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Exhibit A
(Affidavit of Thomas B. Cochran

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RADIATION STANDARDS FOR HOT PARTICLES

A REPORT ON THE INADEQUACY OF
EXISTING RADIATION PROTECTION STANDARDS
RELATED TO INTERNAL EXPOSURE OF MAN TO INSOLUBLE PARTICLES
OF PLUTONIUM AND OTHER ALPHA–EMITTING HOT PARTICLES.

FEBRUARY 14, 1974

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FOLDER BIKINI - MOTION FOR PRELIM INJUNCTION AFFADAVIT OF T.B. COCHRAN (EXHIBIT A)

ARTHUR R. TAMPLIN
THOMAS B. COCHRAN

Natural Resources Defense Council
1710 N Street, N.W.
Washington, D. C.
20036

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TABLE OF CONTENTS

I Introduction 1

II Plutonium Use and Public Health 3

III Existing Standards for Plutonium Exposure 6

IV Calculating the Dose Due to Insoluble Alpha-Emitters . . 11

A The Dose Equivalent. 11

B Modifying Factors 13

C The Hot Particle Problem 18

V Biological Data Related to the Cancer Risk from Insoluble Plutonium Exposure 21

A The Geesaman Hypothesis 22

B Related Human Experience 26

C Related Lung Experiments 29

VI Critical Particle Activity 32

A Exposure at Rocky Flats 34

B Manhattan Project Workers 38

C Weapons Test Fallout 41

VII Exposure Standards for Hot Particles 41

A Occupational Exposure 42

B Exposure of the General Public 44

C Exposure from Accidental Releases 46

D Surface Contamination 48

E As Low as Practicable Hearings 50

VIII Summary of Recommendations 51

Appendix A Radiation Standards Setting Organizations and Their Roles

Appendix B Statement Submitted to Attorneys for Mr. Edward Gleason

Glossary

I. Introduction

This report is written in support of a petition by the Natural Resources Defense Council to the Environmental Protection Agency (EPA) and the Atomic Energy Commission (AEC) requesting (1) a reduction of the existing radiation protection standards applicable to the internal exposure of man to insoluble alpha-emitting hot particles and (2) the establishment, with respect to such materials, of standards governing the maximum permissible concentrations in air and maximum permissible surface contamination levels in unrestricted areas.

Before proposing modifications to existing radiation protection standards related to plutonium exposure[1], we review in the following section the gravity of the public health concern as plutonium becomes a principal article of commerce in the nuclear power industry.

1/ While much of this report focuses narrowly on plutonium-239, the discussion is, nevertheless, germaine to all radionuclides in insoluble particles with a high specific activity. (The definition of specific activity and other technical terms in this report are given in the Glossary). The justification for focusing on plutonium has been aptly stated by the International Commission on Radiological Protection (ICRP): "the emphasis on plutonium is clearly a reflection of the general consensus that, in terms of amount available, projected usage, extent of anticipated accidental human exposure, and radiotoxicity, plutonium is the most formidable radionuclide in the periodic table." [ICRP Publication 19, "The Metabolism of Compounds of Plutonium and Other Actnides," Pergamon Press, 1972, p.1.]

This is followed in Section III by a review of the specific radiation protection regulations that are in force in the United States today and which are at issue. This section focuses on the existing guidelines for Pu-239, but it is to be understood that, in this and subsequent sections, it should be applied to all alpha-emitting radionuclides that meet the hot particle criteria developed in this report. Before reading Section III, those unfamiliar with the national and international organizations which have primary responsibility for recommending or establishing radiation protection standards, may find it useful to read Appendix A, where these organizations and their authority are reviewed.

Section IV presents assumptions inherent in the existing radiation protection standards and identifies those assumptions that are inappropriate when applied to insoluble alpha-emitting particulates. The biological data which demonstrate that these assumptions are inappropriate when applied to hot particles are discussed in Section V.

Utilizing the data presented in Section V, the criteria that define a hot particle are developed in Section VI. Recommendations for exposure standards for hot particles are then developed in Section VII and summarized in Section VIII.

II. Plutonium Use and Public Health

Plutonium occurs in nature, although in such small amounts that it does not constitute a practical source of the element[2]. Plutonium is bred in nuclear reactors by the capture of neutrons in uranium-238. To date, the nuclear weapons program has been the principal source of plutonium. However, it is anticipated that the commercial nuclear power industry will become the principal source of this material within the next two decades. In today's commercial reactors plutonium is produced as a by-product in the production of electricity.

As a result of the growth of the nuclear power industry, the AEC estimates that the total cumulative production of plutonium in the commercial sector of the United States will be some 4.5 million kilograms by the year 2000[3]. Since plutonium, like uranium, can serve as a reactor fuel, both are recovered from spent reactor fuel in anticipation that they will be recycled. The reactor together with the variety

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2/ The ratio of the concentrations of plutonium-239 to uranium in ores varies from 4×10^{-13} to 1.5×10^{-11}. Katz, J.J., Chapter VI, The Chemistry of Actinide Elements, Methuen and Co., Ltd., London, 1957, pp. 239-330.

3/ Environmental Statement, Liquid Metal Fast Breeder Reactor Demonstration Plant, USAEC, WASH-1509, April 1972, p. 149.

of support activities required both to provide raw fuel and to recover and recycle the uranium and plutonium make up what is known as the nuclear fuel cycle. The AEC has projected that over 4 million megawatts of nuclear capacity will be installed between 1970 and 2020[4]. Over the lifetimes of these plants this installed capacity could result in a cumulative flow of approximately 200 million kilograms of plutonium through the nuclear fuel cycle.

In today's commercial reactors the plutonium is in oxide form, PuO_2[5]. At various facilities in the nuclear fuel cycle, aerosols of PuO_2 are released to the environment on a routine basis. In addition, there are numerous points in the fuel cycle where accidents, particularly those associated with fire or explosions, can release significant amounts of PuO_2 as aerosols that can be inhaled by man.

These small aerosol particles of PuO_2 are highly radioactive. An appreciable fraction of the inhaled PuO_2 particles are trapped in the deep respiratory tissue of the lung, where, because they are insoluble in human tissue,

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4/ Updated (1970) Cost-Benefit Analysis of the U. S. Breeder Reactor Program, USAEC, WASH-1184, January 1972, p. 34. Four million megawatts (Mw) corresponds to 4000 nominal-size nuclear reactors -- 1000 Mw each.

5/ Some advanced reactors of the future may use fuel in carbide and nitride, rather than oxide, form.

they can remain for long periods of time and deliver a very intense radiation dose to the surrounding lung tissue.

Plutonium is one of the most potent cancer producing agents known to man. A machinist of plutonium metal carried 0.08 micrograms of plutonium-239 imbedded at the site of the puncture wound in the palm of his hand. Within the four year period before it was excized, it produced a nodule which displayed precancerous changes[6]. There is little doubt from experimental animal studies that inhaled plutonium is one of the most potent respiratory carcinogens known. There is experimental and observed evidence that plutonium concentrations in the lungs of dogs as low as 0.2 microcuries (3 micrograms of plutonium-239) produce cancer[7]. Hence, the flow of 200 million kilograms of plutonium represents a flow of over 10^{17} cancer doses, a staggering number which, as will be demonstrated subsequently, may be an underestimate of the cancer doses by several orders of magnitude.

The persistance of this toxic material, once lost to the environment, is measured in terms of thousands of years. Roughly two-thirds of the plutonium flowing in the nuclear

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6/ Lushbaugh, C.C. and J. Langham, "A Dermal Lesion from Implanted Plutonium," Archives of Dermatology, 86, October 1962, pp. 121-124.

7/ There are 0.061 curies per gram of plutonium-239. Two-tenths of a microcurie of plutonium-238 would have a mass of only 0.01 micrograms since plutonium-238 has a much higher specific activity, 17.47 curies per gram.

fuel cycle will be plutonium-239 which has a 24,400 year half-life. In other words, in 240,000 years the inventory of this hazardous material would be reduced by only a factor of 1000 due to natural radioactive decay. This material must be isolated from the environment in perpetuity.

III. Existing Standards for Plutonium Exposure

Radiation exposure standards have been established because radiation is known to produce cancer and genetic mutations in individuals irradiated. The mutations can in turn cause genetic defects in subsequent generations. The intent of the exposure standards is to limit this biological damage. The magnitude of the biological effect has been shown to be related to the radiation dose. The higher the dose the greater the effect. Therefore, the primary radiation exposure standard is one that limits the radiation dose. This primary standard is generally referred to as the maximum permissible dose and is given in units of rem/yr. We shall discuss the nature of this unit subsequently.

An individual can be exposed to radiation from sources that are external to his body as, for example, an X-ray machine or from radionuclides which emit X-ray like radiation deposited on the ground (this occurred with fallout from nuclear weapon tests). Alternately, an individual can be

irradiated by internal sources; that is, by radionuclides incorporated in body tissues. These radionuclides gain entrance into the body through inhalation or through contaminated food or water. Once inside they behave like their non-radioactive counterparts. Radioactive iodine, for example, accumulates in the thyroid gland in the same fashion as stable iodine, and radioactive strontium or calcium accumulate in the bone similar to their naturally occurring non-radioactive counterparts. The radioactive iodine will thus deliver a dosage to the thyroid gland that is many times larger than that to the other organs or to the whole body, and the radioactive strontium and calcium will mainly irradiate the bone.

Because of the uneven distribution of radionuclides in the body organs, radiation exposure standards have been developed not just for the whole body, but also for individual organs. In this report we will be referring to the maximum permissible whole body and lung doses.

Largely as a matter of convenience, secondary or derived radiation standards have been developed. These secondary standards, which limit radionuclide concentrations or organ burdens, are often more easily employed than the primary dose standards. We shall examine two secondary standards in this

report; the maximum permissible lung burden (MPLB) and the maximum permissible concentration in air (MPC_a). The MPLB is the total amount of a given radionuclide in the lung of an average size man that will result in the lung being irradiated at the maximum permissible lung dose (MPLD). The MPC_a is the concentration in air that will result in an average adult male obtaining a MPLB and hence a MPLD by breathing the air.

It is important to recognize that the MPLD is the primary standard; it applies to all radionuclides and radiation sources. The MPLB and the MPC_a are derived standards and are specific for a radionuclide. These derived standards are related to the biological properties of a radionuclide and to the form of radiation it emits.

Table I lists the existing exposure standards for employees of the nuclear industry that apply to Pu-239 in insoluble form. The MPLD of 15 rem/yr is included in the recommendations of the International Commission on Radiological Protection (ICRP)[8], the National Council on Radiation Protection and Measurements (NCRP)[9], and the Federal Radiation Council

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8/ ICRP Publication 9, Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1966), Pergamon Press, New York, 1966, p. 14.

9/ NCRP Report No. 39, Basic Radiation Protection Criteria, NCRP Publications, Washington, D. C., Jan. 15, 1971, p. 106.

(FRC)[10]. The MPC_a is included in the ICRP recommendations[11] and is also an AEC radiation standard[12]. Of the standards in Table I only the MPC_a is designated in the AEC regulations. However, this MPC_a corresponds to that tabulated in ICRP Publication 2[13] which is derived on the basis of the MPLD listed in Table I. The MPLB is also derived on the basis of the MPLD[14]. The MPLB is not included in either the recommendations of ICRP, NCRP, the guidelines of FRC, or the AEC regulations. In summary, in Table I the MPC_a (designated in AEC regulations) is consistant with the MPLD and MPLB. In Table I the MPLD applies to all forms of ionizing radiation. The MPLB and MPC_a apply specifically to Pu-239 in insoluble form[15].

10/ FRC Report No. 1, Op. cit., p. 38. The FRC has been abolished and its duties transferred to EPA.

11/ ICRP Publication 2, Report of Committee II on Permissible Dose for Internal Radiation, Pergamon Press, New York, 1960. [Appeared in Health Physics, Vol. 3, Pergamon Press, June 1960.]

12/ 10 CFR 20, Appendix B.

13/ ICRP Publication 2, Op. cit.

14/ Mann, J.R. and A.R. Kirchner, "Evaluation of Lung Burden Following Acute Inhalation of Highly Insoluble PuO_2," Health Physics, Vol. 13, 1967, pp. 877-882.

15/ The MPLB could apply to most other alpha-emitting radionuclides with long half-lives, since the alpha particle energies do not differ appreciably from the Pu-239 alpha energy.

TABLE I

Existing Occupational Exposure Guidelines

that Apply to Pu-239 in Insoluble Form*

MPLD	(ICRP, NCRP, FRC)	15 rem/yr
MPLB		0.016 uCi
MPC_a	(ICRP, AEC)	4×10^{-11} uCi/ml

*Note: See Glossary for definitions of symbols.

The exposure guidelines for Pu-239 that apply to non-occupational exposure of the general public are tabulated in Table II. Two guidelines are applied here. One is for the limiting exposure to an individual and the other is for the average exposure of a population sample. These two guidelines differ by a factor of 3. The ICRP recommendations include only the guidelines for individuals. The MPLD values within the parentheses in Table II correspond to the latest recommendation of the NCRP[16]. These latest recommendations of the NCRP have not, at this time, been incorporated into either the AEC or EPA regulations.

16/ NCRP Report No. 39, Op. cit., p. 95.

TABLE II

Existing Exposure Guidelines for Non-Occupational Exposure that Apply to Pu-239 in Insoluble Form*

	Individual	Population Average
MPLD (ICRP, NCRP, FRC)	1.5 (0.5) rem/yr	0.5 (0.17) rem/yr
MPLB	0.0016 (0.0005) uCi	0.0005 (0.00017) uCi
MPC_a (ICRP, AEC)	10^{-12} ($3x10^{-13}$) uCi/ml	$3x10^{-13}$ (10^{-13}) uCi/ml

* The MPLD values in parentheses refer to the latest recommendations of the NCRP. The MPLB and MPC_a values in parentheses correspond to the new NCRP dose recommendations.

IV. Calculating the Dose Due to Insoluble Alpha-Emitters

The purpose of this section is to examine the assumptions in the radiation standards above that are inappropriate when applied to insoluble alpha-emitting particulates such as aerosols of PuO_2. The assumptions are introduced through a review of basic definitions of radiation dose and the factors used to calculate the dose.

A. The Dose Equivalent

When an X-ray or the radiation emitted by a radionuclide passes through tissue it transfers energy to the cells in

these tissues. This energy produces chemical changes in the molecule of the cells; for example, such a chemical change could be a mutation in a gene. The radiation dose is actually a measure of the energy transferred to or absorbed by the tissue. The basic unit of dose is the rad (one rad represents the absorption of 100 ergs of energy per gram of material).

In addition to X-rays, radionuclides emit gamma rays (high energy X-rays), beta particles (electrons), and alpha particles (helium nuclei). In radiobiological experiments, it was determined that, while these various types of radiation produced the same biological effects, such as cancer, the magnitude of the effect was not the same per rad. For example, it was found that 100 rad of alpha radiation would produce roughly 10 times as many cancers as 100 rad of X-rays. Moreover, it was found that because of the special way in which Pu-239 deposits in the bone, its alpha particles were 5 times more effective in producing bone cancer than the alpha particles from radium[17]. To account for these differences in the magnitude of the observed effects at the same absorbed dose in rad, the maximum permissible dose limits are given in rem rather than rad.

