NCRP REPORT No. 46

ALPHA-EMITTING PARTICLES IN LUNGS

Recommendations of the
NATIONAL COUNCIL ON RADIATION PROTECTION
AND MEASUREMENTS

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Preface

In recognition of the fact that the potential for release of particulate plutonium and transplutonium alpha-emitting elements from nuclear power generation facilities poses questions concerning spatial distribution of dose and the resultant biological effects, the NCRP initiated a new NCRP activity concerned with alpha-emitting particles in lungs. It was not intended that a complete review of the subject would be prepared. On the other hand, the study was not to be directed exclusively to recently promulgated controversial proposals relating to standards for alpha-emitting particles in the lung. Rather, the new effort was intended to be a limited-scope study aimed at the development of a statement on the question of whether the current practice of averaging over the lungs the absorbed dose from particulate alpha-emitting radionuclides is a defensible procedure in the practice of radiation protection, and whether exposure limits derived on this basis are more, or less, conservative than those that might result from a precise consideration of the spatial distribution of the dose.

The present report was prepared by the *Ad Hoc* Committee on "Hot Particles". Serving on the Committee during the preparation of this report were:

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The Council wishes to express its appreciation to the members of the Committee for the time and effort devoted to the preparation of this report.

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Contents

I. Introduction and Definition of Scope

The potential for release of particles of plutonium and transplutonium alpha-emitting elements from nuclear power generating facilities poses urgent questions concerning spatial distribution of dose and the resultant biological effects. These questions are of special importance in the setting of standards for radiation exposure. The major problems, and the pertinent information bearing on them, have been summarized in several recent compendia (ICRP, 1969; NCRP, 1971; ICRP, 1972; Hodge, *et al.*, 1973; Bair, 1974; Bair and Thompson, 1974; Bair, *et al.*, 1974; Tamplin and Cochran, 1974).

We make no attempt, here, to review the general problem, but focus our attention on the specific problem of the biological effects of alpha-emitting particles in the lung. We address the question of whether the current practice of averaging over the lung the absorbed dose from particulate alpha-emitting radionuclides is a defensible procedure in the practice of radiation protection and whether exposure limits derived on this basis are more or less conservative than those that might result from a precise consideration of the spatial distribution of dose.

II. Distribution and Mobility of Alpha-Emitting Particles in Lungs

We must first consider the biological behavior of inhaled particles as deduced from observations on experimental animals and man (Hodge, *et al.*, 1973). Inhaled radionuclides, especially the alpha-emitting heavy elements are seldom distributed uniformly throughout the lungs. Material inhaled as insoluble particles is initially deposited nonuniformly, and may be further aggregated by cellular action. Countering this tendency to aggregation are processes leading to solubilization of the particles. Also contributing to the dispersion of radiation dose are pulmonary clearance processes which move particles around within the lung as well as removing them from the lungs.

All of the above processes are influenced by such factors as the chemical nature, the physical size and shape, and the specific radioactivity of the material inhaled; also, by the many factors that may influence the biological functioning of the respiratory system. One cannot hope to define precisely the behavior of an individual particle in such a complex system. It has been reported, however, that plutonium tends to accumulate in the lung parenchyma, often in subpleural regions in apparent association with the subpleural lymphatics, and, to a lesser extent, in peribronchiolar and perivascular regions (Morrow and Casarett, 1961; Clarke and Bair, 1964; Yerokhin *et al.*, 1971; Moskalev, 1972; and Sanders, 1973). These regions receive the major part of the long-term radiation exposure and are also the sites of cancer induction in experimental animals that inhale plutonium (Clarke and Bair, 1964; Howard, 1970; Sanders, 1973; and Lafuma *et al.*, 1974).

III. Distribution of Energy from Alpha-Emitting Particles in Lungs

In view of the complex behavior of particles in lungs, estimates of absorbed dose must be based on simplifying assumptions. One possible simplification is to average the absorbed dose over the total organ. Another possibility is to calculate the absorbed dose at the particle surface. These two approaches lead to widely divergent numerical values and both can be informative in the proper context. An approach intermediate between these extremes considers the maximum absorbed dose obtained by averaging over a "significant tissue volume". The NCRP has suggested that one cubic centimeter be considered the smallest significant tissue volume (NCRP, 1971). Such an approach has not been used regularly, however, because of the practical difficulty of application, and because of the absence of biological evidence that the absorbed dose averaged over one cubic centimeter is more relevant than the average over the total organ, the absorbed dose at the particle surface, or any of several other possible alternatives.

