	1.0E-07	6.2E-08	0.10	Kidney
Pb-210	6.3E-07	7.4E-06	1.9E-06	12	8.8E-06	1.1E-04	3.4E-05	12	Bone surface
Th-228	1.9E-04	4.9E-04	4.9E-04	2.6	4.6E-03	1.2E-02	1.2E-02	2.6	Bone surface
Th-232	6.2E-04	3.6E-03	3.6E-03	5.81	1.6E-02	9.2E-02	9.2E-02	5.8	Bone surface
Cm-240	7.0E-06	1.3E-05	1.3E-05	1.9	1.2E-04	2.2E-04	2.2E-04	1.8	Bone surface

A, B, and C correspond to assumptions listed in text.

A METHOD FOR ESTIMATING THE SYSTEMIC BURDEN OF PLUTONIUM FROM URINALYSES

R. W. Leggett and K. F. Eckerman

INTRODUCTION

For many years Langham's urinary excretion model (1950) served as the primary tool for estimating intakes and systemic burdens of plutonium from urinalyses, but it has gradually become evident that this model substantially overestimates systemic burdens at extended times after exposure. In this study we compare three recent approaches that are believed to produce more accurate bioassay models: (1) modification of Langham's model using autopsy and urine data for former plutonium workers, (2) empirical curve fitting to updated data for humans injected with plutonium, and (3) modeling of the retention and excretion of plutonium using general physiological considerations combined with plutonium-specific radiobiological data. These three approaches are shown to yield fairly consistent estimates of the urinary excretion rate for several decades after contamination of blood. Estimates from the three approaches are combined to obtain a set of predicted urinary excretion rates for 1 to 20,000 days after contamination of blood. A simple method is developed for using these excretion rates to calculate intake rates and systemic burdens from exposures in which the general pattern of intake to blood is known.

COMPARISON OF THE THREE APPROACHES

Over the past few years it has become evident that Langham's equation underestimates the rate of excretion of Pu by a factor $F(t)$ that increases with time t . This conclusion is based in part on comparative autopsy and excretion data for plutonium workers exposed several years ago (Norwood and Newton 1975; McInroy 1976). While fraught with uncertainties, these data indicate that Langham's equation may underestimate the urinary excretion rate for Pu by a factor of perhaps 2-3 at 4-8 years, by a factor of perhaps 4-7 at 10-15 years, and roughly by an order of magnitude at 25-30 years. A revision of Langham's model based on our analysis of these data is indicated in Fig. 1.

Updated information on Langham's Pu-injected subjects also indicates that the urinary excretion of Pu is greater than originally projected. In particular, Rundo and coworkers (1976) collected samples of excreta from two of Langham's subjects at 27 years after injection. The measurements indicate that Langham's equation underestimates the urinary excretion rate by a factor of 7-13 at 27 years.

Recently S. R. Jones (1985) reanalyzed the human injection data and developed an empirical model (curve fit) from the adjusted data. This curve is compared with the Langham model in Fig. 2. The version of Jones' model shown here includes the tentative adjustment (approximately a 25% increase at times greater than 3 years) that Jones added 'in proof' after learning of W. D. Moss' expected corrections and additions to Langham's published data on the Pu-injected subjects.

Another estimate of the urinary excretion of Pu over an extended period can be made using a recent model of Leggett (1985) which was constructed using general physiological considerations and Pu-specific radiobiological information related to the retention, translocation, and excretion of Pu that has reached the bloodstream. A comparison of estimates derived from the model of Leggett, the model of S. R. Jones, and the revision of the Langham model based on recent data for former Pu workers is made in Fig. 3.

A SUGGESTED METHOD

The values derived from the three different approaches have been combined in Table 1 to yield a urinary excretion model that gives consideration to plutonium injection data, autopsy and urinary data for plutonium workers, and physiological and radiobiological information on the behavior of plutonium in the body. The weighting of the three approaches for a given period of time was based primarily on judgments concerning the relative strength of the logical support for each approach over that period.

The last column in Table 1, the total fraction of the injected Pu excreted in urine and feces by the end of the given interval, is based

on the estimates given in Table 1 for urinary excretion together with the time-dependent urine-to-feces ratio R predicted by the model of Leggett. These ratios are in close agreement with measured values of R at 1-138, 340, and 10,000-12,000 days after exposure.

To estimate systemic burdens and intakes of plutonium, the values $B(i)$ in Table 1 may be used in the following formula:

$$U = \sum_i A(i)B(i)L(i)$$

where

U is the measured urinary excretion of plutonium at some reference time $t=0$,

i is the index for the intervals indicated in Table 1,

$L(i)$ is the length of interval i ,

$A(i)$ is the average inflow rate of plutonium to blood from the respiratory or gastrointestinal tract or wounds during interval i .

Note that time is measured backwards from the time that the urine sample is taken.

The expression for $A(i)$ should contain only one unknown, which is to be found by solving this equation. Usually it is assumed that the 'shape' of the curve for inflow to blood is known, perhaps because of a knowledge of relative changes in the environment of the exposed person. The equation would then be used to estimate the magnitude of the intake function, which would in turn be used together with the last column of Table 1 to estimate the current body burden.

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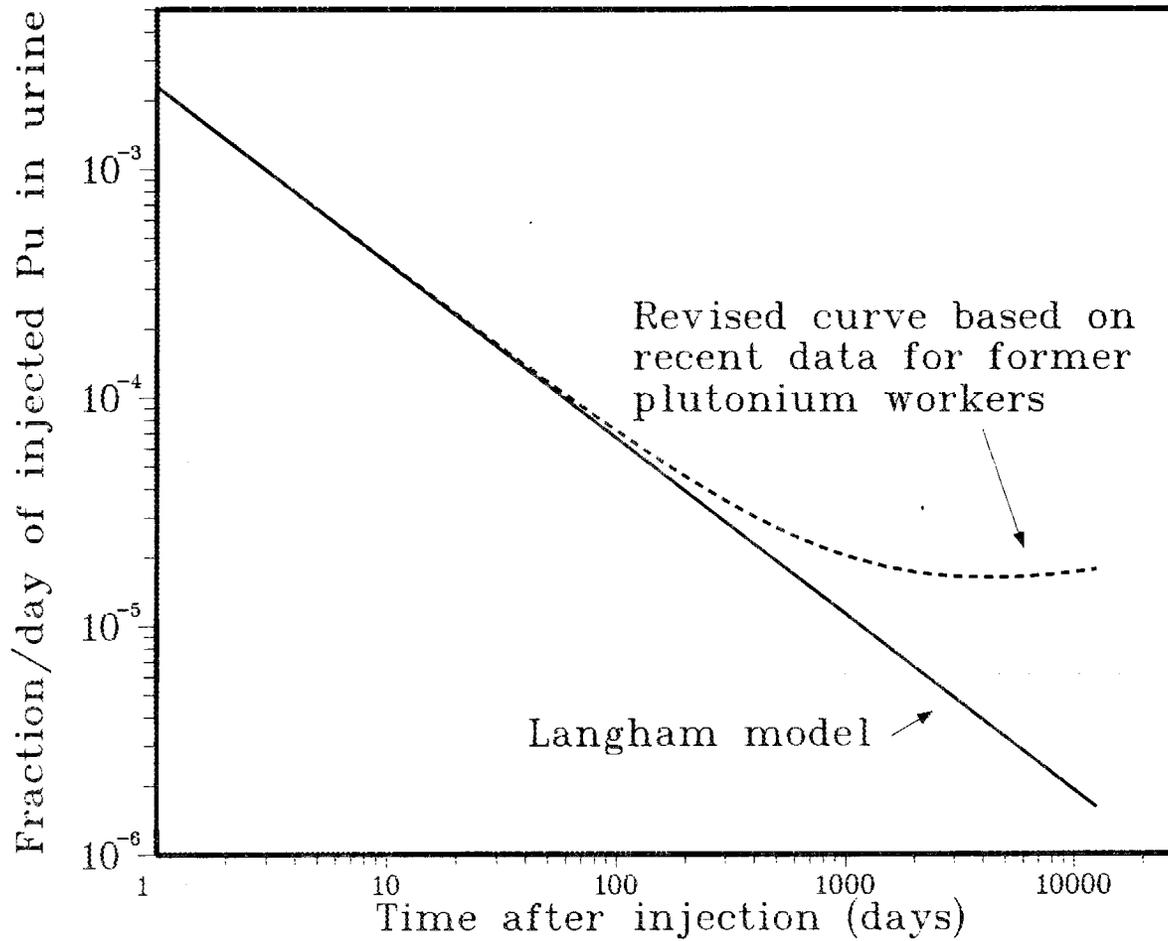


Figure 1. Comparison of Langham curve and revision based on data for Pu workers.

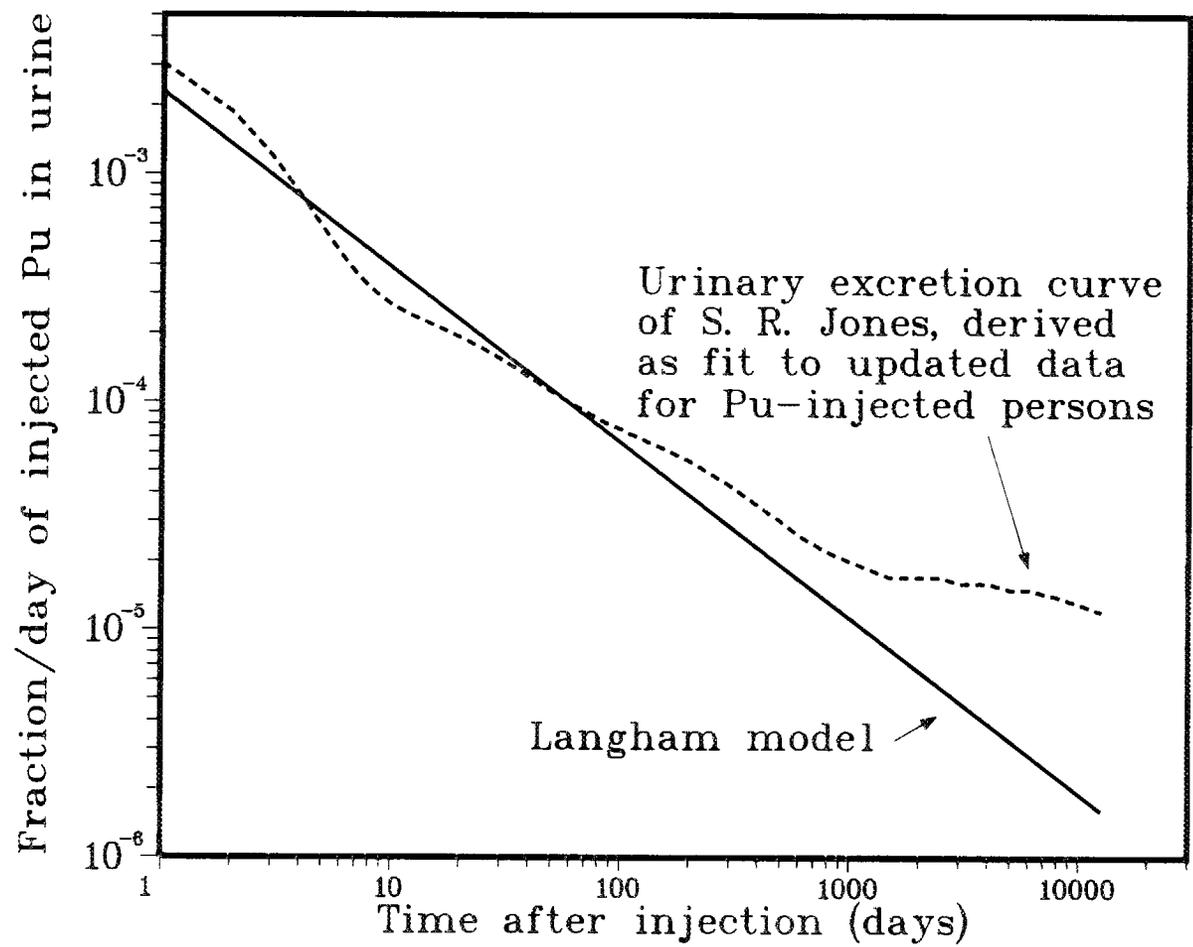


Figure 2. Comparison of Langham curve and curve of Jones based on updated information for Langham's subjects.

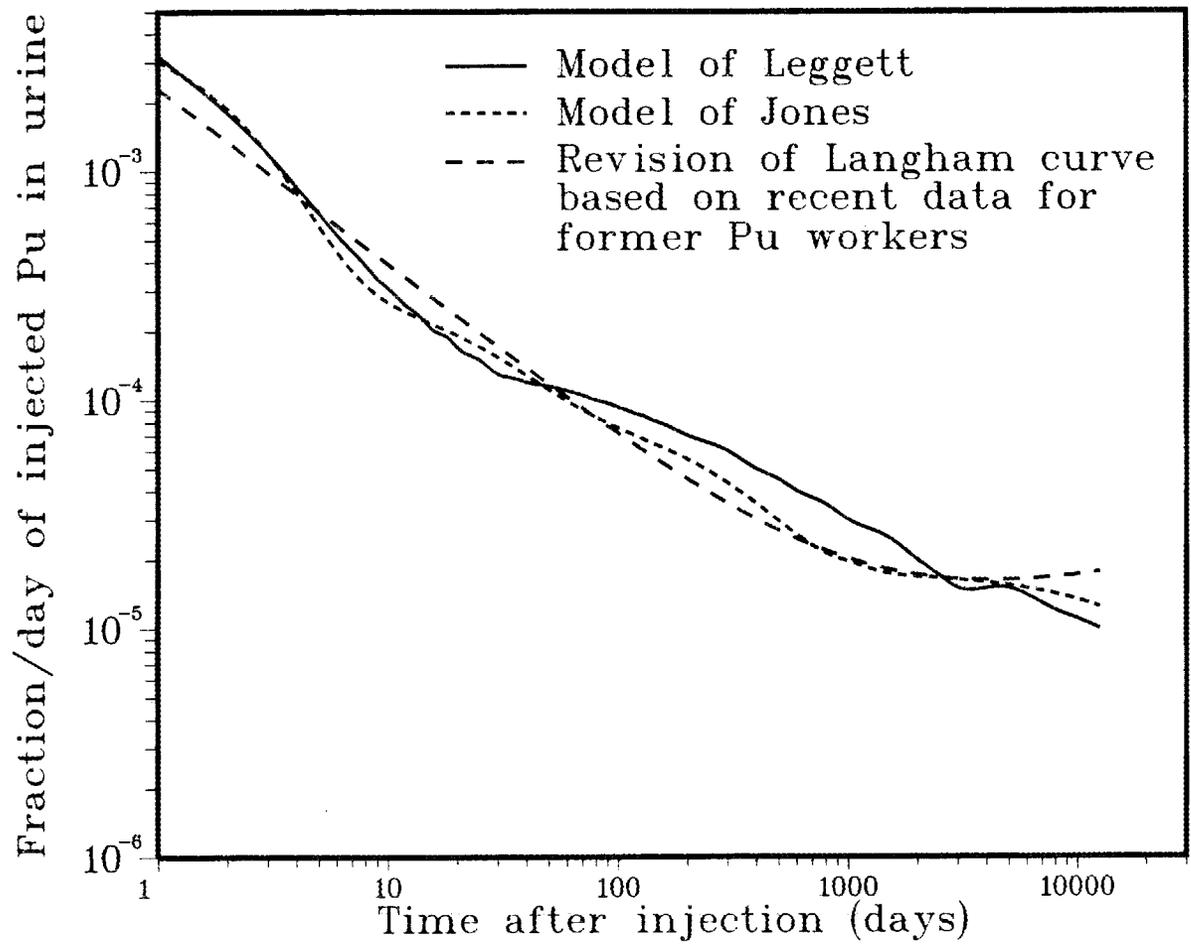


Figure 3. Comparison of model of Leggett, curve of Jones, and revision of Langham curve based on data for Pu workers.

Table 1. Values for the urinary and total excretion rates for plutonium recommended for estimating intakes and systemic burdens. Values are related to an initial unit activity in blood at time zero.

Interval number (i)	Period prior to urine sample (days)	Length of interval in days (L(i))	Average urinary excretion rate per day (B(i))	Fraction excreted by end of interval (urine plus feces)
1	0-1	1	0.004	0.0075
2	1-2	1	0.002	0.012
3	2-3	1	0.0015	0.015
4	3-4	1	0.0010	0.017
5	4-5	1	0.0007	0.019
6	5-6	1	0.0006	0.020
7	6-7	1	0.0005	0.022
8	7-8	1	0.00045	0.023
9	8-9	1	0.00040	0.024
10	9-10	1	0.00035	0.025
11	10-12	2	0.00030	0.026
12	12-14	2	0.00028	0.028
13	14-16	2	0.00026	0.029
14	16-18	2	0.00023	0.031
15	18-20	2	0.00020	0.032
16	20-25	5	0.00018	0.034
17	25-30	5	0.00016	0.036
18	30-35	5	0.00015	0.038
19	35-40	5	0.00013	0.039
20	40-50	10	0.00012	0.041
21	50-60	10	0.00011	0.043
22	60-70	10	0.000105	0.044
23	70-80	10	0.000100	0.046
24	80-90	10	0.000095	0.047
25	90-100	10	0.000090	0.049
26	100-110	10	0.000085	0.050
27	110-120	10	0.000080	0.051
28	120-130	10	0.000075	0.052
29	130-140	10	0.000070	0.053
30	140-150	10	0.000065	0.054
31	150-200	50	0.000060	0.058
32	200-300	100	0.000050	0.064
33	300-400	100	0.000045	0.069
34	400-500	100	0.000040	0.074
35	500-600	100	0.000035	0.079
36	600-800	200	0.000030	0.086
37	800-1000	200	0.000025	0.092
38	1000-1500	500	0.000022	0.11
39	1500-2000	500	0.000020	0.12
40	2000-2500	500	0.000018	0.13
41	2500-3000	500	0.000016	0.14
42	3000-4000	1000	0.000015	0.16
43	4000-5000	1000	0.000015	0.18
44	5000-6000	1000	0.000015	0.21
45	6000-8000	2000	0.000015	0.25
46	8000-10,000	2000	0.000015	0.29
47	10,000-12,500	2500	0.000015	0.34
48	12,500-15,000	2500	0.000012	0.38
49	15,000-17,500	2500	0.000011	0.42
50	17,500-20,000	2500	0.000010	0.46

ABSORBED FRACTION IN ACTIVE MARROW FOR ELECTRONS WITHIN TRABECULAR BONE

K. F. Eckerman

INTRODUCTION

Because the structure of trabecular bone could not be described in simple geometrical terms, Spiers and coworkers (Spiers 1969; Whitwell and Spiers 1978) introduced a method of calculating the energy deposition in which the geometries of the trabeculae and marrow cavities are represented by measured distributions of the chord-lengths across them. If the track of a particle is assumed to be straight then the total track in trabeculation is represented by path-lengths alternately selected in a random manner from the chord-length distribution for trabeculae and cavities. The energy loss of the electrons in the trabeculae and cavities can be computed from the range-energy relationship.

DISTRIBUTION OF CHORD-LENGTHS

There are many ways in which randomness of chords may arise in convex bodies, however only two are of interest here:

Mean-free-path randomness (or μ -randomness). A chord of a convex body is defined by a point in space and a direction. The point and direction are chosen randomly from independent, uniform distributions. This kind of randomness results, for example, if a convex body is exposed to a uniform, isotropic field of straight lines.

Interior radiator randomness (or I-randomness). A chord is defined by a point within the interior of the convex body and a direction. The point and direction are chosen randomly from independent, uniform distributions. This kind of randomness results, for example, if the convex body contains a uniform distribution of point sources, each of which emits radiation isotropically.

If charged particles (electrons) originate in a uniform-isotropic manner outside of a convex body (trabeculae or marrow cavity) one is dealing with μ -randomness, which is the situation under which chord-length distributions were measured by Spiers and coworkers (Spiers 1969; Beddoe et al. 1976; and Beddoe 1977). However, for particles

originating within a convex body I-randomness is applicable. The chord distributions under μ - and I-randomness have been shown to be related as (Kellerer 1971);

$$f_I(x) = \frac{x}{\langle x \rangle_\mu} f_\mu(x) \quad , \quad (1)$$

where

$f_I(x)$ and $f_\mu(x)$ denote the probability density functions for chord-lengths under I- and μ -randomness, respectively,

$\langle x \rangle_\mu$ denotes the mean value of the $f_\mu(x)$ distribution.

Equation (1) refers to the full chord; however, we are interested in 'half-chords' or rays formed by particles originating within the convex body. The probability density function for the ray-length distribution, $f_i(x)$, can be shown to be given by

$$f_i(x) = \frac{1}{\langle x \rangle_\mu} \left[1 - F_\mu(x) \right] \quad , \quad (2)$$

where $F_\mu(x)$ is the cumulative distribution function given as $F_\mu(x) = \int_0^x f_\mu(s) ds$.

From the preceding it is apparent that the mean ray-length for particles emitted internally to a convex body is one-half the mean chord-length for I-randomness, i.e., $\langle x \rangle_i = \frac{1}{2} \langle x \rangle_I$. The mean chord-length under μ -randomness, $\langle x \rangle_\mu$, is related to the volume, V , and surface area, S , of a convex body by Cauchy's theorem: $\langle x \rangle_\mu = 4 \frac{V}{S}$.