The MPLD is given in rem in Tables I and II. The

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17 ICRP Publication 11, "A Review of the Radiosensitivity of the Tissues in Bone," Pergamon Press, New York, N. Y., 1967, p. 21.

rem is the unit of Dose Equivalent (DE)[18]. The DE is obtained by multiplying the absorbed dose in rad by modifying factors to correct for these observed differences in the magnitude of the effect. As a consequence, the magnitude of the effect will be the same for a given DE regardless of the nature of the radiation or the manner of radiation.

B. Modifying Factors

At the present time, two modifying factors are employed. One is the Quality Factor (QF) which accounts for differences in producing biological effects among various forms of radiation. The other is the Distribution Factor (DF) which accounts for the modification of the biological effects when a radionuclide is nonuniformly distributed in an organ. For example, the DE for X-ray to bone tissue is determined by using QF=1 and DF=1, while that for Pu-239 in the bone is determined by using a QF=10 (to account for the greater effectiveness of alpha particle irradiation) and a DF=5 (to account for the peculiar distribution of Pu in the bone)[19]. A DE=50 rem from X-rays or Pu-239 would thus induce the same number of cancers in bone but the absorbed dose from the X-rays would be 50 rad while that from Pu-239 would be only 1 rad.

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18 NCRP Report No. 39, Op. cit., p. 81.

19 ICRP Publication 11, Op. cit., p. 21.

In obtaining the derived values in Tables I and II, MPLB and MPC_a for Pu-239, a QF=10 was employed. This QF implies, as mentioned above, that the particles of Pu-239, which emit alpha particle radiation, are 10 times more effective in inducing cancer than X-rays. Although the irradiation of tissue by insoluble plutonium particles is highly nonuniform, no DF value has been assigned to these particles and hence, a DF=1 was employed in determining the derived values in Tables I and II. Ideally, the DF should be determined by the ratio of the observed effects in an organ following uniform and nonuniform radiation of the tissue with the same radionuclide; for example:

$$DF = \frac{\text{Number of cancers (nonuniform irradiation)}}{\text{Number of cancers (uniform irradiation)}}$$

Since direct experimental data are not available, it is necessary to derive the DF for insoluble Pu-239 particles from collateral data. In a subsequent section, we shall present the biological evidence that strongly suggests that a DF=1 grossly underestimates the DE for insoluble particulates of Pu-239 and, consequently, that the derived standards, MPLB and MPC_a for this radionuclide, are greatly in error.[20] In fact, it will be shown that the biological data strongly suggests that for such particles one should use a DF=115,000.

20/ This applies as well to other alpha-emitting actinides in insoluble particulate form.

Before turning to the biological data it is appropriate to discuss first the radiation field around a particle of PuO_2 and thereby define the fundamental questions that need to be answered by the collateral data from radiobiological studies.

The unique form of tissue irradiation displayed by insoluble particles of Pu-239 occurs because, when Pu-239 decays, it emits an alpha particle with an energy of 5.1 MeV. This particle has a range (produces biological damage) of only some 40-45 u (0.004 cm) in human tissue. In other words, a Pu-239 particle in tissue will only irradiate a volume of tissue enclosed in a sphere of 45 u radius. As one moves inward from the surface of this sphere, the radiation intensity increases geometrically. About half of the alpha particle energy is dissipated at 20 u (that is, with a volume that is 1/8 the total volume). This means that the average dose delivered in the first 20 u is 8 times that delivered in the remaining 20 u. The first column of Table III describes the radiation field around such a particle in soft tissue; e.g., the skin. Since the lung is a spongy tissue with a large air volume, the range of alpha particles is longer in the lung and consequently the mass of irradiated tissue is larger. Professor Donald Geesaman made a detailed analysis of plutonium

particle irradiation of deep respiratory tissue[21]. The last two columns in Table III describe the radiation field around such a particle in the lung using Geesaman's lung model[22]. The dose rate to the entire organ is given in column 2 of Table III for comparison. From Table III it is significant to note that with an assumed DF=1, the lung dose from the same particle varies by more than 8 orders of magnitude depending on whether one averages the dose over the entire lung or calculates it on the basis of the tissue exposed.

TABLE III

Radiation Dose Rate Due to a Pu-239 Particle

(1 u in diameter, 0.28 pCi[23])

	Soft Tissue Irradiated[24]	Lung: Entire Organ	Lung: Tissue Irradiated[25]	Lung: Closest 20 Alveoli[26]
Mass of Tissue	0.4 ug	1000 g[27]	65 ug	19 ug
Dose Rate (rem/yr)	730,000	0.0003	4000	11,000

21/ Geesaman, Donald P., An Analysis of the Carcinogenic Risk from an Insoluble Alpha-Emitting Aerosol Deposited in Deep Respiratory Tissue, UCRL-50387 and UCRL-50387 Addendum, Lawrence Livermore Laboratory, Livermore, Calif., 1963.

It would take 53,000 particles of the size illustrated in Table III to reach the MPLB of 0.016 uCi which results in 15 rem/yr to the entire (1000 g) lung. However, as Table III indicates, these particles would irradiate only 3.4 g of this 1000 g to the lung, but at a dose rate of 4000 rem/yr[28]. Thus, as Table III indicates, these particles result in an intense but highly localized irradiation. A fundamental question is, then: is this intense but localized irradiation more or less carcinogenic than uniform irradiation? Alternatively, is the DF for this particular form of irradiation equal to, greater than, or less than one? In the remainder of this section, we review the guidance, or more appropriately lack of guidance, for dealing with this hot particle problem.

22/ Geesaman, Donald P., UCRL-50387, pp. 8, 15.

23/ Langham, Wright H., The Problem of Large Area Plutonium Contamination, U. S. Dept. of H. E. W., Public Health Services, Seminar Paper No. 002, Dec. 6, 1968, p. 7.

24/ Long, A.B., "Plutonium Inhalation: The Burden of Negligible Consequence," Nuclear News, June 1971, p. 71.

25/ Geesaman, Donald P., UCRL-50387, pp. 8, 15. Based on Geesaman's model for a lung at one-half maximum inflation. Geesaman estimates a total of 68 alveoli at risk, each 8×10^{-6} cm^3 in volume, and deep respiratory zone tissue density of 0.12 g/cm^3.

26/ See footnote 23.

27/ Based on a lung mass of a standard man = 1000 g.

28/ This assumes that the radiation field of the 53,000 particles do not overlap.

C. The Hot Particle Problem

It is important to recognize that the ICRP has given no guidance with respect to nonuniform irradiation of the lung by insoluble alpha-emitters such as insoluble plutonium particles. In its Publication 9, the ICRP states:

> ...In the meantime there is no clear evidence to show whether, with a given mean absorbed dose, the biological risk associated with a non-homogeneous distribution is greater or less than the risk resulting from a more diffuse distribution of that dose in the lung.[29]

In effect, the ICRP is saying that there is no guidance as to the risk for non-homogeneous exposure in the lung, hence the MPC_a and the MPLB are meaningless for insoluble plutonium particles.

The NCRP offers the following and similar statement with respect to these particles:

> (210) The NCRP has arbitrarily used 10 percent of the volume of the organ as the significant volume for irradiation of the gonads. There are some cases in which choice of a significant volume or area is virtually meaningless. For example, if a single particle of radioactive material fixed in either lung or lymph node may be carcinogenic, the averaging of dose either over the lung or even over one cubic centimeter may have little to do with this case.[30]

This hot particle problem is also well recognized in the biological community. The following is extracted from a

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29/ ICRP Publication 9, Op. cit., p. 4.

30/ NCRP Report No. 39, Op. cit., pp. 79-80.

paper by Professor Donald P. Geesaman:

> So there is a hot particle problem with plutonium in the lung, and the hot particle problem is not understood, and there is no guidance as to the risk. I don't think there is any controversy about that. Let me quote to you from Dr. K. Z. Morgan's testimony in January of this year before the Joint Committee on Atomic Energy, U.S. Congress. [a] Dr. K. Z. Morgan is one of the United States' two members to the main Committee of the International Commission on Radiological Protection; he has been a member of the committee longer than anyone; and he is director of Health Physics Division at Oak Ridge National Laboratory. I quote: "There are many things about radiation exposure we do not understand, and there will continue to be uncertainties until health physics can provide a coherent theory of radiation damage. This is why some of the basic research studies of the USAEC are so important. D. P. Geesaman and Tamplin have pointed out recently the problems of plutonium-239 particles and the uncertainty of the risk to a man who carries such a particle of high specific activity in his lungs." At the same hearing, in response to the committee's inquiry about priorities in basic research on the biological effects of radiation, Dr. M. Eisenbud, then Director of the New York City Environmental Protection Administration, in part replied, "For some reason or other the particle problem has not come upon us in quite a little while, but it probably will one of these days. We are not much further along on the basic question of whether a given amount of energy delivered to a progressively smaller and smaller volume of tissue is better or worse for the recipient. This is another way of asking the question of how you calculate the dose when you inhale a single particle." [b] He was correct; the problem has come up again.

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[a] Morgan, K. Z., "Radiation Standards for Reactor Siting," in Environmental Effects of Producing Electrical Power Phase 2. Testimony presented at Hearings before the Joint Committee on Atomic Energy, 91st Congress, 1970. Washington, D. C., U. S. Government Printing Office.

[b] Eisenbud, M. Panel Discussion. In: Environmental Effects of Producing Electrical Power, Phase 2. Testimony presented at Hearings before the Joint Committee on Atomic Energy, 91st Congress, 1970. Washington, D. C., U. S. Government Printing Office.

In the context of his comment it is interesting to refer to the National Academy of Sciences, National Research Council report of 1961 on the Effects of Inhaled Radioactive Particles. [c] The first sentence reads, "The potential hazard due to airborne radioactive particulates is probably the least understood of the hazards associated with atomic weapons tests, production of radioelements, and the expanding use of nuclear energy for power production." A decade later that statement is still valid. Finally let me quote Drs. Sanders, Thompson, and Bair from a paper given by them last October. [d] Dr. Bair and his colleagues have done the most relevant plutonium oxide inhalation experiments. "Nonuniform irradiation of the lung from deposited radioactive particulates is clearly more carcinogenic than uniform exposure (on a total-lung dose basis), and alpha-irradiation is more carcinogenic than beta-irradiation. The doses required for a substantial tumor incidence, are very high, however, if measured in proximity to the particle; and, again, there are no data to establish the low-incidence end of a dose-effect curve. And there is no general theory, or data on which to base a theory, which would permit extrapolation of the high incidence portion of the curve into the low incidence region." I agree and I suggest that in such a circumstance it is appropriate to view the standards with extreme caution.[31]

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[c] U. S. NAS-NRC Subcommittee, Effects of Inhaled Radioactive Particles. Report of the Subcommittee on Inhalation Hazards. Committee on Pathologic Effects of Atomic Radiation. National Academy of Sciences - National Research Council, Washington, D. C. 1961. Publication 848. NAS-NRC/PUB-848, 1961.

[d] Sanders, C.L., R.C. Thompson, and W.J. Bair, "Lung Cancer: Dose Response Studies with Radionuclides." In: Inhalation Carcinogenesis. Proceedings of a Biology Division, Oak Ridge National Laboratory, conference held in Gatlinburg, Tennessee, October 8-11, 1969. M.G. Hanna, Jr., P. Nettesheim, and J.R. Gilbert, eds., U. S. Atomic Energy Commission Symposium Series 18, 1970. pp. 285-303. (CONF-691001).

31/ Geesaman, Donald P., "Plutonium and Public Health," Lawrence Livermore Laboratory, Calif., CT-121-70, April 19, 1970, reproduced in Underground Uses of Nuclear Energy, Part 2, Hearings before the Subcommittee on Air and Water Pollution of the Committee on Public Works, U. S. Senate, 91st Congress, 2nd Session August 5, 1970, pp. 1530-1532.

To these comments, referenced by Geesaman, can be added the comments of Dr. A. B. Long:

> ". . . there is an urgent need to dispell the sense of security and certainty that the present limits for the maximum permissible lung burden and the maximum permissible air concentration bring . . . the public should be informed of the uncertainties that exist in these limits."[32]

V. Biological Data Related to Cancer Risk from Insoluble Plutonium Particles

We have shown that insoluble alpha-emitting particles result in intense but localized radiation. They can irradiate at very high doses without being organism- or organ fatal. We said that the available biological data strongly suggests that a DF=1 grossly underestimates the DE for insoluble particulates of Pu-239, and consequently, the derived standards MPLB and MPC_a for this radionuclide are greatly in error. We now turn to the experiments involving cancer induction by intense local exposure, since these are especially relevant in judging whether or not insoluble alpha-emitting particles constitute a unique risk. Geesaman collected and analyzed the pertinent experiments, and what follows

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32 Long, A.B., Op. cit., p. 73.

is essentially a review of his analysis[33], which has become known as the "Geesaman hypothesis."

A The Geesaman Hypothesis

Dr. Roy E. Albert and co-workers performed a number of experiments on the induction of cancer in rat skin[34-36]. Albert's study of radiation-induced carcinoma in rat skin gives some quantitative description of a high-dose carcinogenic situation. A skin area of 24 cm^2 was exposed to electron radiation with various depths of maximum penetration. The dose response curves are reproduced in Figure 1. In all cases the response at sufficiently high doses (1000-3000 rem) was large, ~1-5 tumors per rat by 80 weeks post exposure. It was noted by Albert that when the dose was normalized to a skin depth of 0.27 milimeters, the three response curves became continuous (See Figure 2). Since this

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33/ Geesaman, D.P., UCRL-50387 Addendum, Op. cit.

34/ Albert, R.E., F.J. Burns, and R.D. Heimbach, "The effect of penetration depth of electron radiation on skin tumor formation in the rat," Radiation Res. 30, 1967, pp. 515-524.

35/ Albert, R.E., F.J. Burns, and R.D. Heimbach, "Skin damage and tumor formation from grid and sieve patterns of electron and beta radiation in the rat," Radiation Res. 30, 1967, pp. 525-

36/ Albert, R.E., F.J. Burns, and R.D. Heimbach, "The association between chronic radiation damage of the hair follicles and tumor formation in the rat," Radiation Res. 30, 1967, pp. 590-599.

depth is near the base of the hair follicle which comprises the deepest reservoir of epithelial cells of the germinal layer, it was suggestive that this might be a critical region in the observed carcinogenesis. The suggestion gained significance from the observations that most of the tumors are similar to hair follicles, and that in the non-ulcerogenic dose range the number of tumors per rat was in nearly constant ratio (1/2000-1/4000) with the number of atrophied hair follicles. Thus the carcinogenesis in this experiment was remarkably correlated with the dose to and specific damage of a particular skin structure. When exposures were made with stripe and sieve patterns of roughly 1 mm scale, geometrical effects were observed: most notably the cancer induction in the sieve geometry was suppressed at doses of 1700 rad but not at doses of 2300 rad. The reduction, however, was again consistent with the reduction in damage as characterized by atrophied hair follicles.

To summarize this important experiment, a high incidence of cancer was observed after intense local doses of radiation, and the carcinogenesis was proportional to the damage or disordering of a critical architectural unit of the tissue, the hair follicles.

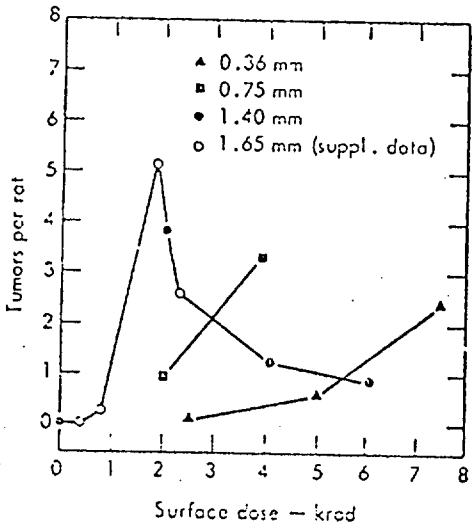

Fig. 1. Tumor incidence with respect to surface dose at 80 weeks for three penetration depths of electrons.