On biological grounds it would seem that the relevant parameter is the energy actually deposited in a cell or in a critical group of cells. Absorbed dose is a statistical concept. It does not measure the energy concentration actually deposited in individual cells. In fact, for alpha radiation, the concept of absorbed dose loses all significance at the dimensions of single cells and must be replaced by the quantity "specific energy" which is defined as the energy imparted to a microscopic region, divided by its mass (Rossi, 1967; ICRU, 1971). A 12-μm-diameter cell traversed by a single alpha particle receives a specific energy of about 20 rad and a nucleus of 5 to 6 μm-diameter traversed by a single alpha particle receives a specific energy of about 100 rad. It has been found in cell culture experiments that one alpha-particle traversal is frequently sufficient to kill a cell and that a cell traversed by more than one alpha particle will be even less likely to survive (Barendsen, 1964, 1967). The most relevant parameters at the cellular level are, therefore, the number

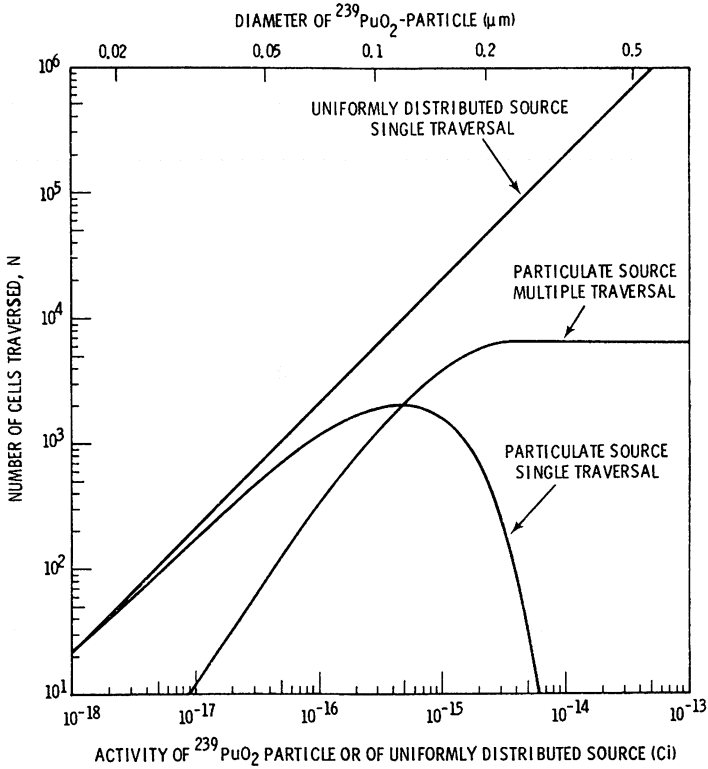

Fig. 1. Number of lung cells traversed by alpha particles during 1400 days exposure to a single, immobile $^{239}PuO_2$ particle, or to the same activity uniformly distributed. For the particulate source, curves are shown for the number of singly traversed cells and for the number of multiply traversed cells. For the uniformly distributed source, the number of multiply traversed cells is insignificant. Derivation of these curves is discussed in Appendix A. (Upper abscissa does not apply to uniformly distributed source.)

of cells traversed by a single alpha particle and the number of cells traversed by more than one alpha particle (James and Kember, 1970). Figure 1 shows the relationship of these two parameters to the activity and to the diameter of $^{239}PuO_2$ particles. It is assumed that the particles are immobile and that the exposure extends over 1400 days, an average retention time corresponding to a retention half time of about 1000 days (Bair *et al.*, 1973; Bair, 1974; Watts, 1974). Also shown in Figure 1 is the number of cells that would be traversed by a single alpha particle in 1400 days if the activity of the $^{239}PuO_2$ particle of indicated size were distributed uniformly. These relationships are derived in Appendix A and are applied to theoretical considerations in the next section.

IV. Effects of Alpha-Emitting Particles in Lungs

Theoretical Considerations

We will assume that cancer production is the most significant risk of plutonium exposure. This seems a reasonable assumption, judging from the experimental animal data (Bair, *et al.*, 1973; Bair, 1974). Much of the following argument would also be applicable, however, to other radiation effects.