As an example, consider a sphere whose chord-length distribution for μ -randomness is simple and well-known, i.e., $f_\mu(x) = 2 \frac{x}{d^2}$, with mean chord-length $\langle x \rangle_\mu = \frac{2}{3} d$. From Eq. (1), the distribution for I-randomness in a sphere is $f_I(x) = \frac{3x^2}{d^3}$, for which the mean chord length is $\langle x \rangle_I = \frac{3}{4} d$. The probability density function of ray-lengths, obtained from Eq. (2), is

$$f_i(x) = \frac{3}{2d} \left[1 - (x/d)^2 \right] \quad , \quad (3)$$

with mean ray-length of $\langle x \rangle_i = \frac{3}{8} d$. The various distributions of chord- and ray-lengths in a sphere are depicted in Fig. 1.

Mean chord and ray-lengths for the trabeculae and marrow cavities of several trabecular bones of the skeleton of man are summarized in Table 1. Note that the parietal bone appears to be distinct from the other bones in that its thick trabeculae and small marrow cavities lead to a high $\langle t \rangle_{\mu} : \langle c \rangle_{\mu}$ ratio.

ABSORBED FRACTIONS FOR MONOENERGETIC ELECTRONS

The absorbed fraction in v from r , $\varphi(v \leftarrow r)$, is defined as

$$\varphi(v \leftarrow r) = \frac{\text{energy absorbed in target region } v}{\text{energy emitted by source region } r} . \quad (4)$$

Thus φ embodies the transport of the radiation under consideration as well as the geometric relationship of the regions. The absorbed fraction data developed here are for monoenergetic electrons emitted uniformly (by mass) and isotropically within the trabeculae and cavities of trabecular bone. The target region of interest is the active or red marrow (denoted as RM) for which we average the energy deposition over the marrow cavities.

The representation of paths for an electron of energy E and range in marrow R_{RM} are illustrated in Fig. 2. By use of chord-length distributions the three-dimensional geometry has been reduced to one dimension. Furthermore the two media (bone and marrow) nature of the problem can be reduced to a single media as the ratio of the range of electrons in marrow (RM) to that of bone (TB) is nearly constant over electron energies of interest here, that is: $R_{RM} \cong 1.75 R_{TB}$.

For irradiation of the active marrow by electrons originating within trabeculae, Monte Carlo sampling is used to select a chord-length, t , from the probability density function, $f_I(t)$, for the bone under consideration. A ray-length, t' , is then determined as $t' = \xi t$, where ξ is a random number uniform on the region $0 < \xi < 1$. The electron is tracked as it alternately passes through marrow cavities along lengths c_1, c_2, \dots , and trabeculae along chords t_1, t_2, \dots , selected by Monte Carlo sampling of the probability density functions $f_{\mu}(c)$ and $f_{\mu}(t)$, respectively. The electron is tracked until

$$1.75(t' + t_1 + t_2 + \dots) + (c_1 + c_2 + \dots) \geq R_{RM} \quad , \quad (5)$$

i.e., its energy has been deposited. The energy deposition in trabeculae (t's) and marrow cavities (c's) is calculated as the difference between the energy on entering and leaving a trabecula or cavity, in each case being determined from the residual range of the electron at that point in its track. The range-energy relationship was taken from Berger (1973). By tracking a large number of electrons in this manner, the absorbed fraction is obtained by dividing the total energy deposited in marrow cavities by the total energy of electrons simulated.

For electrons emitted within marrow cavities, the calculations proceed as above with first selection of a chord-length from the probability density function $f_I(c)$ and determination of a ray-length c' as noted above. The electron is tracked until

$$(c' + c_1 + c_2 + \dots) + 1.75(t_1 + t_2 + \dots) \geq R_{RM} \quad . \quad (6)$$

The energy deposition in the marrow cavities and the absorbed fraction are determined as discussed above. Typically, ten- to seventy-thousand electrons were tracked in each of the two absorbed fraction calculations. The statistical errors in the Monte Carlo calculations were less than 1%.

RESULTS

The absorbed fraction data for the parietal bone and lumbar vertebra of the skeleton of a 44-year-old male are shown in Fig. 3. At low electron energies, $\phi(RM \leftarrow TB)$ approaches zero and $\phi(RM \leftarrow RM)$ approaches unity. This limiting behavior reflects the fact that at low energy the range of electrons is small relative to the mean ray-lengths, $\langle t \rangle_i$ and $\langle c \rangle_i$, and thus the energy is locally deposited. At high energies, $\phi(RM \leftarrow RM) \cong \phi(RM \leftarrow TB)$ and the behavior is described as

$$\lim_{E \rightarrow \infty} \phi(RM \leftarrow TB) \cong \phi(RM \leftarrow RM) \cong \frac{\langle c \rangle_{\mu}}{\langle c \rangle_{\mu} + 1.75 \langle t \rangle_{\mu}} \quad , \quad (7)$$

i.e., the absorbed fraction is simply the fractional track length in the marrow cavities. The equality of the absorbed fractions at high energy arises as electrons traverse multiple trabeculae and cavities thus establishing an energy deposition pattern which is largely independent of the electron's origin.

The results of the calculations for the parietal bone of the skull and the lumbar vertebra are given in Tables 2 and 3 for a 44-year-old male and a 20-month-old child, respectively. The atypical structure of the parietal bone is reflected in the absorbed fraction data for either the child or the adult, however, difference with age appears to be less pronounced.

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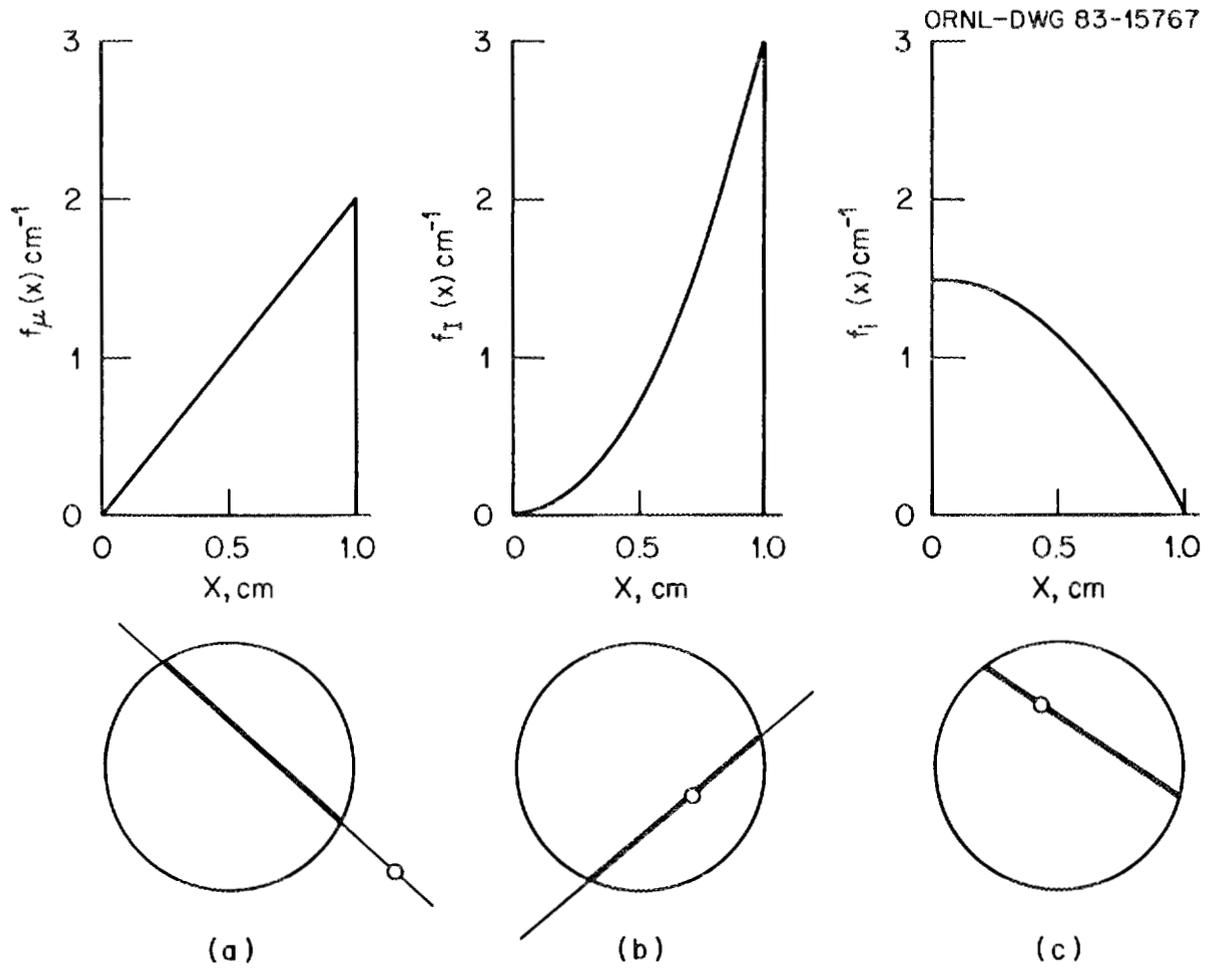


Figure 1. For a sphere of unit diameter, chord-length distributions for μ -randomness (a), I-randomness (b), and ray-length distribution (c).

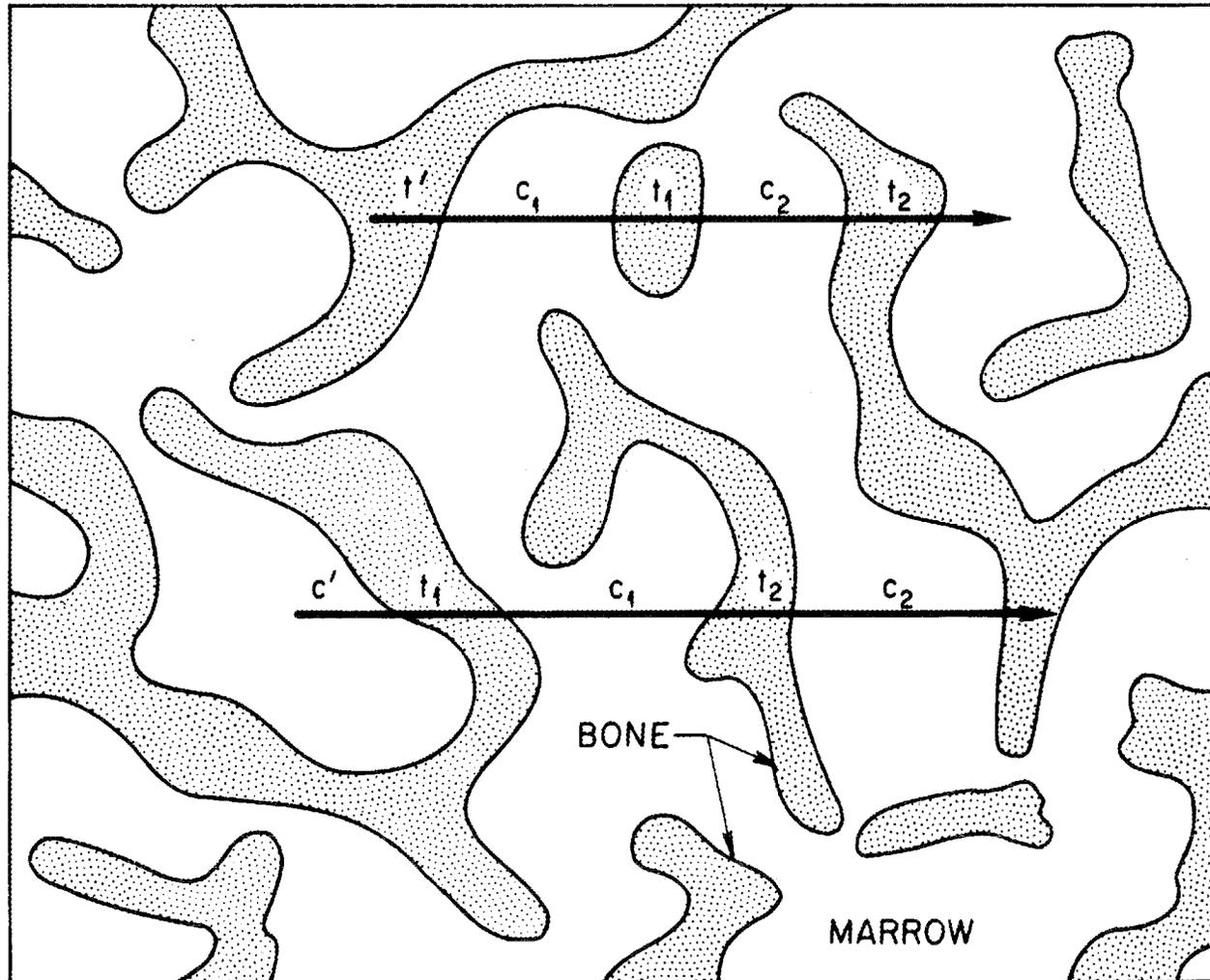


Figure 2. Schematic illustration of the track of an electron through trabecular bone. Monte Carlo sampling of chord-length distributions is used to determine the t 's and c 's defining the total track.

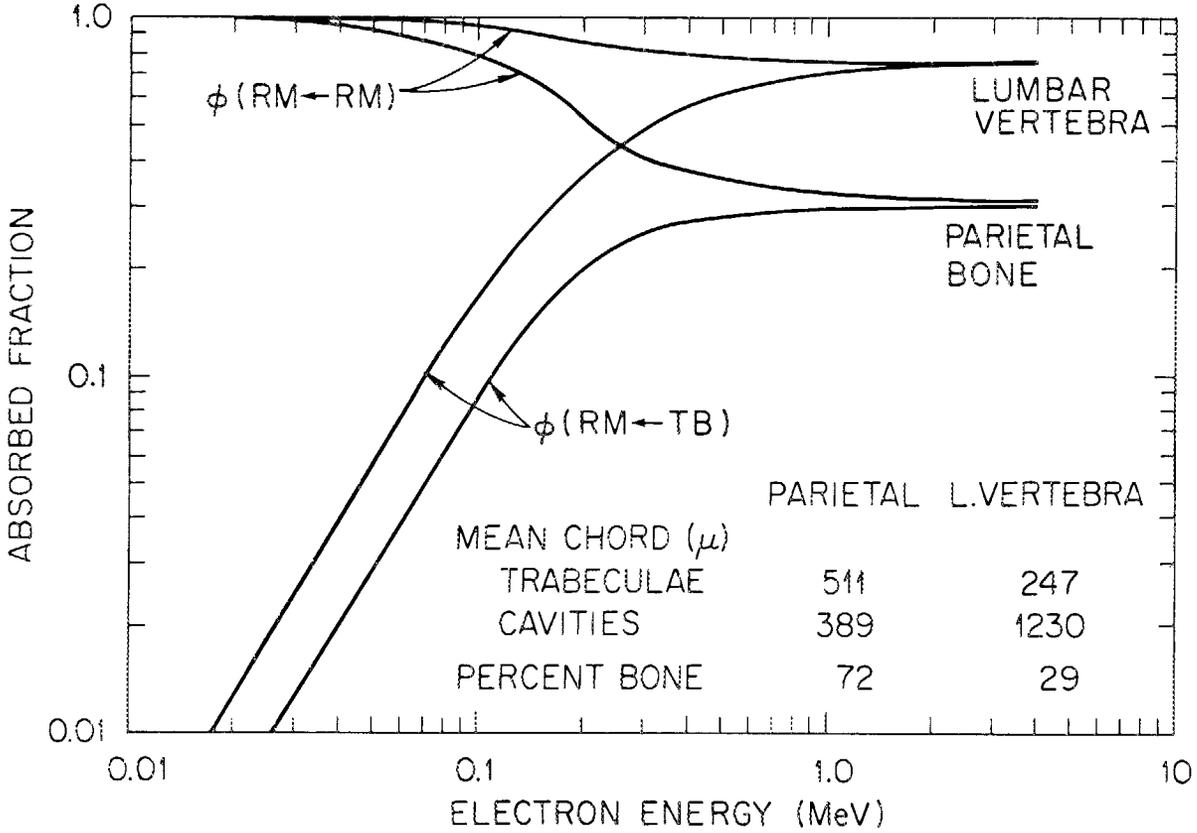


Figure 3. Absorbed fractions in active marrow RM for a monoenergetic electron source uniformly distributed in the trabeculae TB and the active marrow of the lumbar vertebra and parietal bone of the adult.

Table 1. Mean chord- and ray-lengths (μm) for trabeculae and marrow cavities in various bones of man.

Bones	Trabeculae ^a			Marrow Cavities ^a			
	$\langle t \rangle_{\mu}$	V_{μ}	$\langle t \rangle_i$	$\langle c \rangle_{\mu}$	V_{μ}	$\langle c \rangle_i$	$\langle t \rangle_{\mu} : \langle c \rangle_{\mu}$
44-year-old male ^b							
Parietal	511	0.570	401	389	0.784	347	1.31
Cervical vertebra	279	0.719	240	910	0.894	861	0.307
Lumbar vertebra	247	1.11	260	1228	1.12	1299	0.201
Rib	265	1.49	330	1706	1.09	1786	0.155
Iliac crest	242	0.675	203	904	0.647	745	0.268
Femur head	232	0.665	193	1157	0.901	1099	0.200
Femur neck	314	0.914	301	1655	0.905	1576	0.190
9-year-old child ^c							
Parietal	539			306			0.272
Cervical vertebra	162			906			0.179
Lumbar vertebra	168			857			0.196
Rib	231			1123			0.204
Iliac crest	180			744			0.242
Femur, head & neck	249			616			0.404
20-month-old child ^b							
Parietal bone	566	1.21	625	255	2.90	500	2.22
Lumbar vertebra	188	1.04	192	736	0.987	731	0.255
Rib	191	1.22	212	559	1.04	569	0.342
Iliac crest	181	1.43	206	575	0.873	539	0.315
Femur	197	0.865	184	789	1.10	830	0.250

^aNotation: $(\langle t \rangle_{\mu}, V_{\mu})$ and $(\langle c \rangle_{\mu}, V_{\mu})$ denote the mean and the fractional variance under μ -randomness for the trabeculae and marrow cavities, respectively. $\langle t \rangle_i$ and $\langle c \rangle_i$ denote the mean ray-length for trabeculae and cavities, respectively. Lengths are in units of μm .

^bComputed from the chord-length distributions of Whitwell (1973).

^cSee Tables 1 and 3 of Beddoe (1977).

Table 2. Absorbed fraction, ϕ , in active marrow, RM, from a uniformly distributed source of monoenergetic electrons in trabeculae, TB, and marrow of the parietal bone and lumbar vertebrae of a 44-year-old male.

Electron energy (MeV)	Parietal bone		Lumbar Vertebrae	
	ϕ (RM \leftarrow TB)	ϕ (RM \leftarrow RM)	ϕ (RM \leftarrow TB)	ϕ (RM \leftarrow RM)
0.010	1.95(-3)	0.994	3.94(-3)	0.999
0.015	3.29(-3)	0.990	7.81(-3)	0.997
0.020	5.77(-3)	0.983	1.29(-2)	0.996
0.030	1.23(-2)	0.969	2.59(-2)	0.991
0.040	1.98(-2)	0.950	4.34(-2)	0.985
0.050	2.94(-2)	0.927	6.26(-2)	0.979
0.060	4.03(-2)	0.901	8.25(-2)	0.971
0.080	6.34(-2)	0.854	1.31(-1)	0.953
0.10	8.80(-2)	0.794	1.83(-1)	0.935
0.15	1.53(-1)	0.654	3.12(-1)	0.888
0.20	1.99(-1)	0.538	4.17(-1)	0.848
0.30	2.58(-1)	0.415	5.47(-1)	0.808
0.40	2.71(-1)	0.376	5.97(-1)	0.793
0.50	2.76(-1)	0.358	6.25(-1)	0.779
0.60	2.82(-1)	0.346	6.48(-1)	0.767
0.80	2.88(-1)	0.335	6.74(-1)	0.765
1.0	2.93(-1)	0.327	6.90(-1)	0.757
2.0	2.97(-1)	0.317	7.17(-1)	0.747
3.0	3.00(-1)	0.311	7.22(-1)	0.747
4.0	3.01(-1)	0.308	7.27(-1)	0.744

Table 3. Absorbed fraction, ϕ , in active marrow, RM, from a uniformly distributed source of monoenergetic electrons in trabeculae, TB, and marrow of the parietal bone and lumbar vertebrae of a 20-month-old child.