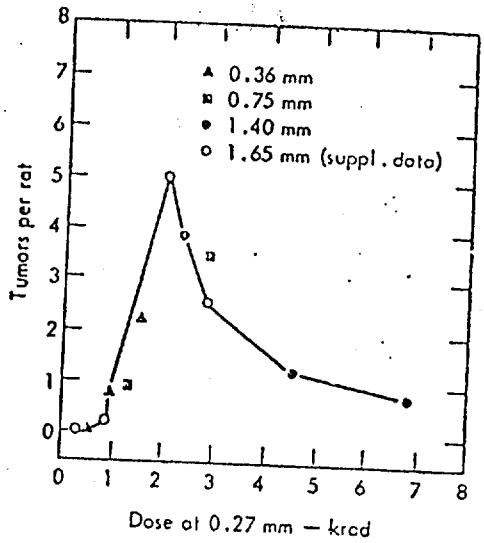

Fig. 2. Tumor incidence with respect to the dose at a depth of 0.27 mm in the skin at 80 weeks for three penetration depths of electrons.

Source of Figures: Albert, R. E., et al., Radiation Res. 30, Op. cit., pp. 515-524, Figures 5 and 7; reproduced in Geesaman, UCRL-50387 Addendum, Op. cit., p. 2.

Others have observed carcinomas and sarcomas in rats and mice after intense exposure of the skin to ionizing radiation[37-43]. Cancer induction is generally a frequent event in these experiments. Even at elevated doses, such as 12,000 rad of 1 MeV electrons, Boag and Glucksmann induced .5 sarcomas/100 cm^2 in rats[37].

A few results for rabbits, sheep, and swine were obtained at Hanford[38-41]. Despite the small number of animals

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37/ Withers, H.R., "The dose-survival relationship for irradiation of epithelial cells of mouse skin," Brit. J. Radiol. 40, 1967, pp. 187-194.

38/ Hulse, E.V., "Tumours of the skin of mice and other delayed effects of external beta irradiation of mice using ^{90}Sr and ^{32}P," Brit. J. Cancer 16, 1962, pp. 72-86.

39/ Boag, J.W. and A. Glucksmann, "Production of cancers in rats by the local application of Beta-rays and of chemical carcinogens," Progress in Radiobiology, J.S. Mitchell, B.E. Holmes, and C.L. Smith, eds. Proceedings of the Fourth International Conference on Radiobiology held in Cambridge, 14-17 August 1955. Edinburgh, Oliver and Boyd, 1956, pp. 476-479.

40/ George, L.A. and L.K. Bustad, "Gross effects of beta rays on the skin," Hanford Atomic Products Operation, Biology Research Annual Report for 1956, HW-47500, 1957, pp. 135-141.

41/ George, L.A. II, R.L. Pershing, S. Marks, and L.K. Bustad, "Cutaneous fibrosarcoma in a rabbit following beta irradiation," Hanford Atomic Products Operation, Biology Research Annual Report for 1959, HW-65500, 1960, pp. 68-69.

42/ Ragan, H.A., W.J. Clarke and L.K. Bustad, "Late effects of skin irradiation," Battelle-Northwest Laboratory Annual Report for 1965 in the Biological Sciences, BNWL-280, 1966, pp. 13-14

43/ Karagianes, M.T., E.B. Howard and J.L. Palotay, Battelle-Northwest Laboratory Annual Report for 1967 to the USAEC Division of Biology and Medicine, Vol. I, Biological Sciences, BNWL-714, 1968, pp. 1.10-1.11

involved, surface doses of 16,000 rad from a P^{32} plaque induced an average of 1 cancer/animal which is indicative that larger mammals are similarly susceptible to skin cancer after intense radiation insult. Again, these gross observations demonstrate that enhanced tumor incidence does occur after very high doses.

Intense localized radiation of the subcutaneous and intraperitoneal tissue of animals by Pu-239 has also been shown to cause a high frequency of cancer induction[43-45].

Now what are these experiments trying to tell us? Certainly a reasonable interpretation of these experimental results is: when a critical architectural unit of a tissue (e.g., a hair follicle) is irradiated at a sufficiently high dosage, the chance of it becoming cancerous is approximately 10^{-3} to 10^{-4}. This has become known as the "Geesaman hypothesis."

B Related Human Experience

Since the above experiments relate to cancer induction in animals, it is pertinent to ask whether man is more or less

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44/ Sanders, C.L. and T.A. Jackson, "Induction of Mesotheliomas and Sarcomas From 'Hot Spots' of PuO_2 Activity," Health Physics, Vol. 22, No. 6, June 1972, pp. 755-759.

45/ Lisco, Harman, et al, "Carcinogenic Properties of Radioactive Fission Products and of Plutonium," Radiology, Vol. 49, No. 3, Sept. 1947, pp. 361-363.

sensitive to such intense localized radiation. C. C. Lushbaugh reported on a lesion that developed as the result of residual Pu-239 from a puncture wound[46]. The particle contained 0.08 ug (0.005 uCi) of Pu-239. Commenting on the histological examination of the lesion, the authors state, "The autoradiographs showed precise confinement of alpha-tracks to the area of maximum damage and their penetration into the basal areas of the epidermis, where epithelial changes typical of ionizing radiation exposure were present. The cause and effect relationship of these findings, therefore, seemed obvious. Although the lesion was minute, the changes in it were severe. Their similarity to known precancerous epidermal cytologic changes, of course, raised the question of the ultimate fate of such a lesion should it be allowed to exist without surgical intervention..." In this case, less than 0.1 ug of Pu-239 produced precancerous changes in human tissue. The dose to the surrounding tissue was very intense. There is every reason to believe that a smaller quantity of Pu-239 would have produced similar changes. This precancerous lesion indicates that a single Pu-239 particle irradiates a significant (critical) volume of tissue and is capable of inducing cancer. The Lushbaugh study was

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46/ Lushbaugh, C.C. and J. Langham, Op. cit., pp. 461-464.

published in 1962. At that time the total number of puncture wounds in man was less than 1,000[47]. The treatment of such wounds was excision so that the total number of wounds displaying residual contamination by plutonium particles was certainly less than 1,000. Therefore, this wound data would suggest that insoluble plutonium particles could offer a risk of cancer induction in man that is even greater than 1/1000 per particle. In other words, when a critical unit of tissue is irradiated, man may be more susceptible to cancer than the Albert data as analyzed by Geesaman would suggest.

A second case of plutonium particle induced cancer is that of Mr. Edward Gleason. He was not associated with the nuclear industry but was a freight handler who unloaded, rotated and reloaded a crate that was contaminated by the leaking carboy of Pu-239 solution which it contained. He subsequently developed an infiltrating soft tissue sarcoma on the left palm which eventually resulted in his death. Although this case is not as clear cut as the case of the plutonium worker, there is an overwhelming medical probability that his cancer was induced by plutonium. Mr. Gleason's unfortunate contact with Pu-239 lead to a lawsuit,

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47/ Vanderwalk, J.W., "Plutonium in Puncture Wounds," HW-66172, Hanford Laboratories Operation, July 25, 1960.

Edward Gleason, et al v. NUMEC. This suit was eventually settled out-of-court. A discussion of the evidence in this case by one of the authors is presented in the Appendix B of this report.

These two cases, drawn from the relatively small number of individuals so contaminated, strongly suggest that Pu-239 particles offer a unique carcinogenic risk. They indicate that a single particle is capable of delivering an intense radiation dose to a critical volume of tissue and that this disruptively irradiated tissue, like an atrophied hair follicle, has a high probability (maybe as high as 1/1000) of becoming cancerous.

C. Related Lung Experiments

The skin experiments with animals are remarkable in that a highly disruptive dose of radiation to a small portion of repairable mammalian tissue produced frequent carcinogenesis. The chance of producing one cancer per animal is essentially unity. It is reasonable to expect that a comparable development could occur in lung tissue. While a number of radioactive substances have been used to induce lung cancers in mice and rats[48], it is difficult to derive any characterization of carcinogenesis from these experiments.

48/ Cember, H., "Radiogenic lung cancer," Progress in Experimental Tumor Research, F. Hamburger, ed. New York, Hafner Publishing Company, Inc., Vol. 4, 1964, pp. 251-303.

The work of Laskin, et al, though not specifically involving deep respiratory tissue, does demonstrate a source intensity-response curve for lung tissue[49]. A Ru-106 cylindrical source was implanted in the bronchi of rats, and cancers were observed to arise from the bronchial epithelium. The response curve indicates a substantial response (7 percent) even at 0.008 uCi burden, and a slow, approximately logarithmic increase of tumor incidence over three orders of magnitude in the source intensity. Corresponding first-year doses to adjacent bronchial epithelium varied from 10^3 rad to 10^6 rad[50]. Animals were followed until death and it was observed that the tumor incidence generally increased with the dose accumulated at death. The lowest accumulated dose associated with a cancer was 1400 rad. For an accumulated dose of the order of 10^6 rad the incidence was approximately two-thirds. Cember fortified glass beads (0.3 u diameter) with several microcuries of Sr-90, and single beads were implanted in the lungs of rats. Tumors were observed in 7 of 23 animals. In a second experiment Cember exposed rat lungs to Ce-144 particles. For

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49/ Laskin, S., M. Kuschner, N. Nelson, B. Altshuler, J.H. Harley and M. Daniels, "Carcinoma of the lung in rats exposed to the beta-radiation of intra-bronchial ruthenium-106 pellets. I. Dose response relationships," J. Natl. Cancer Inst. 31, 1963, pp. 219-231.

50/ Altshuler, B., "Dosimetry from a Ru^{106}-coated platinum pellet," Radiation Res. 9, 1958, pp. 626-632.

a burden range of 0.5 uCi to 50 uCi the observed tumor incidence fluctuated between 0.04 and 0.3[51].

All of these lung experiments involved intense exposures and a significant level of carcinogenesis. Severe damage and disruption of tissue were associated with the exposures.

The most relevant lung experiment is Bair's $Pu^{239}O_2$ inhalation study with beagles[52-54]. Exposure was to particulates of 0.25 u or 0.5 u median diameter; burdens were in the uCi range. Twenty of the 21 dogs that survived more than 1600 days post exposure had lung cancer. Many of these cancers were multicentric in origin. The cancers again appeared in conjunction with severe lung injury. Since the natural incidence of the disease is small, it appears that at this level of exposure the induction of lung cancer is a certainty during the normal beagle life span. At the same

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51/ Cember, H., Op. cit.

52/ Bair, W.J., J.F. Park, and W.J. Clarke, "Long-term study of inhaled plutonium in dogs," Battelle Memorial Institute (Richland), AFWL-TR-65-214, 1966 (AD-631 690).

53/ Park, J.F., W.J. Clarke and W.J. Bair, "Chronic effects of inhaled $^{239}PuO_2$ in beagles," Battelle-Northwest Laboratory Annual Report for 1967 to the USAEC Division of Biology and Medicine, Vol. I, Biological Sciences, BNWL-714, 1968, pp. 3.3-3.4.

54/ Park, J.F., et al, "Progress in Beagle Dog Studies with Transuranium Elements at Battelle-Northwest," Health Physics, Vol. 22, No. 6, June 1972, pp. 803-810.

time, since the pathological response is saturated in this experiment, it is inappropriate to draw any inference about the magnitude of the response at smaller burdens. The smallest burden (at death) in a dog showing lung cancer was 0.2 uCi. Presumably this would correspond to a particle burden of about 10^7 particles. Burdens which are smaller by orders of magnitude may still induce a substantial incidence of cancer. Indeed, the cancer risk may, as for skin and soft tissues, correspond to a risk per particle in the neighborhood of 1/1000 to 1/10,000.

VI. Critical Particle Activity

Not all particles would be expected to result in these high cancer probabilities. As the particle size or specific activity per particle is reduced so is the dosage to the surrounding tissue. Indeed, at sufficiently small particle size or specific activity, one would expect the radiation insult to behave similar to uniform irradiation. The study of Albert on induction of cancer in rat skin indicates a precipitous change in the dose response curve as the dosage exceeds 1,000 rem[55]. (See Figure 2). This suggests that a particular level of tissue damage must occur before this unique carcinogenic response occurs. The experiments of

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55 Albert, R.E., et al, Radiation Res. 11, Op. cit., pp. 515-524 Figure 7; reproduced in Gessaman, UCRL-50387 Addendum, Op. cit., p. 2.

Laskin, et al, indicate a significant carcinogenic response in the lung at 1400 rem, suggesting a comparable sensitivity of lung tissue[56]. Geesaman indicates that the tissue repair time in the lung is of the order of one year[57]. It therefore seems appropriate, but not necessarily conservative, to accept as guidance that this enhanced cancer risk occurs when particles irradiate the surrounding lung tissue at a dose rate of 1000 rem/yr or more.

TABLE IV

Particle Activity and Size to Give a Dose of 1000 rem/year to the Surrounding Lung Tissue[58]

	Particle Activity (pCi)	Particle Diameter (u)[59]	
		$^{239}PuO_2$	$^{238}PuO_2$
3/4 max inflated (138 alveoli)	0.14	0.8	0.12
1/2 max inflated (68 alveoli)	0.07	0.6	0.09
Closest 20 alveoli	0.02	0.4	0.06

56/ Laskin, et al, Op. cit.

57/ Geesaman, Donald P., UCRL-50387, Op. cit., p. 11.

58/ Ibid

59/ Based upon specific activity given by Langham, W.H., Op. cit., p. 7.

As seen from Table IV, using Geesaman's lung model, a particle with an alpha activity between 0.02 pCi and 0.14 pCi is required to give a dose of 1000 rem/yr to irradiated lung tissue. For purposes of establishing a maximum permissible lung particle burden we will use 0.07 pCi from long half-lived (greater than one year) isotopes as the limiting alpha activity to qualify as a hot particle. Thus, throughout the remainder of this report, hot particle will imply a particle with at least this limiting alpha activity which is insoluble in lung tissue.

A. Exposures at Rocky Flats

The AEC has a plutonium facility associated with its nuclear weapons program at Rocky Flats, Colorado. This facility is operated under contract to the AEC by the Dow Chemical Company. The employees, the environment and undoubtedly the surrounding population have been contaminated with plutonium particles as a result of the operation of this plant.[60-62] It is, therefore, pertinent here to examine the information

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60/ Mann, J.H. and A.R. Pirchnev, Op. cit.

61/ Poet, S.E. and E.A. Martell, "Plutonium-239 and Americium-241 in the Denver Area," Health Physics, Vol. 23, 1972, pp. 537-549.

62/ Richmond, Chet, Transcript of Plutonium Information Meeting of the Advisory Committee on Reactor Safeguards, Los Alamos, N. Mex., 5 January 1974, pp. 319-320.

available on the exposure of employees of the Rocky Flats facility and to relate this to the hot particle problem.

J. R. Mann and R. A. Kirchner discuss the exposures that resulted from a plutonium fire at Rocky Flats on 15 October 1965.[63] Some 400 employees were working in the room at the time the fire occurred. These employees were subsequently placed in a whole body counter to determine their lung burdens of Pu-239. However, Mann and Kirchner reported only on those 25 employees who were exposed above the MPLB of 0.016 uCi.

