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PROCEEDINGS OF PUBLIC HEARINGS: PLUTONIUM AND THE OTHER TRANSURANIUM ELEMENTS

VOLUME 3
ADDITIONAL MATERIALS RECEIVED

U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Radiation Programs

PROCEEDINGS OF PUBLIC HEARINGS: PLUTONIUM AND THE OTHER TRANSURANIUM ELEMENTS

VOLUME 3
ADDITIONAL MATERIALS RECEIVED

U. S. ENVIRONMENTAL PROTECTION AGENCY
Office of Radiation Programs
Criteria and Standards Division
Washington, D.C. 20460

CONTENTS

page

Environmental Protection Agency
Hearing Announcement 1
Federal Register Notices 2

National Audubon Society, letter 5

Bruce W. von Zellen, letter 6

Catherine Quigg, letter and article 7

Atlantic County Citizens Council on
Environmental, letter 13

Colorado Department of Health, letter 14

Linda Meader, letter 17

Freihofer et. al, letters 18

Natural Resources Defense Council,
letter and articles 31

Biophysical Society, letter and article 232

J. W. Healy, letter and articles 255

Elmer Glueck, letter 383

National Radiological Protection Board,
letter and article 384

Chester R. Richmond, letter 439

Annette Cottrell, letter 445

L. R. Anspaugh, letter and article 446

Frederick Forscher, letter 467

William Lipton, letters and article 468

General Electric, letter 490

[FRL 284-2]

PLUTONIUM AND THE TRANSURANIUM ELEMENTS

Public Hearing

In the September 23, 1974 issue of the FEDERAL REGISTER, 38 FR 24098, the Environmental Protection Agency published a notice of intent to evaluate the environmental impact of plutonium and the other transuranium elements and to consider whether new guidelines or standards under the authorities of this Agency are needed to assure adequate protection of the general ambient environment and of the public health from potential contamination of the environment by radionuclides of the transuranium elements. This notice requested information from interested parties relevant to the development of standards and guidelines.

In accordance with the above request, the Office of Radiation Programs of the Environmental Protection Agency will hold public hearings beginning on December 10, 1974, at 9:30 a.m. in the GSA Auditorium, 18th & F Streets, NW., Washington, D.C. Further hearings will be held in other locations if deemed advisable. Announcement of such hearings will be made in the FEDERAL REGISTER. The Office of Radiation Programs will consider the information derived from these hearings, along with other available information, in determining the adequacy of current guidelines for plutonium and the other transuranium elements and in developing any new standards deemed necessary. Specific information is desired in the following categories:

(1) General: to include considerations of general concern, including the public and social implications of plutonium utilization, and the factors involved in balancing of costs vs. benefits.

(2) Dosimetry, Health, and Environmental Effects: to include consideration of available data concerning conversion of ambient concentrations to exposure data, on the health effects of plutonium exposure in humans and animals, as well as of other possible adverse environmental effects. Discussions of research needs are also desired.

(3) Environmental Levels and Pathways: to include consideration of available monitoring data, of the precision, accuracy and completeness of available data, of theoretical models developed to predict transport through the ecosystem, and of experimental verification of such models.

(4) Applications Using Plutonium: to include consideration of current and projected uses of plutonium and other transuranium elements, the estimated quantities in each such application, and the magnitude of possible releases to the environment.

(5) Control and Cleanup Technology: to include consideration of currently available or near-term projected engineered safeguard devices and installations to minimize and restrict releases to the environment and of technology available to restore contaminated areas, but not to include waste storage facilities.

A detailed agenda and schedule will be made available on request from the Office of Radiation Programs on December 6, 1974.

The entire proceeding will be open to the public, and attendance by interested persons is encouraged. Persons wishing to make a statement at the hearing will be afforded an opportunity to do so. In addition, written comments as requested in the FEDERAL REGISTER notice of September 23, 1974, (39 FR 34098) will continue to be received. The following procedures and requirements shall apply to the hearing:

(a) The hearings will be conducted informally. Technical rules of evidence will not apply. Discovery and cross-examination of participants will not be permitted.

(b) A Hearing Panel, to be appointed by the Deputy Assistant Administrator for Radiation Programs and consisting of a Chairman and three or more technical experts in the field of radiation protection, will conduct the hearings.

(c) The Chairman of the hearing panel is empowered to conduct the meeting in a manner that in his judgment will facilitate the orderly conduct of business, to schedule presentations by participants, and to exclude material which is irrelevant, extraneous, or repetitious.

(d) Persons wishing to present an oral statement shall give written notice to the Director, Criteria and Standards Division (AW-560), Office of Radiation Programs, U.S. Environmental Protection Agency, Washington, D.C. 20460, no later than November 23, 1974, in order to be placed on the agenda. Such notice shall include the name, address and affiliation (if any) of the participant, the amount of time required, and a reasonably detailed summary of the statement to be presented at the hearing. The time allotment for such oral statements shall be at the discretion of the Chairman, but shall not ordinarily exceed 20 minutes.

(e) Persons wishing to submit written statements regarding the agenda items may do so either in advance or during the hearings. If practicable, at least 20 copies should be provided. Such persons may also request an opportunity to present an oral statement in accordance with paragraph (d) above.

(f) Oral presentations may be presented by panel discussion of technical experts chosen to present a particular viewpoint if notice is given in accordance with paragraph (d) above. The time allotment for such panel discussions shall be at the discretion of the Chairman, but shall not ordinarily exceed 60 minutes.

(g) Requests at the time of the hearings for the opportunity to make oral statements, with no previous notice, shall be ruled on by the Chairman of the Hearing Panel, who is empowered to apportion the time available, but not ordinarily to exceed 5 minutes.

(h) Questions may be propounded only by members of the Hearing Panel or its consultants. At the discretion of the Chairman, a procedure may be made available for submission of pertinent questions from other persons to participants.

(i) Seating for the public will be available on first-come, first-served basis.

(j) The use of still, motion picture, and television cameras, the physical installation and presence of which will not interfere with the conduct of the meeting, will be permitted both before and after the hearing and during any recess. The use of such equipment will not, however, be allowed while the hearing is in session.

(k) A transcript of the hearing will be made and a copy of the transcript, together with copies of all documents presented at the hearing, will constitute the record of the hearing. A copy of the transcript of the hearing will be available for public inspection and copying within 30 days after conclusion of the hearings at the U.S. Environmental Protection Agency Freedom of Information Office, 2nd Floor, West Tower, 401 M Street, SW., Washington, D.C. 20460.

Dated: October 17, 1974.

ROGER STRELOW,
Assistant Administrator for Air and Waste Management.

[FR Doc.74-24733 Filed 10-23-74;8:45 am]

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October 31, 1974

Mr. W. D. Rowe, Ph.D.
Deputy Asst. Administrator, Radiation Programs
United States Environmental Protection Agency
Washington, D. C. 20460

RE: Plutonium and the transuranium elements

Dear Mr. Rowe,

I would like to make one brief statement for the upcoming hearing.

Could not the great majority of problems related to the storing, handling, transporting, etc. of the referenced elements be eliminated by reducing the volume (mass) of these elements which reactors are now generating as waste?

Specifically, I am referring to a transmutation process whereby waste plutonium is bombarded creating lighter and shorter lived isotopes. Nuclear scientists insist this is technically possible though a paucity of funding appears as the main obstacle.

Sincerely,

Robert K. Turner LP

Robert K. Turner
RKT:lbp

cc: Mr. John Quarles, Acting Administrator of EPA

008

FOR: Mr. Gordon Burley, Ph.D.
Chief, Environmental Standards Bureau
U.S. Environmental Protection Agency
Washington, D.C. 20460

FROM: Catherine T. Quigg (Mrs.)
Vice president
Pollution & Environmental Problems, Inc.
54 S. Brockway
Palatine, Illinois 60067

SUBJECT: PLUTONIUM

As a representative of Pollution & Environmental Problems, Inc., with members in the north and northwest suburbs of Chicago, I urge your consideration of the awesome dangers to the general population from the proliferation of plutonium in our environment.

For compelling evidence against plutonium, one need only look to the Atomic Energy Commission's own laboratories.

"Plutonium is toxic beyond human experience," according to Donald Geesaman of the AEC's Lawrence Radiation Laboratory at Livermore, California. He warned it was "demonstrably carcinogenic to animals in microgram quantities" stating that an injection of a millionth of a gram has caused cancer in mice and dogs. Moreover, he said, "our transition to plutonium as a major energy source will inextricably involve our society with the large-scale commercial production of a substance that is a suitable nuclear explosive." Gessaman estimated that by the year 2000 annual production of plutonium would exceed 100 tons. Can such quantities be protected from internal subversion? Gessaman thinks not. He has stated that

becomes widespread, the possibility must be faced, of awful accidents, either accidental or deliberate, that will cause wide regions of our earth be becomes forever uninhabitable."

We should heed Dr. Watson's ominous words now - and ban the fast breeder - before it bans us from this earth.

###

ATLANTIC
COUNTY
CITIZENS
COUNCIL ON
ENVIRONMENT 2 Old Turnpike ∘ Pleasantville, N. J. 08232 ∘ Phone (609) 646-6604

November 6, 1974

013

Mr. W. D. Rowe, Ph.D.
Deputy Assistant Administrator
Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Mr. Rowe:

The Atlantic County Citizens Council on Environment hereby registers a request that Atlantic City, New Jersey be considered one of the cities at which the Environmental Protection Agency hearings on the impact of transuranium elements be held. The nation's first offshore floating nuclear power plant is planned to be located 2.8 miles off the Atlantic County shore near here. A county referendum this week resulted in an overwhelming vote against a nuclear power plant being located off our shores.

The ACCCE is a non profit non funded citizens organization of several hundred members including associate organizations membership. The primary interest is the environment of Atlantic county. This Council, along with the City of Brigantine, N.J. and the Atlantic County Board of Chosen Freeholders, is an official intervenor in Atomic Energy Commission hearings on the offshore nuclear power plant held here in Atlantic City and in Washington, D. C. The AEC hearings held here are heavily attended by concerned citizens and officials from this area.

One of our major concerns is the deleterious effects of transuranium elements or materials. Of particular interest, are the effects of plutonium releases, especially into the ocean off our shores. Since this is the first location of an ocean sited floating nuclear power plant, we are concerned with its impact upon our total environment if such transuranium materials should be deposited or released into the sea as a result of abnormal operation or from a major accident wherein nuclear core meltdown could occur into the ocean waters. The consequential Poseidon syndrome occurring in the marine environment is feared to be much more disastrous than the land based nuclear counterpart major accident referred to as the China syndrome.

The ACCCE wishes to present testimony at the upcoming EPA hearings on adverse environmental impact of plutonium and other transuranium materials. Response will be made to the Director, Criteria and Standards. Meanwhile, we urge the EPA to designate Atlantic City as one of the hearing locations.

Sincerely yours,

John Williamson

John Williamson
President, ACCCE

c/c
Commissioner, N.J. DEP
Secretary, USAEC

"WE ARE WHAT WE ALLOW OUR ENVIRONMENT TO BECOME"

014

COLORADO DEPARTMENT OF HEALTH

4210 EAST 11TH AVENUE · DENVER, COLORADO 80220 · PHONE 388-6111

Edward C. Dreyfus, M.D., M.P.H. Executive Director

November 8, 1974

Dr. W. D. Rowe
Deputy Assistant Administrator
for Radiation Programs
United States Environmental
Protection Agency
Washington, D.C. 20460

Dear Mr. Rowe:

Reference is made to your announcement of October 25, 1974, concerning hearings on the environmental impact of releases of plutonium and other transuranium elements.

Because of the location of the USAEC's Rocky Flat Plant, upwind from a large metropolitan area, we have considerable interest in the proposed hearings. There being no federal or international standards for plutonium in soil, the Colorado Department of Health held an open hearing in Denver on February 14, 1973. On March 21, 1973, the Colorado Board of Health established a standard for plutonium in soil, a copy of which is enclosed for your information.

It is presumed that most organizations and individuals who participated in the 1973 hearing would desire to present their opinions in a hearing that might result in the establishment of federal standards. Therefore, we recommend that you schedule a hearing in Denver, preferably early in January 1975.

A spokesman for the Colorado Department of Health will present the Department's opinion on this subject. We request that 30 minutes be allowed for our presentation.

Sincerely,

Albert J. Hazle, Director
Occupational & Radiological
Health Division

AJH/lc
Enclosure: (1) Plutonium Standard

The State of Colorado
DEPARTMENT OF LAW
OFFICE OF THE ATTORNEY GENERAL
104 STATE CAPITOL
DENVER, COLORADO 80203

JOHN P. MOORE
TTORNEY GENERAL

JOHN E. BUSH
DEPUTY ATTORNEY GENERAL

April 6, 1973

Dr. Roy L. Cleere
Director
Department of Health
4210 East 11th Avenue
Denver, Colorado 80220

RE: Permissible Levels of Radioactive Material in Uncontrolled Areas (Plutonium)

Dear Doctor Cleere:

Pursuant to the request of Mr. Alfred L Capra, we have examined the Amendment to the above-captioned rules and regulations which were adopted by the State Board of Health on March 21, 1973, to become effective May 1, 1973.

Pursuant to Section 3-16-2, C.R.S 1963 (1967 and 1969 Perm. Cum. Supp.), you are hereby advised that it is the opinion of this office that said rules and regulations are constitutional and within the authority granted to the State Board of Health by the legislature.

You are further advised that your office must file two copies of this letter of approval along with two copies of said rules and regulations with the Secretary of State's office.

Very truly yours,

JOHN P. MOORE
Attorney General

JPM:WT:ms

AMENDMENT TO THE

State of Colorado Rules and Regulations Pertaining to Radiation Control

Subpart RH 4.21 is added:

RH 4.21 Permissible Levels of Radioactive Material in Uncontrolled Areas

4.21.1 Plutonium. Contamination of the soil in excess of 2.0 disintegrations per minute of Plutonium per gram of dry soil or square centimeter of surface area (0.01 microcurie plutonium per square meter) presents a sufficient hazard to the public health to require the utilization of special techniques of construction upon property so contaminated. Evaluation of proposed control techniques shall be available from the Department of Health upon request.

Adopted: Colorado State Board of Health
March 21, 1973

Effective: May 1, 1973

93 Main street
Byfield, Ma. 01922
November 11, 1974

Dear Sir(s)

I'm writing on behalf of your article on the EPA setting Plutonium standards. My opinion is I don't think that any plutonium what so ever should be let out of the Nuclear power plants, because when the plutonium gets into the soil it is then carried along by water which is later evaporated into the atmosphere and when it is inhaled it causes instant death. It has these effects on animals as well as people and does not stop killing until 24,000 years after it is released. A small amount of plutonium about the size of a folf-ball could kill a city the size of Pittsburg. Everytime this little amount of plutonium is released it adds up and we'll all be wiped out before we ever got a chance to use this type of energy. I feel this very strongly. Thank-you.

Sincerely,

Linda Reader

FREIHOFER, COOK, HECHT, OOSTERHOUSE & DEBOER, P.C.
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STUART F. CHENEY
MARK H. VERWYS

OF COUNSEL:
FRED N. SEARL
EDWARD C. McCOBB

November 13, 1974

Director, Criteria and Standards
Division (AW-560)
Office of Radiation Programs
Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

This letter is written on behalf of West Michigan Environmental Action Council, Inc., 822 Cherry Street, S.E., Grand Rapids, Michigan 49506 (WMEAC), and responds to a letter WMEAC has received dated October 25, 1974, together with two attached Federal Register notices entitled Plutonium and the Transuranium Elements, over the signature of W. D. Rowe, Ph.D., Deputy Assistant Administrator for Radiation Programs (AW-558).

Pursuant to the requests set forth in the letter, this is to notify you that WMEAC desires to submit a written statement at the hearings which you contemplate for plutonium and the transuranium elements. WMEAC is unable to make an oral presentation due to limited funds, and, therefore, no time for an oral presentation is requested at this time.

WMEAC intends to file its written statement prior to the public hearing scheduled for Washington, D.C., on December 10, 1974. WMEAC would appreciate being notified of the schedule of hearings and the agenda for other cities.

WMEAC's statement will set forth its viewpoints as they pertain to the problems of plutonium and the environment, and the statement will be limited both in length and in scope. WMEAC, in conjunction with other organizations, may submit additional technical comments.

Very truly yours,

FREIHOFER, COOK, HECHT,
OOSTERHOUSE & DE BOER, P.C.

Peter W. Steketee

PWS/jmc

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OF COUNSEL:
FRED N. SEARL
EDWARD C. McCOBB

December 12, 1974

Dr. William A. Mills, Director
Criteria and Standards Division (AW-560)
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

This letter relates to EPA's hearings on Plutonium and the Transuranium Elements. Please refer to my letter to you dated November 13, 1974, and to your response to me of November 26, 1974. This letter is submitted on behalf of West Michigan Environmental Action Council, Inc., 822 Cherry Street, S.E., Grand Rapids, Michigan 49506 ("WMEAC"), and is intended to be WMEAC's written statement for submission at the hearings. It is my understanding that the hearing record will be kept open for several weeks after December 10, 1974, and, consequently, I would appreciate it if you would include this statement of WMEAC in the hearing record.

As I indicated in my letter of November 13, 1974, this statement does not address any technical points relating to plutonium and the transuranium elements. Instead, for our technical comments we rely on the submissions of the Natural Resources Defense Council, Inc.

Also, EPA's October 17, 1974, public notice requested information on "contamination of the environment by radionuclides of the transuranium elements", apparently suggesting that EPA would not be concerned in these hearings with the full range of the hazards raised potentially by plutonium. In other words, EPA appears to us to be primarily concerned in these hearings with the public health problems (that is, with the radiation protection problems, including the "hot particles" question) posed by plutonium rather than with the nuclear safety, nuclear proliferation, safeguards and civil liberties issues, even though all of these issues are interrelated. On this assumption, we will limit our comments to the public health issues. However, for a truly excellent review of the broader implications of plutonium, we refer you to an article by Speth, Tamplin and Cochran entitled, Plutonium Recycle:

The Fateful Step, November, 1974, Bulletin of the Atomic Scientists, a copy of which is enclosed for your convenience.

Turning, then, to the public health issues posed by plutonium, WMEAC will try to outline as clearly and briefly as it can those considerations which it considers to be of primary importance:

Although it may be possible still for more light to be thrown on the subject at this time, WMEAC believes that the exchange of technical views which has taken place recently between NRDC scientists and the AEC on the "hot particle" issue has brought the disputed technical questions, as well as the resulting political, legal and moral questions, into clear focus.

Specifically, we are referring to the NRDC report of Tamplin and Cochran, 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; to the AEC's response by Blair, Richmond and Wachholz, A Radiological Assessment of the Spatial Distribution of Radiation Dose from Inhaled Plutonium, WASH 1320, September, 1974; to the NRDC Comments on WASH 1327, Draft Generic Environmental Impact Statement on Mixed Oxide Fuels (GESMO), Re: The Hot Particle Discussion in Volume 3, Chapter IV, Section J.1. and Section J, Appendix D, Pages IV J-7, IV J(D)-1 to 41, September, 1974, Tamplin and Cochran; and to the NRDC rebuttal to WASH-1320, The Hot Particle Issue: A Critique of WASH 1320 as it Relates to the Hot Particle Hypothesis, November, 1974, Tamplin and Cochran.

Tamplin and Cochran state the hot particle thesis as follows in their November, 1974, rebuttal of WASH 1320 (p. 5):

> "If a particle deposited in the deep respiratory tissue is of such activity as to expose the surrounding lung tissue to a dose of at least 1000 rem in 1 year, this particle represents a unique carcinogenic risk. The biological data suggest that such a particle may have a cancer risk equal to 1/2000."