From Figure 1 it is apparent that plutonium distributed uniformly throughout the lungs will irradiate many more cells than will the same quantity of plutonium distributed nonuniformly as particles. This is true except for very small particles, e.g., particles of $^{239}PuO_2$ of diameter less than about 0.1 μm and of activity less than about 3×10^{-16} Ci. Despite the greater number of cells-at-risk in the case of uniformly distributed plutonium, one can postulate mechanisms that would lead to enhanced risks from particulate sources. For example, one might postulate (1) that the cells irradiated by particles have a greater potential for cancer induction than the average carcinogenic potential of all lung cells, or (2) that the rate of induction of cancer increases with dose more than linearly. With regard to the first possibility, there is no evidence to indicate that plutonium particles in lungs concentrate in regions of exceptional radiosensitivity. This is in contrast to bone, where plutonium deposits on endosteal surfaces within alpha-particle range of sensitive cells (Vaughan, 1973). Plutonium-oxide particles accumulate in peribronchiolar and subpleural regions of the lung parenchyma (Clarke and Bair, 1964). Observations on experimental animals and atomic bomb survivors have not shown these regions to have a greater inherent potential for cancer induction than other lung regions (Severi, 1966; Nettesheim *et al.*, 1970; NAS-NRC, 1972; UNSCEAR, 1972).

With regard to the second possibility—that the rate of induction of cancer increases with dose more than linearly—one observes generally that cellular effects produced by alpha-rays are proportional to absorbed dose at low and intermediate absorbed doses and decline at higher absorbed doses due to a saturation effect. This is in contrast to the situa-

tion with low-LET beta or gamma irradiation, where cytotoxic effects often increase more than linearly with absorbed dose. This difference is explained by the presumed single-hit nature of alpha-particle damage at the cellular level as compared to the multi-hit nature of most low-LET radiation effects (Lea, 1955). The above consideration applies to individual cells. One can readily conceive of indirect mechanisms involving the interaction of undamaged, partially damaged and inactivated cells that might lead to a higher order dose dependency. Our limited understanding of the mechanisms of radiation carcinogenesis precludes quantitative prediction of such possibilities.

While no precise quantitative treatment is possible on theoretical grounds, it would seem reasonable to assume that carcinogenic potential must be in some manner related to the number of non-lethally damaged cells, and that the number of such cells is a constant fraction of singly traversed cells (James and Kember, 1970). According to Figure 1, the maximum number of singly traversed cells around one particle is reached at a particle activity of somewhat less than 10^{-15} Ci. Increase of activity beyond this level has the effect of very rapidly decreasing the number of singly traversed cells, which should result in decreased carcinogenic risk per particle and, of course, greatly decreased risk per unit activity. Thus, the enhancement of carcinogenic risk by local aggregation of damaged cells would have to be very large indeed, if for a particle of activity greater than 10^{-14} Ci, it is to outweigh the reduction in the number of cells at risk. Smaller particles of perhaps 10^{-16} Ci might seem to offer the optimum situation for interaction of singly traversed cells.

It is biologically unrealistic, however, to consider cellular interactions only over a 1400-day time period. Particles move within the lungs, and cells will be replaced by normal biological processes on a time scale much shorter than 1400 days. If we consider an exposure time of 14 days, which may approximate the average turnover time of the lung cells involved in cancer induction (Bertalanffy, 1968; Shorter, 1970), the numbers on the abscissa of Figure 1 will be increased by a factor of 100; i.e., a 100-fold more active particle will be required to achieve a specific effect. In this case, particles of about 10^{-14} Ci might offer the optimum situation for interaction of singly traversed cells and the 32 disintegrations per day from a 10^{-14} Ci particle might be sufficiently high to afford a significant opportunity for such interaction.

Whatever time period is chosen for the integration of effects, it seems clear that there must be a rather narrow range of particle activity between that which is too high to produce significant single traversals and that which is too low to produce disintegrations close enough together in time to allow interaction between traversed cells.

Despite the interesting and potentially significant implications of the above, it must be held that our understanding of the mechanisms of alpha particle-induced carcinogenesis is clearly inadequate to the task of providing a definitive answer to the question of the relative hazard of particulate versus uniformly-distributed alpha emitters on theoretical grounds. The conclusions of the above analysis do, however, provide a useful background for consideration of experimental results.