Electron energy (MeV)	Parietal Bone		Lumbar Vertebrae	
	ϕ (RM \leftarrow TB)	ϕ (RM \leftarrow RM)	ϕ (RM \leftarrow TB)	ϕ (RM \leftarrow RM)
0.01	1.62(-3)	0.990	4.66(-3)	0.997
0.015	3.44(-3)	0.981	9.90(-3)	0.995
0.02	6.09(-3)	0.969	1.65(-2)	0.992
0.03	1.24(-2)	0.947	3.39(-2)	0.984
0.04	1.91(-2)	0.920	5.67(-2)	0.973
0.05	2.80(-2)	0.889	8.01(-2)	0.962
0.06	3.46(-2)	0.858	1.12(-1)	0.949
0.08	5.12(-2)	0.789	1.74(-1)	0.922
0.10	7.09(-2)	0.724	2.34(-1)	0.892
0.15	0.106	0.591	3.74(-1)	0.829
0.20	0.130	0.501	4.70(-1)	0.786
0.30	0.154	0.401	5.58(-1)	0.750
0.40	0.168	0.354	5.94(-1)	0.730
0.50	0.176	0.328	6.26(-1)	0.722
0.60	0.179	0.308	6.38(-1)	0.718
0.80	0.181	0.283	6.51(-1)	0.706
1.0	0.188	0.267	6.62(-1)	0.708
2.0	0.196	0.238	6.78(-1)	0.698
3.0	0.199	0.224	6.81(-1)	0.696
4.0	0.201	0.221	6.84(-1)	0.696

ORGAN DOSIMETRY FOR JAPANESE A-BOMB SURVIVORS

G. D. Kerr, K. F. Eckerman, J. S. Tang, J. C. Ryman, and M. Cristy

INTRODUCTION

Studies for reassessment of A-bomb radiation dosimetry in Hiroshima and Nagasaki are underway both in Japan and the U.S. (Bond and Thiessen 1982; Thompson 1983; Kato et al. 1984). The U.S. effort involves the Oak Ridge National Laboratory, other national laboratories, the University of Utah, and several private consulting firms. One of our group's main tasks is the reassessment of various organ-dose parameters related to a survivor's exposure to neutrons and gamma rays. The medical follow-up studies of the Hiroshima and Nagasaki populations by the Radiation Effects Research Foundation provide data on dose-related parameters such as the location of survivors and their shielding by surrounding structures at the times of the bombings. Only very preliminary results are available from reassessment studies concerning the delayed fireball radiations from the weapons and the shielding by houses and other structures. Hence, our calculations of organ doses are presently limited to the prompt radiations from the weapons in the idealized situation of an A-bomb survivor exposed in the open (or in the absence of any shielding by structures or terrain).

CALCULATIONAL METHODS

Absorbed doses in selected organs have been investigated relative to in-air tissue kerma. The following three components of the absorbed doses are considered: prompt gamma rays (g), prompt neutrons (n), and autogammas produced by prompt neutron interactions in the body (n,g). In our organ-dose calculations, we are currently using a series of six mathematical phantoms of the body and principal internal organs (Cristy 1980). This series has total-body masses ranging from 3.5 kg for a newborn infant to 70 kg for an adult Caucasian male as defined by the International Commission on Radiological Protection. The mathematical

phantom with a total-body mass of 55 kg was used in our organ-dose calculations for A-bomb survivors exposed as adults.

Our organ-dose calculations employ adjoint Monte Carlo techniques embodied in our computer code, MORSE-SGC/PHANTOM, which is a modified version of the MORSE-SGC code (Fraley 1976). Compilation speed and precision are improved by using (a) fictitious scattering in the Monte Carlo tracking of gamma rays and neutrons (Cramer 1978) and (b) mathematical equations, instead of the standard combinatorial-geometry package of the MORSE-SGC code, to describe the geometrical configuration of the total body and various internal organs (Ryman et al. 1985). Our computer code, FOLD, is then used to couple the adjoint Monte Carlo results to the differential energy and angular fluence of a radiation field of interest by the use of a surface integral approximation (Hoffman et al. 1972). The FOLD code first calculates the spectral fluence of unscattered and scattered neutrons or photons within a specific organ of interest, and then uses fluence-to-dose response factors to obtain the absorbed dose in that organ for the radiation field of interest (Kerr 1982; Kerr and Eckerman 1985). The radiation fields of interest here come from air transport calculations for the prompt radiation from the A-bomb explosions in Hiroshima and Nagasaki (Kerr, Pace, and Scott 1983).

Examples from our calculations of organ doses in adult survivors are given in Table 1 for a deeply seated organ (small intestines), a widely distributed organ (active bone marrow), and a superficial organ of the body (female breasts). Effects of body orientation relative to the hypocenters of the explosions appear to be important only in the determination of the gamma component (g) and neutron component (n) of the absorbed dose in superficial organs of the body (e.g., the female breasts). The autogamma component (n,g) of absorbed dose in all organs is found to be insensitive to body orientation, and the neutron component (n) and gamma component (g) of absorbed dose in deeply seated organs of an adult (e.g., small intestines) are found to be insensitive to both body orientation (Table 1) and body size (Fig. 1). Hence, the Japanese-adult phantom developed by Mark Cristy (1985) will be adopted for calculations of organ dose for survivors above the age of 12 years at the times of the bombings. For survivors aged 4 to 12 years, we will

use the 5-year-old phantom developed by Cristy (1980) and his 1-year-old phantom for survivors less than 3 years of age at the time of the bombings. The use of the two additional mathematical phantoms will take reasonable account of the age-dependence of organ doses at young ages, while not overly complicating the dosimetry system for the A-bomb survivors.

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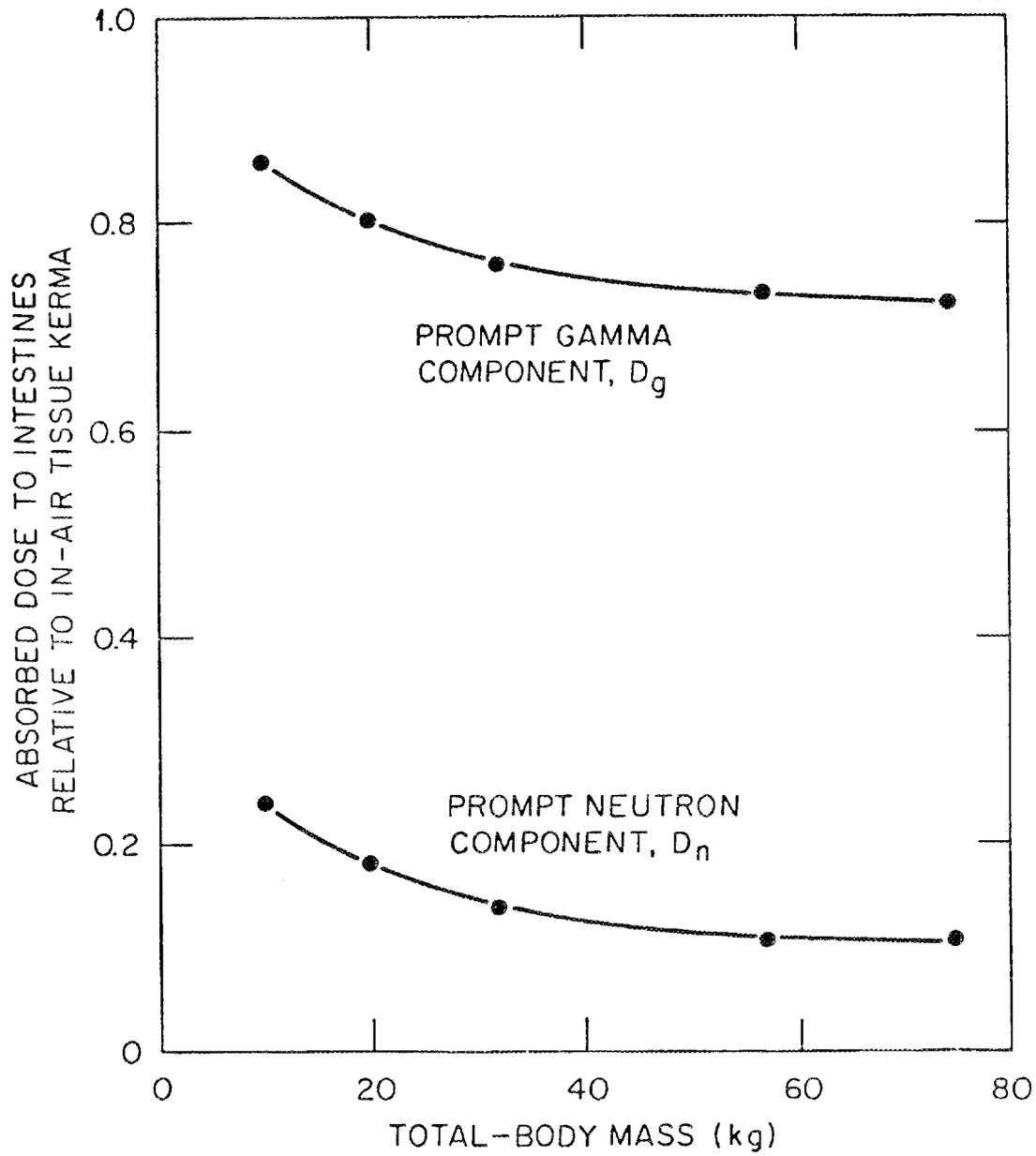


Figure 1. Effect of body size on absorbed dose in small intestines relative to in-air tissue kerma at a ground distance of 1100 m in Hiroshima.

Table 1. Absorbed dose (D) relative to in-air tissue kerma from prompt radiation in Hiroshima and Nagasaki

Organ	City	Distance from hypocenter in meters	Orientation to hypocenter of explosions						
			Facing toward		Facing away		Unknown ^a		
			D _g	D _n	D _g	D _n	D _g	D _n	D _{n,g}
Breasts	Hiroshima	700	0.92 ^b	0.55 ^c	0.68 ^b	0.32 ^c	0.83 ^b	0.43 ^c	0.41 ^c
		1100	0.95	0.59	0.68	0.33	0.84	0.46	0.26
		1500	0.97	0.63	0.68	0.34	0.85	0.49	0.18
	Nagasaki	700	0.93	0.62	0.66	0.35	0.82	0.50	0.13
		1100	0.96	0.64	0.67	0.35	0.83	0.51	0.13
		1500	0.96	0.66	0.67	0.35	0.85	0.52	0.12
Active marrow	Hiroshima	700	0.77	0.22	0.82	0.28	0.79	0.25	0.50
		1100	0.79	0.24	0.84	0.32	0.81	0.28	0.33
		1500	0.80	0.27	0.87	0.36	0.83	0.31	0.23
	Nagasaki	700	0.77	0.27	0.83	0.35	0.79	0.31	0.18
		1100	0.79	0.28	0.84	0.37	0.81	0.32	0.17
		1500	0.79	0.30	0.87	0.39	0.83	0.34	0.16
Intestines	Hiroshima	700	0.75	0.099	0.71	0.080	0.70	0.083	0.51
		1100	0.78	0.14	0.75	0.11	0.72	0.11	0.33
		1500	0.78	0.18	0.78	0.14	0.74	0.14	0.24
	Nagasaki	700	0.76	0.17	0.72	0.14	0.69	0.14	0.19
		1100	0.77	0.20	0.75	0.16	0.72	0.15	0.18
		1500	0.77	0.22	0.78	0.18	0.74	0.17	0.17

^aAll standing orientations of body relative to hypocenter are considered equally likely.

^bApply these factors to in-air tissue kerma from gamma rays.

^cApply these factors to in-air tissue kerma from neutrons.

EQUATIONS FOR TOTAL AND PARTIAL DENSITIES OF MOIST AIR

G. D. Kerr and J. V. Pace, III

INTRODUCTION

A set of equations is given which can be used to calculate the total density of moist air and the partial densities of dry air and water vapor. The total and partial densities of moist (natural) air are needed in a variety of atmospheric radiation transport problems, especially those involving fast neutrons (Banks, Klem, and Lichtenstein 1978; Kerr 1981; Pace, Knight, and Bartine 1982).

MIXING RATIO OF MOIST AIR

The equation of state for an ideal gas can be written as:

$$P = \rho RT , \quad (1)$$

where P is the pressure, T is the temperature, R is the specific gas constant (or universal gas constant divided by the molecular mass, M , of the gas), and ρ is the density of the gas under consideration. If we assume that the equation of state for an ideal gas can be applied to moist (natural) air, then we can use Dalton's law of partial pressures to write (Iribarne and Godson 1973):

$$\rho_m R_m = \rho_d R_d + \rho_v R_v , \quad (2)$$

where the subscripts m , d , and v refer to the moist air, dry air, and water vapor, respectively. A more convenient form of the above equation is

$$R_m = R_d [1 + r/\epsilon]/[1 + r] , \quad (3)$$

where

$$\epsilon = R_d/R_v = M_v/M_d = 0.622 , \quad (4)$$

$$r = \rho_v / \rho_d . \quad (5)$$

The mixing ratio, r , defined as the ratio of the partial densities of water vapor to dry air, can be written as (Iribarne and Godson 1973; Houghton 1977):

$$r = [e_v/R_v]/[(P - e_v)/R_d] = \varepsilon [e_v/(P - e_v)] , \quad (6)$$

where P is the barometric pressure, e_v is the partial pressure of water vapor, and $P - e_v$ is the partial pressure of dry air. To calculate the partial densities of dry air and water vapor in moist air, we use the relationship

$$\rho_m = \rho_d + \rho_v , \quad (7)$$

and the definition of the mixing ratio to write

$$\rho_d = \rho_m / (1 + r) , \quad (8)$$

$$\rho_v = r\rho_m / (1 + r) . \quad (9)$$

TOTAL DENSITY OF MOIST AIR

Note from Eq. (3) that the specific gas constant of moist air is not really a constant but a variable since the water vapor in moist air is variable. It is, therefore, convenient to write the total density of moist air as:

$$\rho_m = P/R_m T = P/R_d T^* , \quad (10)$$

where

$$T^* = T [1 + r/\varepsilon]/[1 + r] . \quad (11)$$

The virtual temperature, T^* , is defined as the temperature of dry air having the same pressure and density as the moist air. We can simplify Eqs. (10) and (11) by ignoring small second-order corrections involving

either the mixing ratio, r , or partial pressure of water vapor, e_v , and by writing the virtual temperature as:

$$T^* = T [1 + r/\epsilon] [1 - r] , \quad (12)$$

and the total density of moist air as:

$$\rho_m = P [1 - (1 - \epsilon)e_v/P]/R_d T = (P - 0.378e_v)/R_d T . \quad (13)$$

Finally, we can use the standard atmospheric conditions for dry air at mean sea level to write (Valley 1965; NACA 1955):

$$\rho_m = \rho_0 [T_0/T] [(P - 0.378e_v)/P_0] , \quad (14)$$

where T_0 is equal to 15° C (288.16°K), P_0 is equal to 760 mm of mercury (1013.25 millibar), and ρ_0 is equal to 1.225 kg m⁻³. This equation is consistent with the equation found in CRC Handbooks for the total density of moist air (Weast 1965).

The above set of equations outlines a convenient means of calculating the total and partial densities of moist air (Kerr, Pace, and Scott 1983). In practical applications, we first calculate the mixing ratio and total density of moist air by use of Eqs. (6) and (14) and then calculate the partial densities of dry air and water vapor by use of Eqs. (8) and (9).

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ORGAN DOSES FROM EXPOSURE TO ISOTROPIC FIELDS OF GAMMA RAYS
WITH AN EMPHASIS ON ACTIVE MARROW AND OSTEOGENIC TISSUE OF THE SKELETON

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INTRODUCTION

Most recent estimates of absorbed dose in internal organs of the body are derived from Monte Carlo calculations of the radiation transport within mathematical phantoms of Reference Man (Snyder et al. 1969; Cristy 1980). For ease of application in the Monte Carlo calculations, the total body and internal organs of Reference Man are modeled by the use of simple geometrical shapes, and three distinct regions of varying composition and density are defined: skeleton, lungs, and other soft tissues (i.e., total body minus the skeleton and lungs). The densities of the skeleton, lungs, and other soft tissues of the body are assumed to be 0.296, 1.4, and 1.04 gm cm⁻³, respectively. Various complexities involved in modeling the intricate geometry of the soft tissue in bone have led to the use of a homogeneous mixture approximation for the skeleton (i.e., connective tissues, bone, and soft tissues in bone). The homogenized skeleton provides appropriate radiation transport characteristics, but it ignores the effects of the microstructure of bone on absorbed dose in soft tissues of the skeleton (Ashton and Spiers 1979; Kerr 1980). We have completed a new set of state-of-the-art calculations for exposure to isotropic fields of gamma rays with an emphasis on improving the estimates of absorbed dose in active marrow and osteogenic tissue of the skeleton. Organ doses from isotropic fields of gamma rays are of special interest in assessing risks from both low-level exposures to environmental radiation and high-level exposures to nuclear-weapon fallout.

CALCULATIONAL METHODS AND RESULTS

Our organ-dose calculations employ Monte Carlo transport techniques embodied in our computer code, MORSE-SGC/PHANTOM, which is a modified version of the MORSE-SGC computer code (Fraley 1976). Compilation speed

and precision are improved by the use of (a) mathematical equations, instead of the standard combinatorial geometry package, to represent the total body and internal organs (Ryman, Warner, and Eckerman 1985), and (b) fictitious scattering in the Monte Carlo transport of the photons (Cramer 1978). The Monte Carlo transport of photons is based on the 38 energy-group set of cross sections from the Vitamin-E (ENDF/B-V) Library (Weisbin et al. 1979), but the final results are collapsed into a somewhat smaller 31 group set of photon energies. We first use our MORSE-SGC/PHANTOM code to calculate the spectral fluence of unscattered and scattered photons in a specific organ from an isotropic field of gamma rays, and then use fluence-to-dose response factors to obtain the absorbed dose in that organ (Kerr 1982; Kerr and Eckerman 1985; Eckerman and Cristy 1984). The dose response factors for active marrow and osteogenic cells are derived from microdosimetric considerations of the effect of bone on absorbed dose to the soft tissues in bone. A complete set of results from our state-of-the-art calculations of absorbed dose in twenty organs and tissues of a Reference Man phantom are summarized in Table 1. The fractional standard deviations associated with our Monte Carlo calculations vary from about 2% at the highest photon energies to about 5% at the lowest photon energies.

VERIFICATION STUDIES

We have attempted to verify our organ doses by making extensive comparisons with results from a variety of other theoretical and experimental studies. For example, we find reasonably good agreement between the various theoretical data on absorbed dose in active marrow at photon energies of more than several hundred keV, but there is a general lack of agreement at lower photon energies as shown in Fig. 1. Several different homogeneous mixtures of the soft tissues and bone in the skeleton were used in the active-marrow calculations of Poston and Snyder (1974), O'Brien (1980), and Kaul (1982), while T. D. Jones (1977) simply used a soft-tissue approximation in his active marrow calculations. Note that we show two sets of calculated values for the absorbed dose in active marrow from exposure to isotropic fields of gamma rays. One set is based on dose response factors for the

homogeneous skeleton approximation used in the mathematical phantoms of Reference Man (Kerr 1980) and reproduces the well-known results of Poston and Snyder (1977), while the other set is based on our recently published dose response factors for active marrow in bone cavities of the skeleton (Kerr and Eckerman 1985). Our calculated values for active marrow in bone cavities are also compared in Fig. 2 to measured values of Ashton and Spiers (1979), A. R. Jones (1978), Beck et al. (1969, 1971) and Clifford and Facey (1970). The various measured values are in close agreement with our calculations for gamma rays incident isotropically on the body from all directions in space. Only gamma rays incident isotropically from directions above the horizon to a standing phantom were considered in the measurements by Clifford and Facey (1970), but their measured values provide important verification of our calculated energy response for absorbed dose in active marrow. Our calculated energy-response curves for active marrow and several other organs of the body have shapes that are significantly different at low photon energies than the recently proposed energy-response curves of Ashton and Spiers (1979).

DISCUSSION

One surprising result of our organ-dose calculations is that the osteogenic tissue of the skeleton appears to be the most highly irradiated tissue or organ of the body. The International Commission on Radiological Protection (ICRP) has recommended a system of dose limitation which is based on dose equivalent to various organs weighted by a given set of risk factors (ICRP 1977; Grovas and Goodard 1981; Kramer and Drexler 1982). We have used the results of our organ-dose calculations (Table 1) to investigate the effective dose equivalent from exposures to isotropic fields of gamma rays (Table 2). The weights for the risk factors assigned to various organs are as follows: skin (0.01), active marrow (0.12), osteogenic tissue (0.03), thyroid (0.03), lungs (0.12), testes (0.12), ovaries (0.12), female breasts (0.15), kidneys (0.06), liver (0.06), spleen (0.06), stomach (0.06), and thymus (0.06). Another surprising result is that exposure to isotropic fields of gamma rays with energies between 0.1 and 20 MeV give values for the

effective dose equivalent (Table 2) and active-marrow dose (Table 1) which agree to within approximately $\pm 5\%$. The active marrow has always been of special interest in assessing risks from both low-level exposures to environmental radiation and high-level exposures to nuclear-weapon fallout.