WASH 1320 discusses a number of animal experiments which, it claims, rebut the hot particle thesis. However, we believe that Tamplin and Cochran have shown that WASH 1320 is not successful in disproving the hot particle thesis. Their November, 1974, rebuttal of WASH 1320 succinctly states the public health consequences of WASH 1320's failure (pp. 28-29):

> ". . . Thus, these experiments do not set aside the hot particle hypothesis. Rather, they suggest additional experiments involving longer lived animals to determine whether this histological change progresses

THE bulletin OF THE ATOMIC SCIENTISTS

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Plutonium Recycle: The Fateful Step

Impending move to reprocess fuel would escalate the risks of nuclear power

> *I fear that when the history of this century is written, that the greatest debacle of our nation will be seen not to be our tragic involvement in Southeast Asia but our creation of vast armadas of plutonium, whose safe containment will represent a major precondition for human survival, not for a few decades or hundreds of years, but for thousands of years more than human civilization has so far existed.*
>
> *James D. Watson*
> *Nobel Laureate, Medicine*

J. GUSTAVE SPETH, ARTHUR R. TAMPLIN and THOMAS B. COCHRAN

The Atomic Energy Commission, if unchecked, is about to sow the seeds of a national crisis. The Commission now proposes to authorize the nuclear power industry to proceed to use plutonium as fuel in commercial nuclear reactors around the country. The result of a decision approving this commercial use of plutonium will be the creation of a large civilian plutonium industry and a dramatic escalation in the risks posed by nuclear power.

This decision to launch what the AEC calls the plutonium economy is the conclusion of the AEC's recently released draft environmental impact statement for *plutonium recycle*: the recycling of plutonium as fuel in the present generation of light water reactors [1, 2]. The final version of the impact statement, which is expected to confirm the decision to authorize plutonium recycle, is due in a few months.

Plutonium is virtually unknown in nature; the entire present-day inventory is man-made, produced in nuclear reactors. Plutonium-239, the principal isotope of this element, has a half-life of 24,000 years, hence its radioactivity is undiminished within human time scales. It is perhaps the most toxic substance known. One millionth of a gram has been shown capable of producing cancer in animals [3]. Plutonium is also the material from which nuclear weapons are made. An amount the size of a softball is enough for a nuclear explosive capable of mass destruction. Scientists now widely recognize that the design and manufacture of a crude nuclear explosive is no longer a difficult task technically, the only real obstacle being the availability of the plutonium itself [4].

We believe that the commercialization of plutonium will place an intolerable strain on our society and its institutions. Our unrelenting nuclear technology has presented us with a possible new fuel which we are asked to accept because of its potential commercial value. But our technology has again outstripped our institutions, which are not prepared or suited to deal with plutonium. Those who have asked what changes in our institutions will be necessary to accommodate plutonium have come away from that enquiry profoundly concerned. And the AEC's environmental impact statement does not allay these concerns. It reinforces them.

The AEC concedes that the problems of plutonium toxicity and nuclear theft are far from solved and indicates that they may not be for some years. Yet it concludes, inexplicably, that we should proceed. Whether stemming from blind faith in the technology it has fostered or from callous promotion of the bureaucratic and industrial interests of the nuclear power complex, the AEC's proposal cannot be justified in light of what we know and, just as important, what we do not know.

The fuel now used in present-day reactors, the light water reactors, is uranium which has been enriched; the uranium-235 content is increased from 0.7 percent present in natural uranium to about 3 or 4 percent. Uranium-235 is a fissionable isotope of uranium, the remainder being non-fissile uranium-238. Unlike plutonium, uranium fuel is not extremely toxic, and it is not sufficiently rich in uranium-235 to be fashioned into nuclear weapons. The uranium

J. Gustave Speth (attorney), Arthur R. Tamplin (biophysicist) and Thomas B. Cochran (nuclear physicist) are on the staff of the Natural Resources Defense Council in Washington, D. C. Dr. Tamplin is on leave of absence from the Lawrence Livermore Laboratory of the University of California.

The keeping of police dossiers will not be limited to nuclear industry personnel. The New York Times reported August 11 that Texas state police maintain files on nuclear power plant opponents. How much more government investigation into the private lives of individuals can be tolerated by a free society? Security and surveillance procedures at best infringe upon the privacy of families and their friends. At worst, they are the instruments of repression and reprisal.

A second AEC safeguards proposal is the creation of a federal police force for the protection of plutonium plants and shipments. The draft impact statement for plutonium recycle justifies such a federal force in the following terms:

> A federal security system would be less apt to have the variations in staff and capability that would be encountered in use of private security guards. In addition, it should be noted that the consequences of a successful theft or diversion of plutonium would undoubtedly have nationwide impacts and could best be handled by Federal authorities; certainly, with Federal participation, there is the potential for a larger force, more effective weapons, and better communications [2, chap 5, p. 42].

How large would such a force be? What standards should govern and restrain its operations? The Washington Post reported in October 1973 that the AEC issued shoot-to-kill orders to personnel directing the production, shipment and storage of atomic weapons at the height of the Yom Kippur War.

Once a significant theft of plutonium or other weapons material has occurred, how will it be recovered? To prevent traffic in heroin, police have asked for no-knock search laws. This infringes upon one of our most cherished freedoms. To live with plutonium we may have to abandon this freedom along with others. In the presence of nuclear blackmail threats, the institution of martial law seems inevitable. It has been said that the widespread availability of weapons material and terrorists targets in the nuclear fuel cycle will radically alter the power balance between large and small social units (De Nike [16]). It should be added that the threatened society will undoubtedly attempt to redress that balance through sophisticated and drastic police action.

In sum, to accommodate plutonium we shall have to move toward a more intimidated society with greatly reduced freedoms. In this respect the following passage from the report of the distinguished international group of scientists attending the 23rd Pugwash Conference on Science and World Affairs is instructive:

> The problem of theft of nuclear material by internal groups of individuals intent on sabotage, terrorism or blackmail was agreed to be a very serious one, although there was some sentiment expressed that the possibility of such activity was much smaller in socialist states.

We believe that sentiment to be true. It is also apparent that that is the direction in which we must move to accommodate the nuclear industry. After having spent billions of dollars for our nuclear deterrent, our civilian nuclear industry might well accomplish that which our defense system is trying to prevent.

Alvin Weinberg is one of the few persons closely associated with the nuclear power complex who has looked carefully at the political and regulatory institutions that will be necessary to support a plutonium-based nuclear power economy, and his views on this subject merit close attention [19]. Weinberg's basic premise is that nuclear power will place unprecedented strains on our society. In an unpublished paper circulated prior to a conference in June 1973 at the Woodrow Wilson International Center for Scholars in Washington, D.C., Weinberg set out his views on the type of new institutions required to cope with the plutonium economy:

> One suggestion (proposed by Sidney Siegel) that is relevant to the situation in the United States would be to establish a national corporation patterned after COMSAT to take charge of the *generation* of nuclear electricity. Such an organization would have technical resources that must exceed those available to even a large utility: and a high order of technical expertise in operating reactors and their sub-systems is essential to ensuring the continued integrity of these devices. [Here Dr. Weinberg suggests nationalization of the industry.]
>
> Each country now has its own AEC that sets standards or, in some cases, actually monitors or operates reactors. Perhaps this will be sufficient forever. Yet no government has lasted continuously for 1,000 years: only the Catholic Church has survived more or less continuously for 2,000 years or so. Our commitment to nuclear energy is assumed to last in perpetuity—can we think of a national entity that possesses the resiliency to remain alive for even a single half-life of plutonium-239? A *permanent* cadre of experts that will retain its continuity over immensely long times hardly seems feasible if the cadre is a national body.
>
> It may be that an International Authority, operating as an agent of the United Nations, could become the focus for this cadre of expertise. The experts themselves would remain under national auspices, but they would be part of a worldwide community of experts who are held together, are monitored, and are given long-term stability by the International Authority. The Catholic Church is the best example of what I have in mind: a *central authority* that *proclaims* and to a degree *enforces* doctrine, maintains its own long-term social stability, and has connections to every country's own Catholic Church. (Emphasis added.)

These are far-reaching concepts presented by Weinberg. The basic question they pose is: Will the plutonium economy raise socio-political problems of such magnitude that their resolution will be unacceptable to society? In attempting to do the impossible—live with plutonium—we may create the intolerable.

Super-Human Requirements

The commercialization of plutonium will bring with it a major escalation of the risks and problems already associated with nuclear power. Plutonium will further strain the already weakened regulatory fabric of the nuclear industry.

Hannes Alfven, Nobel laureate in physics, has described the regulatory imperatives applicable to the nuclear industry:

> Fission energy is safe only if a number of critical devices work as they should, if a number of people in key

BEFORE THE
ENVIRONMENTAL PROTECTION AGENCY
AND
ATOMIC ENERGY COMMISSION

In the Matter of

RADIATION PROTECTION STANDARDS AS THEY APPLY TO HOT PARTICLES	))	EPA Docket No. AEC Docket No. RM

PETITION TO AMEND
RADIATION PROTECTION
STANDARDS AS THEY
APPLY TO HOT PARTICLES

The NATURAL RESOURCES DEFENSE COUNCIL, INC. ("NRDC"), on its own behalf and on behalf of its members hereby, petitions the Environmental Protection Agency and the Atomic Energy Commission to amend their radiation protection standards as they apply to insoluble particles of plutonium and other alpha-emitting hot particles as more fully described in the Report prepared by Arthur R. Tamplin, Ph.D. and Thomas B. Cochran, Ph.D., entitled Radiation Standards for Hot Particles, submitted herewith. This request is filed pursuant to 5 U.S.C. § 553(e) and 10 CFR § 2.802.

Identification of Petitioner

Petitioner NATURAL RESOURCES DEFENSE COUNCIL, INC. ("NRDC") is a non-profit, membership corporation organized under the laws of the State of New York. NRDC is a charitable organization exempt from taxation under Section 501(c)(3) of the Internal Revenue Code. NRDC's principal office and place of business is located at 15 West 44th Street, New York, New York. It maintains other offices at 1710 N Street, N. W., Washington, D. C., and at 664 Hamilton Avenue, Palo Alto, California. NRDC has a nationwide membership composed of scientists, lawyers, educators, and other citizens dedicated to the defense and preservation of the human environment and the natural resources of the United States. Other persons support NRDC's objectives by financial contributions and personal efforts.

The objectives of NRDC include:

(a) to maintain and enhance environmental quality;

(b) to monitor federal departments and regulatory agencies to ensure that environmental values are fully considered in decisionmaking, and, in particular, to ensure that federal statutes designed to protect and enhance the environment are fully and properly implemented;

(c) to improve federal agency decisionmaking which affects the environment by commenting, furnishing information, and initiating and participating in administrative proceedings;

(d) to select and undertake environmental lawsuits which have a potential for establishing widely applicable precedent for saving or reclaiming some important aspect of our national endowment, and, in particular, which require federal agencies to meet legal obligations established in federal statutes designed to protect and enhance the environment; and

(e) to provide a central, national focus for scientists, lawyers, and educators, and concerned citizens in an effort to make our courts and administrative agencies effective instruments of environmental protection.

In pursuit of its objectives, NRDC has been involved in many proceedings involving the Environmental Protection Agency and the Atomic Energy Commission.

Petitioner's Interests in the Proceeding

NRDC's basic interest in the captioned proceeding is to ensure that the public and radiation worker are adequately protected from exposure to hot particle radiation by establishing radiation standards governing permissible exposure to hot particles which carry a risk comparable to existing radiation standards governing uniform exposure to whole body radiation. It is the view of NRDC that the present radiation standards when applied to hot particles are too high by a factor of 115,000. Each of NRDC's individual numbers is a potential victim of exposure to hot particles. As an organization NRDC is dedicated to preservation of the public health and safety as part of its responsibility to maintain and enhance the environment.

Supporting Statement

Attached to this Petition is a Report prepared by Arthur R. Tamplin, Ph.D. and Thomas B. Cochran, Ph.D. entitled Radiation Standards for Hot Particles, dated February 14, 1974. This Report and the documents referenced therein provide the principal support for and elaboration of this Petition. The hot particle problem underlying this Petition can be briefly summarized as follows:

The existing biological evidence indicates that exposure to airborne hot particles of plutonium at the levels permitted by existing guidelines is extremely likely, indeed almost certain, to lead to lung cancer in the exposed individuals. Such exposures have occurred at the AEC's plutonium facility at Rocky Flats, Colorado. Moreover, it has been shown that the environment around the Rocky Flats facility has also been contaminated with plutonium from the facility. As a consequence some members of the general public have also been exposed to this material.

These exposures involved minute particles of plutonium oxide (PuO_2). These particles can become lodged in the deep respiratory tissue where, because they are insoluble, they remain fixed for a year or longer. During this time, they subject the surrounding lung tissue to an intense radiation dose. For this reason they are called hot particles -- that is, radiologically intense. While a single particle of Pu-239 oxide (one-millionth of a meter in diameter) in the lung of an average man will deliver a dose of only 0.3 mrem per year when averaged over the entire lung, the dosage to the tissue actually irradiated (65 ug)* is 4,000,000 mrem per year (4,000 rem per year). By comparison the same tissue would receive a dose of only 90 mrem due to natural background radiation. This highly non-uniform hot particle irradiation poses a unique cancer risk. For the purposes of establishing radiation exposure standards for hot particles, the risk of cancer from a single hot particle in the lung should be considered equal to one chance in 2,000. As a result, Petitioner proposes that, when hot particles are involved, the existing radiation standards governing plutonium exposure should be reduced by a factor of 115,000.

* A ug is one-millionth of a gram.

Proposed Action

The action requested by Petitioner is detailed in Chapter VII of the attached Report and summarized in Chapter VIII. The Report, for the sake of clarity, is written with reference to the hot particle formed from plutonium-239. However, the discussion in the Report and the standards sought to be established are intended to be applicable to all radionuclides or mixtures thereof capable of forming hot particles.

As the Report and subsequent discussion on jurisdiction indicate, several of the actions sought here are at least partially within the jurisdiction of both EPA and the AEC. Other proposals are apparently exclusively within AEC jurisdiction. It is not our purpose to postpone resolution of this problem by jurisdictional squabbles. We jointly petition both agencies in order to underscore our intent that between them we believe the requested action must be taken. Each proposal in the Petition should be deemed addressed to the agency which will most quickly implement the recommendation.

Jurisdiction

This Petition is addressed jointly to the Environmental Protection Agency and the Atomic Energy Commission. Under the Reorganization Plan No. 3 of 1970, Sections 2(a)(3)(ii)(C), 2(a)(6) and 2(a)(7), 42 U.S.C. § 4321, the Environmental Protection Agency became responsible for a broad range of regulations related to radiation protection. Most significantly, EPA is required to establish standards which are to be "generally applicable...for the protection of the general environment from radioactive materials". Section 2(a)(6). Three of the standards proposed in the Report limit off-site exposures to hot particles and are intended for the protection of the general environment. Two of these seek to establish the maximum permissible concentrations in air (MPC_a) and on land (MPSC) respectively.*/ The third standard limits the maximum permissible exposure resulting from nuclear facility accidents.

*/ Both of these standards are derived from the recommended maximum permissible lung particle burden (MPLPB), which should also be established as a standard.

Pursuant to the provisions of the Atomic Energy Act of 1954, 42 U.S.C. §§ 2013(d), 2133(d), and 2201(b), the AEC is mandated to develop regulations and take other actions needed to protect the health and safety of the public. The AEC has adopted radiation standards which establish maximum permissible concentration of plutonium-239 in air (see 10 CFR Part 20) and has established limits for radiation exposure due to reactor accidents (see 10 CFR Part 100). These standards fail to address the hot particle problem or provide limitations which will protect exposed persons from the risk of lung cancer attendant upon exposure to hot particles, as described more fully in the accompanying Report. The AEC's radiation protection standards in these two areas are thus inadequate and insufficient. They fail to implement the agency's mandate to protect the health and safety of the public.*/

*/ In 10 CFR Parts 20, 30, 31, 40, 50, 70, 71, 73, 100, and 115, the AEC regulates in whole or in part activities which include the reprocessing and storage of nuclear wastes, plutonium enrichment, transportation and storage of plutonium and other activities from which the normal or accidental release of hot particles could occur. None of these regulations have their own limitations applicable to hot particle releases, although Section 115.30 does make Part 20 and Part 100 applicable to all activities licensed under Part 115. It is Petitioner's position that the AEC should provide specific regulations implementing the recommendations and underlying principles of the Report filed herewith for each relevant stage of the nuclear fuel cycle.

In two areas relevant to the Petition, the AEC may have exclusive jurisdiction. First, the AEC must establish and enforce provisions for occupational exposure to radiation. 42 U.S.C. §§ 2133, 2201(b) and (p); 29 U.S.C. § 653(b)(1); 10 CFR Part 19 and §§ 20.101 et. seq. Specific limits for worker exposure to hot particles are recommended in the Report. These limits are consistent with the risks inherent in the existing levels adopted for uniform whole body exposure. Second, the AEC has now recognized the principle that all radioactive releases should be kep as far below permissible levels as is practicable. Thus, the AEC should, after adoption of the standards recommended here, move quickly to limit further hot particle releases to as low as practicable levels.

Time for Action

We believe that matters of importance to the public health and safety require prompt action. The matters we raise here are based on data generally known and available to both agencies for some time. Allowing a reasonable period for public comment we recommend that the standards requested be set within six months.

Pending and Proposed Action

We request that all approvals for construction or operation of any facilities for the handling, processing or reprocessing of plutonium or other radionuclides or mixtures thereof capable of forming hot particles or for use of such radionuclides be held in abeyance until final resolution of the matters raised by this Petition. As to projects already using or processing such radionuclides, we request that no increase in the quantity of such radionuclides processed or used be approved until final resolution of the matters raised by this Petition.

Conclusion

The requested action represents minimum steps which must be taken to protect the public from the dangers of hot particle releases. Further actions consistent with and in furtherance of the Report submitted herewith, including the adoption of additional standards and guidelines, should be considered by EPA and the AEC to assure that the public is provided the full protection to which it is entitled.

Respectfully submitted,

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J. Gustave Speth
NATURAL RESOURCES DEFENSE COUNCIL
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Attorneys for Petitioner

February 14, 1974

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July 19, 1974

Dr. William D. Rowe
Deputy Assistant Administrator for
Radiation Programs
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Re: NRDC Petition To Amend Radiation Protection Standards As They Apply To Hot Particles

Dear Dr. Rowe:

This letter is in response to your letter to me of March 26, 1974, in which you indicate that hearings will be held on the rulemaking actions which we have proposed regarding hot particles. We are pleased with this decision and look forward to participating in these hearings.

We have several requests we would like to make at this time regarding these hearings. First, we request that we be granted certain procedural rights in connection with these hearings including at least discovery and cross-examination, as well as assistance from you in making agency witnesses available. We believe that the quasi-adjudicative procedures such as these are guaranteed by International Harvester v. Ruckelshaus, 478 F.2d 615, 4 ERC 2041 (D.C. Cir. 1973); Appalachian Power Company v. EPA, ___ F.2d ___, 5 ERC 1222 (4th Cir. 1973), and elsewhere. In this regard we ask that we have an opportunity to discuss appropriate hearing procedures with you in greater detail prior to decision on your part regarding such procedures.

Second, we believe that during the procedures EPA intends to initiate regarding plutonium the issues raised by our petition should be treated distinctly so that a discrete and separable record regarding our petition can be made.

Finally, we would appreciate hearing from you regarding the question of who has jurisdiction over the various rules and standards requested in our petition, EPA or AEC. We would also like to know who will chair the hearings that will be held on our petition and what role the AEC will play in those hearings.

I would appreciate hearing from you regarding these matters at your earliest convenience.