Table V presents the information on the exposure of these 25 employees. Utilizing the other information presented by Mann and Kirchner, we have also estimated in Table V the fraction of the lung burden activity (uCi) associated with hot particles and the number of hot particles that this represents.

63/ Mann, J.R. and R.A. Kirchner, Op. cit.

TABLE V

Rocky Flats Exposure*

Number of Cases	Total Lung Burden (uCi)	Hot Particles Lung Burden (uCi)	Number of Hot Particles
1	0.272	0.033	137,000
1	0.160	0.019	79,000
1	0.111	0.013	54,000
3	0.064	0.008	33,000
19	0.024	0.003	12,500

* Mann and Kirchner presented the lung burdens as number of MPLB. These have been converted to uCi in column two using MPLB=0.016 uCi. (For the groups with 3 and 19 cases, we selected the midpoint of the reported range.) The hot particle burden in column three was estimated by multiplying the total burden by 0.17, the fraction of the activity on particles above 0.6 u, and 0.70, the fraction of initial deposited activity that was involved in long term retention in the lung. Based on particle size data reported by Mann and Kirchner, we estimate the average hot particle activity is about 0.24 pCi. The numbers of hot particles in the last column were obtained by dividing the hot particle burdens in column three by the average hot particle activity (0.24 pCi).

Allowing a risk of cancer equal to 1/2000 per hot particle, suggests that the individuals whose exposures are presented in Table V stand a very high chance of developing lung cancer -- the probability is essentially unity. In this respect, it is significant to note that in the experiments

reported by Park, et al, the beagle dog with the smallest lung burden, i.e., 0.2 uCi, developed lung cancer.[64] The highest burden in Table V is comparable to the lowest beagle exposure; the lowest exposure in Table V, the 19 cases with lung burdens in the 0.024 uCi range are only an order of magnitude less than the lowest beagle exposure. We would suggest that this is potentially a serious situation.

As of this time, none of these individuals has developed lung cancer.[65] However, it is only 9 years since the exposure and there is good reason to suggest that the latent period (the time between exposure and the development of cancer) is much longer than this. In the beagle dog experiments, the lowest lung burden was associated with a latent period of 11 years. The latent period may be longer in man and particularly at these lower dosages and the small number of cases involved. Therefore, while these exposed individuals will be expected to supply pertinent data relative to this hot particle cancer risk over the next 10 to 20 years, these exposures give us no information at this time that would warrant modifying the risk per particle or the critical particle activity.

64/ Park, J.F., et al, Health Physics, Op. cit. p. 805.

65/ Richmond, Chet, Op. cit., p. 320.

B. Manhattan Project Workers

Another study of human respiratory exposure to plutonium relates to 25 young men exposed to plutonium during the Manhattan Project.[66] The latest examination of this group found them to be free of lung cancer although the report states, "The bronchial cells of several subjects showed moderate to marked metaplastic changes, but the significance of these changes is not clear." Such metaplastic changes are a possible indicator for detecting incipient or actual lung cancer. In one case the report indicates that the subject was a heavy smoker (3 packs/day) and undoubtedly this contributed to the changes. Nevertheless, these findings suggest that lung cancer may become manifest in some of these subjects in the future. Indeed, one would not be surprised to find one lung cancer even in such a group of non-exposed subjects. During the latest examination of these workers, *in vivo* measurement of the plutonium lung burdens were conducted with these results:

- An average MDA for a 2000-sec counting time is about 7 nCi if one uses the 95% confidence level.[67] For the 63% confidence level and a similar counting time, the comparable value is about 3.5 nCi.

66/ Hemplemann, L.H., *et al*, "Manhattan Project Plutonium Workers; A Twenty-Seven Year Follow-Up Study of Selected Cases."

67/ MDA refers to the minimum detectable amount.

> Positive counts were obtained for 14 of 21 persons measured. These counts suggested chest burdens ranging from 3 to about 10 nCi. However, in no case did the estimated chest burden exceed the MDA at the 95% confidence level. Seven of the 14 subjects with positive chest counts had estimated chest burdens of 7 nCi or greater and may be considered (at the 68% level of confidence) to have statistically significant chest burdens of from 7 to 10 nCi.[68]

Since the plutonium is still in the lung cavity, 27 years post-exposure, it is correct to assume that it was initially in the insoluble form and hence pertinent here.[69] At the time of this measurement, however, most of the material would be expected to be in the lymph nodes. Nevertheless, we could estimate the initial particle burden in these subjects from these data if we knew the initial particle size at the time of contamination. This particle size data is unavailable.

The nature of the contaminating events suggest that the particle size might have been somewhat larger than those that result from plutonium fires where most of the respirable activity resides on particles in the size range of 0.1 u to 0.5 u in diameter.[70] Much of the contamination of the

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68/ Hemplemann, L.H., Op. cit., p. 474.

69/ ICRP Publication 19, The Metabolism of Compounds of Plutonium and Other Actnides, Pergamon Press, New York, 1972, p. 7.

70/ Mann, J.R. and A.R. Kirchner, Op. cit., p. 880.

Manhattan workers resulted from aspiration of droplets of liquid solutions of plutonium into the air wherein much larger particle sizes would result. At the same time, the activity of the plutonium in the particle would be considerably less than that for a particle of PuO_2. For example, it is stated that 14 of the 25 subjects with measurable body burdens of plutonium worked in the recovery operation and that this occurred when working with solutions containing 1-40 g/liter of plutonyl nitrate to which H_2O_2 was being added with vigorous stirring in an open hood. This resulted in considerable fizzing and the discharge of droplets into the air outside the hood. A droplet 1 u in diameter (0.5 u^3) from the solution with the highest concentration (40 g/liter) would therefore contain only 6×10^{-4} pCi compared with a 0.07 pCi particle of PuO_2 [71] (a specific activity that is lower by a factor of 100).[72] In other words, the particles involved in this study do not qualify as hot particles. They are delivering dosages lower than 1000 rem/yr to the

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71/ Recall from Table IV that a 0.07 pCi, the limiting activity for a hot particle, would give a dose of 1000 rem/yr to the surrounding tissue in a lung inflated to 1/2 maximum.

72/ Of the particles of an inhaled aerosol that are deposited in the deep respiratory zone of the lung, virtually all are less than 5 u in diameter (Geesaman, UCRL-50397, Op. cit., p. 3). A 5 u droplet from the 40 g/liter solution would correspond roughly to the limiting activity of a hot particle.

surrounding tissue (roughly 10 rem/yr).

C Weapons Test Fallout

Another source of human contamination that is suggested as being pertinent to this problem is the plutonium in the fallout from nuclear weapon tests. The plutonium from weapon tests is incorporated in or deposited on particles that contain other materials and, like that for the Manhattan workers, the specific activity in these particles is much smaller than that in hot particles.

VII Exposure Standards for Hot Particles

Thus the existing biological evidence strongly suggests that an insoluble particle of Pu-239 deposited in deep respiratory tissue represents a risk of cancer induction between 1/1000 and 1/10,000. Prudent public health practices should assess the risk associated with environmental plutonium and establish exposure guidelines on the basis of these probabilities.

The existing standards for uniform radiation exposure of the whole body or lung can be used as the basis for establishing particle exposure standards by equating the risk of cancer induction between the two types of exposure (uniform vs. grossly non-uniform). The most recent assessment of the risk associated with uniform irradiation of

man was performed by the NAS-NRC Advisory Committee on the Biological Effects of Radiation. Their report, published in 1972, is referred to as the BEIR Report.[73]

A. Occupational Exposure

The existing occupational exposure standard for uniform whole body irradiation is 5 rem/yr and for the lung, 15 rem/yr. the BEIR Report estimates that exposure of the whole body of an individual to 5 rem/yr would lead to a cancer risk between 4.5×10^{-4} and 2.3×10^{-3}/yr.[74] Their best estimate is 10^{-3}/yr.[75] Their estimate of the risk of cancer to the individual from a lung exposure of the 15 rem/yr is 3×10^{-5}/yr.[76] Allowing a risk of cancer induction between 1/1000 and 1/10,000 per particle, Table V presents the maximim permissible lung particle burdens (MPLPB) that result in risks comparable to these uniform radiation standards for occupational exposure.

The MPLPB values in Table V represent a very substantial reduction in the MPLB. A hot particle of Pu-239 at the lower limit activity contains only 0.07 pCi while the MPLB for occupational exposure is 1.6×10^{4} pCi. Thus the

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73/ NAS-NRC, "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," (BEIR Report), NAS-NRC, Washington, D. C., Nov. 1972.

74/ Ibid, p. 91.

75/ Ibid, p. 91.

76/ Ibid, p. 156.

TABLE V

Occupational Exposure Guidance for Insoluble Alpha Emitters, Maximum Permissible Lung Particle Burden (MPLPB)[77]

Cancer risk due to 5 rem/yr whole body exposure[78]	Assumed Risk in Particle		
	1/1000	1/2000	1/10,000
4.5×10^{-4}	0.45	0.9	4.5
10^{-3} (best estimate)	1.	2.	10.
2.3×10^{-3}	2.3	4.6	23.

largest MPLPB in Table V, 23 particles, represent a reduction of the existing MPLB and MPC_a by a factor of 10,000. It is recommended here that the best estimate of the effects of uniform exposure by the BEIR Committee be used together with a risk of cancer induction of 1/2000 per hot particle in determining the MPLPB for insoluble alpha-emitting radionuclides in hot particles. This is a somewhat arbitrary compromise and is not the most conservative value that could be recommended. Thus, the recommended MPLPB for occupational exposure from hot particles of alpha-

77/ The number of particles required to give a cancer risk equal to that from uniform radiation.

78/ Source: BEIR Report, Op. cit., p. 91. The MPLPB corresponding to a lung cancer risk of 3×10^{-5} due to 15 rem/yr lung dose [BEIR Report, Op. cit., p. 156] are 0.03, 0.06 and 0.3 for assumed particle risks of 1/1000, 1/2000 and 1/10,000 respectively.

emitting radionuclides in the deep respiratory zone is 2 particles. This corresponds to a MPLB of 0.14 pCi and represents a reduction of 115,000 in the existing MPLB. This implies that the DF for hot particles is 115,000. Moreover, it requires a reduction of the MPC_a for Pu-239 by 115,000 to a value of 3.5×10^{-16} uCi/ml unless it is determined that the plutonium is not in hot particles.

B. Exposure of the General Public

As indicated in Table II, the MPLB for non-occupational exposure (members of the public) is tenfold less than that for occupational exposure. Such an exposure limit for a hot particle would be 0.2 particles. Exposure at this level implies that on the average one out of five individuals would be contaminated by a particle and the other four would not. Obviously the exposed invididuals would be assuming a disproportionate fraction of the risk. In fact, since an individual is exposed to whole particles, any non-occupational exposure to hot particles would be an overexposure. This condition does not meet the recommendations and admonitions of the FRC, ICRP and NCRP.

> Under certain conditions, such as widespread radioactive contamination of the environment, the only data available may be related to average contamination or exposure levels. Under these circumstances, it is necessary to make assumptions concerning the relationship between

> average and maximum doses. The Federal Radiation Council suggests the use of the arbitrary assumption that the majority of individuals do not vary from the average by a factor greater than three. Thus, we recommend the use of 0.17 rem for yearly whole-body exposure of average population groups. (It is noted that this guide is also in essential agreement with current recommendations of the NCRP and the ICRP.) It is critical that this guide be applied with reason and judgment. Especially, it is noted that the use of the average figure, as a substitute for evidence concerning the dose to individuals, is permissible only when there is a probability of appreciable homogeneity concerning the distribution of the dose within the population included in the average.[79]

Strict adherence to these guidelines implies that the ambient air standard should be zero particles.[80] While a variety of suggestions could be proposed, we recommend a slight deviation from these guidelines and the acceptance of the disproportionate risk implicit in the 0.2 particle standard. This is a workable solution since best estimates of lung burdens can be fractional quantities. Thus, we recommend that the MPLPB for members of the public be 0.2 hot particles, and the average lung burden for members of the public be 0.07 hot particles, a factor of 3 less than the maximum.

79/ FRC Report No. 1, Op. cit., p. 27.

80/ Had we based the standard on a 1/10,000 risk per particle (See Table V), the MPLPB would have been one particle and this problem would not exist.

The MPLPB=0.2 particles implies that the existing MPCa for non-occupational exposure to Pu-239 should also be reduced by a factor of 115,000 to a value of 9×10^{-18} uCi/ml unless it is determined that the plutonium is not in hot particles.

C. Exposure from Accidental Releases

There are no direct statements by standard-setting organizations regarding an "acceptable" exposure associated with release of radioactivity in an accident.[91] For purposes of evaluating sites for nuclear reactors, establishing site boundaries, and preparing safety analysis reports, however, the AEC has adopted specific criteria. The reactor site boundary (surrounding the exclusion area) must meet the followin criteria (10 CFR 100.11(a)(1)):

> (1) An exclusion area of such size that an individual located at any point on its boundary for two hours immediately following onset of the postulated fission product release would not receive a total radiation dose to the whole body in excess of 25 rem[2] or a total radiation dose in excess of 300 rem[2] to the thyroid from iodine exposure.

91/. Fish, B.R., G.W. Keilhalte, W.S. Snyder, and S.D. Swisher, Chapter 7 of early draft version of B.R. Fish, et al, "Calculation of Doses Due to Accidental Released Plutonium from an LMFBR," ORNL-NSIC-74 (Nov. 1972), p. 113. This chapter was deleted from the final version at the direction of AEC-Division of Reactor Development and Technology because it was judged to be not directly applicable to the objective of the study, and the information base from which it was developed was already available in other documents. AEC-DRDT further stated that it was not removed because of the quality of the work.

[2]The whole body dose of 25 rem referred to above corresponds numerically to the once in a lifetime accidental or emergency dose for radiation workers which, according to NCRP recommendations may be disregarded in the determination of their radiation exposure status (see NBS Handbook 69 dated June 5, 1959). However, neither its use nor that of the 300 rem value for thyroid exposure as set forth in these site criteria guides are intended to imply that these numbers constitute acceptable limits for emergency doses to the public under accident conditions. Rather, this 25 rem whole body value and the 300 rem thyroid value have been set forth in these guides as reference values, which can be used in the evaluation of reactor sites with respect to potential reactor accidents of exceedingly low probability of occurrence, and low risk of public exposure to radiation.

Fish, et al, made the following comments regarding the applicability of these criteria to the case of plutonium release. These comments are also applicable to hot particle case.

> First, the wording of sections 100.11(a)(1) clearly limits the application to the irradiation of the whole body and the thyroid; no other organ or tissue is mentioned or implied. Furthermore, only fission products in general and iodine in particular are identified as reference substances. Finally, footnote (2) states unequivocally that the guides are not to be considered as acceptable limits for emergency doses to the public under accident conditions.[32]

Without addressing whether the guideline values, 25 rem to the whole body and 300 rem to the thyroid, should

32/ Ibid, p. 129.

be considered as acceptable limits, or whether design basis accidents that are currently evaluated under these criteria are "of exceedingly low probability of occurrence," we recommend that 10 CFR 100.11(a)(1) be modified as follows in order to establish a hot particle standard that is equivalent to the risk associated with 25 rem whole body irradiation:

> (1) An exclusion area of such size that an individual located at any point on its boundary for two hours immediately following onset of the postulated fission product <u>or other radionuclide</u> release would not receive a total radiation dose to the whole body in excess of 25 rem[2] or a total radiation dose in excess of 300 rem[2] to the thyroid from iodine exposure, <u>or receive a lung particle burden in excess of 10 hot particles.</u>[3]

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[2] (Unchanged from original text)

[3] A hot particle is a particle that contains sufficient activity to deliver at least 1000 rem/yr to the surrounding lung tissue. For isotopes having half-lives greater than one year, this would correspond to particles containing at least 0.07 pCi of alpha activity.