Observations in Experimental Animals

Many animal experiments have demonstrated the carcinogenic effect of plutonium in lungs; unfortunately for the problem at hand, the distribution of absorbed dose has been a controlled variable in few of these experiments. However, there is some pertinent information. Experiments are now in progress where a variety of sizes and numbers of plutonium-containing particles have been deposited in the lung capillaries of hamsters (Richmond and Sullivan, 1974). More than 2000 of these animals have lived out their normal life span. While these experiments are incomplete, and certain control data are still lacking, there is no indication of an enhanced carcinogenic effect of particulate compared to more uniformly distributed plutonium.

Many other experiments have employed particulate plutonium, and substantial aggregation of plutonium is known to occur within the lungs regardless of the form inhaled (Bair, 1974). In none of these experiments has the incidence of lung cancer been so high as to suggest a markedly enhanced effectiveness of the particulate exposure relative to more uniform irradiation of lung.

In animal studies with plutonium, significantly increased cancer incidence has not been observed at average absorbed doses of alpha radiation of less than about 30 rad, or dose equivalents less than about 300 rem (Bair, 1974).[1] While these absorbed doses are somewhat lower than those usually associated with radiation carcinogenesis, they cannot be regarded as evidence for a markedly enhanced effect of plutonium in particulate form.

Where a direct experimental comparison of less uniform versus more uniform alpha irradiation of lung tissue has been made, the less uniform

[1] In comparison to the same total absorbed dose in the lung from beta- or gamma-emitting radionuclides, alpha emitters are about 2 to 20 times more effective per unit of absorbed dose in causing lung cancer. This corresponds to the range of quality factors recommended for alpha radiation relative to low-LET radiation (NCRP, 1971).

exposure has always proved less hazardous. Thus, it was found that plutonium aggregated in asbestos-induced lesions was less carcinogenic than more uniformly distributed plutonium (Sanders, 1975). Rats exposed to relatively non-particulate soluble ^{238}Pu, which was highly dispersed during its relatively short residence time in the lungs, developed a higher incidence of lung cancer per μCi than was observed for more particulate ^{238}Pu and ^{239}Pu sources (Sanders, 1973; Sanders and Dagle, 1974). Curium-244, which was found to be more uniformly distributed throughout the lungs of rats than plutonium and americium, was also more effective in reducing survival time and inducing lung cancer than the more particulate sources (Lafuma *et al.*, 1974). The interpretation of these results is that the dispersed radionuclides exposed more cells to the carcinogenic action of the alpha radiation than the more particulate radionuclides. Similar experiments with the alpha-emitter polonium-210, uniformly distributed, or aggregated on ferric oxide particles, showed the particulate polonium to be less carcinogenic than the uniformly distributed polonium (Little and O'Toole, 1974).

Experiments on the induction of skin tumors in rats (Albert *et al.*, 1967a, 1967b, 1967c; Burns *et al.*, 1968; Heimbach *et al.*, 1969) are considered by Tamplin and Cochran (1974) to support the concept of an enhanced effectiveness of alpha irradiation from particulate sources. These experiments provided evidence for the existence of an especially sensitive substructure within the skin of certain strains of rats. They provide no evidence for the existence of similarly sensitive substructures within lungs, nor do these experiments offer any explanation for how particles might selectively irradiate such hypothetical substructures if they did exist in lungs. In fact, the experiments on skin indicated that the carcinogenic response required irradiation of the entire hair follicle, which could not be accomplished by a single alpha-emitting particle.

Observations in Man

The experience with plutonium in human beings does not support an argument for enhanced carcinogenic effectiveness of particulate plutonium (Hempelmann *et al.*, 1973; Bair, *et al.*, 1974; Healy, *et al.*, 1974). Despite the hundreds of workers that have been exposed to plutonium, mostly in particulate form, no tumors have been reported that are clearly attributable to plutonium exposure. Some of these workers received exposures in excess of present exposure limits and a large proportion of these high exposures occurred as long as 25 years ago. Thus, while nega-

tive evidence must always be viewed with caution, a substantially enhanced carcinogenic effectiveness of particulate plutonium should have become apparent by now. The presence of histologic lesions in a plutonium worker who had a small piece of plutonium metal removed from his hand (Lushbaugh and Langham, 1962), and a case of synovial sarcoma allegedly associated with the handling of a leaky carboy (Tamplin and Cochran, 1974), are not grounds for reinterpreting the effectiveness of plutonium as a lung carcinogen. If these effects were indeed due to plutonium, they would only emphasize the fact that plutonium is a potent carcinogen, which is well known from animal experiments. They provide no evidence concerning an enhanced effectiveness of the particulate source.