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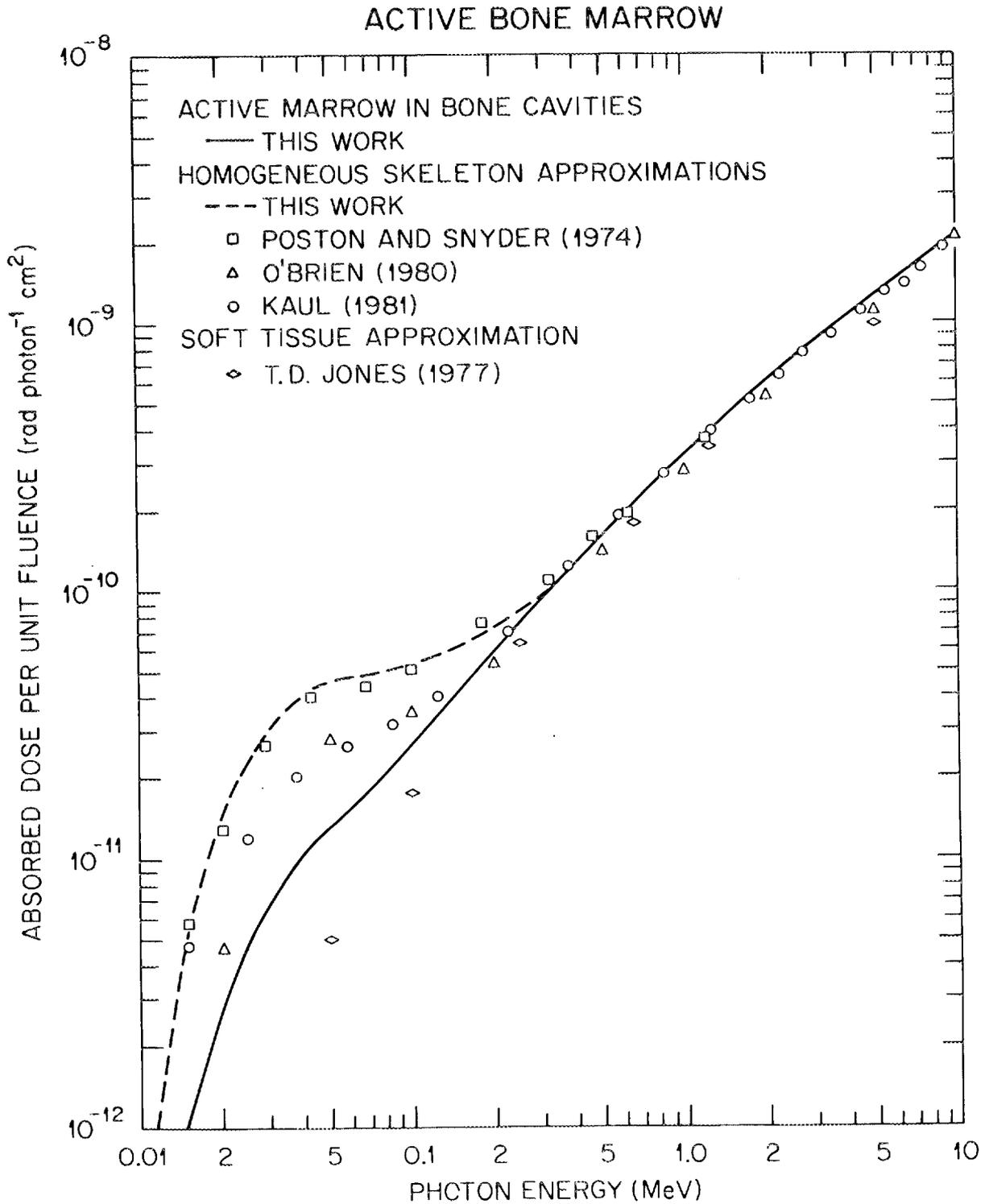


Figure 1. Comparison of calculated active-marrow doses in Reference Man from exposure to isotropic fields of gamma rays.

ACTIVE BONE MARROW

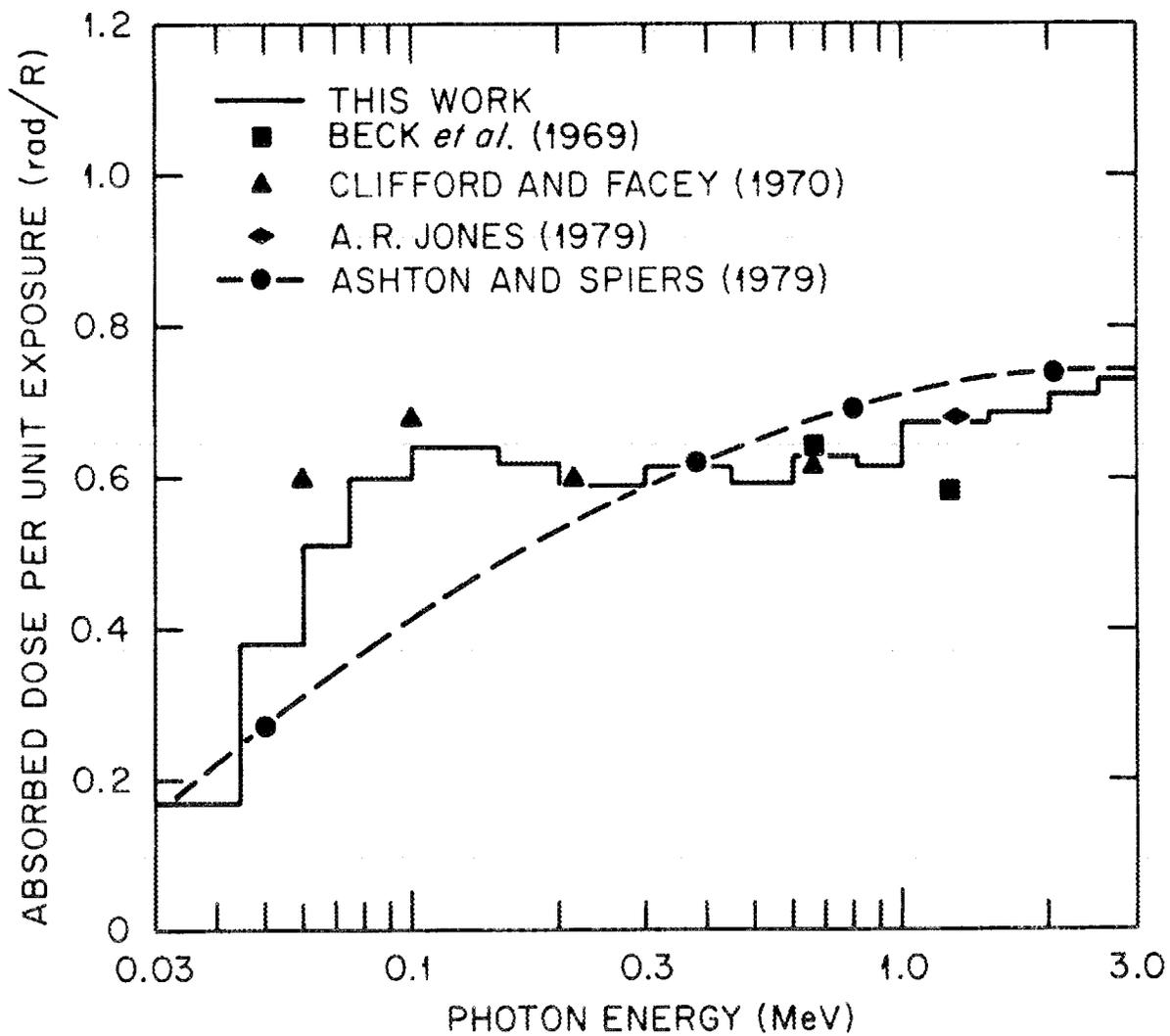


Figure 2. Comparison between experimental and theoretical data on absorbed dose in active marrow of a Reference Man-type phantom from exposure to isotropic fields of gamma rays.

Table 1. Calculated organ doses in Reference Man from exposure to isotropic fields of gamma rays

Gamma energy group	Upper bound (MeV)	Absorbed dose per unit isotropic-field fluence (rad/photon/cm ²)									
		Skin	Brain	Small intestines	Active marrow	Osteogenic tissue	Thyroid	Lungs	Testes	Ovaries	Female breasts
1	20.0	3.49E-09	3.49E-09	3.06E-09	3.47E-09	4.20E-09	3.37E-09	3.44E-09	3.29E-09	2.98E-09	3.30E-09
2	14.0	2.77E-09	2.76E-09	2.48E-09	2.68E-09	3.35E-09	2.75E-09	2.65E-09	2.59E-09	2.36E-09	2.71E-09
3	12.0	2.36E-09	2.35E-09	2.11E-09	2.42E-09	2.77E-09	2.25E-09	2.27E-09	2.30E-09	2.13E-09	2.35E-09
4	10.0	2.04E-09	1.95E-09	1.80E-09	2.01E-09	2.35E-09	1.93E-09	1.93E-09	1.91E-09	1.79E-09	2.04E-09
5	8.0	1.83E-09	1.81E-09	1.64E-09	1.77E-09	2.06E-09	1.77E-09	1.74E-09	1.72E-09	1.56E-09	1.77E-09
6	7.5	1.78E-09	1.69E-09	1.48E-09	1.63E-09	1.89E-09	1.67E-09	1.68E-09	1.64E-09	1.46E-09	1.66E-09
7	7.0	1.61E-09	1.62E-09	1.42E-09	1.56E-09	1.78E-09	1.58E-09	1.57E-09	1.52E-09	1.42E-09	1.62E-09
8	6.5	1.57E-09	1.54E-09	1.33E-09	1.47E-09	1.73E-09	1.51E-09	1.48E-09	1.42E-09	1.33E-09	1.49E-09
9	6.0	1.50E-09	1.43E-09	1.23E-09	1.37E-09	1.62E-09	1.40E-09	1.40E-09	1.32E-09	1.25E-09	1.44E-09
10	5.5	1.31E-09	1.29E-09	1.16E-09	1.30E-09	1.47E-09	1.27E-09	1.29E-09	1.28E-09	1.17E-09	1.34E-09
11	5.0	1.25E-09	1.21E-09	1.05E-09	1.23E-09	1.41E-09	1.18E-09	1.18E-09	1.15E-09	1.08E-09	1.21E-09
12	4.5	1.14E-09	1.16E-09	9.91E-10	1.09E-09	1.29E-09	1.12E-09	1.10E-09	1.05E-09	1.01E-09	1.10E-09
13	4.0	1.04E-09	1.04E-09	8.53E-10	1.01E-09	1.12E-09	9.82E-10	9.86E-10	9.40E-10	8.75E-10	1.02E-09
14	3.5	9.68E-10	9.40E-10	8.06E-10	8.82E-10	1.03E-09	9.35E-10	8.83E-10	8.68E-10	8.02E-10	9.45E-10
15	3.0	8.45E-10	8.27E-10	7.34E-10	7.76E-10	8.76E-10	8.21E-10	7.97E-10	7.80E-10	7.10E-10	8.36E-10
16	2.5	7.32E-10	7.03E-10	6.02E-10	6.58E-10	7.51E-10	6.99E-10	6.77E-10	6.64E-10	5.84E-10	7.22E-10
17	2.0	5.98E-10	5.95E-10	4.73E-10	5.33E-10	6.42E-10	5.76E-10	5.45E-10	5.44E-10	4.75E-10	5.89E-10
18	1.5	4.63E-10	4.41E-10	3.51E-10	4.02E-10	4.83E-10	4.32E-10	4.14E-10	4.02E-10	3.46E-10	4.57E-10
19	1.0	3.46E-10	3.36E-10	2.62E-10	2.89E-10	3.51E-10	3.18E-10	3.01E-10	2.92E-10	2.48E-10	3.32E-10
20	0.80	2.81E-10	2.61E-10	1.94E-10	2.33E-10	2.90E-10	2.50E-10	2.46E-10	2.47E-10	1.96E-10	2.73E-10
21	0.60	2.11E-10	1.95E-10	1.49E-10	1.70E-10	2.22E-10	1.91E-10	1.75E-10	1.89E-10	1.53E-10	2.05E-10
22	0.45	1.46E-10	1.35E-10	1.02E-10	1.24E-10	1.59E-10	1.36E-10	1.23E-10	1.24E-10	9.96E-11	1.47E-10
23	0.30	9.56E-11	8.03E-11	6.71E-11	7.54E-11	1.17E-10	9.38E-11	8.22E-11	8.68E-11	6.49E-11	9.13E-11
24	0.20	6.28E-11	5.79E-11	4.44E-11	5.18E-11	8.47E-11	5.58E-11	5.29E-11	5.33E-11	4.23E-11	6.08E-11
25	0.15	4.44E-11	3.94E-11	3.13E-11	3.56E-11	7.58E-11	4.04E-11	3.81E-11	3.72E-11	3.06E-11	4.09E-11
26	0.10	2.97E-11	2.76E-11	2.11E-11	2.29E-11	7.19E-11	2.69E-11	2.65E-11	2.69E-11	2.05E-11	2.93E-11
27	0.075	2.57E-11	2.23E-11	1.60E-11	1.79E-11	7.12E-11	2.29E-11	2.22E-11	2.25E-11	1.52E-11	2.50E-11
28	0.060	2.50E-11	1.79E-11	1.15E-11	1.37E-11	7.16E-11	2.13E-11	1.91E-11	2.10E-11	1.19E-11	2.49E-11
29	0.045	3.25E-11	1.15E-11	6.99E-12	9.74E-12	5.54E-11	2.09E-11	1.46E-11	2.26E-11	5.69E-12	2.85E-11
30	0.030	5.19E-11	2.21E-12	1.50E-12	4.80E-12	2.93E-11	1.90E-11	5.56E-12	2.21E-11	5.42E-13	3.39E-11
31	0.020 ^a	1.04E-10			1.03E-12	5.41E-12	2.80E-12		6.20E-12		1.83E-11

^aLower bound for the gamma energy of 31st group is 0.010 MeV.

Table 1. Continued

Gamma energy group	Upper bound (MeV)	Absorbed dose per unit isotropic-field fluence (rad/photon/cm ²)									
		Kidneys	Liver	Lower large intestines	Pancreas	Spleen	Stomach	Thymus	Upper large intestines	Urinary bladder	Uterus
1	20.0	3.18E-09	3.24E-09	3.15E-09	3.04E-09	3.26E-09	3.19E-09	3.31E-09	3.09E-09	3.01E-09	3.05E-09
2	14.0	2.54E-09	2.52E-09	2.53E-09	2.42E-09	2.52E-09	2.44E-09	2.57E-09	2.45E-09	2.51E-09	2.54E-09
3	12.0	2.20E-09	2.14E-09	2.13E-09	2.06E-09	2.20E-09	2.17E-09	2.23E-09	2.14E-09	2.13E-09	2.02E-09
4	10.0	1.85E-09	1.78E-09	1.76E-09	1.75E-09	1.79E-09	1.79E-09	1.87E-09	1.78E-09	1.76E-09	1.73E-09
5	8.0	1.63E-09	1.66E-09	1.60E-09	1.62E-09	1.68E-09	1.61E-09	1.67E-09	1.56E-09	1.68E-09	1.55E-09
6	7.5	1.53E-09	1.54E-09	1.52E-09	1.48E-09	1.51E-09	1.49E-09	1.63E-09	1.54E-09	1.52E-09	1.48E-09
7	7.0	1.48E-09	1.52E-09	1.44E-09	1.40E-09	1.47E-09	1.45E-09	1.51E-09	1.39E-09	1.43E-09	1.39E-09
8	6.5	1.35E-09	1.39E-09	1.36E-09	1.35E-09	1.39E-09	1.41E-09	1.45E-09	1.38E-09	1.39E-09	1.31E-09
9	6.0	1.32E-09	1.34E-09	1.26E-09	1.23E-09	1.32E-09	1.30E-09	1.34E-09	1.25E-09	1.24E-09	1.23E-09
10	5.5	1.19E-09	1.22E-09	1.12E-09	1.14E-09	1.19E-09	1.21E-09	1.23E-09	1.14E-09	1.20E-09	1.16E-09
11	5.0	1.11E-09	1.05E-09	1.08E-09	1.11E-09	1.09E-09	1.12E-09	1.15E-09	1.08E-09	1.08E-09	1.05E-09
12	4.5	1.01E-09	1.02E-09	9.77E-10	9.62E-10	1.03E-09	9.86E-10	1.06E-09	1.03E-09	1.01E-09	9.96E-10
13	4.0	9.04E-10	9.08E-10	8.92E-10	8.73E-10	9.22E-10	9.13E-10	9.51E-10	9.03E-10	9.16E-10	8.87E-10
14	3.5	8.40E-10	8.33E-10	8.30E-10	8.20E-10	8.31E-10	8.24E-10	8.76E-10	7.99E-10	8.05E-10	8.21E-10
15	3.0	7.48E-10	7.39E-10	7.06E-10	7.01E-10	7.49E-10	7.19E-10	7.73E-10	6.97E-10	7.21E-10	7.00E-10
16	2.5	6.22E-10	6.21E-10	5.94E-10	5.90E-10	6.40E-10	6.37E-10	6.49E-10	6.01E-10	6.04E-10	5.95E-10
17	2.0	5.08E-10	5.09E-10	4.91E-10	4.78E-10	5.27E-10	5.02E-10	5.30E-10	4.82E-10	4.91E-10	4.75E-10
18	1.5	3.86E-10	3.78E-10	3.51E-10	3.52E-10	3.86E-10	3.73E-10	4.05E-10	3.49E-10	3.59E-10	3.57E-10
19	1.0	2.62E-10	2.82E-10	2.68E-10	2.53E-10	2.80E-10	2.63E-10	2.90E-10	2.69E-10	2.59E-10	2.52E-10
20	0.80	2.22E-10	2.18E-10	2.07E-10	1.96E-10	2.17E-10	2.16E-10	2.35E-10	2.06E-10	2.07E-10	1.93E-10
21	0.60	1.64E-10	1.67E-10	1.51E-10	1.45E-10	1.59E-10	1.68E-10	1.73E-10	1.49E-10	1.53E-10	1.49E-10
22	0.45	1.13E-10	1.15E-10	1.05E-10	1.02E-10	1.16E-10	1.12E-10	1.24E-10	1.04E-10	1.07E-10	9.70E-11
23	0.30	7.37E-11	7.54E-11	6.72E-11	6.45E-11	7.57E-11	7.33E-11	7.85E-11	6.74E-11	6.74E-11	6.45E-11
24	0.20	4.92E-11	4.88E-11	4.61E-11	4.36E-11	4.87E-11	4.86E-11	5.28E-11	4.50E-11	4.61E-11	4.16E-11
25	0.15	3.50E-11	3.49E-11	3.25E-11	3.07E-11	3.44E-11	3.38E-11	3.72E-11	3.22E-11	3.34E-11	3.00E-11
26	0.10	2.50E-11	2.42E-11	2.16E-11	2.14E-11	2.41E-11	2.44E-11	2.56E-11	2.15E-11	2.31E-11	2.09E-11
27	0.075	1.89E-11	1.95E-11	1.65E-11	1.60E-11	1.96E-11	1.88E-11	2.15E-11	1.70E-11	1.85E-11	1.63E-11
28	0.060	1.67E-11	1.62E-11	1.21E-11	1.27E-11	1.56E-11	1.59E-11	1.84E-11	1.32E-11	1.60E-11	1.23E-11
29	0.045	1.35E-11	1.12E-11	7.22E-12	6.16E-12	1.09E-11	1.20E-11	1.46E-11	8.56E-12	1.08E-11	6.85E-12
30	0.030	7.67E-12	3.95E-12	1.20E-12	5.46E-13	3.88E-12	4.49E-12	7.30E-12	2.17E-12	4.37E-12	1.09E-12
31	0.020										

Table 2. Effective dose equivalent from exposure to isotropic fields of gamma rays

Gamma energy group	Upper bound (MeV)	Effective dose equivalent (rem/photon/cm ²)
1	20.0	3.31E-09
2	14.0	2.59E-09
3	12.0	2.28E-09
4	10.0	1.92E-09
5	8.0	1.71E-09
6	7.5	1.60E-09
7	7.0	1.53E-09
8	6.5	1.44E-09
9	6.0	1.36E-09
10	5.5	1.26E-09
11	5.0	1.16E-09
12	4.5	1.07E-09
13	4.0	9.61E-10
14	3.5	8.75E-10
15	3.0	7.76E-10
16	2.5	6.59E-10
17	2.0	5.37E-10
18	1.5	4.04E-10
19	1.0	2.92E-10
20	0.80	2.37E-10
21	0.60	1.78E-10
22	0.45	1.24E-10
23	0.30	8.07E-11
24	0.20	5.29E-11
25	0.15	3.76E-11
26	0.10	2.67E-11
27	0.075	2.21E-11
28	0.060	1.96E-11
29	0.045	1.69E-11
30	0.030	1.27E-11
31	0.020 ^a	4.94E-12

^aLower bound for gamma energy of 31st group is 0.010 MeV.

NEUTRON AND PHOTON FLUENCE-TO-DOSE CONVERSION FACTORS
FOR ACTIVE MARROW OF THE SKELETON

G. D. Kerr and K. F. Eckerman

Most estimates of absorbed dose in various organs of the body are derived from radiation transport calculations in anthropomorphic phantoms with a homogeneous representation of the soft tissues and bone in the skeleton. The homogenized skeleton is adequate for radiation transport purposes since it provides correct scattering and absorption properties. However, the effects of the microstructure of bone must be considered in deriving realistic estimates of the absorbed dose in soft tissues of the skeleton, particularly the active (red) marrow, which is one of the most radiosensitive tissues of the body.

We have investigated the effects of the microstructure of bone on absorbed dose in active marrow for a variety of active marrow sites in the skeleton and a wide range of neutron and photon energies. In previous calculations for neutrons and photons, specific geometrical models have been used for the marrow cavities (e.g., thin slabs, cylinders, and spheres). These studies indicate clearly that the presence of bone alters the absorbed dose in active marrow from photons with energies less than several hundred keV and neutrons with energies greater than several MeV, but the results cannot be applied generally. We avoid the assumption of a special geometry by using measured chord-length distributions to represent the microstructure of the trabecular bones containing the active marrow (e.g., parietal bone, lumbar vertebra, cervical vertebra, iliac crest, rib, and both the head and neck of the femur).