We also recommend that similar criteria be established limiting hot particle releases for nuclear facilities not now covered under 10 CFR 100.

D. <u>Surface Contamination</u>

Hot particles deposited on land surfaces can be resuspended into the air by any number of means, including wind, automobile traffic, human or animal movements. Following

an accident wherein surfaces are contaminated with hot particles, it is necessary to have a standard to apply to decontamination measures.

The number of particles that can be resuspended from surfaces has been the subject of a number of experiments. These experiments have usually resulted in the determination of a resuspension factor (RF). The RF is defined by:

$$RF\ (m^{-1}) = \frac{\text{concentration in air } (uCi/m^3)}{\text{concentration on surface } (uCi/m^2)}$$

R. L. Kathren has reviewed the data obtained on RF values.[83] He indicates that, "reported [RF] values for plutonium and its compounds range over 11 orders of magnitude." This 11 orders corresponds to values between 10^{-1} to 10^{-11} m^{-1}. Kathren indicates that, "an RF of 10^{-4} m^{-1}, although conservative is appropriate."[84] Langham indicates that a member of the Danish scientific team used an RF=10^{-3} m^{-1} during the Thule deliberation.[85] We would recommend that

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83/ Kathren, R.L., "Towards interim acceptable surface contamination levels for environmental PuO_2," BNWL-SA-1510, Battelle Northwest Laboratory, Richland, Washington, April 1968, pp. 3-4.

84/ Ibid, p. 4.

85/ Langham, Wright H., Op. cit., p. 5. The Thule Deliberations refer to the deliberations following the accidental crash of a B-52 bomber carrying nuclear weapons near Thule Air Force Base in Greenland. The high explosives in the weapons detonated and dispersed the plutonium.

the value selected by Kathren be used when the RF is unknown to determine the ambient ground contamination standard. Applying an RF=10^{-4} m^{-1} to the ambient MPC_a standard recommended in the previous section, we obtain a maximum permissible surface contamination (MPSC) level for hot particles of 9×10^{-8} uCi/m^2.[86] This is roughly 1 hot particle/m^2. In areas where an RF greater or less than 10^{-4} m^{-1} could be shown to apply, the MPSC could be altered appropriately.

E. As Low as Practicable Hearings

It is to be understood that the above recommendations do not represent endorsement on our part of the risk inherent in the existing radiation protection guidelines upon which these recommendations are based. Rather, we offer the admonition that the exposures should be kept as far below these guidelines as is practicable. Therefore, we further recommend that these guidelines be incorporated into the existing regulations without delay and that the appropriate agency or agencies convene hearings to determine for the regulations what constitutes as low as practicable limits for exposure to hot particles.

86/ This value is derived as follows: The recommended MPC_a for hot particles is 9×10^{-18} uCi/ml which corresponds to 9×10^{-12} uCi/m^3. The maximum ground contamination level, using RF=10^{-4} m^{-1}, is $9 \times 10^{-12}/10^{-4} = 9 \times 10^{-8}$ uCi/m^2.

VIII Summary of Recommendations

The following recommendations apply to alpha-emitting hot particles where a hot particle is defined as a particle that contains sufficient activity to deliver at least 1000 rem/yr to the surrounding lung tissue. For isotopes having half-lives greater than one year, this would correspond to particles containing at least 0.07 pCi of alpha activity.[87]

It is recommended that:

1. For occupational exposure

   MPLPB = 2 hot particles

   MPC_a for Pu-239 = 3.5×10^{-16} uCi/ml[88]

2. For non-occupational exposure

   MPLPB = 0.2 hot particles

   MPC_a for Pu-239 = 9×10^{-18} uCi/ml[89]

87/ These particulates would consist of compounds of Pu and the other actnides which fall into Class Y material in the ICRP Task Group Lung Model. These materials would be retained for years in the lung. See for example, ICRP Publication 19, Op. cit., p. 6. Since only particles in the size range of 5 u and below in diameter would be deposited in the deep respiratory tissue, this in effect sets an upper limit for the particle size of interest here. If the half-life is less than or close to 1 year the limit of 0.07 pCi can be adjusted upward through appropriate calculations.

88/ This MPC_a applies for particles containing 0.07 pCi of Pu-239. For particles containing more than 0.07 pCi the MPC_a could be increased proportionately. For particles containing less than 0.07 pCi the existing $MPC_a = 4 \times 10^{-11}$ pCi/ml would apply. The MPC_a for hot particles of other isotopes and mixtures of isotopes should be established on a similar basis with consideration given to the half-life of the isotope.

89/ Ibid.

3. For accidental releases exposure (10 CFR 100.11(a)(1))

   MPLPB (2 hours exposure) = 10 hot particles

4. For unrestricted areas

   MPSC = 1 hot particle/m^2 [90]

5. Hearings should be convened to determine as low as practicable regulations.

90/ This value is meant for guidance with respect to decontamination of an unrestricted area that has been contaminated with hot particles. In areas where an RF greater or less than 10^{-4} m^{-1} could be shown to apply, the MPSC could be altered appropriately.

APPENDIX A

Radiation Standards Setting Organizations and Their Roles

The organization which recommends basic radiation criteria and standards at the international level is the International Commission on Radiological Protection (ICRP). It was established in 1928 under the auspices of the Second International Congress of Radiology. During the early period and until 1950, the ICRP was concerned primarily with recommendations designed to provide protection to members of the medical profession in their diagnostic and therapeutic use of X-rays and gamma radiation from radium. However, since the advent of atomic energy, and radiation uses on a large scale, it has extended its efforts to include studies of radiation protection matters covering the whole gamut of radiation applications. It works together with its sister commission, the International Commission on Radiation Units Measurements (ICRU), and relies on the ICRU for background knowledge on radiation measurements.

The National Council on Radiation Protection and Measurements (NCRP) was organized in 1929, a year after the ICRP, as a combined effort of several radiation protection committees in the United States to consolidate their scattered efforts and to present a unified voice at meetings of the ICRP.[1] The ICRP and NCRP are private groups whose recommendations are purely advisory.

In 1934 the NCRP adopted the simple level of 0.1 roentgen per day, measured in air as the tolerance dose. In 1940, it recommended a permissible body burden of 0.1 microgram for ingested radium. The latter standard, still in effect today, corresponds to an average dose to the skeleton of about 30 rem/yr or a dose to the critical endosteal tissue out to a distance of 5-10 microns of about 10 rem/yr.

1/ Initially the NCRP was known as the Advisory Committee on X-rays and Radium Protection; in 1946 the name was changed to the National Committee on Radiation Protection and Measurements, and in 1964 it received a Federal charter and took its present name.

In 1949, the maximum permissible dose for radiation was lowered to 0.3 roentgen per week. It was lowered again in 1957 to 5 rem/yr as the permissible dose for radiation workers. This standard is still in effect.

The AEC has also played a significant role in setting radiation standards. However, the AEC's regulatory authority over materials was, and still is, limited by the Atomic Energy Act of 1954, as amended, to source, by-product, and special nuclear material. Before the Federal Radiation Council (FRC) was formed, the AEC, when setting radiation standards, generally followed closely the recommendations of the NCRP, which in turn paralleled the ICRP recommendations.

In 1959, after the advent of the atomic age had aroused public fears over fallout from nuclear weapons, the U. S. government, because of uncertainty of government influence over radiation protection standards, organized the FRC. It was authorized by Congress to "...advise the President with respect to radiation matters directly or indirectly affecting health, including guidance for all federal agencies in the formulation of radiation standards and in establishment and execution of programs in cooperation with the states..."[2] The final authority with respect to radiation standards rested not with the FRC but with the President. Such a subordinate agency as the AEC, for example, had to make its rules, e.g., those governing licensed reactors, compatible with the overall guides developed by the FRC.

Throughout the 1950's the ICRP and NCRP continued to revise and refine the basic recommendations concerning permissible radiation exposure standards. Standards were recommended for some non-occupational groups and for the whole population. Maximum permissible body burdens and maximum permissible concentrations of radionuclides in the air and in water were recommended as secondary standards. Most of these recommendations were incorporated by the FRC and the AEC.

In 1970 the FRC was abolished and its duties were transfer to the EPA. Since that time, the setting of population exposure standards has resided in EPA. Population standards,

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2/ FRC Report No. 1, Background Material for the Development of Radiation Protection Standards, Government Printing Office. Washington, D. C., May 13, 1960, p. 1.

in this case, mean exposure to persons "outside the fence" of an AEC (or AEC-licensed) facility. Criteria, required to meet these standards, for plant operation and design remained with the AEC. Hence, present responsibility for assessment of health effects resides in EPA, while the responsibility for developing technology to control emissions resides in AEC. The Office of Management and Budget (OMB) in a recent letter to EPA and AEC clarified the delegation of responsibility between these agencies for promulgating regulations to limit the radioactivity that may be emitted from facilities in the nuclear power industry. OMB stated:

> AEC should proceed with its plans for issuing uranium fuel cycle standards, taking into account the comments received from all sources, including EPA; that EPA should discontinue its preparations for issuing, now or in the future, any standards for types of facilities; and that EPA should continue, under its current authority, to have responsibility for setting standards for the total amount of radiation in the general environment from all facilities combined in the uranium fuel cycle, i.e., an ambient standard which would have to reflect AEC's findings as to the practicability of emission controls.[3]

There are other agencies and groups which are concerned with radiation standards and in some cases have regulatory authority. These include, but are not limited to, the Department of Health, Education and Welfare, Department of Labor, Bureau of Mines, the American National Standards Institute, and state agencies. The radiation standards of these organizations are not at issue here. For the most part they play a secondary role, or where applicable, follow the guidance of the NCRP, EPA and AEC.

3/ Memorandum for Administrator Train and Chairman Ray from Roy L. Ash, Dec. 7, 1973.

APPENDIX B

Statement Submitted to Attorneys for Mr. Edward Gleason

Re: Edward Gleason, et al vs. NUMEC

by: Arthur R. Tamplin

The following is my analysis of the origin of Mr. Edward Gleason's soft tissue sarcoma that ultimately resulted in his death and of the Consultation Report, submitted by Dr. Niel Wald, dated Jan. 29, 1973.

Mr. Gleason unloaded, rotated, and loaded a crate containing a leaking carbov of plutonium-239 (Pu-239) solution. This could not have occured without contaminating the palmar surface of his left hand, which was bare. The question is: did this Pu-239 contamination cause Mr. Gleason to develop a sarcoma? Since radiation induced cancers are identical with those that occur spontaneously, it is necessary to consider the relative chances that the cancer was spontaneous or Pu-239 induced.

The United States Vital Statistics, record a death rate for malignant neoplasms (other than melanoma) of the skin in the upper extremity of less than one per million per year. Since synovial sarcoma is a rare form that often metastasizes and hence has a poor prognosis, its occurrence rate is certainly less than the total skin cancer death rate of one per million per year. Thus it is highly unlikely that anyone who handled this crate would spontaneously develop this sarcoma on the contaminated hand (less than one chance in a million).

Now let us consider what the chances are of the development of cancer as a result of plutonium contamination of the skin. Experimental data from plutonium contaminated animals demonstrate that injection of 1 microgram of Pu-239 into the skin of rats promptly produced cancer in up to 5% of the animals (Exhibit 1). The particular tumors are fibrosarcomas.

Now the analysis done by LASL indicated that the Pu-239 concentration was about 160 micrograms per milliliter. This is reason to suspect, since the volume of liquid was reduced, the Pu was actually more concentrated in 1963. But setting that aside, one drop would be expected to contain between 8 and 16 micrograms of Pu-239. One-one hundredth of a milliliter (a very small amount of liquid) would have been sufficient to

produce sarcomas in animals. There is little reason to doubt that this small amount of liquid (0.01 milliliter) or even more found its way below the surface of Mr. Gleason's palm. In this event, his chance of developing cancer would be one in twenty. This is at least 50,000 times higher than his chances of developing the cancer spontaneously. In other words, the evidence is overwhelming in favor of the tumor resulting from Pu-239 contamination.

The above relative probability is based upon data from animals. It is quite possible that man is more sensitive than animals to cancer induction by Pu-239. In fact, the biological evidence strongly suggests that man is more sensitive. Exhibit 2 is a case report of a nodule removed from a man. This nodule contained only 0.08 ug of Pu-239. Commenting on the histological examination of the lesion, the authors states,"The autoradiographs showed precise confinement of α-tracks to the area of maximum damage and their penetration into the basal areas of the epidermis, where epithelial changes typical of ionizing radiation exposure were present. The cause and effect relationship of these findings, therefore, seemed obvious. Although the lesion was minute, the changes in it were severe. Their similarity to known precancerous epidermal cytologic changes, of course, raised the question of the ultimate fate of such a lesion should it be allowed to exist without surgical intervention..." In this case, less than 0.1 ug of Pu-239 produced precancerous changes in human tissue. The dose to the surrounding tissue was very intense. There is every reason to believe that a smaller quantity of Pu-239 would have produced similar changes.

When I consider the above human and animal data together with the relative probability of 50,000, I can come to no other conclusion than that this sarcoma was a direct result of the contamination of Mr. Gleason's left palm by Pu-239.

Turning now to Dr. Wald's Consultation Report, it can be stated that he has presented no evidence to disprove the claim that this sarcoma was caused by Pu-239 contamination. I shall discuss Dr. Wald's report in the order that it was written.

According to the Division of Inspection Report submitted by Anson M. Bartlett on April 11, 1963, pages 29-30, the January 19 examination was conducted not on Mr. Gleason, but on his home, clothing and automobile. The single urine and feces

samples collected subsequent to January 20 gave negative results. The only thing that this demonstrates is that no detectable level of Pu-239 was found. Even following the injection of large volumes of Pu-239 solution into the skin and muscle of animals, the Pu-239 is slowly absorbed and appreciable fractions, up to 70%, remain at the site of injection. Moreover, of the quantity absorbed only a small fraction appears in the urine or feces (see page 3, Exhibit 3 and Exhibit 4). In Mr. Gleason's case we are concerned with only a very small volume of solution and hence we should not be surprised if we obtain negative results in an individual urine or feces sample. (See also Exhibit 5)

The physical examination performed by Dr. Roy E. Albert on January 23, 1963, has no relevance. One would expect no overt signs of radiation injury at this early date from the small quantity of Pu-239 which is at issue here. We are concerned here with the long term effects, not the acute effects.

The medical history of Mr. Gleason as recorded by Dr. Wald appears to be accurate, however, he omitted the conclusions of the Pathology Report of the Hospital for Special Surgery wherein the unanimous opinion of the pathologists was stated to be that this lesion was a synovial sarcoma.

The negative findings in the feces and urine in April of 1970 are of no more relevance than the similar findings in the January 1963 samples. The whole body counter has a detection limit of 0.3 u Ci of Pu-239. At issue here are quantities below 0.06 u Ci and, hence, well below the detectable limit.