V. Conclusions

The substantial body of experimental animal data available at the present time indicates that particulate plutonium in the lung is no greater hazard than the same amount of plutonium more uniformly distributed throughout the lung. This is consistent with the theoretical analysis of the microscopic distribution of energy absorption in the two cases. It is therefore concluded that the current NCRP practice of averaging over the lung the absorbed dose from particulate alpha-emitting radionuclides is a defensible procedure when employed in conjunction with appropriate dose limits. A more precise consideration of the spatial distribution of absorbed dose cannot be utilized to advantage in the derivation of permissible exposure limits without a better understanding of the mechanisms involved in the translation of dose to effect. It should be stressed that these conclusions relate only to the problem of spatial distribution of absorbed dose. They are neither an endorsement nor a commentary on the absolute numerical adequacy of present NCRP standards for plutonium or other alpha-emitting radionuclides.

APPENDIX A

Estimation of Cell Traversals

If the diameter of the lung cell is taken as 12 μm, the average size of Type II pneumocytes (Hampton, 1975), then the mean traversal length is 8 μm. With its total tissue range of about 40 μm a ^{239}Pu alpha particle will, therefore, on the average, traverse five cells. If the activity is uniformly distributed throughout the lung, and if it is small enough so that the absorbed dose is much less than 20 rad, the probability of multiple traversals can be neglected. In this case one finds that the number of cells traversed during t days is:[2]

$$N = 1.6 \times 10^{16}\, tA$$

where A is the activity in Ci. The straight line in Figure 1 represents this function for $t = 1400$ days.

For the case of alpha particles arising from a fixed point (particulate plutonium), we assume an average density of 0.2 g/cm^3 for lung tissue, and consequently a spherical domain of radius 200 μm in which the fluence of alpha particles is inversely proportional to the square of the distance, r, from the center. One should note that the cells at the border of the domain may be only partly traversed by alpha particles. The energy deposition in these cells is reduced and might lead to the kind of non-lethal damage which is of particular relevance to possible mechanisms of tumor development.

We assume a population of 6600 cells in the spherical domain of radius 200 μm around a $^{239}PuO_2$ particle; this corresponds to a cell diameter of about 12 μm and a density of 0.2 g/cm^3. The fraction of these "cells at risk" that are actually traversed by alpha particles must increase as the activity of the $^{239}PuO_2$ particle increases. The calculation of the actual number of cells traversed, once or several times, is complicated because

[2] The number, ν, of decays produced by the activity A in t days is:

$$\nu = 3.7 \times 10^{10} \times 3600 \times 24 \times tA$$
$$= 3.2 \times 10^{15}\, tA$$

where A is measured in Ci. Since it is assumed that five cells are traversed by each alpha particle, the total number of cell traversals produced by the activity A in t days is $5\,\nu = 1.6 \times 10^{16}\, tA$.

one deals with a variable fluence of alpha particles in the vicinity of the $^{239}PuO_2$ particle. This fluence of alpha particles is proportional to $1/r^2$, where r is the distance from the particle. Accordingly, the expected number of traversals, μ, is:

$$\mu = k/r^2$$

if the cell is at the distance, r, from the particle. If one averages this number over the sphere of radius, R, equal to the alpha particle range, one obtains the mean number of traversals per cell in this sphere:

$$\bar{\mu} = \int_0^R r^2 k/r^2 dr \Big/ \int_0^R r^2 dr = 3k/R^2$$

According to the formula of Poisson, the probability for zero traversals at distance, r, is:

$$p_0 = e^{-k/r^2}$$

and the average of this probability over the whole sphere is:

$$\bar{p}_0 = \int_0^R r^2 e^{-k/r^2} dr \Big/ \int_0^R r^2 dr = \frac{3}{R^3} \int_0^R r^2 e^{-k/r^2} dr$$

The probability for 1 traversal at distance, r, is:

$$p_1 = k/r^2 e^{-k/r^2}$$

and the average of this probability over the whole sphere is:

$$\bar{p}_1 = \int_0^R r^2 k/r^2 e^{-k/r^2} dr \Big/ \int_0^R r^2 dr = \frac{3k}{R^3} \int_0^R e^{-k/r^2} dr$$