Results of our calculations for neutrons and photons with energies up to 20 MeV are provided as fluence-to-dose conversion factors, for application in radiation transport calculations of absorbed dose in active marrow from photons and neutrons externally incident on the body, and photons produced by neutron interactions within the body. Our

results suggest that the neutron and photon fluence-to-dose conversion factors for lumbar vertebra can be used as a representative example for all active marrow sites except those in parietal bone of the skull.

These results are to be published in the proceedings of the Fifth Symposium on Neutron Dosimetry, Neuherberg/Munich, West Germany, September 17-21, 1984.

SPECIFIC ABSORBED FRACTIONS OF ENERGY
AT VARIOUS AGES FROM INTERNAL PHOTON SOURCES

M. Cristy and K. F. Eckerman

INTRODUCTION

A series of reports to be published later this year tabulates specific absorbed fractions (Φ 's) for monoenergetic photon sources uniformly distributed in organs of mathematical phantoms representing humans of various ages and describes the methods used to compute them (Cristy and Eckerman 1985a,b,c,d,e,f,g). Procedures for choosing the 'best' estimates of Φ from the estimates generated by the various methods are described in the first volume, and the Φ 's calculated by three methods and the 'best' estimates recommended by us are tabulated in the remaining volumes for the newborn, for ages 1, 5, 10, and 15 years, for an adult female, and for an adult male. These Φ 's will be used in calculating dose equivalent rates from radionuclides in the body at various ages, which will be published separately.

The methods used to calculate Φ 's are similar to those used by Snyder, Ford, Warner, and Watson (1974) for their adult phantom. However, more use is made of the converse Monte Carlo estimate, $\Phi(S \leftarrow T)$ (S = source organ, T = target organ), as an approximation to the direct Monte Carlo estimate, $\Phi(T \leftarrow S)$. More extensive use is made of empirical correction factors for the estimates generated by the point kernel (or buildup factor) method. Also, a better method to calculate the fraction of energy deposited in the active marrow and the endosteal cells has been employed.

The phantoms described previously by Cristy (1980) are designed like the adult phantom of Snyder et al. (1974) and have different densities and chemical compositions for lung, skeletal, and soft tissues. ('Soft tissues' are all near-unit-density tissues, i.e.,

density = 1 g/cm³.) The age 15 phantom has been redesigned so that it represents both a 15-year-old male and an adult female.

METHODS OF CALCULATING Φ

Three methods are used to calculate the Φ for a given source organ-target organ pair at a given initial photon energy. (1) $\Phi(T \leftarrow S)$ is calculated with the Monte Carlo radiation transport computer program. (2) $\Phi(S \leftarrow T)$ is calculated with the Monte Carlo computer program, and this value is used to estimate $\Phi(T \leftarrow S)$, sometimes after applying a correction factor. (3) $\Phi(T \leftrightarrow S)$ is calculated with the point kernel (or buildup factor) method. A correction factor may also be applied to this estimate. For the special case of the active marrow or the endosteal cells as the target organ, a fourth method is employed. This method is a refinement of method (1).

Monte Carlo Radiation Transport Computer Program

A radiation transport computer program employing Monte Carlo techniques, similar to that of Snyder et al. (1974), simulates the transport of photons of any given initial energy originating in a given organ (source organ). The photon emission is uniformly distributed in the source organ. The specific absorbed fraction, i.e., the energy absorbed in another organ (target organ), normalized as the fraction of emitted energy and per kilogram of target organ, is calculated, and the reliability of the Φ is calculated as a coefficient of variation. The details of the method and the computer program may be found in Ryman, Warner, and Eckerman (1985a).

For a given source-target pair, we obtain two numbers: the direct estimate, $\Phi(T \leftarrow S)$, obtained when the photon emission is in the organ labeled 'source,' and the converse estimate, $\Phi(S \leftarrow T)$, obtained when the photon emission is in the organ labeled 'target.' Each of these numbers is from a Monte Carlo computer run: what is labeled the direct estimate and what is labeled the converse estimate depend upon which organ we label the target organ. According to the reciprocal dose theorem, the converse estimate should be a good approximation to the direct estimate

under ideal conditions. The usefulness of this theorem in providing more reliable estimates of Φ has been documented (Cristy 1983).

Point Kernel Method

In this method, the equation describing the absorption of energy at a distance r from a point source of monoenergetic photons in an infinite homogeneous medium (water) is employed:

$$\Phi(r) = \frac{\mu_{en}}{\rho} \cdot \frac{1}{4\pi r^2} \cdot e^{-\mu r} \cdot B(\mu r) ,$$

where

$\Phi(r)$ = point isotropic specific absorbed fraction at r ,

μ_{en} = linear energy-absorption coefficient at the source energy,

μ = linear attenuation coefficient at the source energy,

ρ = density of medium,

$B(\mu r)$ = energy absorption buildup factor, a factor which corrects for the contribution from scattered radiation.

Equations describing $B(\mu r)$ for point photon sources in water have been published by Spencer and Simmons (1973).

This equation is integrated over the volumes of the source and target organs, with numerical methods, to yield $\Phi(T \leftrightarrow S)$. Note the double arrow: the conditions of the reciprocal dose theorem are met, and the reciprocal doses are equal.

In this method, the phantoms are composed of water throughout and are embedded in an infinite water medium. In the Monte Carlo radiation transport method, the phantoms have different densities and chemical compositions for lung, skeletal, and soft tissue and are embedded in vacuum. Thus there may be systematic errors in the point kernel estimates of Φ . These errors are reduced by applying empirical correction factors. Point kernel estimates are necessary when the Monte Carlo estimates are statistically unreliable.

Details of the point kernel computer program are given in Ryman, Warner, and Eckerman (1985b).

Special Case:

Active Marrow and Endosteal Cells as Target Organs

The Monte Carlo transport code assigns the average energy lost by photons in interactions in the body to the organ in which the interaction occurred, i.e., the transport of energy by secondary electrons is not treated. This approach is reasonable if electronic equilibrium is established such that the transport of energy by secondary electrons out of the organ is balanced by transport into the organ. However, in the region of discontinuities in tissue compositions such as that between bone and soft tissues within bone, significant errors may be introduced.

In each phantom the skeleton is represented as a uniform mixture of its component tissues, namely, cortical bone, trabecular bone, active marrow, fatty marrow, and various connective tissues. The tissues of interest as target regions are the active marrow of trabecular bone and osteogenic cells adjacent to the surfaces of both cortical and trabecular bone; this target is referred to as 'bone surface'. To estimate the energy deposition in these regions, one must consider the energy transport by secondary electrons arising from photon interactions within the regions and from electrons entering the regions from skeletal components in the immediate vicinity of the target, e.g., bone adjacent to the target region.

The energy deposition by photon radiation in soft tissue regions of the skeleton was first studied in 1949 by Spiers and later by Spiers and others using simple geometrical models (e.g., thin slabs, cylinders, and spherical cavities) to approximate the interface geometry (Woodard and Spiers 1953; Charlton and Cormack 1962; Aspin and Johns 1963; Howarth 1965). These studies demonstrated that for photon energies less than about 200 keV electronic equilibrium does not exist and electrons liberated in mineral regions may contribute significantly to the absorbed dose in soft-tissue regions of the skeleton.

The problems in formulating the absorbed dose in skeletal tissues are similar to those encountered in the dosimetry of beta emitters incorporated in bone. For beta emitters Spiers and co-workers reduced the intractable three-dimensional geometry to one dimension through use

of measured distributions of chord-lengths in trabeculae and marrow cavities of trabecular bone (Spiers 1969; Beddoe et al. 1976; Beddoe 1977). Clearly this approach to the geometry can be applied to secondary electrons liberated by photon interactions in the skeleton.

We have developed a computational approach using available information on the microscopic structure of bone as a means of deriving realistic estimates of the absorbed dose. The approach is formulated in a manner which retains the homogeneous representation of the skeleton and requires only a minor modification to the Monte Carlo transport code to yield the additional information. The basic feature of the approach is a separation of the photon-to-electron transfer of energy from the dissipation of energy by the electrons.

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MATHEMATICAL PHANTOMS FOR USE IN REASSESSMENT OF
RADIATION DOSES TO JAPANESE ATOMIC-BOMB SURVIVORS

M. Cristy

In 1972 committees of the United Nations and the U.S. National Academy of Sciences emphasized the need for organ dose estimates on the Japanese atomic-bomb survivors. These estimates were then supplied by workers in Japan and the U.S., and they were used with the so-called T65D estimates of a survivor's radiation exposure to assess risk from radiation. Recently the T65D estimates have been questioned, and programs for reassessment of atomic-bomb radiation dosimetry have been started in Japan and the U.S. As a part of this new effort a mathematical analogue of the human body (or 'mathematical phantom'), to be used in estimating organ doses in adult survivors, has been developed. Recommendations on organ dosimetry for juvenile survivors have also been made. This work will appear as Oak Ridge National Laboratory Report ORNL/TM-9487, 1985.

A COMPARISON OF VARIOUS SKELETAL DOSE QUANTITIES

K. F. Eckerman

INTRODUCTION

With the issuance of ICRP Publication 30 (1979), the dosimetry of bone-seeking radionuclides has changed from direct comparison with radium in the skeleton, as used in ICRP Publication 2 (1960), to consideration of the dose equivalent in specific cell populations within the skeleton. The stage for this change had been set with the issuance of ICRP Publication 11 (1968); however, it was not until the issuance of Publication 30 that official status was given to these recommendations. In the literature, various dose quantities are found representing the dose in the marrow-free skeleton (7 kg mass) and in mineral bone (5 kg mass), as well as assorted ratios of doses in skeletal tissues employed by researchers in bone dosimetry. The purpose of this work is to compare the dose averaged over mineral bone with the dose in the skeletal tissues addressed in ICRP Publication 30. This is part of an ongoing effort to facilitate the application in the workplace of the radiation protection guidance in ICRP Publication 30 and to put this guidance into perspective with regard to previous guidance that may be more familiar to operational health physicists.

THE DISTRIBUTION OF CELLS AT RISK IN THE ICRP 30 BONE DOSIMETRY MODEL

The bone dosimetry model used in ICRP 30 focuses on two specific cell populations in the skeleton: the hematopoietic stem cells of marrow and the osteogenic cells. It has been assumed that the hematopoietic stem cells are uniformly distributed within the marrow space of trabecular bone, and the dose equivalent in these cells is taken as the average dose equivalent in the marrow space. For the osteogenic cells, it is recommended that the dose equivalent be calculated as an average over endosteal tissues up to a distance of 10 μm from bone surfaces. Thus, although the dose to the osteogenic cells commonly is referred to as the bone surface dose, it is actually averaged over the soft-tissue mass (120 g in the adult) adjacent to the bone-soft tissue interface.

The active marrow space is contained within the trabecular bone of the skeleton, and the endosteal tissues are associated with both cortical and trabecular bone. (Cortical bone is the hard mineral region on the exterior of the bones of the skeleton while trabecular bone is the soft, spongy mineral lying in the interior of bone, particularly the vertebrae, ribs, flat bones, and the ends of the long bones of the skeleton.) To implement the dosimetric formulations for these two target regions, it is necessary to consider nuclear transformations occurring within both cortical and trabecular bone and the distribution of these transformations within the volume or along the bone surfaces.

EQUATION FOR ESTIMATION OF DOSE EQUIVALENT

Following the notation of ICRP Publication 30, we express the committed dose equivalent in target organ T by the equation

$$H_{50,T} = \frac{k}{M_T} \sum_s U_s \sum_i Y_i E_i AF(T \leftarrow S)_i Q_i \quad (1)$$

where

M_T = mass of target organ T (g),

U_s = number of transformations in source region s,

Y_i = yield of radiation i per transformation,

E_i = average or unique energy of radiation i (MeV),

$AF(T \leftarrow S)_i$ = fraction of energy absorbed in target organ T from radiation i originating in s,

Q_i = quality factor appropriate for radiation i.

The numerical constant k has a value of 1.6×10^{-10} if the desired unit of dose equivalent is the sievert. It is convenient to consider the sum over i in Eq. (1), divided by the target mass M_T , as a separate quantity called the specific effective energy, $SEE(T \leftarrow S)$. Thus Eq. (1) can be rewritten as

$$H_{50,T} = k \sum_s U_s \text{SEE}(T \leftarrow S) \quad (2a)$$

where

$$\text{SEE}(T \leftarrow S) = \frac{1}{M_T} \sum_i Y_i E_i \text{AF}(T \leftarrow S)_i Q_i \quad (2b)$$

Our discussion here is restricted to alpha and beta radiations and we assume that only one radiation type is present. Thus, the sum over radiation i in Eq. (1) will be neglected in the discussion. It should be noted that Eq. (1) applies only to a single nuclide; an additional summation is needed to consider each member of a decay chain.

In principle, a value for U_s , where s is cortical bone or trabecular bone, is required in evaluation of Eq. (1). For most radionuclides, however, the metabolic information is not sufficient to yield explicit estimates of these quantities. The following estimating procedures have been employed in ICRP 30. (We denote trabecular bone as TB, cortical bone as CB, and mineral bone as B.)

- a. $U_{TB} = U_{CB} = 0.5 U_B$ for radionuclides assumed to remain on bone surfaces, i.e., surface seekers;
- b. $U_{TB} = 0.2 U_B$ and $U_{CB} = 0.8 U_B$ for radionuclides assumed to be uniformly distributed throughout the volume of mineral bone, i.e., volume seekers.

The above fractional values arise from considering trabecular and cortical bone to be of equal surface area and their masses to be 1000 g and 4000 g, respectively; the total mass of bone is taken to be 5000 g.

The absorbed fraction data, $\text{AF}(T \leftarrow S)$, recommended for use in Eq. (1) are given in Table 1. Those working in bone dosimetry historically have not used the absorbed fraction quantity, but rather the ratio of the dose in the relevant target tissue to the dose in bone mineral. In ICRP Publication 30 (see Chapter 7 of that publication) values for the absorbed fraction were extracted from ratios reported in the literature. Care must be taken here as individual researchers often

define quantities in slightly different manners, and definitions evolve in the course of research. For example, Spiers and coworkers (1968, 1976, 1978) initially reported the dose to red marrow and endosteal tissues as normalized to a unit dose in bone, while in later work they reported values relative to the dose in a soft-tissue cavity within bone with infinite dimensions. Furthermore, the reported ratios for endosteal tissues often addressed only the endosteal tissues of the bone type being considered. Thus, in Table 7.2 of Publication 30, the endosteal tissue absorbed dose ratios for beta emitters in bone volume cited to be from Spiers and coworkers are actually one-half the authors' values. Spiers and coworkers formulated their ratio only for the endosteal tissue of the bone type in question, which is one-half of the total endosteal tissue of the skeleton. No note to this effect is included within the discussion in Publication 30. We point this out only to indicate that one must be careful in deriving relationships from the literature. It appears that such relationships can best be defined by consideration of the dosimetric formulations, as below.

RELATIONS OF DOSES IN RED MARROW AND BONE SURFACES TO THE DOSE IN BONE

The dose to bone for alpha and beta radiations is computed assuming the emitted energies will be absorbed within the mass, M_B , of bone. The dose to bone, H_B , from radiation i is given as

$$H_B = \frac{k}{M_B} U_B Y_i E_i Q_i \quad (3)$$

Thus the ratios of interest here are given by Eq. (1) and Eq. (2) as

$$\frac{H_T}{H_B} = \frac{M_B}{M_T} \sum_s f_s AF(T \leftarrow S)_i \quad (4)$$

where f_s denotes the appropriate fraction of the transformations in bone assigned to source region s as discussed above. Eq. (4) and the

absorbed fraction data of Table 1 are used to establish the ratios given in Table 2. The masses of the target organs T were taken as 1500 g and 120 g for the red marrow and bone surfaces, respectively, and 5000 g for mineral bone. Although we have reported the ratios using the symbol for dose equivalent, it should be noted that these values can be applied to absorbed dose as well as to absorbed dose rates and dose equivalent rates.

Application of the data of Table 2 to bone dose estimates in the literature is quite simple, in principle; however, the resultant values will be meaningful only if bases for the original estimate are understood. That is, one must know what mass the bone dose was based on and, if dose equivalent is reported, what modifying and quality factors were used to compute the reported value.

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Table 1. Absorbed fractions recommended by Committee 2 for the dosimetry of radionuclides in bone*

Class of radionuclide	AF(RM ← TB)	AF(BS ← TB)	AF(BS ← CB)
α emitters			
Volume seekers	0.05	0.025	0.01
Surface seekers	0.5	0.25	0.25
β emitters			
Volume seekers	0.35	0.025	0.015
Surface seekers			
$\bar{E} < 0.2$ MeV	0.5	0.25	0.25
$\bar{E} \geq 0.2$ MeV	0.5	0.025	0.015

*RM, BS, TB, and CB denote red marrow, bone surfaces, trabecular bone, and cortical bone, respectively. Note that AF(RM ← CB) is taken as zero for all radiations.

Table 2. Ratio of the dose to red marrow and bone surfaces to the dose to bone mineral*

Class of radionuclide	$H_{\text{red marrow}}:H_{\text{bone}}$	$H_{\text{bone surface}}:H_{\text{bone}}$
α emitters		
Volume seekers	0.033	0.54
Surface seekers	0.83	10
β emitters		
Volume seekers	0.23	0.71
Surface seekers		
$\bar{E} < 0.2$ MeV	0.83	10
$\bar{E} \geq 0.2$ MeV	0.83	0.83

*The mass of bone is assumed to be 5 kg. If the bone dose was computed for the 7 kg-marrow-free skeleton, the table entries should be multiplied by 1.4.

COMPARISON OF LUNG DOSE ESTIMATES FROM VARIOUS MODELS

K. F. Eckerman

INTRODUCTION

A substantial increase in the estimated dose to the lung is noted when comparing dosimetric data of ICRP Publications 2 and 30. The purpose of this note is to examine the origin of that increase.

In Publication 2 of the International Commission on Radiological Protection (ICRP, 1959) a simple model of the lung was used to define the entrance of inhaled material into the body and the dose to the lung. That model did not reflect the dependence of lung deposition on particle size; 75% of the inhaled activity was assumed to be deposited in the lung, regardless of the sizes of particles in the inhaled material. The physical-chemical nature of the aerosol, which determines its clearance rate from the lung, was classified in a relative manner by the terms 'soluble or insoluble.' Soluble materials were considered to clear rapidly from the lung so that the dose to the lung could be ignored. On the other hand, insoluble materials were assumed not to be absorbed by the body, so that only the dose to the lung and segments of the gastrointestinal tract were considered.

The limitations of the inhalation model of Publication 2 were recognized and the ICRP established a Task Group on Lung Dynamics which formulated and published (in 1966) a more detailed deposition and clearance model. That model (referred to as the Task Group Lung Model - TGLM) considers the respiratory system to consist of three regions, the nasopharyngeal (NP), tracheobronchial (TB), and pulmonary (P), which are interconnected with one another as well as connected with body fluids (blood), the gastrointestinal tract, and the lymphatic system. The fraction of inhaled activity deposited in each region is a function of the activity median aerodynamic diameter (AMAD) of the aerosol. Clearance of the deposited activity is defined in terms of three classes, corresponding to clearance from the pulmonary region on the order of days (D), weeks (W), and years (Y).

In 1978, two decades after issuance of Publication 2, the ICRP updated the numerical values of its secondary limits using the TGLM. Although the TGLM was published a decade earlier, it had never received 'official status' as it awaited application in recommendations of the ICRP. (The limbo status applied as well to the efforts of other ICRP Committee 2 task groups, e.g., metabolism of alkaline earth elements [Publication 20] and Reference Man [Publication 23].) Thus, although the TGLM has been available for application in radiological assessments for a decade, it has been used sparingly. This was due not only to the lack of 'official status' but also because the Publication 2 model was easier to use and the TGLM formulations were subject to interpretations. Although the ICRP has outlined and applied their approach in Publication 30, questions concerning interpretation still remain.

APPLICATION OF LUNG MODELS

Various topics related to lung dosimetry are under current investigation. For our purposes here it will suffice to address these issues rather broadly. In general, the issues center around the question, 'Which respiratory tissues (cells) are at risk with regard to lung cancer?' In a practical and very narrow sense, these questions relate to the manner in which one might apply the TGLM or any other lung model.

A fraction of the activity deposited in the lung is transferred to pulmonary lymph nodes from which limited clearance occurs, particularly for insoluble (Class Y) compounds. Since the mass of the pulmonary lymph is small (about 15 g) the dose (absorbed energy per gram of tissue) delivered to this tissue from Class Y aerosols may be many times greater than that received by other lung tissues. In ICRP Publication 26 (see paragraphs 52-54 of that publication) the Commission decided that irradiation of the lung is likely to be more limiting than that of lymphoid tissue for insoluble radioactive particles. Thus, the Commission considered that for radiation protection purposes it would be satisfactory to consider the tracheobronchial region, pulmonary region, and pulmonary lymph nodes as one composite organ of mass 1000 g. This decision has been a matter of controversy, particularly since it was somewhat indecisive regarding pulmonary lymph (the dose to this tissue

is simply 'smeared' over the rest of the lung) and the Commission resorted to a more advanced lung model, in Publication 32, to deal with the short-lived radon daughters where attention is directed to specific cells at risk.*

COMPARISON OF LUNG MODELS OF PUBLICATIONS 2 AND 30

The committed dose equivalent H_{lung} for the lung per unit activity inhaled can be expressed as

$$H_{lung}(\text{Sv/Bq}) = 1.6 \times 10^{-10} U Q E / M,$$

where U is the number of nuclear transformations in the 50 year period following the inhalation of a unit activity (nt/Bq),

Q is the radiation quality factor,

E is the energy emitted per nt (MeV/nt),

M is the mass, in g, over which the deposited energy is averaged.