There are three reasons for setting aside the negative findings in the initial tissue removed from Mr. Gleason. First, since the pathologist report indicated "no evidence of atypical or malignant changes," it is quite possible that this mass was unrelated to the sarcoma. Recall here that the histology of the small nodule in Exhibit 2 showed severe changes that resembled precancerous changes. Third, the site of contamination was not necessarily removed with the mass or it could have trimmed from the mass prior to production of the paraffin blocks and slides. Consider here that the nodule in Exhibit 2 was only 1/10 of a millimeter in diameter. Since Mr. Gleason eventually developed an infiltrating soft tissue sarcoma, and this original tissue removed showed no atypical change, there is no basis for

assuming that the origin of the sarcoma was included in this tissue mass.

The negative results on the clavicle specimen are also equivocal. The issue here is a small quantity of Pu-239 that remained localized in the palmar area of the left hand. This bone specimen indicates only that the amount of systemically absorbed Pu-239 was too small to be detected in this bone specimen.

None of these clinical findings are able to set aside the strong possibility that Mr. Gleason's sarcoma was a direct result of the plutonium contamination. The most likely course of events is that a small quantity of the Pu-239 solution (less the 0.01 milliliter) was deposited in the tissue below Mr. Gleason's palm. This may have occured through a small cut or via a sliver. The body then reacted to this material as a foreign body, and encapsulated it. Eventually, a lesion similar to that discussed in Exhibit 2 developed. This nodule progressed beyond the precancerous stage to become an infiltrating soft tissue sarcoma. The chances are some 50,000 times greater that the sarcoma developed in this fashion than that it occured spontaneously.

I think that it is important to point out that all of the information relevant to this case was available in 1963. Had Mr. Gleason been informed of the potential cancer risk subsequent to the incident, he could have informed his physicians. As a result they would probably have treated him more cautiously and the tradedy could have been substantially mitigated.

Exhibits

1 Lisco, Herman, et al, Radiology, Vol. 49, No. 3, Sept. 1947, pp. 361-363.

2 Lushbaugh, C.C., et al, Arch. of Dermatology, Vol. 86, Oct. 1962, pp. 461-464.

3 Vanderbeck, J. W., HW-66172, Hanford Laboratories Operation, July 25, 1960.

4 Matsuoka, Mr., et al, Health Physics, Vol. 22, June 1972, pp. 713-722.

5 Lisco, Herman and Walter E. Kesiekeski, American J. of Pathology, Vol. 29, No. 1, Jan. - Feb. 1953, pp. 305-321.

GLOSSARY

Absorbed Dose:	The absorbed dose of any ionizing radiation is the energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the rad. One rad is 100 ergs/gram.
AEC:	Atomic Energy Commission.
Ci:	Abbreviation for curie.
Curie:	The quantity of a radioactive nuclide disintegrating at the rate of 3.7×10^{10} atoms per second.
D:	Abbreviation for Absorbed Dose.
DE:	Abbreviation for Dose Equivalent.
DF:	Abbreviation for Dose Distribution Factor.
Dose Distribution Factor:	A modifying factor used in calculating dose equivalent which accounts for non-uniform distribution of radiation.
Dose Equivalent:	The product of absorbed dose D, quality factor (QF), dose distribution factor (DF), and other necessary modifying factors (The dose equivalent is numerically equal to the absorbed dose in rads multiplied by the appropriate modifying factors). The unit of dose equivalent is the 'rem.'
EPA:	Environmental Protection Agency.
FRC:	Federal Radiation Council. The FRC has been abolished, and its functions taken over by EPA.
g:	Abbreviation for gram.
Half-life:	Time required for a radioactive substance to lose 50 percent of its activity by radioactive decay. Each radionuclide has a unique half-life.

ICRP:	International Commission on Radiological Protection.
m:	Abbreviation for meter.
micron:	One-millionth of a meter.
ml:	Milliliter = 0.001 liters.
MPC_a:	Maximum permissible concentration (of a radionuclide) in air. The average concentration above background of a specific radionuclide to which an individual can be exposed without exceeding the guidelines.
MPC_w:	Maximum permissible concentration (of a radionuclide) in water. (See definition above.)
MPLB:	Maximum permissible lung burden.
MPLD:	Maximum permissible lung dose.
NCRP:	National Council on Radiation Protection and Measurements.
nCi:	Abbreviation for nanocurie, which is one-billionth of a curie, or 10^{-9} curie.
pCi:	Abbreviation for picocurie, which is one-millionth of a microcurie, or 10^{-12} curies.
QF:	Abbreviation for Quality Factor, which is assigned on the basis of a number of considerations. A quality factor is a modifying factor used in calculation of dose equivalent which accounts for differences in producing biological effects among various forms of radiation (e.g., alpha, and X-radiation).
Rad:	Unit of absorbed dose (D), which is 100 ergs/gram. The rad is a measure of the energy imparted to matter by ionizing radiation per unit mass of irradiated material at the place of interest.
Radionuclide:	A nuclide of an element that is radioactive.

Rem:	Unit of dose equivalent. When the appropriate modifying factors are used to calculate dose equivalent one rem is the quantity of any type of ionizing radiation which when absorbed in man produces an effect equivalent to the absorbtion of one rad of X- or gamma-radiation at the place of interest.
Roentgen:	The quantity of X- or gamma-radiation such that the associated corpuscular emission per 0.001293 grams of air produces, in air ions carrying one electrostatic unit of electricity of either sign. For the purposes here, the roentgen is roughly equivalent to the rad.
Specific activity:	Total radioactivity of a given material (isotope, element, or compound) per gram of the material -- curies/gram.
u:	Abbreviation for micron, which is one-millionth of a meter.
uCi:	Abbreviation for microcurie, which is one-millionth of a curie.
ug:	Abbreviation for microgram, which is one-millionth of a gram.

Exhibit 8
(Affidavit of Thomas Cochran

Natural Resources Defense Council, Inc.

917 15TH STREET, N.W.
WASHINGTON, D.C. 20005
202 737-5000

Western Office
654 HAMILTON AVENUE
PALO ALTO, CALIF. 94301
415 327-1080

New York Office
15 WEST 44TH STREET
NEW YORK, N.Y. 10036
212 869-0150

RADIATION STANDARDS FOR OCCUPATIONAL WHOLE BODY EXPOSURE

September 25, 1975

Thomas B. Cochran
Arthur R. Tamplin

I. Introduction

This Report is written in support of a petition by the Natural Resources Defense Council (NRDC) to the Nuclear Regulatory Commission (NRC) requesting a reduction in the maximum permissible occupational whole body radiation exposure. The present standards for occupational exposure are based on still current recommendations of the National Council on Radiation Protection and Measurements (NCRP) and the International Commission on Radiological Protection (ICRP) adopted in 1958 and 1959, respectively. The NRDC petition and this Report were prompted by consideration of the latest information on the biological effects of radiation. This information indicates that the biological effects are greater than was assumed in 1958 and 1959 when the existing standards were recommended.

The latest data have been reviewed by a committee of the ICRP and by the BEIR Committee of the U.S. National Academy of Sciences (NAS).[1,2/] The BEIR Committee was principally concerned with the exposure of the general population

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1/ ICRP Publication 14, Radiosensitivity and Spatial Distribution of Dose, Reports Prepared by Two Task Groups of Committee 1 of the International Commission on Radiological Protection, Pergamon Press, Oxford, 1969.

2/ NAS-BEIR Report, National Academy of Sciences, The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. Report of the Advisory Committee on the Biological Effects of Ionizing Radiation (BEIR Report), Washington, D. C., November, 1972.

and, in this regard, indicated that the existing exposure standard was unnecessarily high.[3/] The ICRP Committee, while declining to make any recommendations, presented a calculation to demonstrate how the new data on the biological effects of radiation could be used to lower the existing whole body exposure standards by a factor of ten.[4/] The reduction requested in this Report corresponds closely to this factor of ten in the ICRP Committee analysis.

In January, 1971, while not recommending an overall change, the NCRP recommended that the occupational exposure of pregnant women be limited to one tenth the present exposure limit.[5/] The reduction requested in this Report would also fulfill this NCRP recommendation.

In the following section of this Report, we shall present an analysis of the risk of somatic and genetic injury at the current maximum permissible exposure limit and compare this risk with those encountered in other occupations. This analysis will serve to indicate that the exposure limit is too high. In Section III, we shall present our requested modifi-

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3/ Ibid., p.2.

4/ ICRP - Publication 14, op. cit., Appendix IV.

5/ NCRP Report No.39, Basic Radiation Protection Criteria, National Council on Radiation Protection and Measurement, Washington, D. C., 1971, pp.92-93.

cations of the exposure limits and an analysis of the reduced risk associated with these new limits. In the final section, we shall indicate how these requested reductions relate to the recommendation and suggestions of the ICRP, NCRP, and the NAS Committee on the Genetic Effects of Atomic Radiation.

II. Radiation Induced Risk at the Existing Occupational Whole Body Dose Limit

The latest and most comprehensive review of the biological effects of radiation on man is the NAS's 1972 BEIR Report. The BEIR Committee reviewed both the somatic and genetic risk associated with exposure to low levels of ionizing radiation. We shall discuss first the somatic and then the genetic effects.

A. Somatic Effects

Table 1 summarizes the BEIR Report estimate of the excess annual cancer and leukemia deaths per million people assuming whole body exposure to 5 rem/year (the current occupational standard).[6/]

6/ NAS-BEIR Report, op. cit., p.170.

Table 1

Calculation of the excess annual number of cancer deaths for individuals exposed from 20 to 65 years of age

Exposure Conditions	ABSOLUTE RISK MODEL		RELATIVE RISK MODEL	
	Excess Deaths Due to:		Excess Deaths Due to	
	Leukemia	All Other Cancer	Leukemia	All Other Cancer
10^6 people: 5 rem/yr.	81	(a) 300	181	(a) 601
		(b) 336		(b) 746

(a) 30 year plateau
(b) lifetime plateau

(Plateau region = interval following latest period during which the risk remains estimated).

Source: NAS-BEIR Report, p.170.

The risk estimates in Table 1 incorporate the assumption that the million people have an age and sex distribution identical to that of individuals 20 years and older in the U.S. population (1967 statistics). These figures do not represent a 20 year old individual's chance of eventually dying of radiation induced cancer(assuming exposure at the 5 rem/year limit). This chance can, however, be calculated by using the overall mortality rate for individuals over 20 years of age. This death rate in 1973 was 1500 deaths per 100,000 population. Hence, if this rate is divided into the frequency of cancer deaths given in Table 1, the chance of a worker, exposed at

5 rem/year from age 20, dying from radiation induced cancer is calculated to be from 1 in 16 to 1 in 40. We believe this level of risk is excessive.

A means of illustrating the excessiveness of the radiation risk to workers exposed at the maximum permissible dose rate is to compare this risk with the fatality rate associated with other occupations. This comparison is given in Table 2.

Table 2

Fatality Rate by Occupation

Occupation	Yearly Fatality Rate
Radiation Worker - exposed at the current maximum permissible dose rate [a] (from Table 1)	1 in 1000 to 1 in 2600
United States (1973) [b]	
All Industries	1 in 6000
Mining and Quarrying	1 in 900
England and Wales [c]	
All Occupations (males)	1 in 5000 to 1 in 10,000

a Due to cancer induced by occupational whole body exposure (at 5 rem/yr.) only.

b National Safety Council, Accident Facts, 1974 edition, 1974, p.23.

c Decennial Supplement for England and Wales, Registrar General's 1959-1963. Occupational Mortality. Part III, Vol.2. HMSO London (1958) as referenced in ICRP Publication 8, p.60.

In making the above comparison, we believe that the upper limit of the radiation induced risk should be used. The BEIR Committee cautioned that its estimate may be too high or too low.[7/] One reason for suggesting that it is too low is that the linear hypothesis is used as a basis for extrapolating from high dose-high dose rate data to low dose-low rate situations. Recent evidence suggests that the linear hypothesis may underestimate the effect of low dose-low dose rate irradiation. The latest information has been summarized by Dr. Karl Z. Morgan who concluded:

> Frequently in the literature it is stated that the linear hypothesis is a very conservative assumption. During the past few years, however, many studies have indicated that this probably is not true in general and that at low doses and dose rates somatic damage per rad (and especially that from α-irradiation) probably is usually greater than would be assumed on the linear hypothesis.[8/]

Thus, there is little justification for relying on the lower estimate of the radiation induced risk and prudent health practice would indicate that the upper limit should be used. When this is done, inspection of Table 2 indicates that

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7/ Ibid., p.90.

8/ Morgan, Karl Z., Suggested Reduction of Permissible Exposure to Plutonium and Other Transuranium Elements, Journal of American Industrial Hygiene, August, 1975.

the estimated radiation exposure risk corresponds to that associated with mining and quarrying, a risk that is acknowledged to be far too high. The radiation exposure risk exceeds the average occupational risk by six fold. In this report, we are proposing a reduction in the risk of radiation induced cancer at the maximum allowable whole body exposure by a factor of 6 together with the request that the exposures be kept as far below the proposed new limits as is practicable. In making the above comparison and proposing this reduction, we do not mean to imply that all radiation workers are exposed to the maximum level of the current standards. We only mean to imply that the current exposure standard is an inappropriate guideline against which to apply the as-low-as-practicable rule.

It could be argued that it is not appropriate to set the maximum exposure limit at a level that corresponds to the average occupational fatality rate because the limit applies in practice only to the most exposed individuals. But it is precisely these most exposed workers about whom we must be concerned, and we see no reason why the nuclear industry should subject its workers to an above-average risk, certainly not when that risk is comparable to that in the mining and quarrying industry. Moreover, we believe this approach is appropriate because radiation workers are also

subject to normal non-radiological occupational hazards, and hence the average risk in the industry will still be above the average for all occupations even with the adoption of our proposed changes. Thus, it would even be reasonable to argue that the risk of radiation induced cancer should be further reduced. Consequently, we see no justification for a higher risk, particularly since the above estimate of the cancer and leukemia risk does not include the additional risk associated with radiation induced genetic damage.

B. Genetic Effects

The BEIR Report estimated that the total incidence of all identified serious genetic diseases due to 5 rem per generation to a population of 1 million would be between 300 to 7,500 per year at equilibrium.[9/] In addition, the BEIR Report estimated that this same exposure at equilibrium would eventually lead to an increase of between 0.5% and 5% in the ill health of the population.

The approach for estimating the genetically significant dose (GSD) is to use that exposure accumulated by age 30. The existing exposure limit would allow a worker exposed at 5 rem/year from age 18 to accumulate a dose of 60 rem by age 30. Hence, based on the BEIR Report estimates

9/ NAS-BEIR Report, op. cit., p.51.

above, if one million workers were exposed from age 18 at the current 5 rem/year limit, between 3,600 and 90,000 identified serious genetic disease and a significant increase of ill-health would show up in the progeny of these workers, assuming an average of 2 children per worker. The increased incidence in ill-health would be equivalent to between 6% and 60% of the incidence in a population of 1 million, e.g., the first generation. This genetic risk can be compared with the somatic risk to the workers themselves. Thus, an individual worker exposed at 5 rem/year from 18 to 65 years of age would incur an additional risk of fatal cancer between 1 in 16 and 1 in 40, and an additional risk of between 1 in 10 and 1 in 300 that one of his progeny will incur a serious genetic defect. In terms of the raw numbers, the somatic and genetic risks overlap quantitatively. This simple comparison of the somatic and genetic risks associated with a single worker's lifetime exposure assumes equal weighting of the hurt or suffering associated with the somatic and genetic damage.