Sincerely,

J.G. Speth

cc: L. Manning Muntzing
Peter W. Steketee
Carrie Dickerson
Ilene Younghein

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

AUG 12 1974

Mr. J. G. Speth
Natural Resources Defense Council, Inc.
1710 N Street, N.W.
Washington, D.C. 20036

Dear Mr. Speth:

Thank you for your July 19 letter in which you requested responses to several questions regarding our proposed information hearings in order to obtain information on the environmental impact of transuranium elements and to consider whether new or additional guidelines or standards are needed. We will respond to your questions in the order presented.

Since our proposed hearings are of a legislative nature for information gathering purposes only, we do not intend to allow for discovery or cross-examination.

We consider the issues raised by the NRDC petition and the information contained therein to be important sources of data on the overall problem of the potential environmental impact of the transuranium elements. Therefore, for these particular hearings, we would hope that NRDC would want to introduce the material into the proceedings so that the hearing record would be as complete as possible. Regarding the manner in which EPA treats the NRDC petition, it will, of course, receive specific attention; however, information gained at our proposed hearing may affect the ultimate decision.

As regards to a suggested or possible jurisdictional problem between AEC and EPA over the various rules and standards requested in the NRDC petition, we see none. AEC has the authority, under the Atomic Energy Act of 1954, as amended, and under the general guidance approved by the President of the Federal Radiation Council's (FRC) mandate "to encourage the maintenance of radiation doses as far below the guides as practicable," to establish regulations for controlling occupational exposures, and for specifying effluent limits for its activities. However, EPA through the President's Reorganization Plan No. 3 of 1970 now has the FRC authority and it may consider revision of the basic FRC radiation protection guides and recommend to the President guidance for Federal agencies to

cover occupational radiation standards. Also, EPA has the authority to establish generally applicable environmental standards for the protection of the general environment from radioactive materials. This includes transuranium elements as well as others. As a result of our proposed hearings and the information obtained therein, we will then decide if additional guidance or standards are required.

We have not decided as yet who will chair the proposed hearings or the actual format to be followed. These are matters that are currently receiving serious attention by the ORP staff and a decision should be made by the Agency within the next several weeks. We do not envisage AEC having any role in the hearings different from any other agency, public group, or individual who may wish to present information. We plan to make a serious and honest effort to obtain expressions of thought from all interested persons during the hearings. Therefore, we will publicize on a wide-scale the fact that we will hold such hearings so that no one will be denied an opportunity to present information. At the same time, we may be restricted by time elements or some other unforeseen circumstances; however, no one individual, group, or agency will be permitted to take an unreasonable amount of the hearing time. In summary, we are attempting, through these proposed hearings, to obtain all pertinent information and do this with an appropriate balance and perspective.

If you would care to meet with me to discuss further any of these comments or any other suggestions you may have on our proposed hearings, please let me know.

Sincerely yours,

W. D. Rowe

W. D. Rowe, Ph.D.
Deputy Assistant Administrator
for Radiation Programs (AW-558)

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August 19, 1974

Dr. William D. Rowe
Deputy Assistant Administrator
Office of Radiation Programs
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Re: NRDC Petition Regarding Radiation Standards for Hot Particles

Dear Dr. Rowe:

I have received your letter of August 12 in which you state that "Since our proposed hearings are of a legislative nature for information gathering purposes only, we do not intend to allow for discovery or cross-examination." Naturally, we are disappointed in this decision and urge that you reconsider it.

It is not completely accurate to say that the forthcoming hearings are "for information gathering purposes only." A more basic purpose of these hearings, at least the portion of them responsive to our petition, is to provide a basis for formal rule-making determinations which could have profound impacts on the health and safety of all future generations. It is imperative in this proceeding that every effort be made to probe the expertise and competence of witnesses, to establish as fully as possible the facts as best they are known, and to ventilate completely differences in expert opinion, thus establishing the often implicit bases for these opinions. Cross-examination and other adjudicatory procedures have long been recognized as essential to these purposes. Should EPA or any other federal agency arrogate to itself the full responsibility for the course and outcome of these critical hearings by denying to public groups the opportunity to make an effective presentation?

If it becomes necessary for us to challenge the EPA or AEC disposition of our petition regarding hot particles, we will be forced to raise in such a proceeding any inadequacies in the

procedural rights accorded us at the agency level, including, for example, the denial of the right to cross-examination.

Sincerely,

J.G. Speth

cc: Edson Case

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The Hot Particle Issue:

A Critique of WASH 1320* as it Relates to the Hot Particle Hypothesis

by

Arthur R. Tamplin

Thomas B. Cochran

November 1974

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* "A Radiobiological Assessment of the Spatial Distribution of Radiation Dose from Inhaled Plutonium," WASH 1320, U. S. Atomic Energy Commission [September 1974].

I. Background

On February 14, 1974, the Natural Resources Defense Council (NRDC) petitioned the Atomic Energy Commission (AEC) and the Environmental Protection Agency (EPA) to amend their radiation protection standards applicable to "hot particles" of plutonium and other actinides where hot particles were defined more fully in an accompanying report.[1] The report (referred to herein as the Tamplin-Cochran Report) concluded that the existing radiation protection standards are grossly inadequate to protect workers and the public from the high cancer risk posed by exposure to the atmospheric release of plutonium particulates from the nuclear power and weapons industries. The report recommended (and the petition requested) that the current standards be made more restrictive by a factor of 115,000. In the petition NRDC indicated that matters of importance to the public health and safety such as this require prompt action. Allowing a reasonable period for public comment NRDC recommended that the proposed standards be set within six months (by August 14, 1974).

On March 15, 1974, the AEC released its Draft of the Liquid Metal Fast Breeder Reactor Program Environmental Impact Statement (DRAFT LMFBR EIS). This statement contained a 15-page discussion of the hot particle problem.[2] This discussion, based

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1/ Tamplin, A. R. and T. B. Cochran, "Radiation Standards for Hot Particles," Natural Resources Defense Council, Washington, D. C., 14 February, 1974.

2/ DRAFT LMFBR EIS, Vol. II, Part 2, Section 4.G.5, pp. 4.G-89 to 4.G-105, March 1974.

on an earlier report by John W. Healy (referred to herein as the Healy Report) of Los Alamos Scientific Laboratory,[3] was used as justification for ignoring the approach taken in the Tamplin-Cochran Report for estimating the lung cancer incidence associated with the inhalation of plutonium particulates (hot particles) and using instead the assumption of uniform lung exposure even where hot particles are concerned.

On March 28, 1974, the AEC gave notice in the Federal Register (39 Fed. Reg. 11450) of NRDC's filing of its petition and requested public comments by May 28, 1974.

On April 16, 1974, NRDC submitted to the AEC a critique of the hot particle discussion in the DRAFT LMFBR EIS.[4] Since the hot particle discussion in the DRAFT LMFBR EIS drew heavily from the Healy Report (much of it reproduced verbatim), the NRDC comments were a critique of the Healy Report itself.

On August 5, 1974, the AEC announced that it was releasing a draft Generic Environmental Statement on Mixed Oxide Fuel (DRAFT GESMO), i.e., recycled plutonium in light water reactors. NRDC in a letter of February 21, 1974, had requested that the AEC give in this generic environmental statement a full and candid

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3/ Healy, J. W., "Contamination Limits for Real and Personal Property," Los Alamos Scientific Laboratory, Los Alamos, New Mexico, LA-5482-PR, January 1974.

4/ NRDC Comments on WASH 1535, Draft Environmental Impact Statement, Liquid Metal Fast Breeder Reactor Program, Re: Volume II, Part 2, Section 4.G.5, Particle Lung Dose Effects, pp. 4.G-89 to 4.G-105, 6 May 1974.

discussion of the recommendations and supporting evidence presented in the NRDC petition and accompanying report.

In the DRAFT GESMO, just as in the DRAFT LMFBR EIS, the uniform exposure assumption was used to calculate the lung cancer risk from hot particle exposures. The first paragraph of the following quote from the DRAFT GESMO gives the justification for this assumption. The two remaining paragraphs describe the AEC's treatment of the NRDC petition and the Tamplin-Cochran Report in the DRAFT GESMO.

> Over the past 30 years concern has arisen from time to time about the possibility that radioactivity concentrated in discrete particles might be more potent when in contact with living tissue than the same activity diffusely distributed through the same tissue (hot particle hypothesis). Numerous studies to investigate this hypothesis provide evidence that present standards have been established on a sound basis.[2] The standards setting bodies have not set different limits for these two types of exposure to radioactivity. Diffuse radiation of tissues is used to calculate dose. Hence this approach, that is diffuse irradiation of tissues, has been used in the preparation of this statement.
>
> The AEC has been asked by the Natural Resources Defense Council, Inc. (NRDC) to consider the "hot particle" hypothesis in this generic environmental statement on the use of mixed oxide fuel. Appendix D presents key elements of a report by Arthur R. Tamplin and Thomas B. Cochran[3] submitted by NRDC as well as selections from a report by J. W. Healy.[2] The Healy study is a broad review of investigations on this subject and generally supports the prevailing position of the standards setting bodies.
>
> The Natural Resources Defense Council, Inc. has raised again the question of the effect of "hot particles" in a petition filed with the Atomic Energy Commission, requesting that a reduced limit be imposed upon the concentration of plutonium in air for particles of a specified high activity. This matter is being given careful consideration in a separate proceeding.[5]

5/ DRAFT GESMO, p. IV J-7.

NRDC filed its petition requesting the reduction in the plutonium standards with the agencies charged with the responsibility. In its first official statement on this issue subsequent to the NRDC petition, the AEC presented in the DRAFT LMFBR EIS an argument based on the Healy Report. NRDC responded with a critique (NRDC's comments on the DRAFT LMFBR EIS), setting aside the Healy Report by rebutting each of the points raised in the DRAFT LMFBR EIS and showing why the references cited do not support the hypothesis that hot particles can be analyzed in the same manner as uniform organ exposures, either for purposes of estimating carcinogenic risks or for establishing radiation standards. Four months after submitting those comments, we were presented with the second AEC pronouncement on the hot particle issue (DRAFT GESMO). Here, the AEC used as justification the original Healy Report and made no reference to NRDC's comments. There was absolutely no justification for this aberrant behavior by the AEC.

We are now presented with the third pronouncement on this subject by the AEC in the report by Bair, Richmond and Wachholz (referred to herein as the BRW Report).[6] As we shall show in our critique, it is for the most part an elaboration on the Healy report. Moreover, this report also fails to acknowledge and discuss our comments on the Healy Report submitted some six months

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6/ Bair, W. J., C. R. Richmond and B. W. Wachholz, A Radiobiological Assessment of the Spatial Distribution of Radiation Dose from Inhaled Plutonium, WASH-1320, USAEC, September 1974.

ago on April 16, 1974, relative to the DRAFT LMFBR EIS. In this respect, it is also significant to note that on May 22-24, 1974, the AEC sponsored a symposium on the biological effects of plutonium at Los Alamos, New Mexico. Attendance was by invitation. The authors, Bair, Richmond and Wachholz were invited but we were not invited. When we submitted our report and petition to the AEC, we had hoped that this would lead to a dialogue that would serve to resolve this important issue. However, it appears that the AEC refuses to engage in this dialogue either face-to-face or in writing. It appears to us that the simplest elements of professional responsibility would require that they respond to our refutation of their arguments rather than continually raising the same arguments in successive publications. To this end, we again respond to their arguments. We begin by reviewing the principal elements of the hot particle hypothesis.

II. The Hot Particle Hypothesis

The "hot particle hypothesis" is relatively simple. With respect to alpha-emitting particles in the lung, it is:

> If a particle deposited in the deep respiratory tissue is of such activity as to expose the surrounding lung tissue to a dose of at least 1000 rem in 1 year, this particle represents a unique carcinogenic risk. The biological data suggest that such a particle may have a cancer risk equal to 1/2000.

This hypothesis implies that if a particle exposes the surrounding lung tissue to a dosage greater than 1000 rem in 1 year, the cancer risk is still 1/2000. (This of course causes a larger particle to be less effective on a per μCi basis,

but not on a per particle basis.) The hypothesis implies nothing about particles that expose the tissue to less than 1000 rem in one year.

The basic support for the hypothesis derives from a number of experiments wherein a small volume of tissue was exposed to high dosage. In these experiments cancer was the almost inevitable result. Although it is not explicitly stated, these experiments are relevant to the following NCRP criteria:

> (206) Simplifications in practice hinge largely on reporting a single representative protection dose for a limiting organ system even when the actual irradiation is grossly non-uniform. The representative dose is taken as the highest that can be obtained by averaging over a prescribed significant volume. The implication of this concept, or at least the convention that is followed, is that any redistribution of a given dose within such a volume does not materially alter the radiation response. It is usually assumed that the "significant volume" should be of the order of one cubic centimeter. This will be grossly conservative.
>
> (207) There will be some cases in which selection of a significant volume is inappropriate. Most notably these will include cases where the radiation agent is an alpha particle emitter deposited in thin sheets. As an example, the deposition of radon daughter products on the bronchioepithelial lining of the lungs is a case in which the effective radiation field is virtually two-dimensional only. In such cases, one may plausibly consider a significant area of tissue surface, perhaps equally arbitrarily taken as one square centimeter. Realistic modeling of such cases suggests a much smaller region as the reasonable effective target.[7]

The hypothesis is essentially an extension of these criteria. The quantitative parameters in the hypothesis are derived from a series of experiments conducted by Dr. Roy C. Albert on rat

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7/ NCRP Report No. 39, Basic Radiation Protection Criteria, NCRP Publications, Washington, D. C., January 15, 1971.

skin.[8-10] In these experiments, Dr. Albert observed that the radiation induced cancers were remarkably correlated with the disruption of a critical architectural unit of the skin, the hair follicle. The cancers were induced in the rough proportion of 1 cancer per 2000 atrophied hair follicles when the dosages exceeded some 1000 rem.

The hot particle hypothesis thus suggests that if these skin experiments were performed with small particles, each capable of disrupting a single hair follicle, the observed cancer induction would correspond to one cancer per 2000 particles.

So far as the lung is concerned, the hypothesis contains the corollary that the lung also has such a critical architectural unit that can be disrupted by a single particle and that this also presents a cancer risk of 1/2000.

The potential hazard of a single hot particle embedded in the tissue of humans is illustrated by the observation of Lushbaugh and Langham.[11] They excised a nodule that developed

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8/ 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.

9/ 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. 525-540.

10/ 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.

11/ Lushbaugh, C. C. and J. Langham, "A dermal lesion from implanted plutonium," Archives of Dermatology 86, October 1962, pp. 121-124.

around a Pu-239 particle imbedded in the palm of a machinist. 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....[12]

Considering the above observations, it would be surprising indeed if a physician would not suggest surgical intervention in a case where a patient had a few such imbedded particles. We feel that this lesion alone should cause one to be very cautious in estimating the hazard of hot particles.

That such lesions can develop in lung tissue is supported by the observations of Richmond, et al., on the lesions induced in experiments wherein hot particles were introduced into blood vessels of the lungs of rats:

> Such a lesion with collagenous degeneration and subsequent liquefaction, due to the large local dose of radiation at a high dose rate, has been reported by Lushbaugh et al.,[(9)] whose description of a plutonium lesion found in the dermis is very similar to that observed for plutonium in the lung.[13]

12/ Ibid., p. 463.

13/ Richmond, C. R., et al., "Biological response to small discrete highly radioactive sources," Health Physics 18, 1970, p. 406.

The above represents the distilled essences of the Tamplin-Cochran Report which was an extension of some earlier publications of Professor Donald Geesaman.[14] It is important to restate that the hypothesis suggests that the disruption of a critical architectural unit of a tissue is a significant carcinogenic event.

The actual killing of cells and the development of a fibrotic lesion surrounding the hot particle is the suggested mechanism of carcinogenesis. As Geesaman stated:

> Summing up, intense radiation exposure of mammalian skin and lung tissue commonly results in cancers. Tissue injury and disturbance are a primary consequence of intense radiation insult, and are observed in association with carcinogenesis. Albert has exhibited a simple proportionality between skin carcinomas and atrophied hair follicles. No general description of precarcinogenic injury exists, but in a crude sense the available observations are compatible with the idea of an injury-mediated carcinogenesis. Cancer is a frequent instability of tissue. Since tissue is more than an aggregate of cells, and has a structural and functional unity of its own, it would not be surprising if some disrupted local integrity, a disturbed ordering, comprises a primary pathway of carcinogenesis. The induction of sarcomas with inert discs of Mylar, cellophane, Teflon and Millipore (Brues, et al.[17]) is indicative that such a mechanism exists. Presumably mitotic sterilization is an important factor in any carcinogenesis mediated by radiation-induced tissue injury. The functional relation of this factor in the carcinogenic response may be quite different from a linearity in the surviving mitotic fraction.

14/ Geesaman, D. 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, California, 1968.

> While regrettably unquantitative, the hypothesis of an injury-mediated carcinogenesis is suggestively descriptive. If the respiratory zone of the lung contains a structure analogous to the rat hair follicle, and if a radioactive particulate deposited in the respiratory zone has the capacity to disrupt one or more of these structures and create a pre-cancerous lesion, then cancer risks of the order of 10^{-3} to 10^{-4} per particle can be expected.[15,16]

The lesion excised by Lusbaugh and Langham[17] from human palmar tissue and the observation by Richmond, et al.,[18] that similar lesions are produced in the lung by hot particles strongly argue that a comparable sensitive structure is present in the lung and other tissues. Thus, the uncertainties in the hot particle hypothesis involve these quantitative parameters:

a) Is the risk of cancer per disrupted tissue mass comparable to that per disrupted hair follicle?

b) Is a particle capable of irradiating the surrounding tissue mass at the rate of 1000 rem/year sufficient to produce such a lesion?

The thrust of the NRDC petition to modify the plutonium exposure standards is that, until these uncertainties are resolved, the prudent public health principle is to accept the hot particle hypothesis rather than the less conservative hypothesis that average organ dose from hot particles provides

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15/ Geesaman, D. P., UCRL-50387 Addendum, op. cit., pp. 6-7.

16/ Brues, et al.[17], refers to Brues, A. M., H. Auerbach, G. M. De Roche, and D. Brube, "Mechanisms of carcinogenesis," Argonne National Laboratory, Biological and Medical Research Division Annual Report for 1967, ANL-7409, 1967, pp. 151-155.

17/ Lushbaugh, C. R. and J. Langham, op. cit.

18/ Richmond, C. R., et al., op. cit.

a reasonable basis for protection. The implication is, of course, that while the evidence discussed in the Tamplin-Cochran Report supports the hot particle hypothesis there is no substantial body of scientific evidence that can reject the hypothesis. The purpose of this report is to demonstrate that the evidence is also not to be found in the BRW Report.

III. Points of Analytical Confusion

Before reviewing the BRW Report in detail the following general observations are presented in order to draw clear distinctions among several analytical approaches or concepts that appear to be the source of some confusion to analysts addressing the hot particle issue. These approaches are: (1) The assignment of a risk per hot particle, independent over a range of particle sizes and activities; (2) the comparison of the risk associated with a fixed amount of activity (or absorbed dose) when spread uniformly over tissue with the risk when the same activity (or absorbed dose) is spread non-uniformly over the same tissue; (3) the concept of "wasted radiation" and/or "overkill." It is essential that these three approaches or concepts and their relationships (or distinguishing features) be clearly understood before judging the relevance of experimental data to the hot particle issue. We begin by reviewing each approach or concept and then examine their relationships of (2) and (3) to (1).