The probability for multiple traversals averaged over the sphere is therefore:

$$\bar{p}_{\mu>1} = 1 - \bar{p}_0 - \bar{p}_1$$

The probabilities $\bar{p}_1$ and $\bar{p}_{\mu>1}$ may be converted to absolute numbers of cells traversed on the basis of previously discussed assumptions, i.e., that each alpha particle traverses, on the average, five cells and that 6600 cells are contained in the sphere around the $^{239}PuO_2$ particle. Such values, calculated for an exposure time of 1400 days, and as a function of particle activity, are plotted in Figure 1. Using analogous formulae one can derive the number of cells traversed by up to 2 alpha particles, up to 3 alpha particles, etc. These numbers, too, decrease rapidly with increasing activity, i.e., at higher activities of the plutonium particle, one deals only with cells that are traversed by numerous alpha particles.

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The NCRP

The National Council on Radiation Protection and Measurements is a nonprofit corporation chartered by Congress in 1964 to:

1. Collect, analyze, develop, and disseminate in the public interest information and recommendations about (a) protection against radiation and (b) radiation measurements, quantities, and units, particularly those concerned with radiation protection;
2. Provide a means by which organizations concerned with the scientific and related aspects of radiation protection and of radiation quantities, units, and measurements may cooperate for effective utilization of their combined resources, and to stimulate the work of such organizations;
3. Develop basic concepts about radiation quantities, units, and measurements, about the application of these concepts, and about radiation protection;
4. Cooperate with the International Commission on Radiological Protection, the International Commission on Radiation Units and Measurements, and other national and international organizations, governmental and private, concerned with radiation quantities, units, and measurements and with radiation protection.

The Council is the successor to the unincorporated association of scientists known as the National Committee on Radiation Protection and Measurements and was formed to carry on the work begun by the Committee.

The Council is made up of the members and the participants who serve on the fifty-four Scientific Committees of the Council. The Scientific Committees, composed of experts having detailed knowledge and competence in the particular area of the Committee's interest, draft proposed recommendations. These are then submitted to the full membership of the Council for careful review and approval before being published.

The following comprise the current officers and membership of the Council:

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Currently, the following Scientific Committees are actively engaged in formulating recommendations:

SC-1: Basic Radiation Protection Criteria

SC-7: Monitoring Methods and Instruments
SC-9: Medical X- and Gamma-Ray Protection up to 10 MeV (Structural Shielding Design)
SC-11: Incineration of Radioactive Waste
SC-18: Standards and Measurements of Radioactivity for Radiological Use
SC-22: Radiation Shielding for Particle Accelerators
SC-23: Radiation Hazards Resulting from the Release of Radionuclides into the Environment
SC-24: Radionuclides and Labeled Organic Compounds Incorporated in Genetic Material
SC-25: Radiation Protection in the Use of Small Neutron Generators
SC-26: High Energy X-Ray Dosimetry
SC-28: Radiation Exposure from Consumer Products
SC-30: Physical and Biological Properties of Radionuclides
SC-31: Selected Occupational Exposure Problems Arising from Internal Emitters
SC-32: Administered Radioactivity
SC-33: Dose Calculations
SC-34: Maximum Permissible Concentrations for Occupational and Non-Occupational Exposures
SC-35: Environmental Radiation Measurements
SC-36: Tritium Measurement Techniques for Laboratory and Environmental Use
SC-37: Procedures for the Management of Contaminated Persons
SC-38: Waste Disposal
SC-39: Microwaves
SC-40: Biological Aspects of Radiation Protection Criteria
SC-41: Radiation Resulting from Nuclear Power Generation
SC-42: Industrial Applications of X Rays and Sealed Sources
SC-44: Radiation Associated with Medical Examinations
SC-45: Radiation Received by Radiation Employees
SC-46: Operational Radiation Safety
SC-47: Instrumentation for the Determination of Dose Equivalent
SC-48: Apportionment of Radiation Exposure
SC-49: Radiation Protection Guidance for Paramedical Personnel
SC-50: Surface Contamination
SC-51: Radiation Protection in Pediatric Radiology and Nuclear Medicine Applied to Children
SC-52: Conceptual Basis of Calculations of Dose Distributions
SC-53: Biological Effects and Exposure Criteria for Radiofrequency Electromagnetic Radiation
SC-54: Bioassay for Assessment of Control of Intake of Radionuclides