The genetic risk is different in that the effect is suffered not by the workers but by their offspring and by future generations. As a consequence, one can argue that the genetic risk should be given more weight because it is not assumed by the worker but involuntarily by their offspring and by future generations. Nevertheless, the biological data

indicates that the risk of genetic damage is comparable to the leukemia and cancer risk and, therefore, is also too high regardless of any special weighting that it deserves.

Again, we strongly suggest that the upper limit estimate of the genetic risk be used in this comparison. The BEIR Committee suggested caution in the use of these estimates and began its Discussion section by stating:

> A major concern of the Subcommittee is the possible existence of a class of radiation-induced genetic damage that has been left out of the estimates. By relying so heavily on experimental data in the mouse we may have overlooked important effects that are not readily detected in mice, or the mouse may not be a proper laboratory model for the study of man.10/

As if to reemphasize this, the Committee concluded this section by stating:

> We remind all who may use our estimates as a basis for policy decisions that these estimates are an attempt to take into account only known tangible effects of radiation, and that there may well be intangible effects in addition whose cumulative impact may be appreciable, although not novel.11/

There is reason to suggest that the BEIR Committee should have implied an even more cautious approach to their

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10/ Ibid., p.57.

11/ Ibid.

estimates. In the experiments of Dr. William L. Russell at the Oak Ridge National Laboratory, it was observed that the induced mutation frequency at low dose rates was about 1/3 that observed at high dose rates. The factor of 1/3 was used by the BEIR Committee. However, Dr. Mary F. Lyon, et al., have analyzed the Russell data along with additional data from experiments at low dose rates.[12/] Their analysis shows that as the dose rate drops below some 0.01 r./min., the induced mutation frequency begins to increase. They conclude:

> In future estimates of the genetic hazards of environmental radiation, therefore, it would be prudent to increase this last figure to a value above that seen in mice at 0.01 r./min., for which the maximum likelihood estimate given by the data considered here is 10×10^{-8}.[13/]

The value adopted in the BEIR Report was 2.5×10^{-8} mutations per locus per rem or a factor of 4 lower.

Thus, once again there is little justification for relying on the lower limit estimate and prudent health practices indicate that the upper limit estimate should be employed in establishing radiation protection standards. The upper estimate of the genetic risk (1/10) is comparable

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12/ Lyon, Mary F., D. G. Papworth and Rita J. S. Phillips, "Dose-rate and Mutation Frequency after Irradiation of Mouse Spermatogonia," Nature New Biology, Vol.238, July 26, 1972, pp.101-104.

13/ Ibid., p.104.

to the upper limit estimate of the somatic risk of 1/16, and this genetic risk, like the somatic risk, is excessive. When somatic risk and genetic risk are combined (on an equal weight basis), the combination suggests that the existing exposure standard is at least 10 times too high. In this Report we are proposing a factor of 10 reduction in the genetic risk and a factor of 6 reduction in the somatic risk with the additional request as stated previously that the exposures be kept as far below the proposed new limits as is practicable.

III. Proposed Action

The NRC regulations governing permissible occupational exposure levels to radiation are embodied in the Code of Federal Regulations at 10 CFR 20.101. At present these 10 CFR 20.101 regulations limit the whole body dose to 1-1/4 rem per calendar quarter (5 rem/year), except a licensee may permit an individual to receive up to 3 rem/quarter whole body dose as long as the dose to the whole body when added to the accumulated occupational dose to the whole body, shall not exceed 5 (N-18) rem where "N" equals the individual's age in years.

The objective of the proposed action is to reduce the genetic risk associated with radiation exposure at the current occupational exposure level by a factor of 10 and reduce the somatic risk by a factor of 6. To meet the objective relative to the genetic risk, it is proposed that the current regulations be amended as follows:

1. For individuals under the age of M, where M is not less than 45, the whole body radiation exposure limit shall not exceed 0.5 rem in any calendar year and 0.3 rem in any calendar quarter.

To meet the objective relative to the somatic risk, it is proposed, in addition to the above, that:

2. For individuals equal to or greater than M years of age, a licensee may permit an individual to receive up to 3 rem/quarter whole body dose as long as the dose to the whole body shall not exceed 0.5 (M-18) + X(N-M) rem, where N equals the individual's age in years, and X is calculated to reduce the cumulative somatic risk by a factor of 6 below the cumulative somatic risk associated with exposure at 5 rem/year from age 18. It is proposed that the value of X be calculated using the relative risk model as described more fully in the BEIR Report.[14/]

14/ NAS-BEIR Report, op. cit., p.171. It is proposed that the plateau region, i.e., the interval following the latest period during which the risk remains elevated, for cancers other than leukemia be taken as the lifetime of the individual. The relative risk model with the lifetime plateau assumption gives the upper limit estimate of the risk.

It is further requested that:

3. The NRC institute hearings to determine the as-low-as-practicable extent to which the exposure can be maintained below the proposed new regulations.

The effect of these proposed changes will be to reduce the genetic risk from occupational radiation exposure at the limiting value by a factor of 10 to about 1 in 100 and reduce the risk associated with the induction of fatal cancers to about the same level. Again, it should be recognized that the ordinary occupational risks and the risk associated with other than whole body irradiation must be added to these whole body radiation risks. Nevertheless, the whole body radiation risk is still quite large and therefore, it is essential to maintain the actual exposures as far below these proposed new limits as is practicable.

IV. Additional Justification

The BEIR Committee of the NAS reviewed the more recent data on the biological effects of radiation. They were concerned mainly with the exposure of the general public. In this respect the Committee concluded that the current Radiation Protection Guide was unnecessarily high,[15/] a conclusion

15/ NAS-BEIR Report, op. cit., p.2.

which in our judgment should be equally applicable to occupational exposure standards.

A Committee of the ICRP in 1969 reviewed the same material that formed the basis for the BEIR Report and indicated that the somatic effects of radiation were 5 to 6 times worse than was estimated previously. The ICRP made no recommendations relative to the exposure standards; rather, it stated:

> The choice between no change and a partial and tentative revision will depend, so it seems to us, not only on a scientific assessment of evidence, but also on practical considerations, such as the general desirability of stability in the recommendations over a period of years. The balance between practical considerations and incomplete scientific evidence is a matter for judgement outside the Task Group's frame of reference. Nevertheless, it seemed useful to give an example in Appendix IV of how our conclusions about relative tissue sensitivity to cancer induction by radiation might be used as a basis for setting dose limits for individual tissues and organs and perhaps for the whole body.16/

16/ ICRP - Publication 14, op. cit., p.33.

In Appendix IV, the Committee analysis indicated that, when the somatic and genetic effects are combined, the whole body exposure limit should be reduced by a factor of 10. Thus, the changes proposed here are in accord with this ICRP Committee analysis.

Both the ICRP and NCRP have recommended that special consideration should be given to pregnant and fertile females. In fact, in January, 1971, the NCRP recommended:

> During the entire gestation period the maximum permissible dose equivalent to the fetus from occupational exposure of the expectant mother should not exceed 0.5 rem.17/

The changes proposed in this Report would in effect accomodate this recommendation of the NCRP.

The AEC, while acknowledging the greater sensitivity of the fetus, did not amend the dose limiting sections of the Commission's regulations (10 CFR 20).. So far as pregnant or fertile women are concerned, the AEC noted difficulties in sex discrimination, right-to-work and right-to-privacy as reasons for not changing the limits.18/ The change proposed here, since it applies to both men and women below the age of 45 eliminates these difficulties.

In further justification for not changing the dose limits for pregnant and fertile women, the AEC stated in its

17/ NCRP Report No.39, op. cit., p.92.

18/ Federal Register, Vol.40, No.2, Friday, January 3, 1975, pp.799-800.

Federal Register notice:

> Reduction of the dose limits for all radiation workers in order to avoid discrimination against women does not appear practicable. Such a reduction in the dose limits would cost the nuclear industry large sums of money in the application of design and engineering changes and, in some cases, the employment of additional workers in order to accomplish essential work within the reduced individual dose limits. The latter could even result in a net increase in total man-rems of exposure. Reduction of the dose limit for all workers would aggravate an existing shortage of available manpower in certain key occupations, e.g., radiographers, welders, and pipefitters, that may involve relatively high radiation exposures.19/

While we disagree with the philosophy for setting radiation standards implicit in the reference to the large sums of money it would cost the industry, we note that the changes proposed here do not cause all workers to be limited to 0.5 rem/year. Furthermore, the 3 rem/quarter limit is retained for older workers. Hence, the proposed changes should not place a large burden on the industry. For example, the AEC stated in the Federal Register notice:

19/ Ibid., p.799.

> Data on results of personnel monitoring reported to the Commission pursuant to §20.407, 10 CFR Part 20, for calendar year 1973, indicate that 67,862 individuals were monitored, 29,169 received measurable exposures averaging 0.73 rem for the year, and 3,425 individuals (11.8 percent of those receiving measurable exposures) had estimated exposures in excess of 2 rems.20/

If M in the proposed regulations were set at 55 years and X at 3 rem/year, the necessary reduction in cancer fatalities would be achieved. If the work force has the same distribution as the population, then some 16% would be over 55 years old and the above quotation indicates that only 12% are presently exposed above 2 rem/year. If M were set at 45 and X at 1.5 rem/year, the cancer reduction would be achieved, and some 37% of the work force could be expected to be above 45. In this latter case, by limiting the exposure of workers over 45 to 0.5 rem for 2 years, these same workers (12% of the work force in any one year) could receive 3.5 rem in the third year. Moreover, since there is good reason to believe that the present exposures are not as low as practicable, the industry should not have great difficulty in conforming to these proposed regulations.

20/ Ibid., p.799.

In conclusion, we note that our proposal for limiting the exposure of younger workers while allowing a higher exposure to older workers is not new. It is, in fact, similar to a 1956 recommendation of the NAS Committee on the Genetic Effects of Atomic Radiation in the BEAR Report:

> F) That every effort be made to assign to tasks involving higher radiation exposures individuals who, for age or other reasons, are unlikely thereafter to have additional offspring. Again it is recognized that such a procedure will introduce complications and difficulties, but this committee is convinced that society should begin to modify its procedures to meet inevitable new conditions.21/

We submit that this recommendation is even more appropriate today. Its justification on genetic grounds is undiminished while, at the same time, the cancer inducing potential of radiation is now recognized to be much greater and the high radiosensitivity of the developing embryo and fetus is also now recognized. In the presence of an expanding nuclear industry, the time to implement this recommendation has arrived.

21/ NAS BEAR Report, National Academy of Sciences, The Biological Effects of Atomic Radiation, Summary Reports, Report of the Committee on Genetic Effects of Atomic Radiation, Washington, D. C., 1956, p.29.

Exhibit C
(Affidavit of Thomas B. Cochran)

Table 3-1

Estimated numbers of deaths per year in the U.S. population attributable to continual exposure at a rate of 0.1 rem per year, based on mortality from leukemia and from all other malignancies combined.

Irradiation During Period	ABSOLUTE RISK MODEL[a] Excess Deaths Due to:		RELATIVE RISK MODEL[a] Excess Deaths Due to:	
	Leukemia	All other Cancer	Leukemia	All other Cancer
In Utero	75	75	56	56
0-9 years	164	(a) 73 (b) 122	93	(a) 715 (b) 5,869
10 + years	277	(a) 1,062 (b) 1,288	589	(a) 1,665 (b) 2,415
Subtotal	516	(a) 1,210 (b) 1,485	738	(a) 2,436 (b) 8,340
TOTAL	(a) 1,726 = 0.6% incr. (b) 2,001 = 0.6% incr.		(a) 3,174 - 1.0% incr. (b) 9,078 - 2.9% incr.	

[a] The figures shown are based on the following assumptions:
(1) 1967 U.S. vital statistics can be used for age specific death rates from leukemia and all other cancer, and for total U.S. population
(2) Values for the duration (a or b) of the latent period (the length of time after irradiation befor any excess of cancer deaths occur), duration of risk ("plateau region"), and magnitude of average increase in annual mortality for each group are as shown in Table 3-2.

The Effects on Populations of Exposure to Low Levels of Ionizing Radiation

REPORT OF THE ADVISORY COMMITTEE ON THE
BIOLOGICAL EFFECTS OF IONIZING RADIATIONS

DIVISION OF MEDICAL SCIENCES

NATIONAL ACADEMY OF SCIENCES • NATIONAL RESEARCH COUNCIL
WASHINGTON, D.C. 20006

NOVEMBER 1972

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Preface

This report of the National Academy of Sciences - National Research Council Advisory Committee on the Biological Effects of Ionizing Radiations (BEIR Committee) deals with the scientific basis for the establishment of radiation protection standards and encompasses a review and re-evaluation of existing scientific knowledge concerning radiation exposure of human populations. The present basis of radiation protection is essentially the establishment of single upper limits for individual and population average exposures with the understanding that any biological risks should be offset by commensurate benefits and that these risks should be kept as low as practicable. It has become apparent that these current concepts of radiation protection may not be adequate in a future age of large-scale use of nuclear energy. Inadequacy arises because there is the potential for radiation exposure of entire populations and such exposure may be an alternative to other types of hazards as, for example, the substitution of radioactive contaminants from nuclear power plants for the combustion products from fossil fuel plants. Thus there is a need somehow to make comparisons of biological risks and benefits not only for radiation but for the alternative options. In this report it has not been possible for us to deal with critical interacting factors such as socio-economics, energy needs, and comparative effects of other toxicological agents; nor have we attempted to explore in detail technological matters such as sustained engineering performance of power reactors, large-scale waste disposal, or the problem of catastrophic accidents. Nevertheless, we have felt it urgent to call attention to these issues because ultimately, decisions will have to be made involving them, and public acceptance gained on the basis of providing society with the services that it needs at a minimum risk to health and the environment.

The BEIR Committee has endeavored to ensure that no sources of relevant knowledge or expertise were overlooked in its study and toward this end has established and maintained liaison with appropriate national and international organizations, and has solicited the opinions and counsel of individual scientists. The Committee wishes to express appreciation to those who served on the Subcommittees, and to the many organizations and individuals who have cooperated by providing viewpoints and information. The members of the Committee and Subcommittees acted as individuals, not as representatives of their organizations.

Chapters IV through VII represent the reports of the respective Subcommittees but may have been modified by the Committee. All members of the Committee approve the substance of the report if not necessarily each specific detail.