(1) Risk Per Hot Particle -- The assignment of a risk per hot particle is based on a hypothesis that when the radiation dose to the irradiated tissue mass surrounding a radioactive particle is sufficient to disturb a critical architectural unit of the tissue, such a disrupted tissue mass poses a unique carcinogenic risk. A value is assigned for the tumor risk associated with the disrupted tissue. Since for small particles there is a one to one correspondence between the disrupted architectural unit and the associated radioactive particle, this tumor risk is the risk per particle. In the Tamplin-Cochran Report, a lower limit on the radiation dose (and therefore alpha activity) to disrupt the architecture was assigned (1000 rem to the irradiated tissue) and used to define a hot particle. No opinion was offered with respect to the appropriate risk function for doses (or activities) below this cutoff value. In the lung there is an upper limit on the size of particles that are deposited in the deep respiratory tissue. Hence, in the lung there is a "window" on the hot particle size and activity. In analyzing experimental data vis-a-vis the hot particle hypothesis the relevant parameter is the tumor risk per hot particle.

(2) Uniform Versus Non-Uniform Exposure -- Present radiation standards are based on (i.e., establish limiting values for) the concept of radiation dose equivalent (units of rem) to the whole body and certain critical organs. In the calculation of the rem dose a "dose distribution factor" is assigned in order that the risk associated with a non-uniform distribution of a given type of radiation exposure to the critical organ is

consistent with uniform exposure by the same type of radiation. Consistent with this approach experiments have been designed and analyzed to assess the difference between uniform and non-uniform distributions of dose to critical organs. For internal alpha-emitters the absorbed dose (in rads) to a critical organ is proportional to the total activity in the organ.[19] Hence, tumors per microcurie has been the primary parameter used when comparing tumor risk for uniform versus non-uniform dose distributions.

(3) Wasted Radiation -- The concept of "wasted radiation" or "overkill" has been invoked to describe that fraction of the radiation which kills cells, where these dead cells are assumed not to contribute to tumor production. For example, the dose rate in the immediate vicinity of a single alpha-emitting particle in the lung (or other tissue) can be high enough (given a sufficient particle activity) such that even a limited residence time in the tissue will result in the death of cells within a given radius. Since such cells can not reproduce it has been hypothesized that they would not lead to cancer.[20] An alternative hypothesis, consistant with the hot particle hypothesis, is that the presence of dead cells, cellular products or fibrosis may be required for tumor production.

19/ This is also generally true for beta-emitters.

20/ The concept of "wasted radiation" also has been invoked to describe the radiation dose during the period from the inception of initial malignancy until detection or death. The concepts of overkill and wasted radiation have been used interchangeably.

In order to demonstrate the relationships among the three approaches and concepts described above it is useful to analyze some hypothetical experiments. We do this below:

Tumors/μCi or Tumors/Particle -- Suppose one ran a series of related experiments involving hot particles in tissue where the tissue mass and the total activity were held constant across experiments (e.g., the same number of lungs exposed to 12 nanocuries total activity in each experiment), and the experiments differed only in the number of particles and the activity per particle. Consistent with the hot particle hypothesis (one tumor per 2000 hot particles) suppose one observed a tumor incidence given below in the second column from the right.

Experiment	Number of Hot Particles	Activity per Particle (pCi)	Number of Tumors Observed	Tumors per nCi
1	6000	2	3	0.25
2	4000	3	2	0.17
3	2000	6	1	0.08
4	200	60	0	0.00

From the observed number of tumors and the total activity (12 nCi), the tumors per nanocurie are calculated in the last column. Holding the total activity and tissue mass as constant while increasing the number of particles tends to make the exposure more uniform. Hence the results, when analyzed on a tumor per nanocurie basis (the last column), appear consistent with the view that uniform exposure carries a higher risk than non-uniform exposure. But these same experimental results are exactly consistent with the hot particle hypothesis. What does this tell us? First, it clearly demonstrates that an analysis of

an experiment, or series of experiments, on a tumor per nanocurie or microcurie basis, the results of which appear consistent with the concept that uniform exposure carries a higher tumor risk than non-uniform exposure, is not in itself a refutation of the hot particle hypothesis. In fact, if the hot particle hypothesis is correct, an analysis based on tumor per microcurie is irrelevant. One can just as easily design a series of experiments consistent with the hot particle hypothesis, which when analyzed on a tumor per microcurie basis suggests the opposite, that is, uniform exposure carries a smaller risk than non-uniform exposure, as is the case with respect to the two experiments below.

Experiment	Number of Particles	Total Activity (nCi)	Number of Tumors Observed	Tumors per nCi
1	6000	12	3	0.25
2	4000	6	2	0.33

Again, if the hot particle hypothesis is correct, the analysis based on tumors per microcurie would be irrelevant. If tumor production depends on the number of disrupted architectural units independent of particle activity (over a range of activities), analyzing the data on a tumor per microcurie basis clearly makes no sense. One would not expect, a priori, a correlation between tumors per microcurie and numbers of particles (uniformity of dose). To the contrary one should not be surprised to see conflicting experimental results (i.e., some experiments suggesting uniform exposure carries a higher risk and other experiments

suggesting the opposite). The relevant parameter to judge the hot particle hypothesis is tumors per hot particle, not tumors per microcurie.

At this point we might add that in addressing the hot particle issue, an analysis based on tumors per microcurie (or tumors per rad), where the radiation exposure is from other than hot particles (and therefore a different carcinogenic response mechanism may be controlling), is also irrelevant and is simply a compounding of mistakes.

We do not imply that comparisons of the risks associated with uniform and non-uniform exposure serves no useful purpose. Consider, for example, radium-226 and plutonium-239 which are both alpha-emitters and both bone seekers, that is both are preferentially deposited in the skeleton. The cancer risk per microcurie deposited in the skeleton (or per rad) is about five times higher for plutonium than radium. This suggests that plutonium is preferentially deposited in tissue more sensitive to the development of bone cancer, and that in calculating the dose equivalent (rem) to the skeleton due to plutonium the use of a dose distribution factor of 5 is appropriate. However, this clearly has no relevance to the hot particle hypothesis which is an entirely different effect, aside from the fact that the distribution factor for plutonium in the bone is based on soluble plutonium and not hot particles.

Hot Particles and Wasted Radiation -- Turning next to the concept of wasted radiation, suppose one were to implant one hot particle of alpha activity in a critical organ such as the lung. Under the hot particle hypothesis it would carry a tumor risk

equal to the assigned risk per particle, one in 2000. As long as the particle activity remained above the cutoff limit defining a hot particle, changing the activity, for example doubling it, would not change the lung tumor risk. If the activity and therefore the radiation dose were doubled without a change in the tumor risk, one could invoke the concept of "wasted radiation" or "overkill." At least one-half the activity (more than one-half if the particle activity were greater than twice the minimum defining a hot particle) would be "wasted." The hot particle hypothesis is consistent with the concept of "wasted radiation." But more important, the concept of "wasted radiation" is clearly irrelevant in judging the validity of the hot particle hypothesis. What is important, is the assessment of the risk per particle over the range of particle sizes defining hot particles. The relevant parameter in this assessment is again, the tumor risk per hot particle.

IV. Page by Page Critique of the BRW Report

In this section we will present a page by page critique of the BRW Report. To avoid confusion we will use their method for bibliographic citation. Their bibliography is reproduced at the end of this section.

Page 1. "Summary and Conclusions." We will comment on the conclusions in this section as we review the related material in the main text of the report, only noting here that the conclusions are without merit.

Page 3. "I. Statement of the Problem." We generally agree with this statement of the problem, noting only that the hot particle hypothesis is based on damage to a critical architectural unit as opposed to individual cells. The discussion here is essentially the same as the discussion on pp. 15-17 of the Tamplin-Cochran Report and Table I in the BRW Report is comparable to Table III in the Tamplin-Cochran Report.

Pages 5-7. "II. Background." This is a general discussion of consideration of irradiation from radioactive materials in particulate form by several organizations concerned with radiation protection, including the ICRP, NCRP and National Academy of Sciences--National Research Council (NAS-NRC). The thrust of this discussion is that (1) non-uniformity of dose has been recognized, been of interest, and periodically reviewed since the early days of the Manhattan Project, and (2) organizations with responsibility for recommending radiation standards, such as ICRP, NCRP and NAS-NRC, have never recommended a change from the current practice of basing radiation standards on the mean dose to organ. While the hot particle problem is well recognized in the biological community, and while we agree with the observations above, we do not believe the conclusion reached on page 7 by the authors of the BRW Report is appropriate, namely:

> The fact that these organizations have not changed or recommended changes in the procedures used for calculating dose to the lung as the result of their deliberations is an implication of implicit guidance on this particular problem.

To the contrary, had these organizations intended that this conclusion be drawn, they would have made it explicit. In its Publication 9, the ICRP (1966) states (p. 4):

> ...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.

And the NCRP (1971) offers the similar statement (pp. 79-80):

> (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.

The appropriate interpretation of these remarks by the ICRP and NCRP is that there is no guidance as to the risk for non-homogeneous exposure in the lung. The intent of these remarks is to call attention to exceptions to the general rule, rather than to implicitly advocate averaging the dose over the critical organ when the dose is grossly non-uniform.

Page 7. With regard to the quotation from the ICRP Task Group in Publication 14 (ICRP 1969), it is not at all clear that the Task Group reviewed Geesaman's work before preparing this ICRP report. Moreover, while the opinion of the Task Group may be worth noting, it is important to note that it is only an opinion and is totally unsupported in ICRP Publication 14. Considering this in 1974, it is significant that in the intervening 5 years since the issuance of Publication 14,

adequate support for that opinion has not been forthcoming and as we demonstrate here is not to be found in the BRW Report. Quite the contrary, the analysis of Geesaman and the Tamplin-Cochran Report have emerged to support the opposite. The BRW Report states that new data tend to support the ICRP Task Group's opinion. With this, as we show in this critique, we totally disagree.

Pages 9-23. "III. Animal Studies."

Pages 9,10. "A. Retention of Plutonium in Lung"

This section discusses the long retention time of PuO_2 in human lung. There is no controversy here.

Pages 10-12. "B. Spatial Distribution of Plutonium Within Lung"

This section, while attempting to indicate that Pu particles in the lower respiratory region are not static, does admit on page 12 that autoradiographic evidence demonstrates that such particles are immobilized in scar tissue and possibly in Type I alveolar epithelial cells. The long residence time of Pu particles in the lung suggests that such immobilization must occur.

Pages 12-23. "C. Pulmonary Neoplasia"

These pages present the animal data on Pu induced lung cancers. The data on both soluble and insoluble Pu compounds are presented. It is only those experiments that involve insoluble alpha-emitting hot particles that are of interest here. Of those experiments discussed here, it is only those involving PuO_2 that are pertinent. Since these experiments are recanted in the subsequent section of the BRW Report, we will briefly discuss only a few of them here.

Page 13. Mention is made here of an experiment (Bair, et al., 1962) wherein 800 mice were subjected to inhalation of 0.1 to 2 nCi per gram of lung. At time of death, these animals had retained only 0.1 to 10 pCi in their lung. Moreover, the report states that since so few autopsies were performed, the lung tumor incidence is unknown. In other words, this experiment is of little value to the hot particle problem.

The beagle dog experiment (Park, et al., 1972) (Park and Bair, 1974) did involve Pu hot particles. However, as we indicated in the Tamplin-Cochran Report, since the tumor incidence was essentially 100%, this experiment does little to resolve the uncertainties in the hot particle hypothesis.

Page 15. The Pu-238 experiment by Sanders (1973) involved PuO_2 derived from crushed microspheres. However, Sanders indicates that this material was "soluble" in his experiment and that the irradiation was uniform. The observed rapid clearance from the lungs supports this contention.

The baboon studies (Metivier, et al., 1972) relates to hot particles but at quite large particle concentrations which, as in the beagle experiment, makes it difficult to draw inference relative to lower concentrations.

Pages 16-23. "D. Experiments of Special Relevance to Non-Uniform Dose Distribution"

Page 16. This page is a confusing discussion of "wasted radiation" and "overkill." As we stated in the previous section of this critique, the hot particle hypothesis designates a

minimum particle activity--one that delivers a dose of 1000 rem/year to the irradiated tissue. Such a particle is suggested to have a chance of producing cancer equal to 1/2000. Particles with greater activity have the same chance, hence the concept of "overkill" or "wasted radiation" is included in the hot particle hypothesis.

This page also contains the following sentence and footnote:

> For a single radioactive particle of 239 PuO_2 in the lung (or other tissue), the dose rate near the particle can be high enough to cause the death of all cells within a given radius even if the residence time of the particle is short. Such cells will not be able to reproduce and subsequently result in cancer.*

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*The presence of dead cells, cellular products or fibrosis may be required before a cellular transformation can express itself as a cancer. However, this concept has not been generally accepted.

This same statement and footnote appeared in both the Healy Report and the Draft EIS for the LMFBR with the significant exception of the last sentence in the footnote. Even if this last sentence were true, which we doubt, it is irrelevant because matters of science are not determined by public opinion polls. Nevertheless, we are curious concerning the method employed by the authors of the BRW Report to establish this conclusion.

We have previously indicated that the hot particle hypothesis implies an injury-mediated mechanism of carcinogenesis as the footnote suggests (see pp. 9-10). There is no need to repeat that discussion here. However, we submit that lesion discussed by Lushbaugh and Langham (1962) is by itself so

incriminating of hot particles that we are amazed that the authors of the BRW Report are so reluctant to acknowledge the potential hazard of such particles.

It is, however, obvious that this reluctance led to confusion on their part. For example, the paragraph, from which the above quote was extracted, ends on page 17 with this statement:

> The relevant parameter is tumors per microcurie because the basic question is how the risk from hot particles compares with the risk from uniformly distributed radiation doses.

In the previous section of this critique we demonstrated that the test of the hot particle hypothesis must be on the basis of tumors per particle not tumors per microcurie simply because particles can contain more than the minimum activity (and hence, be "wasteful" on a per μCi basis). If the AEC had chosen to engage in a dialogue with us, this simple but fundamental matter could have been resolved and much of the extraneous material in this BRW Report could have been eliminated (if not the entire report).

Page 17. This page contains the following paragraph:

> Two approaches have been used in skin experiments. The first was to determine whether isolated small areas of irradiated skin gave the same yield of tumors per unit as large-area skin irradiations. The focal irradiation pattern with low LET radiation, electrons (Albert et al., 1967b), was less efficient than the large area exposure in producing tumors. However, with high LET radiation (protons) there was no difference (Burns, et al., 1972). If these results can be extrapolated to alpha radiation, they suggest that the risk from particulate sources is no greater than from uniformly distributed sources.

Apparently the authors of this paragraph do not understand the purpose and significance of the experiment by Burns, et al., (1972)

and this is reflected in the last sentence which makes no sense.

The purpose of the experiment by Burns, et al., was to determine the basis for the lower tumor producing efficiency of electrons where the irradiation was performed in a sieve pattern. Since the electrons are highly scattered, the focal radiation dose was uncertain. With the relatively non-scattering protons, the sieve pattern produced the same number of tumors per area irradiated.

These experiments demonstrate that if 24 cm^2 of rat skin are irradiated to 1000 rem, one tumor will develop per animal. If you irradiated 12 cm^2 to 1000 rem, one tumor will develop per two animals; 6 cm^2 should produce one tumor per four animals and so on. Moreover, the data strongly suggest that as the area irradiated is reduced to that corresponding to a single hair follicle, one tumor will develop per 2000 animals.

The next paragraph discusses the experiments of Albert, et al., and ends with the following discussion:

> A plausible explanation for the experimental results is that each follicle has a population of stem cells at a depth of 0.3 mm that are concerned with the production of sebaceous cells and hair. These stem cells apparently constitute the most sensitive potential oncogenic cell population to ionizing radiation in the rat skin since all the tumors were mainly of hair follicle origin (Albert, et al., 1969). Neoplastic transformation of a significant number of these target cells required large radiation doses which in turn killed most of the target cells and thus caused follicle atrophy.

This is a possible explanation but it does not set aside the hot particle hypothesis. The killing of cells and the consequent disruption of the tissue may well be sufficient by itself for such "neoplastic transformation." The induction of

tumors with mylar film and millipore filters by Brues, et al.,[21] would support this as would the precancerous cytological changes observed around the lesion excised by Lushbaugh and Langham (1962) and around the microspheres in rat lungs by Richmond, et al., (1970).

Page 18. This page goes on to discuss other skin tumor experiments and the first column ends by stating that the evidence does not support the hot particle hypothesis as detailed in the Tamplin-Cochran Report. We offer the above paragraph and this entire critique as refutation of that contention.

The experiments of Richmond, et al., (1970) are discussed. This discussion, however, fails to note that Richmond, et al., stated that the lesions observed in the rat lungs following exposure to these hot microspheres were similar to that observed by Lushbaugh and Langham (1962) in human palmar tissue.

Page 19. The experiment of Passonneau (1952) is mentioned here. It was also discussed on page 17. This experiment is simply a variation of the experiments of Albert, et al., (1967a, 1967c, 1969).

Pages 19-20. These pages discuss the experiments of Richmond with Sullivan and Voelz as reported in:

Richmond, C. R. and G. L. Voelz (eds.)

LA-4923-PR, pp. 18-34 (April 1972),
LA-5227-PR, pp. 1-11 (March 1973),

and Richmond, C. R. and Sullivan, E. M. (eds.)

LA-5633-PR, pp. 1-9 (May 1974).

21/ Brues, A., et al., op. cit.

These are a series of progress reports on experiments wherein microspheres of $^{239}PuO_2$ and $^{238}PuO_2$ incorporated in ZrO_2 particles (10 μ diameter) are injected into the jugular vein of hamsters. These particles lodge in the capillary network of the lung.

The BRW Report suggests that these experiments are a strong argument against the hot particle hypothesis. We shall show that while the experiments raise some questions concerning the quantitative parameter in the hot particle hypothesis, they also support the hypothesis.

In the initial experiment 2000 particles per animal were injected according to the following dosage schedule (60 animals per dosage level).

Isotope	Level	pCi/particle	nCi/animal
Pu-239	1	0.07	0.14
	2	0.22	0.44
	2A	0.42	0.84
	3	0.91	1.82
	3A	1.60	3.20
Pu-238	4	4.30	8.60
	5	13.30	26.60
	6	59.40	119.00

Only two lung tumors developed in the experiments and they occurred in the level 2A exposure group. However, the latest progress report (LA-5633-PR) mentions histological changes occurring in the lungs of long term animals (15-20 months) in the 4-6 exposure levels. Concerning these changes, Richmond and Sullivan (1974, p. 7) stated:

> There has been no increase in frank tumors observed within the past year; however, the epithelial changes described above could be considered as precursors of peripheral adenomas.

This suggests an incipient carcinogenic response to the particles but the life span of the rats and hamsters is too short for the development of a frank tumor.

Similar histological changes were observed in rats injected with these microspheres by Richmond, et al., (1970) who pointed to the similarity of these particle induced lesions in the rat lung to that observed by Lushbaugh and Langham (1962) in human palmar tissue.

For reference, in the beagle dog experiment lung tumors developed (in all animals that survived 1600 days) some 5 to 11 years after the initial alveolar deposition of 3 to 50 nCi/gram of bloodless lung (Park and Bair, 1972). The exposures were by inhalation, not injection.

On a nCi/gram basis, the beagle exposures would correspond to exposure levels 3 and above in the Richmond experiments. But the medium activity per particle in the beagle experiment corresponds to those in exposure levels 1 and 2 in the Richmond experiments which suggests that with longer exposure periods, lower activity particles (corresponding to levels 1 and 2) can produce the histological changes observed in the rat and hamster lung and in human palmar tissue. At the same time, since the beagle exposures involved a spectrum of particle sizes, it must be conceded that the carcinogenic response in the beagles could have been elicited by the larger, higher activity particles.

In either case, the beagle dog data suggest that the induction time for the hot particle mechanism of carcinogenesis exceeds the life span of the hamster by some three years or more.

Thus, the absence of a large carcinogenic response in the hamsters does not set aside the hot particle hypothesis.