In recognition of its responsibility to facilitate and stimulate cooperation among organizations concerned with the scientific and related aspects of radiation protection and measurement, the Council has created a category of NCRP Collaborating Organizations. Organizations or groups of organizations which are national or international in scope and are concerned with scientific problems involving radiation quantities, units, measurements and effects, or radiation protection may be ad-

mitted to collaborating status by the Council. The present Collaborating Organizations with which the NCRP maintains liaison are as follows:

American Academy of Dermatology
American Association of Physicists in Medicine
American College of Radiology
American Dental Association
American Industrial Hygiene Association
American Insurance Association
American Medical Association
American Nuclear Society
American Occupational Medical Association
American Podiatry Association
American Public Health Association
American Radium Society
American Roentgen Ray Society
American Society of Radiologic Technologists
American Veterinary Medical Association
Association of University Radiologists
Atomic Industrial Forum
Defense Civil Preparedness Agency
Genetics Society of America
Health Physics Society
National Bureau of Standards
National Electrical Manufacturers Association
Radiation Research Society
Radiological Society of North America
Society of Nuclear Medicine
United States Air Force
United States Army
United States Energy Research and Development Administration
United States Environmental Protection Agency
United States Navy
United States Nuclear Regulatory Commission
United States Public Health Service

The NCRP has found its relationships with these organizations to be extremely valuable to continued progress in its program.

The Council's activities are made possible by the voluntary contribution of the time and effort of its members and participants and the generous support of the following organizations:

Alfred P. Sloan Foundation
American Academy of Dental Radiology
American Academy of Dermatology
American Association of Physicists in Medicine
American College of Radiology
American College of Radiology Foundation
American Dental Association
American Industrial Hygiene Association
American Insurance Association
American Medical Association
American Mutual Insurance Alliance
American Nuclear Society
American Occupational Medical Association
American Osteopathic College of Radiology
American Podiatry Association
American Public Health Association
American Radium Society
American Roentgen Ray Society
American Society of Radiologic Technologists
American Veterinary Medical Association
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Radiation Research Society
Radiological Society of North America
Society of Nuclear Medicine
United States Energy Research and Development Administration
United States Environmental Protection Agency
United States Public Health Service

To all of these organizations the Council expresses its profound appreciation for their support.

Initial funds for publication of NCRP reports were provided by a grant from the James Picker Foundation and for this the Council wishes to express its deep appreciation.

The NCRP seeks to promulgate information and recommendations based on leading scientific judgment on matters of radiation protection and measurement and to foster cooperation among organizations concerned with these matters. These efforts are intended to serve the public interest and the Council welcomes comments and suggestions on its reports or activities from those interested in its work.

NCRP Reports

NCRP Reports are distributed by the NCRP Publications' office. Information on prices and how to order may be obtained by directing an inquiry to:

NCRP Publications
P.O. Box 30175
Washington, D.C. 20014

The extant NCRP Reports are listed below.

NCRP Report No.	Title
8	*Control and Removal of Radioactive Contamination in Laboratories* (1951)
9	*Recommendations for Waste Disposal of Phosphorus-32 and Iodine-131 for Medical Users* (1951)
10	*Radiological Monitoring Methods and Instruments* (1952)
12	*Recommendations for the Disposal of Carbon-14 Wastes* (1953)
14	*Protection Against Betatron-Synchrotron Radiations Up To 100 Million Electron Volts* (1954)
16	*Radioactive Waste Disposal in the Ocean* (1954)
22	*Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and in Water for Occupational Exposure* (1959) [Includes Addendum 1 issued in August 1963]
23	*Measurement of Neutron Flux and Spectra for Physical and Biological Applications* (1960).
25	*Measurement of Absorbed Dose of Neutrons and of Mixtures of Neutrons and Gamma Rays* (1961)
27	*Stopping Powers for Use with Cavity Chambers* (1961)
28	*A Manual of Radioactivity Procedures* (1961)
30	*Safe Handling of Radioactive Materials* (1964)
31	*Shielding for High-Energy Electron Accelerator Installations* (1964)
32	*Radiation Protection in Educational Institutions* (1966)
33	*Medical X-Ray and Gamma-Ray Protection for Energies Up to 10 MeV—Equipment Design and Use* (1968)
34	*Medical X-Ray and Gamma-Ray Protection for Energies Up to 10 MeV—Structural Shielding Design and Evaluation* (1970)