Note that the above formulation is applicable to particulate radiations, i.e., alpha and beta particles.

In Publication 2 the fraction 0.12 of the inhaled insoluble activity was assumed to be retained in the lung. This activity was assumed to be retained with a half-time of 120 days for all insoluble radionuclides except plutonium and thorium, for which halftimes of 1 and 4 years, respectively, were used. The number of nuclear transformations per unit inhaled activity is proportional to the time integral of the retention, which for this model corresponds to

Radionuclide	U (nt/Bq)
Thorium	2.16E07
Plutonium	5.44E06
Others	1.79E06

In Publication 30, transformations in compartments c through j (comprising the TB, P, and lymph--see Fig. 5.2 of Publication 30) of the lung model are considered in the computation of U, with M taken as

* Paragraph 17 of Publication 32 states, 'In its basic recommendations the Commission has proposed that the total lung (NP+TB+P+L regions) should be considered as a composite organ.' This statement is incorrect.

1000 g. The number of nuclear transformations in each region of the TGLM is shown below. (The assumptions are made that the half-life of the inhaled radionuclide is long relative to biological clearance and the initial deposition is that of a one micron AMAD aerosol.)

Nuclear Transformation per Bq inhaled

Lung Region	Clearance Class		
	D	W	Y
TB	1.94E02	6.03E03	8.21E03
P	1.56E04	9.47E05	9.36E06
L	3.12E03	7.79E04	1.01E07
Total	1.89E04	1.03E06	1.95E07

Note that the number of nuclear transformation in the lung for class Y compounds is about an order of magnitude higher than that for radionuclides in the Note also that about one-half of this increase is associated with the ICRP's decision to smear the nuclear transformation within the pulmonary lymph tissue across the 'composite lung'. The quality factor for alpha emitters increased from 10 to 20 between Publications 2 and 30. This results in dose estimates per unit intake for uranium now being about a factor of twenty higher than those estimated from the simple model of Publication 2. For plutonium the current estimates of the number of nuclear transformations in the lung for class Y compounds is increased by about a factor of 4. For class Y compounds of thorium the new estimates are similar to the earlier values based on a retention half-time of 4 years.

COMPARISON WITH EPA'S APPROACH

EPA averages the dose over the pulmonary region of the lung model, i.e., compartment e through h, to which they assign a mass of 570 g. The ratio of the committed dose equivalent per unit intake as calculated by EPA to that of the ICRP is then

$$\begin{aligned}
 H_{\text{EPA}}: H_{\text{ICRP}} &= (M_{\text{lung}}/M_{\text{P}}) (U_{\text{P}}/U_{\text{total}}) \\
 &= (1000/570) (U_{\text{P}}/U_{\text{total}}),
 \end{aligned}$$

which yields the following values:

Clearance class	$H_{\text{EPA}}: H_{\text{ICRP}}$
D	1.45
W	1.61
Y	0.84

Thus, the EPA approach yields 'lung' dose estimates about 50% higher than those of ICRP for class D and W aerosols and about 20% lower for class Y.

SUMMARY AND CONCLUSIONS

The most significant changes in lung dose estimates have been shown to be associated with the use of the TGLM in place of the simple lung model of Publication 2. The scope of the discussion has been limited, generally, to long-lived radionuclides and insoluble material. The lung dosimetry approach of EPA results in values within 50% of the ICRP values in Publication 30. Although numerous issues in lung dosimetry center around lung models, it is clear that the use of the model of Publication 2 must be abolished and characterization of aerosols in terms of particle size and appropriate clearance class must be pursued. The latter needs particular attention as it can significantly influence lung dose estimates as well as the dose estimates for other organs. Limited information is available in the literature regarding dose estimates for individuals other than adults. Published information on the deposition of aerosols indicates that this aspect is not highly age-dependent; however, little or no information relevant to clearance is available.

STOPPING POWER AND POINT ISOTROPIC SPECIFIC ABSORBED
FRACTION DATA FOR ALPHA PARTICLES IN TISSUE

A. Taner and K. F. Eckerman

INTRODUCTION

Information on the energy loss of alpha particles is fundamental to many areas of radiation dosimetry since it characterizes the transfer of kinetic energy from the particle to the medium. Stopping power estimates are usually obtained from theoretical calculations based on the Bethe formulation; however, a number of empirical considerations, such as the variation in the effective charge of the particle as it slows down, are necessary for an accurate assessment (Harley and Pasternak 1972). Although the theory is quite complete, it is necessary to rely on a combination of experimental and theoretical considerations when estimating stopping powers (Harley and Pasternak 1972; Thorne 1977; Walsh 1970). Recently Ziegler (1977) completed an analysis of the available stopping power information. He tabulated, by element, the coefficients of an empirical function representing the stopping power, thus providing a convenient formulation to estimate stopping power for a wide range of materials. Our application of this information to some fundamental dosimetric considerations involving alpha particles is discussed here.

EMPIRICAL EXPRESSIONS FOR STOPPING POWER

For alpha particles of energy between 1 keV to 10 MeV, Ziegler expressed the elemental electronic stopping power as a five-parameter function of energy. The elemental electronic stopping power, S_e (eV-cm²/10¹⁵ atoms), as a function of energy, E (MeV), is given as

$$\frac{1}{S_e} = \frac{1}{S_1} + \frac{1}{S_2} \quad , \quad (1.a)$$

where

$$S_1 = A_1 E^{A_2} \quad \text{and} \quad (1.b)$$

$$S_2 = A_3/E \ln(1 + A_4/E + A_5 E) \quad . \quad (1.c)$$

At low alpha particle energy (less than 100 keV in tissue) Coulomb interactions with the nucleus contributes significantly to the stopping power. We use the formulation suggested by Ziegler to evaluate the nuclear component of the stopping power, S_n ,

$$S_n = \frac{67.70 Z}{(4+M)(1.587+Z^{2/3})^{3/2}} \begin{cases} 1.593 \varepsilon^{3/2} & \text{if } \varepsilon \leq 0.01 \\ 1.7 \varepsilon^{3/2} \left[\frac{\ln(2.7183+\varepsilon)}{(1+6.8\varepsilon+3.4\varepsilon^2)} \right] & \text{if } 0.01 < \varepsilon < 10 \\ \frac{\ln(0.47)}{2\varepsilon} & \text{if } \varepsilon \geq 10 \end{cases} \quad (2)$$

where ε , the reduced energy of the alpha particle, is given as

$$\varepsilon = \frac{16265 M E}{Z(4+M)(1.587+Z^{2/3})^{3/2}} \quad .$$

In the above equations the atomic and mass number of the element are denoted by Z and M , respectively. The units of the nuclear stopping power are eV-cm²/10¹⁵ atoms.

STOPPING POWER VALUES FOR COMPOUNDS

Assuming Bragg additivity, the mass stopping power for a compound (S/ρ) is evaluated as

$$S/\rho = 602.3 \sum_{i=1}^n \left[\frac{f_i}{MW_i} \right] (S_e + S_n)_i \quad , \quad (3)$$

where f_i is the mass fraction of element i in the compound and MW_i is the molecular weight of element i . The numerical constant, 602.3, represents the product of Avagadro's constant, the conversion from eV to MeV, and the factor of 10¹⁵. The unit of mass stopping power is

MeV-cm²/g. Stopping power estimates from various sources are compared in Fig. 1.

RANGE OF ALPHA PARTICLES

The range of the alpha particle in the medium of interest can be estimated by numerical integration of the stopping power; i.e.,

$$R(E) = \int_{E_0}^E \frac{d\varepsilon}{S/\rho(\varepsilon)} + R(E_0) \quad (4)$$

where the lower integration limit represents an initial energy, E_0 , with a corresponding range $R(E_0)$. In the calculation presented here we have taken E_0 to be the lower range of the empirical relationships, i.e., 1 keV. The residual range was approximated as E_0 divided by $S/\rho(E_0)$. As suggested by Ziegler, the range-energy relationship for various materials can be fitted to a fifth-order polynomial,

$$R(E) = \exp \sum_{i=0}^5 A_i \ln(E)^i \quad (5)$$

Coefficients for several materials of interest are given in Table 1.

POINT ISOTROPIC SPECIFIC ABSORBED FRACTION

The energy deposition per unit mass in a medium as a function of distance from a point isotropic source is referred to as the point isotropic specific absorbed fraction. This is the most basic form of the radiation transport consideration. This quantity has been tabulated for electron and photon radiations, but no such tabulations exist for alpha particles. The point isotropic specific absorbed fraction, $\Phi(r, E_0)$, at distance r from a source of monoenergetic particles of energy E_0 is given as

$$\Phi(r, E_0) = \frac{1}{4\pi r^2} \frac{1}{E_0} S/\rho(E) \quad (6)$$

where S/ρ is the mass stopping power of the particle after traveling a distance r and E_0 is the emitted energy. A tabulation of Φ in soft tissue is given in Table 2 for alpha particles of 4, 6, and 8 MeV; this is illustrated in Fig. 2.

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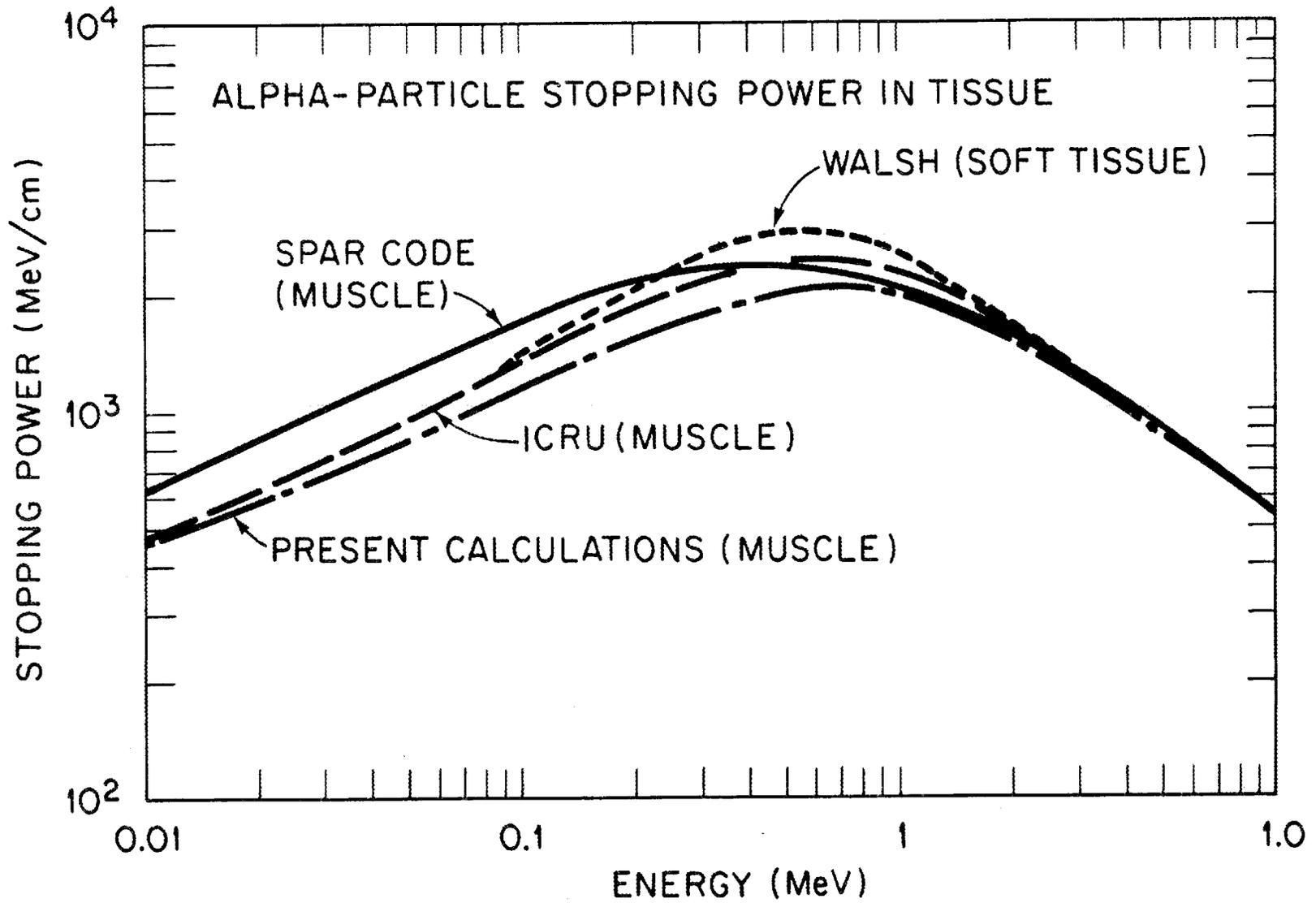


Figure 1. Comparison of stopping power estimates for soft tissue.

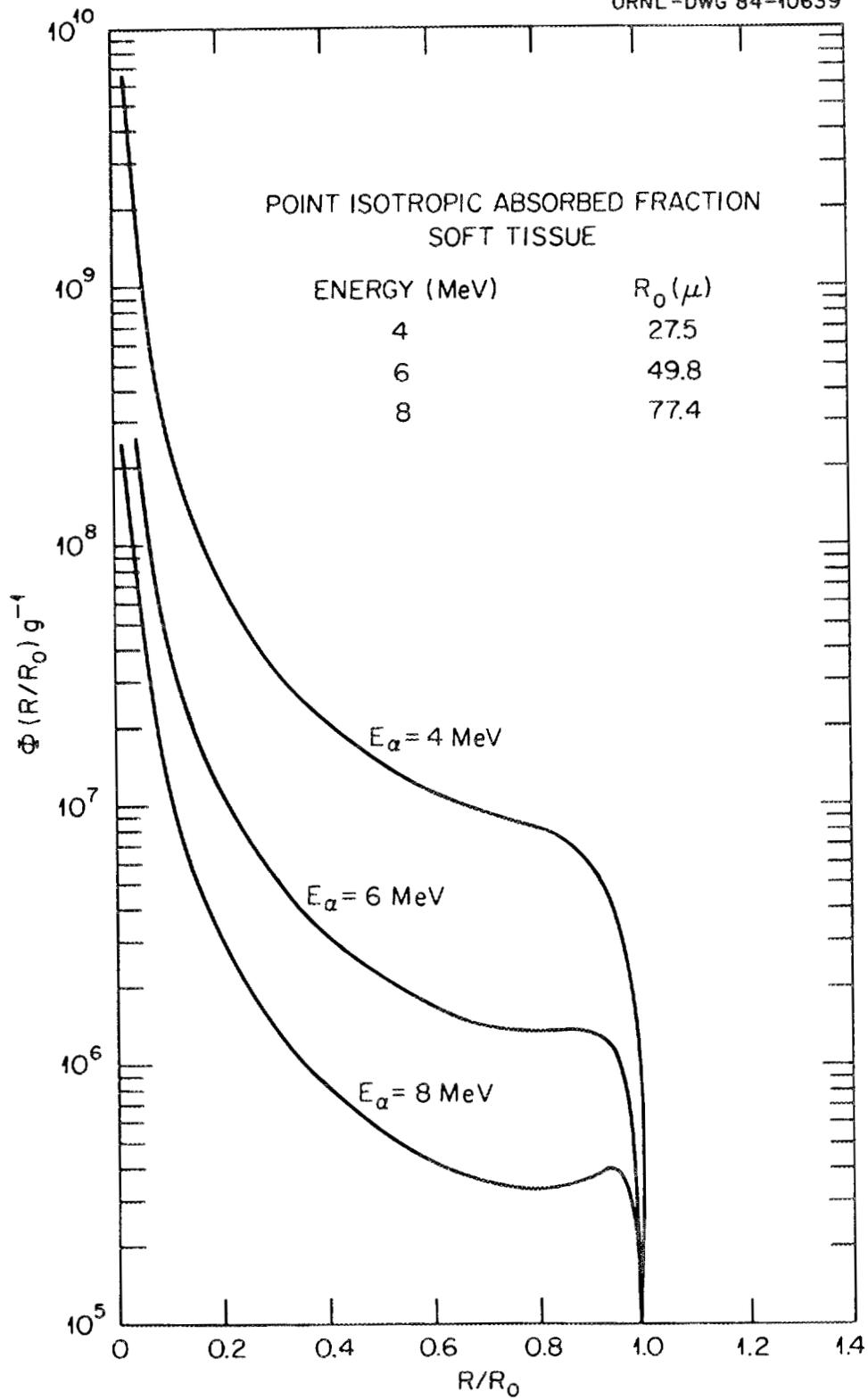


Figure 2. Point isotropic absorbed fraction for alpha particles in soft tissue.

Table 1. Coefficients of the Range-Energy Relationship for Various Materials^o

Coefficients	Compounds					
	S. Tissue		Bone		Dry Air	Water
	(Kerr)	(ICRU)	(Kerr)	(ICRU)		
A ₀	-7.376664	-7.334316	-7.153502	-7.281057	-7.263134	-7.321865
A ₁	0.867641	0.850616	0.847353	0.871201	0.828772	0.839046
A ₂	0.127010	0.126893	0.127340	0.127318	0.144029	0.127250
A ₃	2.226E-2	2.300E-2	2.218E-2	2.160E-2	2.452E-2	2.343E-2
A ₄	1.244E-4	2.267E-4	1.932E-4	7.007E-5	9.687E-5	2.292E-4
A ₅	-1.072E-4	-1.025E-5	-9.663E-5	-1.059E-4	-1.162E-4	-1.057E-4

* Range R (g/cm²) is given as $R = \exp \left[\sum_{i=0}^5 A_i \ln(E)^{i-1} \right]$ where E is the alpha particles energy (MeV).

Table 2. Point Isotropic Absorbed Fraction for Alpha Particles in Soft Tissue*

r/r_0	$\Phi(r/r_0)$ (g^{-1})			r/r_0	$\Phi(r/r_0)$ (g^{-1})		
	4 MeV	6 MeV	8 MeV		4 MeV	6 MeV	8 MeV
0.01	2.62E10	4.14E09	1.06E09	0.52	1.35E07	2.10E06	5.36E05
0.02	6.58E09	1.04E09	2.67E08	0.54	1.28E07	1.99E06	5.06E05
0.04	1.66E09	2.62E08	6.73E07	0.56	1.22E07	1.88E06	4.80E05
0.06	7.45E08	1.18E08	3.02E07	0.58	1.16E07	1.79E06	4.57E05
0.08	4.23E08	6.68E07	1.71E07	0.60	1.11E07	1.72E06	4.36E05
0.10	2.74E08	4.31E07	1.11E07	0.62	1.07E07	1.64E06	4.18E05
0.12	1.92E08	3.02E07	7.76E06	0.64	1.03E07	1.58E06	4.02E05
0.14	1.42E08	2.24E07	5.76E06	0.66	9.95E06	1.53E06	3.87E05
0.16	1.10E08	1.74E07	4.46E06	0.68	9.64E06	1.48E06	3.75E05
0.18	8.80E07	1.38E07	3.56E06	0.70	9.37E06	1.44E06	3.64E05
0.20	7.21E07	1.13E07	2.91E06	0.72	9.13E06	1.41E06	3.55E05
0.22	6.03E07	9.48E06	2.43E06	0.74	8.92E06	1.38E06	3.48E05
0.24	5.13E07	8.05E06	2.07E06	0.76	8.71E06	1.36E06	3.42E05
0.26	4.42E07	6.94E06	1.78E06	0.78	8.50E06	1.35E06	3.39E05
0.28	3.86E07	6.06E06	1.55E06	0.80	8.29E06	1.34E06	3.37E05
0.30	3.41E07	5.34E06	1.37E06	0.82	8.05E06	1.34E06	3.37E05
0.32	3.04E07	4.75E06	1.22E06	0.84	7.75E06	1.35E06	3.40E05
0.34	2.73E07	4.27E06	1.09E06	0.86	7.34E06	1.36E06	3.46E05
0.36	2.47E07	3.86E06	9.88E05	0.88	6.81E06	1.37E06	3.56E05
0.38	2.25E07	3.51E06	8.99E05	0.90	6.11E06	1.36E06	3.70E05
0.40	2.06E07	3.21E06	8.23E05	0.92	5.24E06	1.32E06	3.85E05
0.42	1.90E07	2.96E06	7.58E05	0.94	1.24E06	1.15E06	3.91E05
0.44	1.76E07	2.74E06	7.01E05	0.96	3.16E06	9.28E05	3.56E05
0.46	1.64E07	2.55E06	6.51E05	0.98	1.98E06	5.76E05	2.37E05
0.48	1.53E07	2.38E06	6.08E05	0.99	1.37E06	3.76E05	1.50E05
0.50	1.44E07	2.23E06	5.70E05	1.0	0.0	0.0	0.0

* The range in tissue is denoted as r_0 ; r_0 is 27.54, 49.76, and 77.37 μm for alpha particles of 4, 6, and 8 MeV energy.