The Richmond experiments point out one of the uncertainties in our quantification of the hot particle hypothesis but they do not resolve it. We suggest that a lower limit for a hot particle be one that contain sufficient radioactivity to deliver an average dose of 1000 rem/year to the exposed tissue. For an alpha-emitting hot particle, this limit corresponds to 0.07 pCi. In LA-5633-PR the authors state with respect to this histological change (p. 7), "This lesion has been observed almost entirely in the higher activity levels (levels 4-6 and in animals given relatively small numbers of spheres (2000-6000)." A level 4 particle contained 4.3 pCi, some 60 times our limiting activity. But, at the same time, had these experiments been performed with animals that have longer life spans, it is quite possible that these histological changes would have developed around particles containing our suggested limiting activity.

Nevertheless, a 60 fold increase in activity requires only a 4 fold increase in particle diameter--for Pu-239, a change from 0.6 μ to 2.4 μ; for Pu-238, a change from 0.09 μ to 0.36 μ and for high burn-up nuclear fuel, a change from 0.4 μ to 1.6 μ. These particles are still in the range that permits deposition in the lower respiratory zone. Thus, these experiments do not set aside the hot particle hypothesis. Rather they suggest additional experiments involving longer lived animals to determine whether this histological change progresses into frank tumors and whether lower activity particles also produce these changes.

If an experiment comparable to these with hamsters were initiated with beagles, it would serve to resolve these uncertainties. Such an experiment would take some 15 years to complete. In the meantime, we propose that prudent public health practice dictates that exposure standards should be established on the basis of the hot particle hypothesis.

The experiments of Little, et al., (1970a, 1970b, 1973) are said to add significance to the microsphere experiments. As we show subsequently, the experiments of Little, et al., involved uniform exposure to Po-210 at high dosage (above 8000 rem). These experiments therefore do not involve hot particles and there is no a priori reason for assuming that they involve the same carcinogenic mechanism as hot particles.

Pages 20-21. The experiments of Shubert, et al., (1971) and Brooks, et al., (1974) are discussed here. These experiments made a determination of the frequency of chromosomal aberrations in liver cells following uniform and particulate irradiation. It is important to note that a causal relationship between chromosomal aberrations and subsequent cancer development is only a hypothesis. Moreover, as we have stated previously, the actual killing of cells and the subsequent disruption of the normal tissue architecture may well be the carcinogenic mechanism for hot particles. Thus, these experiments are of little value in resolving this issue.

Pages 21-22. The experiments of Little, et al., (1970a, 1970b, 1973) and Grossman, et al., (1971) are discussed here. In these experiments hamsters were exposed to Po-210 lung doses

ranging from 8,000 to 20,000 rem. In some experiments the Po was absorbed on hematite particles. However, calculations demonstrate that the activity per particle ranged from 10^{-4} to 10^{-3} pCi[22] and, consequently, that these were not hot particles. Therefore, the conclusion of Little, et al., (1973) quoted on page 22 is not relevant to the hot particle issue.

We note in passing, however, the nature of the experiments was that the entire lung was irradiated to very high dosage although there was some aggregation of particles. A large carcinogenic response was initiated in each exposure group. The preliminary data reported here indicate that the life span of the hamster is longer when the dosages are this high and the Po-210 is on particles. However, it is not sufficient to demonstrate a reduction in overall tumor response. Like the beagle experiments, the carcinogenic response in these experiments appears to be saturated because of the high dosage delivered to the whole lung or a major fraction thereof. No conclusions can be drawn relative to lower doses nor relative to hot particles. With respect to lower dosages, the work of Sanders (1973) demonstrates a large tumor incidence in rats at a dosage of 320 rems.

Pages 22-23. These pages discuss the experiments of Cember, et al. The major thrust of the Cember article deals with ^{144}Ce particles in the lung. The ^{144}Ce was introduced admixed with stable Ce as either CeF_3 or $CeCl_3$ in particles of about 1 μ in diameter (0.5 μ^3). ^{144}Ce emits a beta particle

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22/ NRDC Comments on WASH 1535, op. cit., p. 39.

of 0.275 MeV and its daughter product ^{144}Pr emits a beta of 3 MeV. The rate of energy loss for these beta particles in tissue is about 0.2 Kev/μ compared to some 94 Kev/μ for plutonium alpha particles.

This difference in energy loss per micron indicates that the activity of the ^{144}Ce emitters would have to be some 500 times that of the ^{239}Pu in order to deposit the same energy in the tissue irradiated by ^{239}Pu alpha particles. Moreover, since the QF for alpha particles is 10, the ^{144}Ce particles must have an activity (10) x (500) or 5,000 times that of a $^{239}PuO_2$ particle to qualify as a hot particle. Since the limiting activity of a $^{239}PuO_2$ particle is 0.07 pCi, a hot particle of $^{144}CeCl_3$ would have to contain more than 350 pCi. After correcting for the half-life of ^{144}Ce (288 days) a hot particle would have to contain some 500 pCi.

The geometric mean diameter of the particles in these experiments was 1 micron. The highest exposure group received 50 μCi of ^{144}Ce in 30 μg of CeF_3. Allowing a density of 6 g/cm^3 for the CeF_3, the beta-activity per particle of 1 μ diameter is only 5 pCi. In other words, these experiments did not involve hot particles as defined above. The carcinogenesis observed in these Cember experiments, which was considerable, was related to high total and rather uniform organ dosage (1,000-30,000 rad).

Page 23. Here the experiments of Sanders (1973) and Moskalev (1972) are discussed. Large carcinogenic responses were observed in the lungs of rats at doses of 100 to 500 rem

using "soluble" Pu compounds. One conclusion that is justified by the results of these studies is that the exposure standards for plutonium may be much too high (at least 100 times too high) even when hot particles are not involved. The results of Sanders indicate that a uniform dose of 15 rem doubled the natural incidence of lung cancer in the exposed rats. A worker is allowed this dose each year and a member of the population could accumulate this dose in 10 years.

One further point could be made concerning the study of Sanders. It is not at all clear from the description given in the reference that the exposures did not involve a few hundred hot particles. If this were so, these particles could have been partly responsible for the observed cancers.

The preliminary studies by Lafuma (1974) do not appear to be published and we have no copy of the seminar given in France. Indications are, however, that it is not different from the experiments discussed above.

Again we offer the above and this entire critique as refutation of the conclusion reached in the last paragraph of this section.

Pages 25-29. "IV. Human Experience."

This chapter of the BRW Report discusses the exposure of humans to Pu. The major thrust of the chapter involves workers from the Manhattan Project and from the Rocky Flats plutonium facility in Colorado. We discuss these in the Tamplin-Cochran Report but the authors of the BRW Report overlooked or ignored the salient features of our discussion.

Pages 25-26. The Manhattan workers are discussed on these pages. On pages 38 to 40 of the Tamplin-Cochran Report, based upon information from Hempelmann, et al., (1973a, 1973b) we calculated that the exposures of these workers did not involve hot particles. The authors of the BRW Report inexplicably ignored this discussion and made the unjustifiable assumption that the particles here corresponded to those associated with a fire at the Rocky Flats plutonium facility. As a consequence, the discussion of expected cancers on page 26 is without merit.

Pages 26-27. The discussion of chromosome aberrations has no relevance to the hot particle problem.

Pages 27-28. The exposure of employees of the Rocky Flats plutonium facility in October 1965 is discussed here. In the Tamplin-Cochran Report we pointed out that the induction period in man for hot particle carcinogenesis is unknown. In the beagle dog experiment (Park and Bair, 1972) it was 11 years before the dog with the lowest burden developed lung cancer. Thus, although no cancers have developed in the Rocky Flats workers at this time (9 years post exposure) the possibility exists that a number of cancers will appear in the next 10-15 years.

Page 28. The lesion excised by Lushbaugh and Langham (1962) is discussed here. To the extent that a lesion with changes similar "to known precancerous epidermal cytologic changes," that raise the question of its fate without surgical intervention differs from a precancerous lesion, we were remiss in the Tamplin-Cochran Report.

Page 29. As we indicated in the Tamplin-Cochran Report, the Pu in fallout did not occur in hot particles and hence, fallout Pu is irrelevant to the issue.

Pages 31-35. "V. Theoretical Consideration."

At the outset, it is important to note that one hypothesis cannot be used to set aside another. Each hypothesis must stand alone with respect to supporting experimental data.

Pages 31-33. "A. Dosimetry." This is general information about which there is little controversy.

Pages 33-35. "3. Models for Dosimetry and Tumor Probability." We agree with the concluding remarks of this section. The models discussed here relate tumor probability to cellular radiation dose. Depending upon the assumption, they can give a variety of tumor probabilities.

We would simply add that the lesion excised by Lushbaugh and Langham (1962) coupled with the observations of similar lesions induced in the lungs of rats and hamsters should be sufficient to cause anyone to be skeptical of a tumor induction model which indicates a low tumor probability for a hot particle.

Pages 35-39. "B. Radiation Carcinogenesis Relative to Spatial Distribution of Dose."

In the first paragraph of this section, the authors state that one should use experimental data, "meager as it is," rather than models based upon other organ systems. They indicate that this is "particularly true" when rat skin data are used to infer human lung effects. It is doubtful whether anyone would disagree with this. However, in the case of hot particles,

the experimental data are not only meager, they are very disquieting. Since this is a public health matter of importance and not just an academic exercise, prudence dictates that exposure standards should be based upon supportable and conservative hypotheses.

Pages 35-36. The next few paragraphs discuss the concept of "wasted" radiation as it relates to the hypothesis of linear dose-effect response. When uniform irradiation is employed cancer induction is generally shown to be directly proportional to the dose from low doses up to a few hundred rad. The linear hypothesis relates these observations to cellular effects that result from single-track ionizing events. But even with uniform irradiation as one proceeds to higher dosages the response curve changes; for example, the curve steepens or the effects plateau and often decline. Obviously this indicates that other phenomena are becoming dominant. The hot particle hypothesis relates to such a different phenomenon (an injury-mediated mechanism of carcinogenesis). As such, it is not intended to be consistent with the linear hypothesis.

The mechanism of radiation carcinogenesis is not understood even in the range of the linear hypothesis. This is evident in the next several paragraphs of this section of the BRW Report. Actually much of the discussion here is supportive of an injury-mediated mechanism wherein the altered tissue architecture creates a milieu highly favorable to tumor development; for example, the quote of Mayneord (1968).

Page 36. The discussion of contact inhibition as it related to normal or "transformed" cells is again consistent with the hot particle hypothesis. It is the disturbed tissue architecture that can disrupt the normal contact inhibition. As we mentioned earlier in this critique, the induction of cancer by mylar film and millipore filters in the experiments of Brues, et al., supports such a mechanism.[23]

The paragraph that begins, "Thus, both acute and late..." is purely speculative and is no more supported by the previous discussion than is the hot particle hypothesis.

Pages 36-38. The following ten paragraphs in this section are actually a discussion of an injury-mediated mechanism of carcinogenesis.

Page 38. This is followed by the paragraph,

> At present there is no compelling reason to believe that the critical structure or volume required for radiation-induced promotion of cancer arising from cancer-potential cells of hair follicles is limited to the hair follicle. There is also no cogent evidence that the lung has analagous discrete susceptible architectural units with critical tissue volume as small as the sphere of alpha particle range from an isolated "hot particle."

We would propose that there is also no compelling reason for not believing it and that prudent public health practice dictates that such a critical structure should be assumed in establishing exposure standards for hot particles.

Pages 38-39. The next two paragraphs are speculative and are followed by the paragraph:

23/ Brues, A., et al., op. cit.

> Considering the amount of human data available for carcinogenic risk estimates, and the variability and uncertainty concerning dosimetric factors (e.g., relevant doses, differences in spatial and temporal dose distribution, etc.), it has thus far been regarded as necessary to select single values of quantities that characterize the exposure of an organ or that organ in a group of individuals. Mean accumulated tissue dose is the only criterion that can be used practically at present until adequate knowledge of more relevant criteria becomes available. Furthermore, when the energy is deposited non-uniformly and its influence in the exposed organ or a group of individuals is not known, the non-uniformity cannot be dealt with until more adequate data are available. The linear (proportional) hypothesis is the only one that normally permits the use of mean dose as the significant dose factor for conditions of non-uniform exposure and exposure rate in an organ or among individuals, the purposes of estimating risk or setting dose limits in the absence of adequate data on distribution of dose and dose rates.

While this paragraph may have been offered as an explanation for, or even as an excuse for, the present radiation exposure standards, we fail to see how it justifies the standards in the future. So far as hot particles are concerned, we have submitted a supportable hypothesis to supplant the linear hypothesis in establishing hot particle exposure standards. The standards are a practical problem of the moment and should be established on the basis of conservative and supportable hypothesis today. It is irresponsible to leave the health of workers and the public in jeopardy while awaiting more definitive data.

The remaining paragraph is a speculative attempt to set aside the hot particle hypothesis. In this respect, it is interesting to note that this section of the report failed to

recant the observations of Lushbaugh and Langham (1962) wherein a 'precancerous' lesion was induced in the palm of a mechanic by a single plutonium hot particle. Nor did it discuss the observations of Richmond, et al., (1970), Richmond and Voelz (1972, 1973) or Richmond and Sullivan (1974) that similar lesions were induced in the lungs of rats and hamsters by plutonium hot particles. These are observations, not speculation, and they support the hot particle hypothesis.

Pages 39-40. "C. Assessment of Experimental Animal Data."

This section begins with a discussion of a probit transformation of experimental data on animals relating lung cancer and radiation dosage to which the authors correctly ascribe no statistical validity. Nevertheless, so far as the Pu or other alpha data are concerned there is little that is related to hot particles and that which is, such as the beagle data (Park and Bair, 1972), represents a saturated response. The Pu-238 experiments of Sanders (1972) also demonstrate a saturated response at a level of 40 rad or 400 rem. Moreover, Sanders indicates that Pu was soluble in his experiment.

In the second paragraph they indicate that these plots demonstrate a RBE of about 10 for alpha radiation in accord with radiobiological experience. In the third paragraph, they make an assumption concerning the non-uniform distribution of the alpha irradiation and transpose the alpha curve in accord with this assumption. Considering the nature of the alpha experiments (their particle size, solubility, and saturation effects) there is no justification for this assumption and transformation. For example, Sanders states that his irradiation was uniform.

We see little merit to this entire discussion and the conclusions in the 5th and 6th paragraphs that result from it are entirely unjustified.

Page 41. The final 5 paragraphs in the BRW Report discuss a number of animal experiments that supposedly are contrary to the hot particle hypothesis. The first involves the results of Laskin, et al., (1963) wherein Ru-106 pellets were implanted in the bronchi of rats. The results indicated a tumor incidence of 7.3% in animals exposed to a few thousand rads with the incidence rising to 66% in those exposed to 10 rads. This dose was calculated as that delivered to the basal layer of the epithelium. One can readily show that this experiment is consistent with the hot particle hypothesis.

The pellets were some 5000 μ in length. They would therefore be expected to produce lesions larger than the 200 to 300 μ lesions observed around hot particles. The result demonstrated a 7% tumor incidence in the 10^3 rad range with one tumor occurring in an animal exposed to 1400 rad. Thus, the cancer risk associated with this much larger lesion at a dose of some 1000 rad was roughly 1/10 or some 200 times greater than that which we assigned to the smaller lesion around a hot particle. This is entirely consistent with the hot particle hypothesis including the 1000 rem/year activity limit. Moreover, the incidence rose to 66% at higher dosage. The data of Richmond and Voelz (1972, 1973) and Richmond and Sullivan (1974) with Pu microspheres demonstrated that these lesions

develop more rapidly as the particle activity is increased. This suggests that if a sufficient induction period were allowed, the incidence for the large pellet-produced lesion could be unity. Again, this is consistent with the hot particle hypothesis.

The remaining experiments discussed here involved Co-60 implants in a variety of animal species (Warren and Gates, 1968) and whole body x-irradiation of rats (Koletsky and Gustafson, 1955, and Castaneva, et al., 1968). Concerning these experiments, the BRW Report authors state:

> Data in figure V-4 for five species of animals given ^{60}Co wire implanted in their lungs show lung tumor incidences ranging from about 8 to 40%, in all but one instance, for total doses of 10^5-10^6 rad to either the entire lung or to the esophagus. It is of interest that the entire lung is irradiated, including any and all possible "critical architectural units," at high dose rates, yet the tumor incidence is not unity. Also of interest is the similar response shown for the several species used with the possible exception of the rat lung, the highest cancer incidence point. The observation of tumor incidences well below unity is true also for the whole-body exposures to X-irradiation in which the entire lungs and body of rats received doses near 10^3 rad.

All of these experiments involved whole body exposure at fairly high dosage. These exposures elicited a generalized carcinogenic response and a significant life shortening effect. Since lung cancer was competing with this overall response, it is incredible that the authors of the BRW Report expected the lung cancer incidence could have reached 100%.

In the Co-60 experiments, the life shortening effect amounted to 80% in all strains and species except for rabbits

which died earlier. At the same time, 33% of the animals developed cancer in one or more of the three tissues studied: lung, bone, and esophagus. If all tissues had been studied the cancer incidence would have been higher. Nevertheless, in the rat, lung cancer had a competitive edge and reached an incidence of 75%. In the X-ray study of Koletsky and Gustafson (1955) the life shortening approached 50% and the incidence of malignant neoplasms was 35% at a whole body dosage of 660 rad. In the control group the incidence was 8%. The Castaneva, et al., (1968) results showed a malignant tumor incidence of 100% and a 20% life shortening even at a dosage of 430 rad. The control rats in these experiments had a 30% malignant tumor incidence. These experiments are typical of many such experiments and show the overall response to whole body radiation. The relationship to the hot particle problem, if any, is obscure and remote. There is no a priori reason to believe that the same carcinogenic mechanism is involved.

V. Summary and Conclusion

The Tamplin-Cochran Report presented a hot particle hypothesis based on an injury-mediated mechanism of carcinogenic response. In order to assist in setting radiation protection standards we proposed quantative values for 1) the minimum activity defining a hot particle and 2) the carcinogenic risk per hot particle. The "hot particle hypothesis" is relatively simple. With respect to alpha-emitting particles in the lung, it is:

> If a particle deposited in the deep respiratory tissue is of such activity as to expose the surrounding lung tissue to a dose of at least 1000 rem in 1 year, this particle represents a unique carcinogenic risk. The biological data suggest that such a particle may have a cancer risk equal to 1/2000.

The BRW Report has been offered as a refutation of the hot particle hypothesis quantitatively presented in the Tamplin-Cochran Report. The BRW Report cites numerous experimental studies, most of which are not relevant to the hot particle issue. Those which are relevant we have shown to be consistant with our hot particle hypothesis. Thus, the BRW Report is not in any way a refutation of the hot particle hypothesis.

While it must be recognized that there are uncertainties with respect to the quantitative values we have chosen, until those uncertainties can be resolved by appropriate experimental data, it is incumbant upon the AEC and EPA to adopt radiation protection standards comparable to those in the Tamplin-Cochran Report. Furthermore, we submit that these more restrictive standards should be quickly promulgated because it is irresponsible to leave the health of the public and workers in jeopardy while awaiting more definitive data.

VI. BIBLIOGRAPHY

Albert, R. E., (1962), "The Tumorigenic Action of Beta Radiation on the Rat Skin," Progress Report TID-15118.

Albert, R. E., Burns, F. J., and Bennett, P., (1972), "Radiation-Induced Hair-Follicle Damage and Tumor Formation in Mouse and Rat Skin," J. Nat. Cancer Inst. *49:* 1131.

Albert, R. E., Burns, F. J., and Heimbach, R. D., (1967a), "The Effect of Penetration Depth of Electron Radiation on Skin Tumor Formation in the Rat," Radiat. Res. *30:* 515.

Albert, R. E., Burns, F. J., and Heimbach, R. D., (1967b), "Skin Damage and Tumor Formation from Grid and Sieve Patterns of Electron and Beta Radiation in the Rat," Radiat. Res. *30:* 525.