35 *Dental X-Ray Protection* (1970)
36 *Radiation Protection in Veterinary Medicine* (1970)
37 *Precautions in the Management of Patients Who Have Received Therapeutic Amounts of Radionuclides* (1970)
38 *Protection Against Neutron Radiation* (1971)
39 *Basic Radiation Protection Criteria* (1971)
40 *Protection Against Radiation From Brachytherapy Sources* (1972)
41 *Specification of Gamma-Ray Brachytherapy Sources* (1974)
42 *Radiological Factors Affecting Decision-Making in a Nuclear Attack* (1974)
43 *Review of the Current State of Radiation Protection Philosophy* (1975)
44 *Krypton-85 in the Atmosphere—Accumulation, Biological Significance, and Control Technology* (1975)
45 *Natural Background Radiation in the United States* (1975)
46 *Alpha-Emitting Particles in Lungs* (1975)

Binders for NCRP Reports are available. Two sizes make it possible to collect into small binders the "old series" of reports (NCRP Reports Nos. 8–31) and into large binders the more recent publications (NCRP Reports Nos. 32–46). Each binder will accommodate from five to seven reports. The binders carry the identification "NCRP Reports" and come with label holders which permit the user to attach labels showing the reports contained in each binder.

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Volume IV. NCRP Reports Nos. 38, 39, 40, 41
Volume V. NCRP Reports Nos. 42, 43, 44, 45, 46

(Titles of the individual reports contained in each volume are given above.)

The following NCRP reports are now superseded and/or out of print:

NCRP Report No.	Title
1	*X-Ray Protection* (1931). [Superseded by NCRP Report No. 3]
2	*Radium Protection* (1934). [Superseded by NCRP Report No. 4]
3	*X-Ray Protection* (1936). [Superseded by NCRP Report No. 6]
4	*Radium Protection* (1938). [Superseded by NCRP Report No. 13]
5	*Safe Handling of Radioactive Luminous Compounds* (1941). [Out of print]
6	*Medical X-Ray Protection up to Two Million Volts* (1949). [Superseded by NCRP Report No. 18]

7 *Safe Handling of Radioactive Isotopes* (1949). [Superseded by NCRP Report No. 30]

11 *Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water* (1953). [Superseded by NCRP Report No. 22]

13 *Protection Against Radiations from Radium, Cobalt-60 and Cesium-137* (1954). [Superseded by NCRP Report No. 24]

15 *Safe Handling of Cadavers Containing Radioactive Isotopes* (1953). [Superseded by NCRP Report No. 21]

17 *Permissible Dose from External Sources of Ionizing Radiation* (1954) including *Maximum Permissible Exposure to Man, Addendum to National Bureau Of Standards Handbook 59* (1958). [Superseded by NCRP Report No. 39]

18 *X-Ray Protection* (1955). [Superseded by NCRP Report No. 26]

19 *Regulation of Radiation Exposure by Legislative Means* (1955). [Out of print]

20 *Protection Against Neutron Radiation Up to 30 Million Electron Volts* (1957). [Superseded by NCRP Report No. 38]

21 *Safe Handling of Bodies Containing Radioactive Isotopes* (1958). [Superseded by NCRP Report No. 37]

24 *Protection Against Radiations from Sealed Gamma Sources* (1960). [Superseded by NCRP Reports Nos. 33, 34 and 40]

26 *Medical X-Ray Protection Up to Three Million Volts* (1961). [Superseded by NCRP Reports Nos. 33, 34, 35 and 36]

29 *Exposure to Radiation in an Emergency* (1962). [Superseded by NCRP Report No. 42]

The following statements of the NCRP were published outside of the NCRP Report series:

"Blood Counts, Statement of the National Committee on Radiation Protection," Radiology **63,** 428 (1954)

"Statements on Maximum Permissible Dose from Television Receivers and Maximum Permissible Dose to the Skin of the Whole Body," Am. Jr. of Roentgenol., Radium Therapy and Nucl. Med. **84,** 152 (1960) and Radiology **75,** 122 (1960)

X-Ray Protection Standards for Home Television Receivers, Interim Statement of the National Council on Radiation Protection and Measurements (National Council on Radiation Protection and Measurements, Washington, 1968)

Specification of Units for Natural Uranium and Natural Thorium, (National Council on Radiation Protection and Measurements, Washington, 1973)

Copies of the statements published in journals may be consulted in libraries. A limited number of copies of the last two statements listed above are available for distribution by NCRP Publications.

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