Members of the Advisory Committee on the Biological Effects of Ionizing Radiations

Cyril L. Comar, *Chairman*, Department of Physical Biology, New York State Veterinary College, Cornell University, Ithaca, New York

Seymour Abrahamson, Zoology Research Building, University of Wisconsin, 1117 West Johnson Street, Madison, Wisconsin

Victor P. Bond, Brookhaven National Laboratory, Upton, Long Island, New York

J. Martin Brown, Department of Radiology, Stanford Medical Center, Stanford, California

George W. Casarett, Department of Radiation Biology and Biophysics, University of Rochester Medical Center, 260 Crittenden Boulevard, Rochester, New York

James F. Crow, Department of Medical Genetics, Genetics Building, University of Wisconsin, Madison, Wisconsin

John H. Dingle, Department of Community Health, Wearn Research Building, University Hospitals, Cleveland, Ohio

Robert A. Good, University of Minnesota Hospitals, Mayo Box 494, Minneapolis, Minnesota

Samuel P. Hicks, Department of Pathology, University of Michigan Medical Center, Ann Arbor, Michigan

George G. Hutchison, Department of Epidemiology, Harvard School of Public Health, 665 Huntington Avenue, Boston, Massachusetts

Cyrus Levinthal, Department of Biology, Room #754 Schermerhorn Extension, Columbia University, 119th Street & Amsterdam Avenue, New York, New York

Edward B. Lewis, Biology Division, California Institute of Technology, Pasadena, California

Robert W. Miller, Epidemiology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

*Carl V. Moore, Department of Internal Medicine, Washington University School of Medicine, 660 So. Euclid Avenue, St. Louis, Missouri

Edward P. Radford, Department of Environmental Medicine, Johns Hopkins University, School of Hygiene and Public Health, 615 North Wolfe Street, Baltimore, Maryland

Joseph E. Rall, National Institutes of Health, Bldg. 10, Rm. 9N222, Bethesda, Maryland

William L. Russell, Biology Division, Oak Ridge National Laboratory, P. O. Box Y Oak Ridge, Tennessee

Eugene L. Saenger, University of Cincinnati, College of Medicine, Cincinnati, Ohio

Edward L. Tatum, The Rockefeller University, New York, New York

Arthur C. Upton, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, New York

Consultants

Wilfrid J. Dixon, Department of Biomathematics, School of Medicine, University of California, Los Angeles, California

Brian MacMahon, Department of Epidemiology, Harvard School of Public Health, 665 Huntington Avenue, Boston, Massachusetts

Shields Warren, Cancer Research Institute, New England Deaconess Hospital, 194 Pilgrim Road, Boston, Massachusetts

*Deceased

Members of the Subcommittee on Environmental Effects

Cyril L. Comar, *Chairman*, Department of Physical Biology, New York State Veterinary College, Cornell University, Ithaca, New York

Delbert S. Barth, Bureau of Criteria and Standards, National Air Pollution Control Administration, 111 W. Chapel Hill Street, Durham, North Carolina

James H. Carpenter, Macauley Hall, The Johns Hopkins University, Charles and 34th Streets, Baltimore, Maryland

John C. Evans, Box 3275, Duke University Medical Center, Durham, North Carolina

R. John Garner, Office of Radiation Programs, Environmental Protection Agency, Washington, D. C.

Ronald G. Menzel, Water Quality Management Laboratory, U. S. Department of Agriculture, Durant, Oklahoma

Dade W. Moeller, Harvard University School of Public Health, 665 Huntington Avenue, Boston, Massachusetts

Roy E. Nakatani, Fisheries Research Institute, University of Washington, 260 Fisheries Center, Seattle, Washington

Robert B. Platt, Department of Biology, Emory University, Atlanta, Georgia

Edward P. Radford, Department of Environmental Medicine, Johns Hopkins University, School of Hygiene and Public Health, 615 North Wolfe Street, Baltimore, Maryland

H. B. Tukey, Jr., Department of Floriculture and Ornamental Horticulture, Plant Sciences Building, Cornell University, Ithaca, New York

Members of the Subcommittee on Genetic Effects

James F. Crow, *Chairman*, Department of Medical Genetics, Genetics Building, University of Wisconsin, Madison, Wisconsin

Seymour Abrahamson, Zoology Research Building, University of Wisconsin, 1117 West Johnson Street, Madison, Wisconsin

H. John Evans, MRC Clinical and Population Cytogenetics Unit, Western General Hospital, Crewe Road, Edinburgh, EH4, 2XU, Scotland

James V. Neel, Department of Human Genetics, University of Michigan, Ann Arbor, Michigan

Howard B. Newcombe, Biology and Health Physics Division, Atomic Energy of Canada Limited, Chalk River, Ontario, Canada

Dean R. Parker, Department of Biology, University of California, Riverside, California

Klaus Patau, Department of Medical Genetics, University of Wisconsin, Madison, Wisconsin

William L. Russell, Biology Division, Oak Ridge National Laboratory, P. O. Box Y, Oak Ridge, Tennessee

H. Eldon Sutton, Department of Zoology, University of Texas at Austin, Austin, Texas

Sheldon Wolff, Laboratory of Radiobiology School of Medicine, University of California, San Francisco, California

Members of the Subcommittee on Effects on Growth and Development

Samuel P. Hicks, *Chairman*, Department of Pathology, University of Michigan Medical Center, Ann Arbor, Michigan

Jacob I. Fabrikant, *Liaison Representative*, The University of Connecticut Health Center, Hartford, Connecticut

Ernest Furchtgott, Department of Psychology, University of South Carolina, Columbia, South Carolina

Robert W. Miller, Epidemiology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

John J. Mulvihill, Epidemiology Branch, 410 WISCON Bldg., National Cancer Institute, Bethesda, Maryland

Melvin R. Sikov, Battelle-Northwest, Battelle Memorial Institute, P. O. Box 999, Richland, Washington

Members of the Subcommittee on Somatic Effects

Arthur C. Upton, *Chairman*, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, New York

Gilbert W. Beebe, National Academy of Sciences, Medical Follow-up Agency, 2101 Constitution Avenue, N. W., Washington, D. C.

Victor P. Bond, Brookhaven National Laboratory, Upton, Long Island, New York

J. Martin Brown, Department of Radiology, Stanford Medical Center, Stanford California

George W. Casarett, Department of Radiation Biology and Biophysics, University of Rochester Medical Center, 260 Crittenden Boulevard, Rochester, New York

Louis H. Hempelmann, Strong Memorial Hospital, Rochester, New York

Edward B. Lewis, Division of Biology, California Institute of Technology, Pasadena, California

Robert W. Miller, Epidemiology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

Carl V. Moore, Department of Internal Medicine, Washington University School of Medicine, 660 So. Euclid Avenue, St. Louis, Missouri

Edward P. Radford, Department of Environmental Medicine, Johns Hopkins University, School of Hygiene and Public Health, Baltimore, Maryland

Joseph E. Rall, National Institute of Arthritis and Metabolic Diseases, National Institutes of Health, Bethesda, Maryland

Eugene L. Saenger, University of Cincinnati, College of Medicine, Cincinnati, Ohio

William B. Seaman, Department of Radiology, Columbia University, 622 West 168th St., New York, New York

John B. Storer, Biology Division, Bldg. 9207, Oak Ridge National Laboratory, Oak Ridge, Tennessee

Shields Warren, Cancer Research Institute, New England Deaconess Hospital, 194 Pilgrim Road, Boston, Massachusetts

Consultants

Wilfrid J. Dixon, Department of Biomathematics, School of Medicine, University of California, Los Angeles, California

Jacob I. Fabrikant, The University of Connecticut Health Center, Hartford, Connecticut

George B. Hutchison, Department of Epidemiology, Harvard School of Public Health, 663 Huntington Avenue, Boston, Massachusetts

Seymour Jablon, Medical Follow-up Agency, National Academy of Sciences, 2101 Constitution Avenue, Washington, D. C.

Brian MacMahon, Department of Epidemiology, Harvard School of Public Health, 665 Huntington Avenue, Boston, Massachusetts

Members of the Advisory Committee on the Biological Effects of Ionizing Radiations Ad Hoc Committee

Cyrus Levinthal, *Chairman*, Department of Biology, Room #754 Schermerhorn Extension, Columbia University, 119th Street & Amsterdam Avenue, New York, New York

J. Martin Brown, Department of Radiology, Stanford Medical Center, Stanford, California

Cyril L. Comar, Department of Physical Biology, New York State Veterinary College, Cornell University, Ithaca, New York

James F. Crow, Department of Medical Genetics, Genetics Building, University of Wisconsin, Madison, Wisconsin

Samuel P. Hicks, Department of Pathology, University of Michigan Medical Center, Ann Arbor, Michigan

Joseph E. Rall, National Institutes of Health, Bldg. 10, Rm. 9N222, Bethesda, Maryland

Arthur C. Upton, Health Sciences Center, State University of New York at Stony Brook, Stony Brook, New York

Division of Medical Sciences - National Academy of Sciences - National Research Council

STAFF OFFICERS: Albert W. Hilberg
David A. McConnaughey

AFFIDAVIT

TRUST TERRITORY OF THE PACIFIC ISLANDS)
) SS
MARSHALL ISLANDS DISTRICT)

Jormea Leviticus, being duly sworn, states: He is a member of the People of Bikini and one of the named plaintiffs in this action.

He was born on July 7, 1936 at Bikini Atoll, Marshall Islands, and resided at Bikini until the removal of his People from Bikini on March 7, 1946, when he was nine years of age.

Thereafter he lived with his people at Rongerik Atoll for approximately two years where he experienced arthritis at age 10-11 from eating toxic fish.

Thereafter he lived at Kwajalein Atoll for several months and then together with the People of Bikini, was moved to Kili Island where he lived until he came to Majuro Atoll, the District Center of the Marshall Islands to attend Intermediate School in 1953. After completion of Intermediate School, he was trained as a Health Aide at the hospital at Majuro. His training as a Health Aide was completed in 1958.

Affiant speaks and reads both the English and Marshallese languages.

From 1958 to 1959 affiant was employed as a Health Aide at Kili.

In 1959 affiant moved to Ujelang Atoll as a Health Aide for the People of Enewetak who were removed to Ujelang. He remained at Ujelang eight years. Then, after service again at Majuro he moved to Bikini Atoll to serve as a Health Aide in late 1972. He remained as a Health Aide at Bikini until July, 1973, when he returned to Majuro.

Affiant is presently employed at the hospital at Majuro.

For approximately 30 months affiant, together with his wife and seven children lived in the interior of Bikini Island at the place marked in the aerial photograph attached hereto with a red "x" in Janai Weto, where external gamma radiation is between 65 and 75 microroentgens per hour.

Affiant and his family consumed well water and ate locally available foods as well as imported foods.

Affiant and his family consumed bananas, potatoes, papayas, pandanus, coconut, and fish, all from Bikini Island.

Dr. Conard, when he made studies at Bikini in 1974, took urine samples from affiant and his wife, but not from any of their children.

Aside from the urine samples, no other tests were administered to affiant or his family.

Affiant now knows that he and his family, like others who have lived or are living at Bikini, has been placed at risk by exposure to radiation and that to understand the extent of exposure and, if necessary treat results of that exposure, extensive examination will be necessary.

Affiant, as a trained and experienced Health Aide, knows that he and his family require immediate and extensive medical examination and possible treatment.

Dr. Conard knew, when urine samples were taken from affiant and his wife, that they were living in the interior of Bikini Island.

By that time (late 1974) Dr. Conard reasonably must have known of the probability of extremely high radiation risk to affiant and his family.

Nevertheless neither Dr. Conard nor any other ERDA or AEC physician furnished affiant or his family with appropriate examination or care.

Affiant has no confidence in Dr. Conard or other ERDA-AEC health care personnel. Affiant believes he and his family and others who have been placed at risk on Bikini Island should

have a right to select their own physicians for the examination and possible treatment they now require.

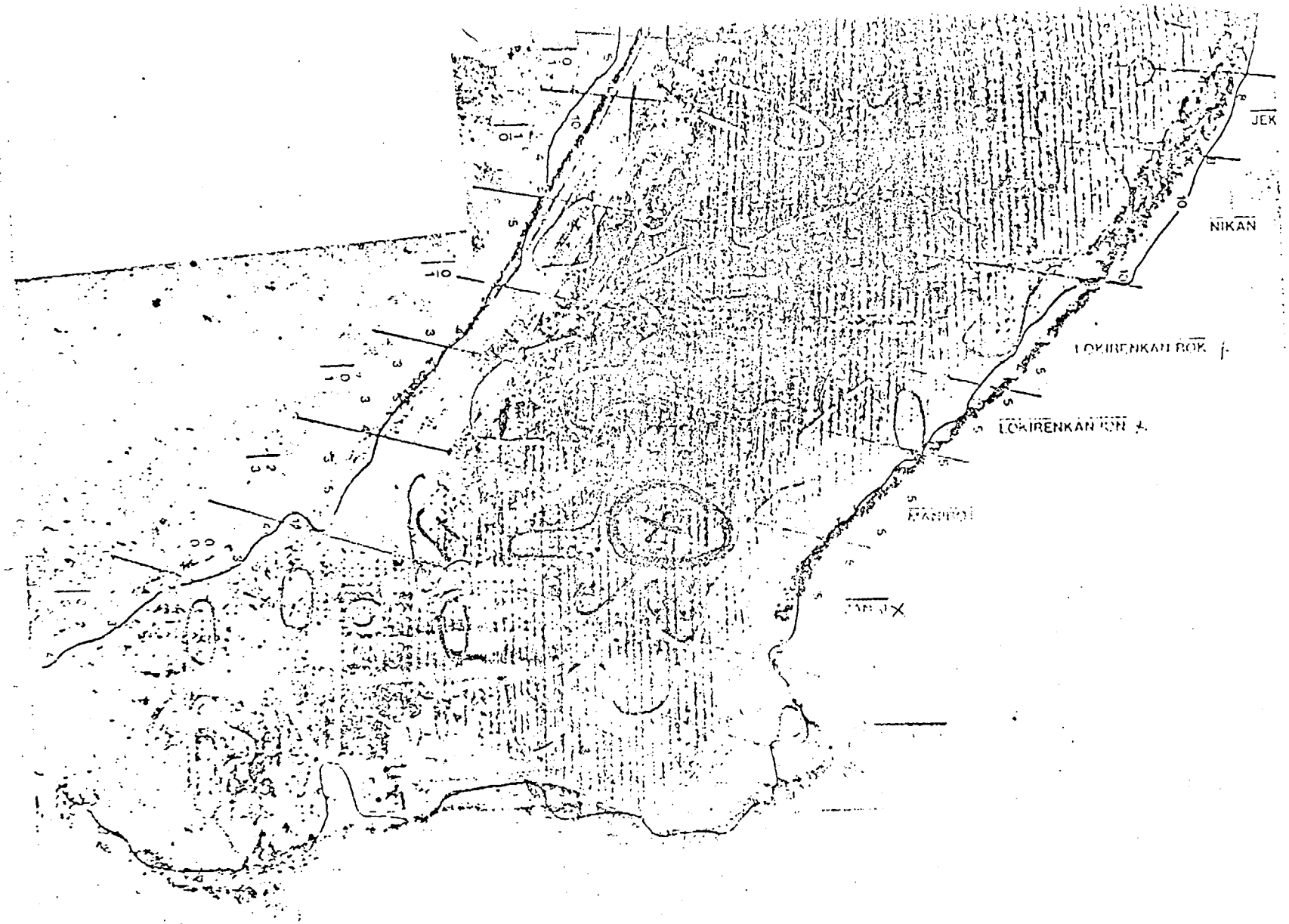
By reason of ERDA's having allowed affiant and his family to have been placed at risk to high radiation (external gamma dosage approximately 20 times greater than in American cities and 35 times greater than elsewhere in the Western Pacific Islands) affiant believes ERDA-AEC should bear the entire expense of examination of himself, his family and others similarly situated, as well as the expense of treatment if remedial treatment should be indicated or possible.

Affiant is willing to have physicians he chooses release results of examination of himself and his family to Dr. Conard and ERDA-AEC but affiant is not willing to entrust his or his family's health care to Dr. Conard or ERDA-AEC as affiant has a reasonable basis to believe, for the reasons set forth above, that Dr. Conard and other physicians employed by ERDA-AEC are concerned with ERDA-AEC and not with the Bikini People.

Jormea Leviticus

Subscribed and sworn to
before me this 7th day of October, 1975.

Notary Public
LILLIAN M. ANDREW, Notary Public
TRUST TERRITORY OF THE PACIFIC ISLANDS
MARSHALL ISLANDS DISTRICT
my commission expires on the
_____ day of _____, 19__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