Albert, R. E., Burns, F. J., and Heimbach, R. D., (1967c), "The Association Between Chronic Radiation Damage of the Hair Follicles and Tumor Formation in the Rat," Radiat. Res. *30:* 590.

Albert, R. E., Newman, W., and Altshuler, B., (1961), "The Dose-Response Relationships of Beta-Ray-Induced Skin Tumors in the Rat," Radiat. Res. *15:* 410.

Albert, R. E., Phillips, M. E., Bennett, P., Burns, F., and Heimbach, R., (1969), "The Morphology and Growth Characteristics of Radiation-Induced Epithelial Skin Tumors in the Rat," Cancer Res *29:* 658.

Archer, V. E. and Lundin, F. E., (1967), "Radiogenic Lung Cancer in Man: Exposure—Effect Relationship," Environ. Res. *1:* 370.

Bair, W. J., (1960), "Radioisotope Toxicity: from Pulmonary Absorption," In: Radioisotopes in the Biosphere (R. S. Caldecott and L. A. Snyder, eds.), 431, publ. by the Center for Continuation Study, University of Minnesota, Minneapolis.

Bair, W. J., (1974), "Toxicology of Plutonium," In: Advances in Radiation Biology, Volume 4, (J. T. Lett, H. Adler and M. Zelle, eds.), Academic Press.

Bair, W. J., (in press), "Considerations in Assessing the Potential Harm to Populations Exposed to Low Levels of Plutonium in Air," In: Seminar on Radiological Safety Evaluation of Population Doses and Application of Radiological Safety Standards to Man and the Environment (L. A. Self, ed.), IAEA-SM-184/14, Portoroz, Yugoslavia.

Bair, W. J., Ballou, J. E., Park, J. F., and Sanders, C. L., (1973), "Plutonium in Soft Tissues with Emphasis on the Respiratory Tract," In: Handbook of Experimental Pharmacology (H. C. Hodge, J. N. Stannard and J. B. Hursh, eds.), Springer-Verlag, Berlin.

Bair, W. J. and McClanahan, B. J., (1961), "Plutonium Inhalation Studies II. Excretion and Translocation of Inhaled $^{239}PuO_2$ Dust," Arch. Environ. Health *2:* 648.

Bair, W. J., Wiggins, A. D., and Temple, L. A., (1962), "The Effect of Inhaled $Pu^{239}O_2$ on the Life Span of Mice," Health Phys. *8:* 659.

Bair, W. J. and Willard, D. H., (1961), "Plutonium Inhalation Studies III. Effect of Particle Size and Total Dose on Retention and Translocation," HW-68645.

Ballou, J. E. and Park, J. F., (1972), "Disposition of Ingested, Injected and Inhaled ^{239}Pu Citrate and ^{239}Pu Nitrate," Pacific Northwest Laboratory Annual Report for 1971 to the USAEC, Division of Biology and Medicine, BNWL-1650, Vol. 1, part 1: 146.

Bennett, B. G., (1974), "Fallout ^{239}Pu Dose to Man," In: Fallout Program Quarterly Summary Report, Health and Safety Laboratory, U. S. Atomic Energy Commission Report HASL-278.

Brooks, A. L., Mewhinney, J. A., and McClellan, R. O., (1972), "The *In Vivo* Cytogenetic Effects of ^{252}Cf in Liver and Bone Marrow of the Chinese Hamster," Health Phys. *22:* 701.

Brooks, A. L., Retherford, J. C., and McClellan, R. O., (1974), "The Effect of $^{239}PuO_2$ Particle Number and Size on the Frequency and Distribution of Chromosome Aberrations in the Liver of the Chinese Hamster," Radiat. Res. (in press).

Brandon, W. F., Bistline, R. W., Bloom, A. D., and Archer, P. G., (1973), "Preliminary Findings: Chromosomes of Plutonium Workers," Mutat. Res. *21:* 211 (abstract).

Buldakov, L. A., Lyubchansky, E. R., Moskalev, Y. I., and Nifatov, A. P., (1969), In "Problemy Toksikologii Plutoniya," Atom Publications, Moscow (translated as "Problems of Plutonium Toxicology," U. S. Atomic Energy Commission LF-tr-41, 1970).

Buldakov, L. A. and Lyubchansky, E. R., (1970), "Experimental Basis for Maximum Allowable Load (MAL) and Pu-239 in the Human Organism, and Maximum Allowable Concentration (MAC) of Pu-239 in Air at Work Locations," Original source unknown, Report ANL-trans-864, Argonne National Laboratory, Argonne, Illinois, (Translated from a Russian Report).

Burk, R. R., (1966), "Growth Inhibitor of Hamster Fibroblast Cells," Nature *212:* 1261.

Burnet, Sir M. B., (1964), "Immunological Factors in the Process of Carcinogenesis," Brit. Med. Bull. *20:* 154.

Burns, F. J., Albert, R. E., Bennett, P., and Sinclair, I. P., (1972), "Tumor Incidence in Rat Skin After Proton Irradiation in a Sieve Pattern," Radiat. Res. *50:* 181.

Burns, F. J., Albert, R. E., and Heimbach, R. D., (1968), "The RBE for Skin Tumors and Hair Follicle Damage in the Rat Following Irradiation with Alpha Particles and Electrons," Radiat. Res. *36:* 225.

Burns, F. J., Albert, R. E., Sinclair, I. P. and Bennett, P., (1973b), "The Effect of Fractionation on Tumor Induction and Hair Follicle Damage in Rat Skin," Radiat. Res. *53:* 235.

Burns, F. J., Sinclair, I. P., and Albert, R. E., (1973a), "The Oncogenic Response of Anagen Phase Rat Skin to Electron Radiation of Various Penetrations," Proc. Amer. Ass. Cancer Res. *14:* 88 (abstract).

Burrows, H. and Horning, E., (1953), "Oestrogens and Neoplasia," Blackwell, Oxford.

Campbell, E. E., Milligan, M. F., Moss, W. D., Schulte, H. F., and McInroy, J. F., (1973), "Plutonium in Autopsy Tissue," Los Alamos Scientific Laboratory Report LA-4875.

Carrano, A. V. and Heddle, J. A., (1974), "The Fate of Chromosome Aberrations," J. Theor. Biol. (in press).

Casarett, G. W., (1965), "Experimental Radiation Carcinogenesis," Progr. Exp. Tumor Res. *7:* 49.

Casarett, G. W., (1973a), "Pathogenesis of Radionuclide-Induced Tumors," In: Radionuclide Carcinogenesis (C. L. Sanders, R. H. Busch, J. E. Ballou, and D. D. Mahlum, eds.), CONF-720505: 1, AEC Symposium Series 29, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Virginia.

Casarett, G. W., (1973b), "Possible Effects of Relatively Low Levels of Radiation," Current Problems in Radiology *3:* 1.

Casarett, L. J. and Milley, P. S., (1964), "Alveolar Reactivity Following Inhalation of Particles," Health Phys. *10:* 1003.

Castanera, T. J., Jones, D. C., Kimeldorf, D. J. and Rosen, V. J., (1968), "The Influence of Whole-Body Exposure to X-Rays or Neutrons on the Life-Span Distribution of Tumors Among Male Rats," Cancer Res. *28:* 170.

Cember, H., (1963), "Further Studies on Lung Cancers from ^{144}CeF$_3$," Health Phys. *9:* 539.

Cember, H., (1964a), "Radiogenic Lung Cancer," In: Progr. Exp. Tumor Res. Vol. 4 (F. Hamburger, ed.), 251, Hafner Publishing Co., Inc., New York.

Cember, H., (1964b), "Empirical Establishment of Cancer-Associated Dose to the Lung from ^{144}Ce," Health Phys. *10:* 1177.

Cember, H., Hatch, R. F., Watson, J. A., and Grucci, T. B., (1955), "Pulmonary Effects from Radioactive Barium Sulfate Dust," AMA Arch. Indust. Health *12:* 628.

Cember, H. and Stemmer, K., (1964), "Lung Cancer from Radioactive Cerium Chloride," Health Phys. *10:* 43.

Cember, H. and Watson, J. A., (1958a), "Carcinogenic Effects of ^{90}Sr Beads Implanted in the Lungs of Rats," American Ind. Hyg. J. *19:* 36.

Cember, H. and Watson, J. A., (1958b), "Bronchogenic Carcinoma from Radioactive Barium Sulfate," AMA Arch. Ind. Health *17:* 230.

Cember, H., Watson, J. A., and Spritzer, A. A., (1959), "Bronchogenic Carcinoma from Radioactive Cerium Fluoride," AMA Arch. Indust. Health *19:* 14.

Clarke, W. J. and Bair, W. J., (1964), "Plutonium Inhalation Studies: VI. Pathologic Effects of Inhaled Plutonium Particles in Dogs," Health Phys. *10:* 391.

Clarke, W. J., Park, J. F., and Bair, W. J., (1966), "Plutonium Particle-Induced Neoplasia of the Canine Lung. II. Histopathology and Conclusions," 345, In: Lung Tumours in Animals, (Lucio Severi, ed.), University of Perugia, Perugia, Italy.

Coleman, J. R. and Perez, L. J., Jr., (1969), "Consideration of a Tumor Probability Function and Micro-Dosimetry for the Lower Pulmonary Compartment," Nuclear Utility Services Report NUS-654, Rockville, Maryland.

Davies, C. N., (1961), "A Formalized Anatomy of the Human Respiratory Tract," In: Inhaled Particles and Vapours, (C. N. Davies, ed.), 82, Pergamon Press, London.

Dean, P. N. and Langham, W. H., (1969), "Tumorigenicity of Small Highly Radioactive Particles," Health Phys. *16:* 79.

Dolphin, G. W., (1971), "Biological Problems in the Radiological Protection of Workers Exposed to ^{239}Pu," Health Phys. *20:* 549.

Erokhin, R. A., Koshurnikova, N. A., Lemberg, V. K., Nifatov, A. P., and Puzyrev, A. A., (1971), In: "Remote Aftereffects of Radiation Damage," (Y. I. Moskalev, ed.), 315, Atomizdat, Moscow (Translated in AEC-tr-7387, 344).

Ettinger, H. J., Elder, J. C., and Gonzales, M., (1973), "Performance of Multiple HEPA Filters Against Plutonium Aerosols," Los Alamos Scientific Laboratory Report LA-5349-PR.

Geesaman, D. P., (1968), "An Analysis of the Carcinogenic Risk from an Insoluble Alpha-Emitting Aerosol Deposited in Deep Respiratory Tissue," USAEC Documents UCRL-50387 and UCRL-50387 Addendum.

Gillette, R., (1974), "Plutonium and the 'Hot Particle Problem': Environmental Group Proposes a Draconian Answer," Science *183:* 834.

Grossman, B. N., Little, J. B., and O'Toole, W. F., (1971), "Role of Carrier Particles in the Induction of Bronchial Cancer in Hamsters by Polonium-210 Alpha Radiation," Radiat. Res. *47:* 253 (abstract).

Harley, N. H. and Pasternack, B. S., (in press), "Experimental Absorption Applied to Lung Dose from Plutonium-239," Health Phys.

Healy, J. W., (1974), "Contamination Limits for Real and Personal Property," Los Alamos Scientific Laboratory Report No. LA-5482-PR.

Heimbach, R. D., Burns, F. J., and Albert, R. E., (1969), "An Evaluation by Alpha Particle Bragg Peak Radiation of the Critical Depth in the Rat Skin for Tumor Induction," Radiat. Res. *39:* 332.

Hempelmann, L. H., Langham, W. H., Richmond, C. R., and Voelz, G. L., (1973a), "Manhattan Project Plutonium Workers: A Twenty-Seven Year Follow-up Study of Selected Cases," Health Phys. *25:* 461; see also Los Alamos Scientific Laboratory report LA-5148-MS (1973).

Hempelmann, L. H., Richmond, C. R., and Voelz, G. L., (1973b), "A Twenty-Seven Year Study of Selected Los Alamos Plutonium Workers," Los Alamos Scientific Laboratory report LA-5148-MS.

Howard, E. B., (1970), "The Morphology of Experimental Lung Tumors in Beagle Dogs," In: Morphology of Experimental Respiratory Carcinogenesis (P. Nettesheim, M. G. Hanna, Jr., and J. W. Deatherage, Jr., eds.), CONF-700501: 147, AEC Symposium Series No. 21, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Va.

ICRU Report 19, (1971), "Radiation Quantities and Units," International Commission on Radiation Units and Measurements, Washington, D.C.

International Commission on Radiological Protection, (1966), "Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965)," ICRP Publication 9, Pergamon Press, Oxford.

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

Kochetkova, T. A. and Avrunina, G., (1957), "Veranderungen in den Lungen und in anderen Organen bec intratrachealev Einfuhrung einiger Radioisotope," Arch. Gewerbepathol. Gewerbehy. *16:* 24.

Kochetkova, T. A., Avrunina, G. A., and Sagaidak-Chernyak, N. D., (1963), "Experimental Lung Cancer Induced with Radioactive Compounds P^{32}, Au^{198} and Fe^{59}," Acta Unio. Intern. Contra Cancrum *19:* 684. (Cited in Morken, D. A., (1955), University of Rochester Report UR-379).

Koletsky, S. and Gustafson, G. E., (1955), "Whole-Body Radiation as Carcinogenic Agent," Cancer Res. *15:* 100.

Koshurnikova, N. A., Lemberg, V. K., and Lyubchansky, E. R., (1971), In: "Remote Aftereffects of Radiation Damage," (Y. I. Moskalev, ed.), 305, Atomizdat, Moscow. (Translated in AEC-tr-7387, 334).

Koshurnikova, N. A., Lemberg, V. K., Nifatov, A. P., and Puzyrev, A. A., (1968), "Pneumosclerosis in Rat Following Intratracheal Introduction of Soluble Pu^{239}Compounds," Gig. Tr. Prof. Zabol. *11:* 27.

Kurshakova, N. N. and Ivanov, A. E., (1962), "A Model of Experimental Lung Cancer Caused by Intratracheal Introduction of Radioactive Cerium," Bull. Exp. Biol. Med. *54:* 787.

Lafuma, J., (1974), "Les Radioelements Inhales," Seminaire de Radiobiologie sur le théme de "La Contamination Radioactive Interne," 18, 19 et 20 Mars 1974-Paris-organisé par la Societé Francaise de Radioprotection.

Lagerquist, C. R., Hammond, S. E., and Hylton, D. B., (1972), "Distribution of Plutonium and Americium in the Body 5 Years after an Exposure via Contaminated Puncture Wound," Health Phys. *22:* 921.

Langham, W. H., (1957), "The Application of Excretion Analyses to the Determination of Body Burden of Radioactive Isotopes," Brit. J. Radiol. Suppl. 7. Part V, 95.

Laskin, S., Kuschner, M., Altschuler, B., and Nelson, N., (1964), "Tissue Reactions and Dose Relationships in Rats Following Intrapulmonary Beta Radiation," Health Phys. *10:* 1229.

Laskin, S., Kuschner, M., Nelson, N., and Altschuler, B., (1963), "Carcinoma of the Lung in Rats Exposed to the Beta Radiation of Intrabronchial Ruthenium-106 Pellets," J. Nat. Cancer Inst. *31:* 219.

Lebel, J. L., Bull, E. M., Johnson, L. J., and Watters, R. L., (1970), "Lymphosarcoma Associated with Nodal Concentration of Plutonium in Dogs: A Preliminary Report," J. of Vet. Res. *31:* 1513.

Lebel, J. L., Watters, R. L., Bistline, R. W., Dagle, G. E., and Gomez, L. S., (1972), "Fourth Technical Progress Report of the Department of Radiology and Radiation Biology," COO-1787-16, Colorado State University, Fort Collins.

Lisco, H., (1959), "Autoradiographic and Histopathologic Studies in Radiation Carcinogenesis of the Lung," Lab. Invest. *8:* 162.

Little, J. B., Grossman, B. N., and O'Toole, W. F., (1970a), "Respiratory Carcinogenesis in Hamsters Induced by Polonium-210 Alpha Radiation and Benzo-(α)Pyrene," In: Morphology of Experimental Respiratory Carcinogenesis, (P. Nettesheim, M. G. Hanna, Jr., and J. W. Deatherage, Jr., eds.), CONF-700501: 383, AEC Symposium Series No. 21, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Va.

Little, J. B., Grossman, B. N., and O'Toole, W. F., (1970b), "Induction of Bronchial Cancer in Hamsters by Polonium-210 Alpha Radiation," Radiat. Res. *43:* 261 (abstract).

Little, J. B., Grossman, B. N., and O'Toole, W. F., (1973), "Factors Influencing the Induction of Lung Cancer in Hamsters by Intratracheal Administration of ^{210}Po," In: Radionuclide Carcinogenesis, (C. L. Sanders, R. H. Busch, J. E. Ballou, and D. D. Mahlum, eds.), CONF-720505: 119, AEC Symposium Series No. 29, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Va.

Lundin, F. E., Wagoner, J. K., and Archer, V. E., (1971), "Radon Daughter Exposure and Respiratory Cancer Quantitative and Temporal Aspects," National Institute for Occupational Safety and Health/National Institute for Environmental Health Sciences, Joint Monograph No. 1, Public Health Service, U. S. Dept. of Health, Education and Welfare.

Lushbaugh, C. C., Cloutier, R. J., Humason, G., Langham, J., and Guzak, S., (1967), "Histopatholgic Study of Intradermal Plutonium Metal Deposits: Their Conjectured Fate," Ann. N.Y. Acad. Sci. *145:* 791.

Lushbaugh, C. C. and Langham, J., (1962), "A Dermal Lesion from Implanted Plutonium," Arch. Dermatol. *86:* 461.

Mann, J. R. and Kirchner, R. A., (1967), "Evaluation of Lung Burden Following Acute Inhalation Exposure to Highly Insoluble PuO_2," Health Phys. *13:* 877.

Marshall, J. H. and Finkel, M. P., (1959), "Autoradiographic Dosimetry of Mouse Bones Containing Ca^{45}, Sr^{90}, and Ra^{226}," Argonne National Laboratory Report ANL-6104: 48.

Marshall, J. H. and Finkel, M. P., (1960), "Autoradiographic Dosimetry of Mouse Bones Containing Ca^{45},

Sr^{90}, and Ra^{226}, II. The Sensitive Region in the Induction of Osteogenic Sarcomas," Argonne National Laboratory Report ANL-6199: 44.

Mayneord, W. V., (1968), "Radiation Carcinogenesis," Brit. J. Radiol. *41:* 241.

Mayneord, W. V. and Clarke, R. H., (1974), "A Mathematical Investigation Into the Carcinogenic Risks Associated with Particulate Sources of Activity," Central Electricity Generating Board, RD/B/N2878, London, England.

McKay, L. R., Brooks, A. L., and McClellan, R. O., (1972), "The Retention, Distribution, Dose and Cytogenetic Effects of ^{241}Am Citrate in the Chinese Hamster," Health Phys. *22:* 633.

McMurtrie, G. E. (Secretary), (1950), "Permissible Doses Conference held at Chalk River, Ontario (Sept., 1949)," Report RH-10, May.

Mercer, T. T., (1967), "On the Role of Particle Size in the Dissolution of Lung Burdens," Health Phys. *13:* 1211.

Metivier, H., Nolibe, D., Masse, R., and Lafuma, J., (1972), "Cancers Provoquoes chez le Singe Babouin (Papio papio) par Inhalation de PuO_2," C. R. Acad. Sci. (Paris), *275:* 3069 (translated by Howath, A. A., LF-tr-80).

Morrow, P. E., Bates, D. V., Fish, B. R., Hatch, T. F., and Mercer, T. T., (1966), "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract," Health Phys. *12:* 173.