GLOBAL ENVIRONMENTAL TRANSPORT MODELS FOR TRITIUM

G. G. Killough and D. C. Kocher

The purpose of this study is to identify some of the obstacles to the construction of credible models of tritium transport for use in dose assessments. These difficulties are illustrated by comparing model predictions of environmental tritium levels with measurements.

Environmental monitoring of tritium has shown that specific activities in precipitation over land are typically higher by a factor of three to four than those in precipitation over the oceans. Experience with modeling CO₂ turnover in the oceans has led to the conclusion that two-box reservoir models of the ocean often give unsatisfactory representations of transient solutions. Failure to consider these factors in global models can lead to distorted estimates of collective dose and create difficulties in validation of the model against real data.

We illustrate these problems with a seven-box model recommended by the National Council on Radiation Protection and Measurements (NCRP), in which we forced the atmospheric compartment to reproduce an exogenous function based on historic observations of HTO in precipitation at 50 degrees N. The fresh water response underestimates data from the Ottawa River by nearly an order of magnitude and the ocean surface response overestimates tritium data from the surface waters of the Northern Pacific by a factor of about three (Fig. 1). Revision of the model to include (1) separate over-land and over-ocean compartments of the atmosphere and (2) a box-diffusion model of the subsurface ocean brings the discrepant responses into good agreement with the environmental data, as the solid curves in Fig. 1 indicate.

In a second exercise, we used a latitudinally disaggregated model and replaced a tropospheric compartment in the northern hemisphere by historic precipitation data (Fig 2.). The model's response greatly underestimates the tritium specific activity in the southern hemisphere. The large discrepancy probably indicates that much of the release from weapons testing occurred in the stratosphere and that a significant

fraction of the release occurred as HT rather than HTO. These exercises lead us to doubt that a proper global transport model for tritium is available at present for collective dose assessment.

These results were presented at the Second Topical Meeting on Tritium Technology in Fission, Fusion and Isotopic Applications, Dayton, Ohio, April 30-May 2, 1985 and will appear in a special supplement to the September 1985 issue of Fusion Technology.

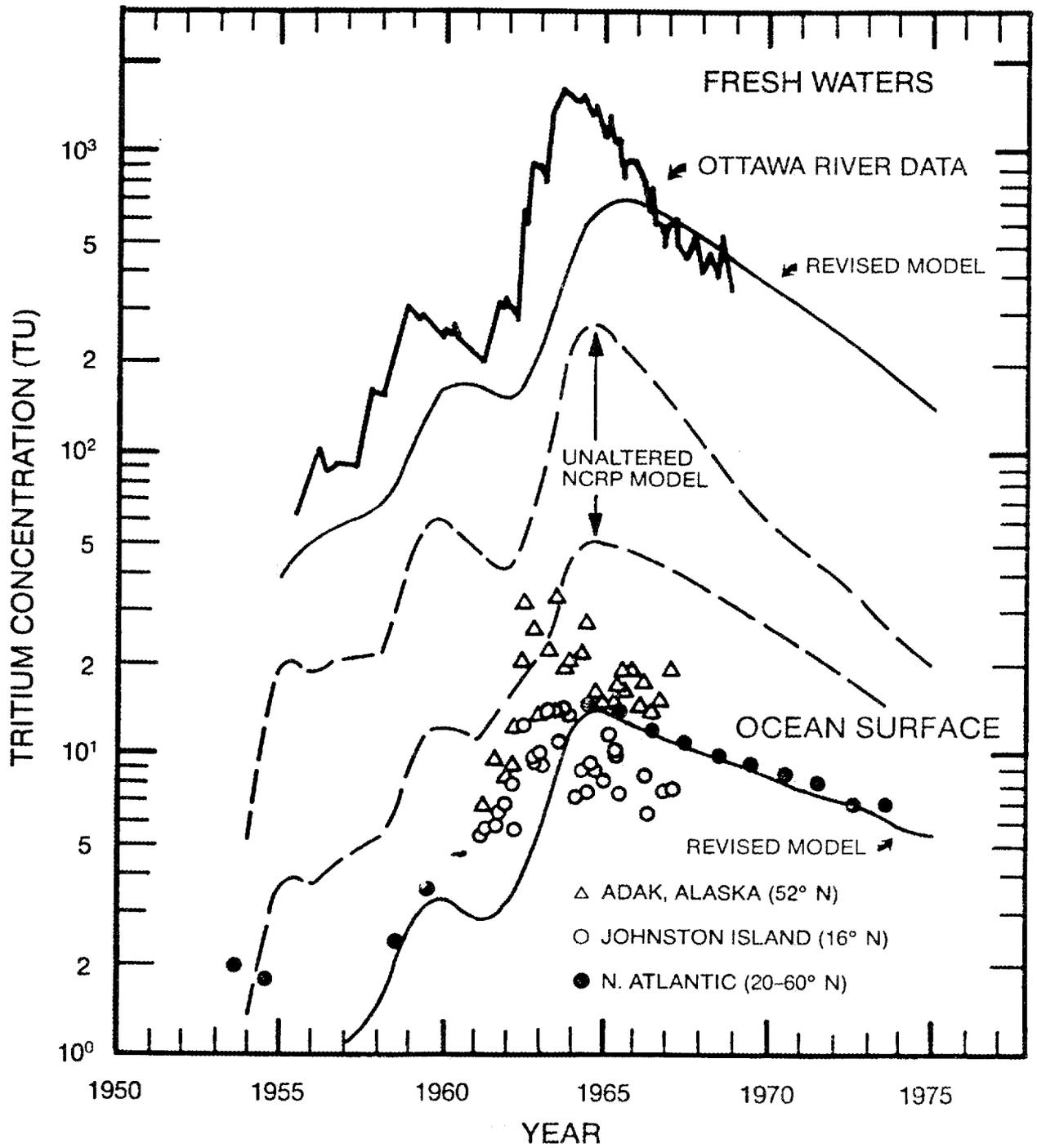


Figure 1. NCRP model predictions compared with observations of environmental tritium levels.

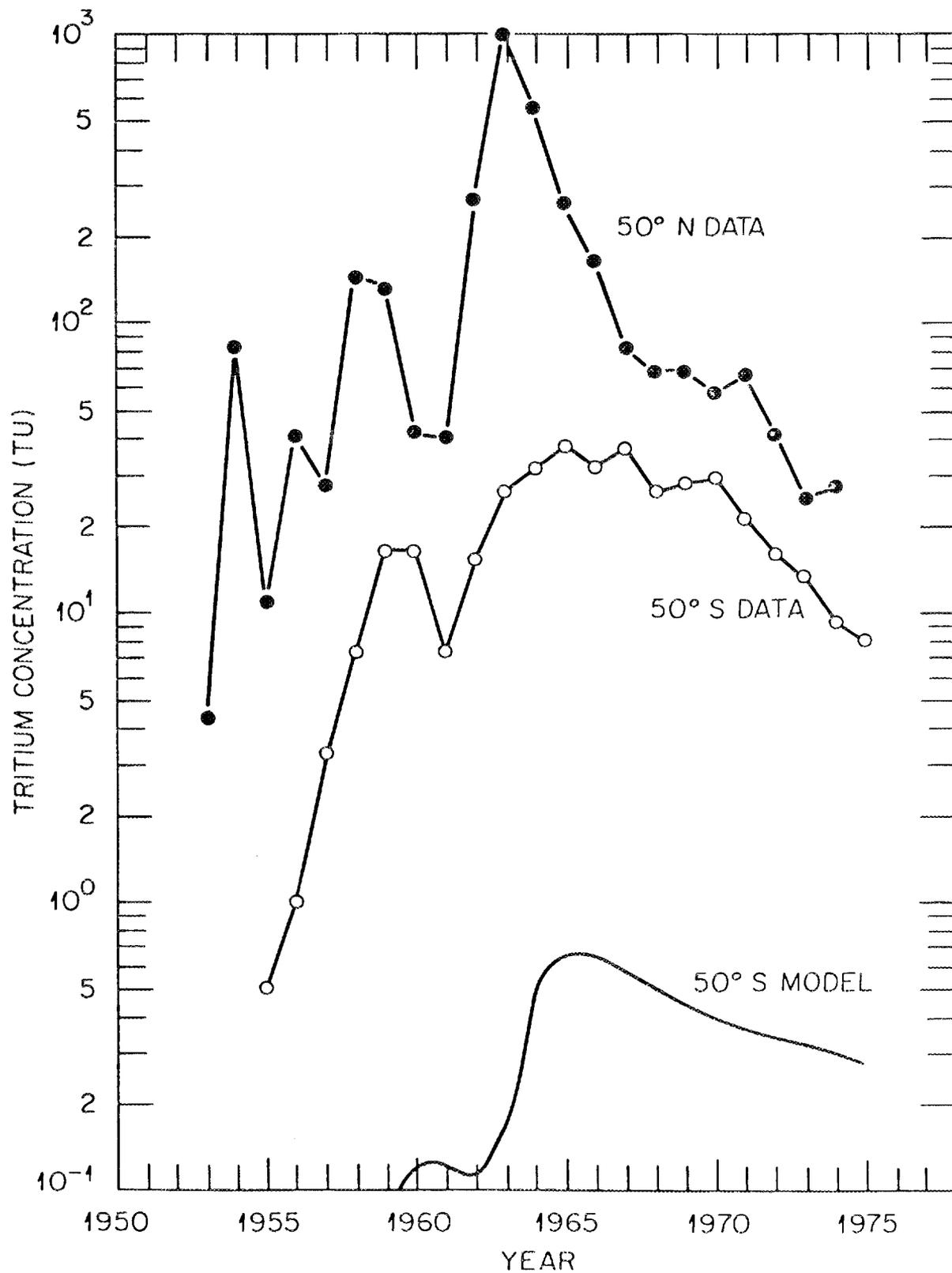


Figure 2. Bergman model predictions compared with observations of tritium levels in precipitation.

PROBLEMS IN ESTABLISHING ANNUAL INTAKE LIMITS FOR AN ANNUAL DOSE SYSTEM

K. F. Eckerman

INTRODUCTION

In Publication 2 of the International Commission on Radiological Protection (1959), restrictions on the intake of radionuclides by workers and members of the public were based on limiting the dose equivalent rate in body organs after 50 years of continuous intake. In the dose limitation system of ICRP Publication 26 (ICRP 1975), intakes are restricted by limiting the committed dose equivalent, as is evident in ICRP Publication 30 (ICRP 1979). It is generally acknowledged that these systems are mathematically equivalent and embody comparable conservatism with regard to cumulative dose equivalent from intakes of tenaciously retained long-lived radionuclides relative to radionuclides briefly retained.

Recently an alternative system has been discussed where intakes are restricted by applying the limit on dose equivalent to the annual rather than the committed dose equivalent. This system and the committed dose equivalent system of the ICRP have been compared by considering the intake of hypothetical radionuclides with very short or very long biological retention times in various organs. For radionuclides briefly retained, the dose equivalent rate following an instantaneous intake decreases rapidly with increasing time and the annual and committed dose equivalent systems are comparable. The dose equivalent rate following the intake of a radionuclide with infinite retention would be constant with time, thus restricting intake of this hypothetical radionuclide to the first year only; i.e., the concept of an annual intake limit has no meaning for such radionuclides. It is assumed that actual radionuclides will be encompassed by these extremes. We note that the dose equivalent rate following intake of some radionuclides is not bound by the hypothetical examples discussed above; the dose equivalent rate may actually be an increasing function of time. We show below that no solution exists to the dosimetric formulations of the annual dose

equivalent system for such radionuclides and illustrate this failure by considering the Pu-241/Am-241 decay chain.

DOSIMETRIC FORMULATIONS

The function describing the dose equivalent rate following an instantaneous intake of a unit activity of a radionuclide at time zero, referred to here as the response function, is a complex function of time. The complexity arises from the kinetics associated with translocation of the radionuclide from the respiratory or gastrointestinal tract and metabolic processes occurring in the organ of interest (and in all other organs in the case of photon emitters). Additionally, the function reflects the kinetics associated with the formation and translocation of daughter radionuclides. Analytical representation of the response functions are generally not available; however, numerical tabulations are readily obtained from a dosimetry computer code formulated in a general manner (see Leggett et al. 1984).

Let $\dot{H}_{I,T}(t)$ represent the dose equivalent rate in organ T at time t following an instantaneous intake of a unit activity at time zero, i.e., $\dot{H}_{I,T}(t)$ is the response function. The committed dose equivalent per unit intake, $H_{50,T}$, of ICRP-30 is simply the integral of the response function over 50 years. The dose equivalent rate in tissue T, $\dot{H}_T(t)$, arising from an arbitrary intake $\dot{I}(t)$ can be expressed in terms of the convolution integral,

$$\dot{H}_T(t) = \int_0^t \dot{I}(\tau) \dot{H}_{I,T}(t-\tau) d\tau \quad (1)$$

The annual dose equivalent in the nth year of intake is*

* The annual system has the desirable aspect that annual dose estimates could, in principle, be based on measurements of the activity residing in the body, assuming the dose equivalent rate is generally directly proportional to the activity present. It is often not appreciated that the dose equivalent rate in an organ may be independent of the activity of the inhaled or ingested radionuclide present in that organ or in the body at the time of interest.

$$H_T(n) = \int_{n-1}^n \dot{H}_T(t) dt \quad . \quad (2)$$

The cumulative dose equivalent $\tilde{H}_T(n)$, for n years of intake, can be obtained from Eq. (2) with a zero lower limit of integration or by summation of the annual dose equivalent for years 1 through n . Restricting the annual dose equivalent $H_T(n)$ in any year to the dose equivalent limit $H_{L,T}$ suggests that intakes must satisfy the following:

$$\int_{n-1}^n dt \int_0^t \dot{I}(\tau) \dot{H}_{I,T}(t-\tau) d\tau \leq H_{L,T} \quad . \quad (3)$$

Equation (3) places no constraints on the form of the intake function $\dot{I}(t)$; e.g., it might be sinusoidal. For the purpose of developing limits on intake, one might assume that the intakes within any year were uniform or that the intake occurred instantaneously on the first day of the year. However, before developing Eq. (3) further one might question whether it is reasonable to expect that a meaningful solution exists, considering the functions involved. In particular, we note that the response function $\dot{H}_{I,T}(t)$ is greater than or equal to zero for all t , may or may not be zero at time $t=0$, and may decrease or increase with time. Any intake function, $\dot{I}(t)$, which satisfies Eq. (3) must be such that $\dot{I}(t) \geq 0$ for $t \geq 0$; i.e., negative intakes, while mathematically permissible in Eq. (3), have no meaning. It is this restriction which results in failure of Eq. (3).

The solution to Eq. (3) will be meaningless (i.e., $\dot{I}(t) < 0$ for some values of t) if the permitted intakes of a radionuclide during a portion of a worker's lifetime by themselves (in the absence of external irradiation and further intakes) result in annual doses in subsequent years exceeding the limit. Prime candidates are radionuclides which head decay chains of dosimetrically significant daughter products while they themselves are of minor dosimetric significance. Such decay chains are well known, e.g., the Pb-210/Po-210 chain; however, we elected to

consider the Pu-241/Am-241 chain. For a discussion of the problems involved in modeling the behavior of decay chains in the body we refer the reader to Leggett et al. (1984).

Pu-241/Am-241 DECAY CHAIN

Plutonium-241 ($T_{1/2} = 14.4$ y) decays by beta decay to the alpha emitting daughter Am-241 ($T_{1/2} = 432.2$ y). The committed dose from Pu-241 intakes is dominated by the alpha emissions of Am-241. Response functions for endosteal tissue (bone surface) for inhalation and ingestion of Pu-241 are shown in Fig. 1. Each curve of Fig. 1 starts at one year; the dose rate at $t=0$ is zero since no Pu-241 activity has transferred from the organ of intake (lung or stomach). The annual doses in the first year per TBq of intake at $t=0$ are 44.1, 48100, and 3610 Sv/TBq, respectively, for oral intake ($f_1 = 10^{-4}$), inhalation of class W compounds, and inhalation of class Y compounds. The form of the response functions is such that the maximum in the annual dose is not reached until about year 38, at which time the values are 1070, 1.28×10^6 , and 5.54×10^5 Sv/TBq of intake at time zero. Thus the annual dose some 38 years after the intake are 24, 25, and 150 times higher than the dose in the year following the intake. If the permitted intakes in the first year represent an annual dose in that year equal to the dose equivalent limit, then in subsequent years the dose limit will be exceeded, even in the absence of further intakes.

CONCLUSIONS

The dose equivalent rate per unit intake of a radionuclide may or may not decrease with increasing time. Thus, one cannot assume the highest annual dose following an intake will occur in the year of intake. For the Pu-241/Am-241 decay chain the annual dose in subsequent years may exceed that for the year of intake by one to two orders of magnitude. This is not an isolated observation; we have identified about 20 decay chains of potential concern in this respect.

Limits on the annual intake of radionuclides cannot be constructed for the annual dose equivalent system, i.e., for some radionuclides no solution exists to the underlying mathematical expressions.

Furthermore, consideration of the annual dose in the present and the past years is not a sufficient basis for managing the radiological protection of workers.

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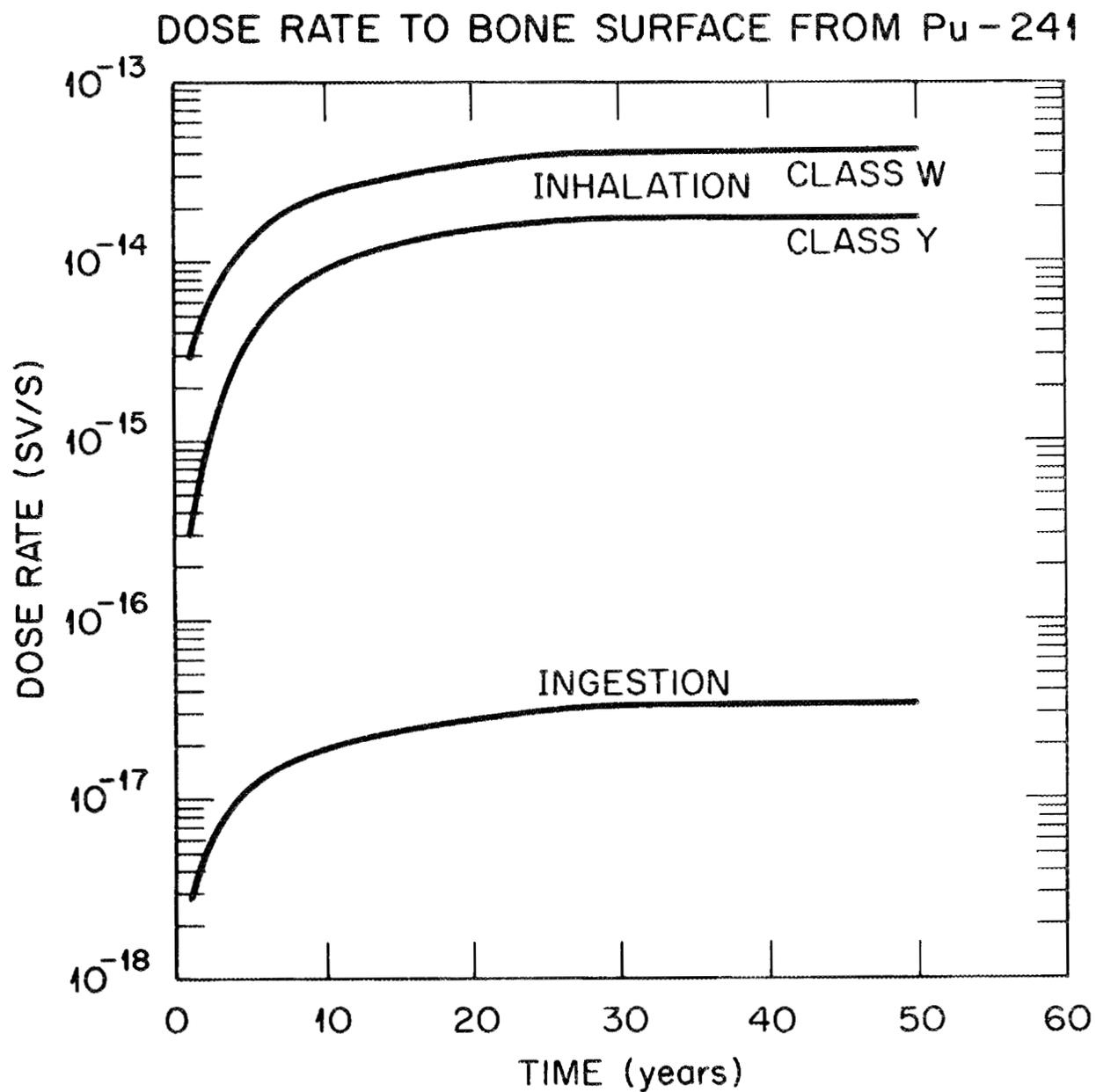


Figure 1. Dose equivalent rate (Sv/s) to bone surface following inhalation and ingestion of a unit activity (1 Bq) of Pu-241.