Morrow, P. E. and Casarett, L. J., (1961), "An Experimental Study of the Deposition and Retention of a Plutonium-239 Dioxide Aerosol," In: Inhaled Particles and Vapors, (C. N. Davies, ed.), Pergamon Press, London.

Moskalev, Y. I., (1972), "^{239}Pu: Problems of its Biological Effect," Health Phys. *22:* 723.

Moss, W. D., Hyatt, E. C., and Schulte, H. F., (1961), "Particle Size Studies on Plutonium Aerosols," Health Phys. *5:* 212.

NAS-NRC Subcommittee on Inhalation Hazards, Committee on Pathologic Effects of Atomic Radiation, (1961a), "Effects of Inhaled Radioactive Particles," Publication 848, U. S. National Academy of Sciences-National Research Council, Washington, D.C.

NAS-NRC Subcommittee on Internal Emitters, Committee on Pathological Effects of Atomic Radiation, (1961b), "Internal Emitters," Publication 883, U. S. National Academy of Sciences-National Research Council, Washington, D.C.

NAS-NRC Report of the Advisory Committee on the Biological Effects of Ionizing Radiations, (1972) "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," National Academy of Sciences-National Research Council, Washington, D.C.

National Council on Radiation Protection and Measurements, (1971), "Basic Radiation Protection Criteria," NCRP Report No. 39, Washington, D.C.

Nelson, I. C., Heid, K. R., Fuqua, P. A., and Mahony, T. D., (1972), "Plutonium in Autopsy Tissue Samples," Health Phys. *22:* 925.

Norcross, J. A. and Newton, C. E., Jr., (1972), "U.S. Transuranium Registry: A Progress Report," Health Phys. *22*, 887.

Park, J. F. and Bair, W. J., (1972), Personal Communication (referenced in Bair, W. J., 1974).

Park, J. F. and Bair, W. J., (1974), Personal Communication.

Park, J. F., Bair, W. J., and Busch, R. H., (1972), "Progress in Beagle Dog Studies with Transuranium Elements at Battelle-Northwest," Health Phys. *22:* 803.

Park, J. F., Catt, D. L., Dagle, G. E., Hackett, P. L., Olson, R. J., Powers, G. J., and Ragan, H. A., (1974), "Late Effects of Inhaled $^{238}PuO_2$ in Dogs," Pacific Northwest Laboratory Annual Report for 1973 to the USAEC Division of Biomedical and Environmental Research, BNWL-1850, Part I.

Passonneau, J. V., Brues, A. M., Hamilton, K. A., and Kisieleski, W. E., (1952), "Carcinogenic Effects of Diffuse and Point Source Beta Irradiation on Rat Skin: Final Summary," USAEC Document ANL-4932: 31.

Raabe, O. G., In Preparation.

Rashevsky, N., (1948), "Outline of a Mathematical Approach to the Cancer Problem," In: Mathematical Biophysics, University of Chicago Press, Chicago.

Richmond, C. R., Holland, L. M., Drake, G. A., and Wilson, J. S., (1974), "Biological Response to Small Discrete Highly Radioactive Sources, III. Effects of Surgically Implanted $^{238}PuO_2$ and $^{239}PuO_2$ Microspheres in Beagles," Abstract P/127, Presented at the Nineteenth Annual Meeting of the Health Physics Society, Houston, Texas, July 1974.

Richmond, C. R., Langham, J., and Stone, R. S., (1970), "Biological Response to Small Discrete Highly Radioactive Sources, II. Morphogenesis of Microlesions in Rat Lungs from Intravenously Injected $^{238}PuO_2$ Microspheres," Health Phys. *18:* 401.

Richmond, C. R. and Sullivan, E. M., (eds.), (1974), Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division for 1973 to the USAEC, Division of Biomedical and Environmental Research, Los Alamos Scientific Laboratory Report LA-5633-PR.

Richmond, C. R. and Voelz, G. L., (eds.), (1972), Annual Report of the Biological and Medical Research Group (H-4) to the USAEC, Division of Biology and Medicine, Los Alamos Scientific Laboratory Report LA-4923-PR.

Richmond, C. R. and Voelz, G. L., (eds.), (1973), Annual Report of the Biological and Medical Research Group (H-4) of the LASL Health Division for 1972 to the USAEC, Division of Biomedical and Environmental Research, Los Alamos Scientific Laboratory Report LA-5227-PR.

Rossi, H. H., (1967), "Energy Distribution in the Absorption of Radiation," Adv. in Biol. and Med. Res. *11:* 27, Academic Press, New York.

Sanders, C. L., (1969), "The Distribution of Inhaled Plutonium-239 Dioxide Particles Within Pulmonary Macrophages," Arch. Environ. Health *18:* 904.

Sanders, C. L., (1972), "Deposition Patterns and the Toxicity of Transuranium Elements in Lung," Health Phys. *22:* 607.

Sanders, C. L., (1973), "Carcinogenicity of Inhaled Plutonium-238 in the Rat," Radiat. Res. *56:* 540.

Sanders, C. L., (1974), "Clearance of PuO_2 from Mediastinal Lymph Nodes," Pacific Northwest Laboratory Annual Report for 1973 to the USAEC, Division of Biomedical and Environmental Research, BNWL-1850, Part I.

Sanders, C. L., (in press), "Effects of PuO_2 Particles Deposited in the Lung Following Intraperitoneal Injection," Health Phys.

Sanders, C. L. and Adee, R. R., (1968), "Phagocytosis of Inhaled Plutonium Oxide—^{239}Pu Particles by Pulmonary Macrophages," Science *162:* 918.

Sanders, C. L. and Adee, R. R., (1970), "Ultrastructural Localization of Inhaled $^{239}PuO_2$ Particles in Alveolar Epithelium and Macrophages," Health Phys. *18:* 293.

Sanders, C. L., Adee, R. R., and Jackson, T. A., (1971), "Fine Structure of Alveolar Areas in the Lung Following Inhalation of $^{239}PuO_2$ Particles," Arch. Environ. Health *22:* 525.

Sanders, C. L. and Dionne, P. N., (1970), "Distribution of Alpha Energy in Alveoli Following Deposition of $^{239}PuO_2$ Particles," Pacific Northwest Laboratory Annual Report for 1968 to the USAEC, Division of Biology and Medicine, BNWL-1050, Part 1: 3.40.

Sanders, C. L. and Park, J. F., (1972), "Pulmonary Distribution of Alpha Dose from $^{239}PuO_2$ and Induction of Neoplasia in Rats and Dogs," In: Inhaled Particles, (C. N. Davies, ed.), Vol. III: 489, Pergamon Press, London.

Sanders, C. L., Thompson, R. C., and Bair, W. J., (1970), "Lung Cancer: Dose Response Studies with Radionuclides," In: Inhalation Carcinogenesis, (M. G. Hanna, Jr., P. Nettesheim, and J. R. Gilbert, eds.), CONF-691001: 285, AEC Symposium Series No. 18, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Va.

Schubert, J. J., Fried, F., Rosenthal, M. W., and Lindenbaum, A., (1961), "Tissue Distribution of Monomeric and Polymeric Plutonium as Modified by a Chelating Agent," Radiat. Res. *15:* 220.

Shorter, R. G., (1970), "Cell Kinetics of Respiratory Tissues, Both Normal and Stimulated," In: Morphology of Experimental Respiratory Carcinogenesis, (P. Nettesheim, M. G. Hanna, Jr., and J. W. Deatherage, Jr., eds.), CONF-700501: 45, AEC Symposium Series No. 21, U. S. Atomic Energy Commission, Office of Information Services, Springfield, Va.

Sivak, A. and Van Duuren, B. L., (1970), "A Cell Culture System for the Assessment of Tumor-Promoting Activity," J. Nat. Cancer Inst. *44:* 1091.

Stoker, M., (1964), "Regulation of Growth and Orientation in Hamster Cells Transformed by Polyoma Virus," Virology *24:* 165.

Stoker, M., (1967), "Contact and Short-Range Interactions Affecting Growth of Animal Cells in Culture," Current Topics in Developmental Biology, (A. A. Moscona and A. Mouroy, eds.), Vol. II: 108, Academic Press, New York.

Tamplin, A. R. and Cochran, T. B., (1974), "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," Natural Resources Defense Council report, Washington, D. C.

Temple, L. A., Marks, S., and Bair, W. J., (1960), "Tumors in Mice After Pulmonary Deposition of Radioactive Particles," Int. J. Radiat. Biol. *2:* 143.

Temple, L. A., Willard, D. H., Marks, S., and Bair, W. J., (1959), "Induction of Lung Tumors by Radioactive particles," Nature *183:* 408.

Thomas, J. M. and Bair, W. J., (submitted for publication), "An Analysis of Experimental Data on Carcinogenic Effects of Inhaled Radionuclides."

Thomas, R. L., Scott, J. K., and Chiffelle, T. L., (1972), "Metabolism and Toxicity of Inhaled ^{144}Ce in Rats," Radiat. Res. *49:* 589.

Wager, R. W., Dockum, N. L., Temple, L. A., and Willard, D. H., (1956), Biology Research Annual Report for 1955, HW-41500, Hanford Atomic Products Operation, Richland, Washington.

Warren, S. and Gates, O., (1968), "Cancers Induced in Different Species by Continuous Gamma Radiation," Arch. Environ. Health *17:* 697.

Watters, R. L. and Lebel, J. L., (1972), "Progress in the Beagle Studies at Colorado State University," Health Phys. *22:* 811.

Weibel, E. R., (1963), "Morphometry of the Human Lung," Springer-Verlag, Berlin.

Zalmanzon, Y. E. and Chutkin, O. A., (1971), "Radiation Absorbed Dose in the Lungs from Radioactive Aerosols," translated from the Russian by The Ralph McElroy Co., Custom Division, 2102 Rio Grande Street, Austin, Texas 78705.

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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

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)	$4x10^{-11}$ 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

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.

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	Tissue Irradiated[25]	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., 1968.

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., GT-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, $\sim$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. 525-540

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.

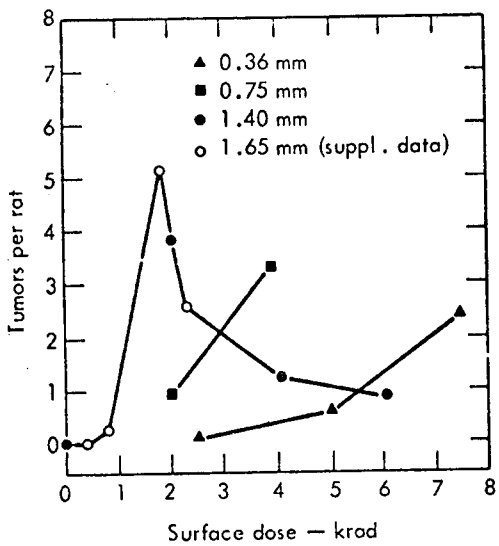

Fig. 1. Tumor incidence with respect to surface dose at 80 weeks for three penetration depths of electrons.

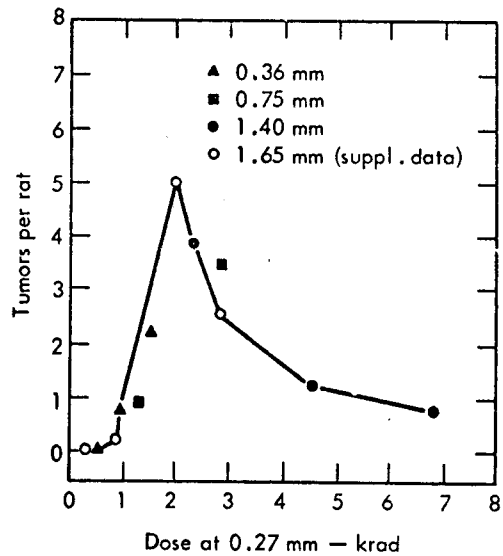

Fig. 2. Tumor incidence with respect to the dose at a depth of 0.27 mm in the skin at 80 weeks for three penetration depths of electrons.

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, 1956,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, Herman, 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/ Vanderbeck, 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. Homburger, 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. 1. 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. 30, Op. cit., pp. 515-524, Figure 7; reproduced in Geesaman, 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.R. and A.R. Kirchnev, 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 68% 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-50387, 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-MRC 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 MPC_a for non-occupational exposure to Pu-239 should also be reduced by a factor of 115,000 to a value of $9x10^{-18}$ 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.[81] 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 following 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.

81/ 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. 128. 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.

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[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.[82]

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

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82/ 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 $9x10^{-8}$ 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 $9x10^{-18}$ uCi/ml which corresponds to $9x10^{-12}$ uCi/m^3. The maximum ground contamination level, using RF=10^{-4} m^{-1}, is $9x10^{-12}/10^{-4}$ = $9x10^{-8}$ 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 transferred 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.

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

Introduction

The draft comments prepared by the Biophysical Society contain a summary by Jane and Richard Setlow, the coordinators of the STAIS Committee on the "Hot Particle Problem," followed by the individual comments of the five committee members. The summary and individual comments are reviewed separately below.

Summary Comments by Jane and Richard Setlow

The summary prepared by Drs. Jane and Richard Setlow indicates the hot particle problem is a valid and serious one. Two of the reviewers felt that the standards should be made more restrictive. There was only one reviewer who stated that there was no reason to change the standard. Except for the latter, most reviewers felt that more information was needed to establish such a standard on a firm basis.

We agree that the available data are not adequate to firmly establish the quantitative parameters in our hypothesis. At the same time, we feel the available data fully support the hypothesis qualitatively. In our report, "Radiation Standards for Hot Particles,"[1] we cited the ICRP and NCRP reservations relative to hot particles. Both this report and "The Hot Particle Issue,"[2] our critique of WASH-1320, present evidence that suggests that hot particles offer a unique carcinogenic risk. This possibility is acknowledged in

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1/ Tamplin, Arthur R. and Thomas B. Cochran, "Radiation Standards for Hot Particles," Natural Resources Defense Council, Washington, D. C. (14 February 1974).

2/ Tamplin, Arthur R. and Thomas B. Cochran, "The Hot Particle Issue: A Critique of WASH 1320 as it Relates to the Hot Particle Hypothesis," Natural Resources Defense Council, Washington, D. C., (November 1974).

the NCRP and ICRP reservations. In other words, there is evidence that suggests that existing exposure standards are not adequate when hot particles are involved. However, to modify these standards, a quantitative estimate of the risk is required. That was the major purpose of our original hot particle report. We petitioned for a modification of the existing standards because there is a present need to protect the workers in -- and the public from -- a rapidly growing plutonium oxide fuel industry. If plutonium were to be banned, like cyclamates, we could await more definitive data, but right now it appears that the nuclear industry is going ahead with its plans.

In attempting to assess our assignment of quantitative values to the hypothesis, it is unfortunate that two of the reviewers[3] attempt to set aside one hypothesis with another. This can only be done with experimental data. Instead of focusing on whether the available data support or contradict our hypothesis, these reviewers proposed totally different hypotheses and estimated quantitative risks based on these different hypotheses. Even if we assume these alternate hypotheses are developed more rigorously and are equally plausible, they still would be only hypotheses and could not be used to set aside ours. Our hypothesis, for which we find support and no contradictory evidence, at present gives a higher risk per particle than the alternate hypotheses. Confronted with different estimates of risk from two or more hypotheses that cannot be set aside, we feel that it would be worthwhile for the committee to express its opinion as to the approach that should be followed in

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3/ Drs. M. L. Randolph and Arthur Cole.

establishing public health standards in such situations. Should the public and workers assume the risk, should the substance be banned, or should the industry be required to develop the technology to reduce the exposure to a level suggested as prudent by the existing but incomplete data? What principle should apply to the practice of public health and safety in such cases?

Comments by M. L. Randolph

A. Overall Views --

We will comment on the conclusions in this section as we review the related material in the subsequent sections.

B. Major Technical Considerations --

Page 2, ¶ 1: Our conviction is found in other experimental evidence detailed in "Radiation Standards for Hot Particles" and "The Hot Particle Issue," our critique of WASH-1320 which we have submitted to the Biophysical Society committee. Perhaps some confusion could have been avoided had we carefully delineated the experimental evidence that supports the hypothesis qualitatively from the evidence used to quantify the hypothesis in order to establish radiation standards.

Page 2, last ¶, beginning 1) (p. 22-26): Albert observed a nearly constant ratio of tumors per atrophied hair follicle in the range (1/2000 to 1/4000). Geesaman, based on the observations of Albert, et al., but allowing for a more liberal margin for error, used somewhat larger (order of magnitude) limits of uncertainty on the tumor risk probability, i.e., 10^{-3} to 10^{-4}. Since we had to quantify this risk in order to recommend a standard, we selected the median between 0.001 and 0.0001, namely 0.0005, or 1/2000.

On the next page (p. 3) Randolph states,

> "I don't understand how the number of hair follicles damaged by large area electron irradiation relates to the volume irradiated by a hot particle . . . One way to estimate the hazard of one micron hot particle would be to assume induction proportional to the volume irradiated."

It is clear that Randolph does not understand our (actually Geesaman's) hypothesis qualitatively, and therefore does not appreciate why the Albert experiments were used to quantify the hypothesis. We will attempt to rephrase the basic hypothesis avoiding the use of the term "critical architectural unit" which may be the source of some confusion.

Qualitatively, the hypothesis is:

> When a critical tissue mass is irradiated at a sufficiently high dose, the probability of tumor production is high.

A corollary to this is:

> When a critical tissue mass in the lung is irradiated by an immobile particle of sufficient alpha activity the probability of a lesion developing approaches unity, and the probability of this lesion developing into a tumor is high.

In order to quantify this hypothesis, we turned to the available biological data to obtain a) the risk of tumor development once the critical tissue structure has been altered through radiation exposure at high doses, and b) the critical particle activity (or local tissue dose) to significantly alter the tissue structure (or with respect to the corollary, produce a lesion in the lung).

There is considerable experimental evidence to support the hypothesis and the corollary qualitatively. However, the only good biological data which quantifies a) the tumor risk per altered tissue structure, is the Albert data. The altered tissue structure

(or "critical architectural unit") in this case is the hair follicle, and the probability of the tumor production once this alteration has taken place is on the order of 1/2000. The same Albert data, and the experiments of Laskin (see discussion on pp. 32-33 of "Radiation Standards for Hot Particles") support the choice of 1000 rem/year to the local tissue as the quantitative value for b) the critical particle activity. The Richmond experiments (see our critique of WASH-1320, pp. 25-29) suggest equivocally that the critical activity may be somewhat higher for particles in the lung. These experiments point out one of the uncertainties in our quantification of the hypothesis.

Randolph's statement on page 3, "one way to estimate the hazard of a one micron hot particle would be to assume tumor induction proportional to the volume irradiated," is a hypothesis clearly distinct from our hypothesis. This "Randolph Hypothesis" is a simplified and probably not a new variation of several tumor production models based on cells at risk. One hypothesis, however, can not set aside another. This can only be done with experimental data and we find no data that are inconsistent with our hypothesis.

In his second major conclusion on page 1 under "A. Overall Views," Randolph questions the relevance of the Albert, et al., comments that no skin tumors were observed with protons, alpha particles, or low energy (0.3 MeV) electrons. These observations simply reflect the fact that these radiation sources did not penetrate the skin sufficiently to disrupt the hair follicle.

Also, on page 1, where Randolph uses the Scottish verdict "not proven," we would substitute "not set aside." This in turn

raises the question: What conservative radiation protection criteria should be adopted to protect the health of worker and the public? We would suggest that the prudent public health principle is to accept the hot particle hypothesis, rather than some less conservative hypothesis, and that our recommended standards provide a reasonable basis for protection.

Page 3, beginning:

2) (p. 25-26): These ^{32}P plaque experiments support our hypothesis qualitatively, namely, a high tumor risk is observed when a small volume of tissue is irradiated at a high dose.