A COMPUTER CODE FOR ANALYSIS OF INCREASED
RISK TO ARBITRARY POPULATIONS

R. W. Leggett

INTRODUCTION

In the late 1970's the computer code CAIRD was developed by the Environmental Protection Agency to be used for estimating risk, in terms of the number of premature deaths and years of life lost, to a hypothetical cohort of persons, all simultaneously liveborn and all subject to the same competing risks throughout life (Cook, Bungler, and Barrick 1978; Bungler, Cook, and Barrick 1981). This code was used, for example, in the development of concentration guidelines for radionuclides in air and water and in population analyses made for the BEIR III document (1980), but it has the important limitation that it cannot be applied in a straightforward way to an arbitrary population or for an indefinite period. This limitation was overcome by the code SPAHR, developed for the Nuclear Regulatory Commission and Department of Energy by Collins and coworkers (1982, 1983). The main drawback of SPAHR is its length; the user must absorb a large amount of documentation before becoming fully conversant with the code.

The purpose of this study was to develop a short, elementary computer code that could be applied to populations with arbitrary age distributions, birth rates, and mortality rates to estimate risk resulting from exposures to radiation.

DESCRIPTION OF THE CODE

The computer code RISKAP (RISK to Arbitrary Populations) was designed to be readily applicable by anyone familiar with elementary Fortran. Ease of application and great flexibility were attained by presenting a concise, easily understood computational package, and allowing the user to alter the mode of input to the code to suit his/her own purposes and preferences. Moreover, the code is sufficiently short and elementary that it may be easily edited to meet special needs.

RISKAP is used to estimate risk to a population exposed to radioactivity. Risk is measured in terms of the expected number of premature deaths resulting from radiogenic cancers, the number of years of life lost as a result of these deaths, the average number of years of life lost per premature death, and, in the special case that the population consists of a single birth cohort, the decrease in life expectancy of the cohort.

One begins with a population having specified size and age distribution at reference time 0. Radiation doses that may vary with age and time, beginning at time 0 or later, are assigned by the user. These doses are used to compute an annual, age-specific risk of premature cancer death, based on a dose-response function selected by the user. The population gains newborns according to an assigned birth rate and loses persons of all ages according to an assigned age-specific mortality pattern, together with the incremental risks from the radiation exposure. Calculations of premature radiation deaths, deaths from all causes, and of the new age distribution of the population are performed for one-year intervals. The population is tracked over any specified period.

An important feature is that the population may be assigned any initial age distribution and any subsequent birth and mortality rates. Thus, age-specific mortality rates may be derived from a single life table that is thought to be representative of the study population, or mortality rates for each age group may be decreased or increased with time in accordance with recent trends or anticipated changes. Although it suffices for many applications to assume that competing risks do not change a great deal during the period of interest, it may sometimes be the case that observed or anticipated changes in non-radiogenic risks during the study period have significant impact on the estimate of the number of premature deaths. In particular, if the assumption is made that the risk of incurring a certain health effect is related to its natural incidence, then changes with time in the incidence of that health effect could be large enough over a few years to alter estimates of premature deaths substantially. A case in point is lung cancer, whose incidence has changed dramatically in some populations during the last few decades.

A pre-publication copy of the code RISKAP, along with user's guide, can be obtained from the author.

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PART II. Abstracts of publications appearing in calendar year 1984

CALCULATION OF ANNUAL LIMITS OF INTAKE OF RADIONUCLIDES BY WORKERS:
SIGNIFICANCE OF BREAST AS AN EXPLICITLY REPRESENTED TISSUE

M. Cristy

ABSTRACT

Specific absorbed fractions (SAF's) given herein provide data useful for calculating radiation doses from internally deposited radionuclides to the breasts, an organ important because of its radiosensitivity. These data were generated using a mathematical phantom which has the dimensions and weight of a reference adult female and has a breast tissue compartment. Dose to the breasts is weighted heavily by the ICRP in its Publication 30 in determining annual limits of intake of radionuclides by workers (ALI's). The mathematical phantom used by the ICRP to estimate the amount of energy absorbed by various target organs from a source of photons in some organ of the body has the dimensions and weight of a reference adult male and does not have a defined breast tissue compartment. Consequently, the ICRP used the breast tissue for the purpose of calculating ALI's. Although the SAF's to the breast compartment of the adult female phantom and the SAF's to the remaining tissues compartment of the phantom used by the ICRP may differ greatly, use of these newer data should not change the ALI's by more than 20% if the SAF's for other target organs from the ICRP phantom are retained. The ALI's thus computed varied from 82 to 104% of the ALI's published by the ICRP. If SAF's from the smaller adult female phantom are used for all target organs, the ALI's varied from 62 to 95% of the ALI's published by the ICRP. These conclusions are based on test calculations with 37 nuclides which are primarily photon emitters; these tests should demonstrate maximal differences. Using metabolic models specifically designed for females and using a different weighting scheme for target organs in females (i.e., not an average for both sexes) could either enlarge or diminish the ALI'S, but these effects were not investigated.

This appeared in Health Phys. 46: 283-291, 1984.

BIOASSAY DATA AND A RETENTION-EXCRETION
MODEL FOR SYSTEMIC PLUTONIUM

R. W. Leggett

ABSTRACT

A model is developed to describe retention, translocation, and excretion of systemic plutonium by humans. The intent was to construct the model in such a way as to improve present estimates of the distribution, retention, and excretion of Pu in the average adult; allow consideration of changes in rate constants as a function of age of the individual during adulthood; allow better characterization of uncertainties regarding retention and excretion of Pu by humans; illuminate areas where further research is needed; and allow improvements of isolated rate constants without alteration of the model framework. In order to meet these goals, it was necessary to construct the model on a mechanistic rather than an empirical basis. That is, the empirical curve fitting procedures that usually characterize retention models for radionuclides were avoided, and compartments and flow of activity were described as much as possible in terms of the primary anatomical and physiological entities involved in the movement of Pu in the body. Plutonium-specific information, primarily from animal studies, was used to identify these entities. Suggested parameter values for soluble Pu were based mainly on information for humans, including general physiological and anatomical information about the compartments involved as well as Pu-specific data for humans. With appropriate modifications in some of the rate constants, the model is applicable to other actinides and to insoluble forms of Pu.

The work described here was published as ORNL/TM-8795, May 1984, and will also appear as a journal article in Health Physics.

A MODEL FOR THE AGE-DEPENDENT SKELETAL RETENTION OF PLUTONIUM

R. W. Leggett and K. F. Eckerman

ABSTRACT

A model of the age-dependent retention of plutonium in the body is described, with particular attention given to the heterogeneous distribution of plutonium in the skeleton. (This is an extension of the model for adults described in the previous abstract.) The main purpose of this paper is to examine the implications of this model concerning the dose to radiosensitive tissues of the skeleton following exposure to plutonium at various ages and to compare model predictions with those obtained using the ICRP metabolic model for plutonium. Integrated doses over 50 years were calculated for various age groups, assuming that one unit of activity of Pu-239 was injected into the bloodstream. It is estimated using this model that about three-fourths of the alpha energy released in the skeleton over a period of 50 years is deposited in non-sensitive tissues, for all ages at injection. For an adult, the ICRP model leads to an estimated dose commitment to active marrow that is about 2 times higher than that estimated using the present model. According to our model, there is little age dependence in 50-year dose commitments to sensitive skeletal tissues, mainly because of the large amount of recycling that occurs over 50 years among the skeleton, liver, and other tissues. These estimates refer only to an initial unit injection and thus do not reflect the potentially large difference with age in the amount of Pu-239 that may reach the bloodstream in a more typical exposure situation, particularly through the ingestion pathway.

This paper appeared in Proceedings of the IRPA, 6th International Congress, May 7-12, 1984, vol. 1, pp. 454-457.

BIBLIOGRAPHY OF LITERATURE RELEVANT TO THE REASSESSMENT
OF A-BOMB RADIATION DOSIMETRY IN HIROSHIMA AND NAGASAKI

G. D. Kerr

ABSTRACT

Radiation doses received by the survivors of the Hiroshima and Nagasaki bombings were estimated in increasingly sophisticated studies between 1950 and 1965. The latest of these estimates, designated as Tentative 1965 Doses or simply T65D values, were used as a basis for risk assessment throughout the 1970's. The T65D values have recently been subjected to critical review as a result of concern over possible changes in radiation protection standards.

A controversial 1978 study of leukemia risks indicated a large relative biological effectiveness (RBE) for neutrons at low radiation doses in Hiroshima. The U.S. National Council on Radiation Protection (NCRP) issued a cautionary statement in February 1980 advising of potential changes in neutron dose limits. At the time, it was believed that health effects observed among the Hiroshima population were primarily due to neutron radiation, while those observed in the Nagasaki population resulted from gamma radiation.

Modern studies of the atomic-bomb explosions in Japan using newer calculational techniques have indicated, however, that the neutron radiation in Hiroshima was grossly overestimated and the RBE for neutrons cannot be obtained from the data on health effects among the A-bomb survivors. Hence, numerous programs for reassessment of A-bomb radiation dosimetry have been instituted recently at national laboratories, universities, and private consulting firms within the U.S. and Japan.

Review and oversight of the reassessment are being provided by committees appointed by the U.S. National Academy of Sciences and Japanese Ministry of Health and Welfare. Their role is to ensure that the programs have a firm scientific basis, that no essential elements are overlooked, and that the final results are formulated for specific application in the epidemiological studies on the A-bomb survivors by

the joint U.S.-Japan institution known as the Radiation Effects Research Foundation (RERF).

The epidemiological data from the RERF studies form the largest resource of information on human radiation responses, particularly leukemogenesis, carcinogenesis, and mutational effects. The value of these data is dependent, however, upon the accuracy of the radiation dose estimates for each individual in the affected populations of the two cities. Thus, it is essential that the reassessment of A-bomb radiation dosimetry is well-documented and that the results are convincing to the scientific community. This bibliography provides a keyword index, author index, and master listing of over 100 published reports dealing with different aspects of the dosimetry reassessment effort.

Although the programs are still in progress, preliminary results suggest that the health effects among the affected populations in both Hiroshima and Nagasaki resulted primarily from gamma radiation. Hence, the current studies address a critical issue of public concern, namely, the risks associated with low levels of exposure to gamma radiation. It was suggested by several 1981 news items in the popular scientific press that the revisions in the A-bomb radiation dosimetry may lead to substantial changes in the dose limits for gamma and x rays. The joint effort of the various studies underway in the U.S. and Japan appears to offer the largest and most significant resource which can be brought to bear on this specific issue.

This abstract is from a report which was published as ORNL/TM-9138 (April 1984).

A CONVERSATIONAL EIGENANALYSIS PROGRAM FOR
SOLVING DIFFERENTIAL EQUATIONS

G. G. Killough and K. F. Eckerman

ABSTRACT

Dynamic models that arise in health physics applications are often expressed in terms of ordinary differential equations. In many cases, such as box models that describe material exchange among reservoirs, the differential equations are linear with constant coefficients, and the analysis can be reduced to the examination of solutions of initial-value problems for such systems. This paper describes a conversational code, DIFSOL, that permits the user to specify the coefficient matrix and an initial vector of the system; DIFSOL prints out closed-form solutions [i.e., expressed as linear combinations of terms of the form e^{-at} , $e^{-at}\cos bt$, and $e^{-at}\sin bt$] and tables of the solution, its derivative, and its integral for any specified linear combination of state variables. The program logic permits menu-driven control. We have operated a FORTRAN IV version of the code on a DEC PDP-10 for several years. A translation into interpreter BASIC has proved practical on Radio Shack TRS-80 Model I and III and IBM PC personal computers for smaller systems of differential equations (fewer than about 12 state variables).

This paper was presented at The Seventeenth Midyear Topical Meeting of the Health Physics Society, Computer Applications in Health Physics, Pasco, Washington, Feb. 5-9, 1984, and was published in the proceedings.

A MEASURE OF MODEL RELIABILITY

L. R. Williams and R. W. Leggett

ABSTRACT

A method for evaluating predictive models is developed by giving a precise and statistically meaningful interpretation to the statement that a model is accurate within a certain factor. This method is applicable to any model for which there is a set $\{y_1, y_2, \dots, y_n\}$ of observations corresponding to a set $\{x_1, x_2, \dots, x_n\}$ of model predictions. The reliability index developed here considers two types of uncertainties that may arise in the evaluation of models. First, there is an observational uncertainty that may arise from variability inherent in the phenomenon being observed and from inaccuracies and imprecisions in the measurement procedure. Second, there is a type of uncertainty characterized in terms of the difference between the predicted value x and a representative or optimal value z ; this uncertainty is associated solely with the model and usually arises from incomplete understanding of the phenomenon being modeled. Our reliability index is an estimate of $\exp[(V_1 + V_2)]^{1/2}$, where V_1 describes the observational variance and V_2 describes the variance of the model predictions from their optimal values. If the observations are made independently and, for a given prediction x , follow a lognormal distribution whose variance does not depend on x , then the probability distribution of the reliability index can be completely specified in terms of V_1 and V_2 . These statistical assumptions are often satisfied by quantities of interest in metabolism and dosimetry of radionuclides, such as radionuclide concentrations in human tissue or external radiation levels from a contaminated environment.

This work appeared in Health Physics 46, 1984, pp. 85-95.

AN ASSESSMENT OF HEALTH RISK
FROM RADIATION EXPOSURES

D. E. Dunning, Jr., R. W. Leggett, and R. E. Sullivan

ABSTRACT

A methodology has been developed to assess potential hazards from low-level exposures to radioactive pollutants. Estimates of dose rates to reference organs from inhalation of contaminated air, ingestion of contaminated food or water, immersion in contaminated air, and exposure to contaminated ground surfaces are computed using current dosimetric models. These dose rates are used in a life-table analysis to estimate the radiation-induced cancer deaths and resulting years of life lost in an exposed cohort of 100,000 persons, all born at the same time and all subject to the same competing risks of death. Radiation risk factors are based on conclusions reached in the 1972 and 1980 BEIR reports and in the 1977 UNSCEAR report. Estimates of health risk are tabulated for approximately 150 radionuclides for each of the exposure pathways.

This work appeared in Health Physics 46, 1984, pp. 1035-1051.

ON ESTIMATING DOSE RATES TO ORGANS AS A FUNCTION OF AGE
FOLLOWING INTERNAL EXPOSURE TO RADIONUCLIDES

R. W. Leggett, K. F. Eckerman, D. E. Dunning, Jr., M. Cristy,
D. J. Crawford-Brown, and L. R. Williams

ABSTRACT

This report describes a method for estimating dose rates as a function of age to tissues of the human body at arbitrary times during or after internal exposure to radioactive material. Essentially any internal exposure pathway may be considered, including inhalation, ingestion, and direct entry into the bloodstream or a body organ through an open wound or an injection. The exposure may be either acute or chronic. In the case of a chronic exposure, variable intake rates as a function of time may be considered.

The methodology allows consideration of differences with age in uptake of radionuclides from environmental sources, metabolism of these radionuclides by the body, and dose to target organs per unit activity in a source organ due to changes with age in the masses and relative geometries of the organs. At present there are few radionuclides for which sufficient information is available to allow full use of all features of the methodology. The intention has been to construct the methodology and the accompanying computer code (called AGEDOS) so that: (1) full use can be made of the relatively sparse age-dependent, nuclide-specific data now available; (2) proper consideration can be given to the generally plentiful age-dependent physiological information; (3) dose rates estimated for adults are at least as accurate as those based on ICRP models for the reference adult; and (4) constantly accumulating metabolic information can be incorporated with minimal alterations in the AGEDOS code.

This work appeared as ORNL/TM-8265 (March 1984).

AGE-DEPENDENT DOSE-CONVERSION FACTORS
FOR SELECTED BONE-SEEKING RADIONUCLIDES

M. Cristy, R. W. Leggett, D. E. Dunning, Jr., and K. F. Eckerman

ABSTRACT

The transuranic elements and the radiostrontiums are potentially important contributors to bone dose from releases from a breeder reactor. Currently available age-specific dose-conversion factors for these nuclides are based on methods of ICRP Publication 2, published in 1959. ICRP Publications 26 and 30, published in 1977 and 1979, outline methodology incorporating new models and new concepts of risk, including consideration of dose to endosteal surfaces and active bone marrow rather than dose to whole bone. This report gives dose-conversion factors for acute intake of a given radionuclide by ingestion or inhalation at various ages from birth to adulthood, using the methodology of ICRP 26 and 30, but modified and extended as appropriate to include age-dependence. Results for 32 isotopes of strontium, plutonium, americium, and curium are tabulated.

This work was published as Oak Ridge National Laboratory Report ORNL/TM-8929 (also NUREG/CR-3535), 1984.

ELECTRON EXTERNAL AND INTERNAL BREMSSTRAHLUNG SPECTRA

L. T. Dillman and K. F. Eckerman

ABSTRACT

Tables of electron external bremsstrahlung spectra are given for thirty-two electron kinetic energies ranging from 1 keV to 10 MeV. These data are based on recent tabulations of exact bremsstrahlung cross sections. At each initial electron energy T , the tables give values of $S'(k,T)$, the number of bremsstrahlung photons per MeV per initial electron multiplied by a scaling factor of $100 k/T$ where k is the photon energy, for a series of 12 k/T ratios ranging from 0.0 to 0.95. Data of this type are provided for absorber media corresponding to each of the atomic numbers $Z=1$ through 92 and for several compounds of interest in internal dosimetry. Methods of obtaining corresponding data for any desired compound are discussed.

Methods for computing the spectrum of internal bremsstrahlung per transformation in the case of beta-minus decay are also discussed and comparisons are made with experimental data and other theoretical calculations. The internal bremsstrahlung calculations of this work show good agreement with experimental measurements for the selection of allowed, non-unique and unique first-forbidden beta decaying radionuclides where comparisons have been attempted; rather poor agreement was observed for the second forbidden beta decay of ^{137}Cs .

This work is presently in review and will be published as Oak Ridge National Laboratory Report ORNL/TM-9125, 1985.

METABOLIC MODELS FOR METHYL AND INORGANIC MERCURY

S. R. Bernard and P. Purdue

ABSTRACT

Following the outbreak of mercury poisoning in Minimata, Japan (1953-60), much work has been done on the toxicology of mercury - in particular, methyl mercury. In this paper we derive compartmental models for the metabolism of methyl mercury and inorganic mercury based upon the data which have been collected since 1960. Simple three- and four-compartment models fit the short-term data very well. However, it was necessary to add long-term compartments so the model would be in keeping with the long-term data as observed in Reference Man (ICRP Publication 23) and in industrial experience. A model incorporating biotransformation of mercury would be useful but it must await further experimental evidence.

This work appeared in Health Physics 46, 1984, pp. 695-699.

FOUR-URN CATENARY MODEL FOR EXCRETION

M. Sobel, S. R. Bernard, V. R. R. Uppuluri, and C. W. Nestor, Jr.

ABSTRACT

A chain-like arrangement of four urns (a catenary system) into which different color balls (white, corresponding to radio-atoms, and black, corresponding to stable atoms) are being transferred is used to simulate the transport of atoms down the GI tract of man and animals. Into the first urn (stomach) are placed w_0 white balls and r black balls while in the 2nd (small intestines) and 3rd (large intestines) urn, only r blacks are put in, with no whites. A sample of size r is transferred from the 1st, 2nd, and 3rd urns to the 2nd, 3rd, and 4th (infinite universe) urns. From the random variable difference equations the first and second moments for the distribution of the number of radio-atoms present in each urn are obtained. The model indicates a higher variance than is indicated by actual data.

This work appeared in Bull. Math. Biol. 46, 1984, pp. 219-229.

**PART III. Publications, presentations, and other professional activities
of group members after January 1, 1984**

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- Eckerman, K. F., "Dosimetry System of ICRP-30," Health Physics Society Summer School, New Orleans, Louisiana, June 9, 1984.
- Eckerman, K. F., and Cristy, M., "Computational Method for Realistic Estimates of the Dose to Active Marrow," Sixth International Congress, International Radiation Protection Association, Berlin, Federal Republic of Germany, May 1984.
- Eckerman, K. F., Ryman, J. C., and Simpson, R. E., "Absorbed Dose Distribution About Point-Isotropic Sources," Twenty-Ninth Annual Meeting of the Health Physics Society, New Orleans, Louisiana, June 3-8, 1984.
- Kerr, G. D. and Eckerman, K. F., "Neutron and Photon Fluence-to-Dose Conversion Factors for Active Marrow of the Skeleton," Fifth Symposium on Neutron Dosimetry, Neuherberg/Munich, West Germany, September 17-21, 1984.
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- Killough, G. G. and Dunning, D. E., Jr., "Analysis of Uncertainties in CRAC2 Calculations: the Inhalation Pathway," Society for Risk Analysis 1984 Annual Meeting, Knoxville, Tennessee, Sept. 30 - Oct. 3, 1984.
- Killough, G. G. and Eckerman, K. F., "A Conversational Eigenanalysis Program for Solving Differential Equations," Seventeenth Midyear Topical Symposium of the Health Physics Society, Pasco, Washington, Feb. 5-9, 1984.

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- Ryman, J. C., Tang, J. S., Eckerman, K. F., Kerr, G. D., Cristy, M., and Warner, G. G., "Comparison of Organ Dose Estimates Derived from Monte Carlo Transport Codes," Annual Health Physics Society Meeting, New Orleans, Louisiana, June 4-8, 1984.
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- Yalcintas, M. G. and Bernard, S. R., "A Metabolic Model for Cobalt," Twenty-Ninth Annual Meeting of the Health Physics Society, New Orleans, Louisiana, June 3-8, 1984.

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