3) (p. 27-28): The Lushbaugh observation lends strong support for the hypothesis qualitatively. While the statistics are obviously poor, this observation is consistent with the quantitative assignment of risk derived from the Albert data and suggests that the hot particle tumor risk that we assigned may even be low.

4) (p. 27-28): While Mr. Gleason's case is equivocal it is also consistent with the hypothesis qualitatively.

5) (p. 30): The work of Laskin, et al., supports the hypothesis qualitatively, and supports the quantitative choice of the minimum critical particle activity as that capable of delivering 1000 rem/year to the tissue at risk (see also p. 39 of our critique of WASH-1320 -- on line 10 of page 39 "10 rads" should read 10^6 rads).

Page 4, beginning:

6) (p. 31-32): With respect to the beagle experiments by Bair, et al., we see no valid basis for assuming "a linear tumor to radioactivity incidence," below 100% incidence at about 0.3 uCi exposure. As we discuss in our hot particle report, the response

in Bair's experiments was saturated and it is impossible to draw any conclusions with respect to lower exposures.

7) (p. 34-37): No one knows the latent period for carcinogenic response to hot particles in human lungs. It could be 20 to 30 years. It is difficult, we submit, to document non-existent information. We did point out (on p. 37 of our hot particle report) however, that in the experiments reported by Park, et al., the beagle with the smallest lung burden (o.2 uCi) developed lung cancer after an 11 year latent period. The highest Rocky Flats worker exposure (9 years ago) is comparable to the lowest beagle exposure.

8) (p. 38-40): We support the suggestion that an attempt be made to reconstruct the Manhattan contamination experiments (sans human lungs) in order to obtain more information about these exposures.

C. Minor Technical Considerations --

1. We find no reference to the activity of a one micron $^{239}PuO_2$ (or $^{238}PuO_2$) particle on page 7. Pu-238 has the 89 year half-life. Langham ["The Problem of Large Area Plutonium Contamination," U. S. Department of HEW, Public Health Services, Seminar Paper No. 002, Dec. 6, 1968, p. 7] lists the activity of $^{238}PuO_2$ and $^{239}PuO_2$ particles as a function of particle diameter. The activity of a one micron $^{238}PuO_2$ particle is given as 8.0×10^{-2} nCi. It is not clear whether these values are measured, or calculated. In either event, assuming Langham's value, and 17.47 curies/gm for Pu-238, the density of Pu-238 in a 1 μ particle of $^{238}PuO_2$ would be slightly less than 10 gm/cm^3.

2. AEC and EPA regulations do include and delineate standards

for both workers and the population. Whether they do so clearly is arguable.

3. DF is defined in "Basic Radiation Protection Criteria," NCRP Report No. 39, p. 84.

4. P. 16. We stand corrected. Assuming some particles in the lung are lodged in areas where they see essentially solid tissue out to 45 μ in any direction, we could have used the soft tissue dose rate value instead of a lung model and obtained $(73 \times 10^4) / (3 \times 10^{-4}) = 2 \times 10^9$, or more than 9 orders of magnitude.

5. The correct number is 57,000. The 53,000 was obtained (with one too many significant digits) from $(16 \times 10^{-9}) / (3 \times 10^{-13})$.

6. P. 24-26. As we noted on p. 5 above, one hypothesis can not be used to set aside another, and we find no data that conflicts with ours.

7. The epithelial cell repair time was used in determining the minimum hot particle activity. This is another of the quantitative uncertainties. The discussion on pp. 25-29 of our critique of WASH-1320 is pertinent to this issue.

8. P. 33. Both the Lushbaugh observation and the Richmond experiments involved dose rates from particles considerably higher than 5000 rem/year. The point is the hot particle can cause severe but highly localized tissue disruption. It is this disrupted tissue mass that we suggest carries a high tumor risk. Above a certain value the total dosage is irrelevant.

9. No comment required.

10. Particle retention and movement is discussed on pp. 9-12 of WASH-1320. On p. 20 of our critique of WASH-1320 we note that

while plutonium particles in the lower respiratory region are not static, auto-radiographic evidence demonstrates that such particles are immobilized in scar tissue and possibly in Type I alveolar epithelial cells. The long residence time of plutonium particles in the lung suggests that such immobilization must occur.

D. Appendix. Tentative Estimate of Maximum Permissible Lung Burden --

Here, Randolph makes a first cut at quantifying a hypothesis based on tumor risk as a function of dose averaged over the entire lung. Referring again back to page 5, one hypothesis can not be used to set aside another hypothesis. We see no data that contradicts our hypothesis. It is interesting to note, however, the quantitative results of Randolph's hypothesis are extremely sensitive to differences in specific activity (^{238}Pu vs. ^{239}Pu). In this regard, had Randolph (at the top of p. 6) noted that a one micron $^{238}PuO_2$ particle gives a dose of 7×10^{-3} rad/year (instead of 2.7×10^{-5} for $^{239}PuO_2$), he would have obtained 2.5 particles versus our 2 particles, instead of 600.

Comments by Louis Hempelmann

Most of the points raised by Hempelmann are discussed in "The Hot Particle Issue," our critique of WASH-1320.

We had no intention of being misleading, nor do we feel that we were so in the three instances cited by Hempelmann.

1. Our report, "Radiation Standards for Hot Particles," was issued on February 14, 1974. We had no way of knowing that in

November, Bair would author the WASH-1320 report. Moreover, our critique of WASH-1320 indicated that the conclusions reached in WASH-1320 are not justified so far as hot particles are concerned.

2. We stated in our hot particle report that the Gleason case was not clear cut. In Appendix B of that report we included the basis for the conclusion that the strong possibility that Gleason's cancer was caused by plutonium. We see no sound reason for concluding that several days after the incident, Mr. Gleason would surely remember having had a sliver puncture in his hand. This would not have had to be a gaping wound as Hempelmann seems to imply.

3. The discussion of the lesion excised by Lushbaugh and Langham indicates that the plutonium particle produced a lesion that was highly suggestive of an incipient carcinogenic response to a single plutonium particle imbedded in soft tissue.

Concerning the assumptions that Hempelmann can not agree with or understand:

1. and 2. The purpose of our report was to develop radiation protection standards for hot particles. To do this it is essential to develop quantitative risk estimates. This work of Albert supplied the only available data on the risk of tumor development as a function of a disordered tissue mass. Rather than make a completely arbitrary assumption concerning the risk per particle we chose the value derived from this biological observation. We pointed out in our critique of WASH-1320 (see pp. 10 and 25-29) that these quantitative values are uncertain. At the same time, it is impossible to set standards without quantitative values.

So far as the architectural structure goes, the lesion of Lushbaugh and Langham and the lesions observed around microspheres

in the lungs of rats and hamsters (see our critique of WASH-1320, pp. 10 and 25-29) suggest that particles placed at random in tissue are capable of inducing a lesion with neoplastic changes similar to precancerous cytological changes.

3. We feel that our discussion of the Rocky Flats workers is valid. It is possible that, if the sample size were as large as the number of exposed uranium miners, some cancers would have appeared already.

4. As we indicated in our hot particle report, the nature of the contaminating events at Los Alamos were described in an article by Hempelmann. The particles (droplets) were aspirated from solutions. As a consequence for the range of concentrations given by Hempelmann the particle size would have had to exceed 5 μ in diameter for the most concentrated solution in order to constitute hot particles. The so-called sophisticated calculations of Anderson relate to particles above 0.6 μ in diameter or a factor of 10 smaller. The reconstruction of these contaminating events, the measurements of the particle size and activity, and the behavior of the inhaled fraction in animals would be a worthwhile experiment.

We have discussed this "good experimental evidence" in our critique of WASH-1320 and have shown this evidence to be either irrelevant to the hot particle hypothesis or supportive of it on the following pages of our critique:

1. Pages 23-25. This experiment was similar to those of Albert wherein the beads corresponded to his sieve patterns.

2. Pages 29-30. These did not involve hot particles.

3. Pages 10 and 25-29. These experiments are supportive.

4. Pages 30-31. Specifically the $BaSO_4$ did not involve hot particles and induced few tumors. The Sr-90 beads (one per animal) induced 7 malignancies in 23 rats. While the dose from the beads (0.3 mm in diameter, 244 nCi) was from a localized source, it irradiated the entire lung. Nevertheless, if one chooses to call these hot particles, the cancer risk was about 1/3 instead of 1/2000 per particle as we propose.

Comments by Andrew M. Rauth

Page 1, ¶ 2: This discussion presents an inadequate synopsis of our report. The discussion under "B. Modifying Factors" (beginning p. 13) in "Radiation Standards for Hot Particles," was meant to serve as background information, namely, a review of basic definitions of radiation dose and factors used to calculate dose. In subsequent sections of the report we estimate a risk per hot particle and the critical particle activity (p. 32). We then recommended radiation standards in terms of hot particle lung burdens which were comparable in risk to the uniform whole body exposure. It is not necessary to go back and calculate what this implies in terms of the Distribution Factor (DF). Calculating the DF is simply an interesting academic exercise. Note that the factor of 115,000 assumes the lung burden consists of hot particles of minimum activity. The DF would be smaller for hot particles with higher activities. Clearly, the concept of DF is not particularly useful if the risk is defined on a per particle, rather than a per microcurie (or per unit dose) basis.

It is inappropriate to say the "DF is based primarily on the two experimental pieces of information, the work of Albert and

coworkers cited on pages 22-24 on electron irradiation of rat skin and the work of Bair on $Pu^{239}O_2$." The basic qualitative support for the hot particle hypothesis derives from a number of experiments wherein small volumes of tissue have been exposed to high doses and where cancer was the almost inevitable result. The experiments by Albert and Bair (and their coworkers) are but two of many such experiments. On the other hand, the quantitative parameters in our hypothesis (as summarized on p. 5 of our critique of WASH-1320) are derived from the Albert experiments but not those by Bair. The quantitative parameters we assigned are supported by other experimental observations, e.g., Laskin, et al., (Reference 56 in our report), Lushbaugh's observation (pp. 27-28 in our report), and the experiments of Richmond, et al., (pp. 25-29 in our critique of WASH-1320).

¶ 3: It is stated that:

> "The authors [Tamplin and Cochran] make no comments on the Albert data on the facts that
>
> 1) This is a microscopic tissue irradiation (24 cm^2) in a single acute dose.
>
> 2) Not only does the tumor incidence go up at 1000 rads, but it also goes down at doses above 2000 rads."

First, it is important to recognize that out of necessity most radiation standards are based on results from acute, as opposed to chronic, exposures. Concerning the second point, at doses above 2000 rads, one is undoubtedly witnessing one or more competing mechanisms. As one moves to higher doses the entire tissue becomes ulcerated and subsequently killed. In the Albert data the ratio of tumor production to atrophied hair follicles persisted to the point where the entire tissue was ablated and there were no hair follicles. It is important to recognize that a single hot particle

can produce this local tissue disruption without being tissue fatal.

The remainder of this paragraph through the first full paragraph on page 2 contains observations with which we agree. We do not share, however, Rauth's conclusions, drawn from what he admits are "superficial considerations."

Comments by Arthur Cole

Concerning Cole's specific points:

> "P. 26. The 'critical architectural unit' is an attractive and simplified hypothesis. However, little evidence is available to support it."

We would suggest there is ample evidence to support this hypothesis qualitatively, although there is much less experimental data from which the hypothesis can be quantified. As demonstrated in our critique of WASH-1320, most of what has been offered as conflicting evidence is not relevant to the hot particle issue. The relevant data support the hypothesis.

> "P. 27-28. The Lushbaugh study of one observed lesion in 1000 puncture wounds provides no statistical basis for estimating a tumor induction probability."

Our hot particle risk estimate was derived from the Albert data. The Lushbaugh observation is consistent with this risk estimate of one tumor per 2000 hot particles. This causal observation by Lushbaugh would suggest that if a comprehensive search had been undertaken other lesions may have been found. While the observed lesion had not progressed into a tumor, the concern that it might was sufficient to have it excised.

> "P. 41. Section VII . . ."

Cole offers two alternative models for estimating the hot particle risk, the first based on Albert's data and the second based on Bair's data. As we have stated previously one hypothesis can not be used to set aside another. This can only be done with experimental data.

It is perhaps worth noting that Cole's first model is similar to the concept of prescribing a significant volume or significant area following NCRP criteria [NCRP Report No. 39, Criteria 206 and 207], as discussed in our critique of WASH-1320 (p. 6).

With respect to the second model Bair's data are not useful for quantifying the hot particle tumor risk, other than establishing a lower limit on the risk per hot particle in the range of 10^{-6} to 10^{-7}. Cole assumed "a reasonable large probability (approaching unity) would still occur for only 10^6 hot particles per dog . . .". Cole could have assumed 10^5, 10^4 . . . and calculated a higher risk. We share Cole's opinion that on the basis of Bair's data alone, the permissible exposure levels should be lowered.

Concerning Cole's final comments, while more experimental work is needed, we submit that our more restrictive standards should be quickly promulgated because it is irresponsible to leave the health of the public and workers in jeopardy while awaiting more definitive data.

Comments by Doris J. Dugas

We are in essential agreement with most of Dugas' comments here. However, they exemplify an approach to this problem that we find quite frustrating, as we discussed in our summary remarks. Standards are required to protect workers and the public from

plutonium exposure. To adopt standards, a quantitative estimate of the risk is required. In the absence of complete data, this risk assessment must be made on the basis of available data.

The only biological observations that we were able to find that allowed an estimate of the risk of cancer given a disrupted tissue mass was the rat skin data. Granted this is uncertain, what is a better value? If the use of plutonium were to be banned pending more definitive data, the public and workers would be protected. But this is apparently not the case. So we selected this approach to quantification because it was an observed biological relationship. As we stated in the summary comments, it would be worthwhile for the committee to propose their approach to this dilemma.

With respect to Dugas' comments on Section V-A, p. 22, the above comments would apply. Moreover, the lesion excised by Lushbaugh and Langham and the lesions and cellular changes observed around microspheres in the lungs of rats suggest that any tissue mass may be the equivalent of a critical volume when hot particles are involved. (See our critique of WASH-1320, pp. 9-10 and 25-29).

The above is also relevant to Dugas' comments on V-B, p. 26 and V-C, p. 29.

As Dugas indicates, VI, p. 32, the epithelial repair time is used to determine the hot particle minimum activity. This is one of the quantitative uncertainties. Our discussion on pages 25-29 of the WASH-1320 critique are pertinent to this issue. If the epithelial turnover time is shorter, the particle activity should be higher.

With respect to Dugas' comments on VI-A, p. 42, the lung seems to be the organ at risk, not the lymph nodes. Although the lymph nodes

I. INTRODUCTION

On February 14, 1974, the Natural Resources Defense Council (NRDC) petitioned the Atomic Energy Commission (AEC) and the Environmental Protection Agency (EPA) to amend their radiation protection standards applicable to "hot particles" of plutonium and other actinides where hot particles were defined more fully in an accompanying report.[1] The report (referred to herein as the Hot Particles Report) concluded that the existing radiation protection standards are grossly inadequate to protect workers and the public from the high cancer risk posed by exposure to the atmospheric release of plutonium particulates from the nuclear power and weapons industries. The report recommended (and the petition requested) that the current standards be made more restrictive by a factor of 115,000 where hot particles are concerned. In the petition NRDC indicated that matters of importance to the public health and safety such as this require prompt action. Allowing a reasonable period for public comment NRDC recommended that the proposed standards be set within six months (by August 14, 1974).

We have requested that EPA hold adjudicatory type hearings on this matter so that the issues could be properly joined.[2-3] Instead EPA held these hearings with a panel format that developed a record which tends to obfuscate the issues. In the first place it is apparent from the transcript of the hearings on December 10 (pages 1-142 to 1-144) that certain members of the hearing panel were not informed as to the purpose of the hearings as detailed in the Federal Register. Moreover, so far as the hot particle issue is concerned,

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1/ Tamplin, A. R. and T. B. Cochran, "Radiation Standards for Hot Particles," Natural Resources Defense Council, Washington, D.C., 14 February, 1974.

2/ Letter from J. G. Speth to Dr. William D. Rowe, dated July 19, 1974.

3/ Letter from J. G. Speth to Dr. William D. Rowe, dated August 19, 1974.

it is evident from the transcript of the hearings of December 10 (pages 1-148 and 1-149) that the EPA had not ascertained that all members of the panel were prepared to discuss this issue. All of the material which we submitted to the AEC as well as the material prepared by the AEC was available to EPA and should have been reviewed by the panel prior to the hearings. In short, we feel these hearings have only served to reenforce the need for the adjudicatory hearing which we requested.

The purpose of this report is to clarify the issues related to the hot particle problem which the hearing record tends to confuse. We shall first discuss the qualitative aspects of hot particle hypothesis and then its quantitative aspects. This will be followed by a discussion of the points raised by Dr. Edward P. Radford, Jr. during the hearings. These discussions will demonstrate that no information capable of rejecting the hot particle hypothesis was presented in the course of the EPA hearing. In fact, together with the hot particle hazard, the recommendations of Dr. Karl Z. Morgan based on a different approach indicate that overall the transuranic standards should be made substantially more restrictive.

II. The Hot Particle Hypothesis

The "hot particle hypothesis" is relatively simple. Qualitatively, the hypothesis is:

> When a critical tissue mass is irradiated at a sufficiently high dose, the probability of tumor production is high.

A corollary to this is:

> When a critical tissue mass in the lung is irradiated by an immobile particle of sufficient alpha activity the probability of a lesion developing approaches unity, and the probability of this lesion developing into a tumor is high.

In order to quantify this hypothesis, we turned to the available biological data to obtain a) the risk of tumor development once the critical tissue structure has been altered through radiation exposure at high doses, and b) the critical particle activity (or local tissue dose) to significantly alter the tissue structure (or with respect to the corollary, produce a lesion in the lung).

In the Hot Particle Report, with respect to alpha-emitting particles in the lung, the hypothesis was quantified on the basis of the available biological data:

> If a particle deposited in the deep respiratory tissue is of such activity as to expose the surrounding lung tissue to a dose of <u>at least</u> 1000 rem in 1 year, this particle represents a unique carcinogenic risk. The biological data suggest that such a particle may have a cancer risk equal to 1/2000.

This hypothesis implies that if a particle exposes the surrounding lung tissue to a dosage greater than 1000 rem in 1 year, the cancer risk is still 1/2000. (This, of course, causes a large particle to be less effective on a per μCi basis, but not on a per particle basis.) The hypothesis implies nothing about particles that expose the tissue to less than 1000 rem in one year.

In the Hot Particle Report we indicated that much of the basic support for the hypothesis derives from a number of experiments wherein in a small volume of tissue was exposed to high dosage. In these experiments cancer was a frequent, almost inevitable, result. One series of experiments that was discussed

in some detail were those conducted by Dr. Roy C. Albert on rat skin.[4-6] In these experiments, Dr. Albert observed that the radiation induced cancers were remarkably correlated with the disruption of an architectural unit of the skin, the hair follicle. The cancers were induced in the rough proportion of 1 cancer per 2000 atrophied hair follicles when the dosages exceeded some 1000 rem.

The hot particle hypothesis thus suggests that if these skin experiments were performed with small particles, each capable of disrupting a single hair follicle, the observed cancer induction would correspond to one cancer per 2000 particles.

A. Qualitative Aspects

In the Hot Particle Report we indicated that there was qualitative support for the hypothesis in terms of two experimental observations related to hot particles embedded in tissue. Since publication of the Hot Particle Report an additional report on hot particles in hamster lungs has been published. We shall discuss each in turn.

The potential hazard of a single hot particle embedded in human tissue is illustrated by the observation of Lushbaugh

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4/ 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.

5/ 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. 525-540.

6/ 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.

and Langham.[7] They excised a nodule that developed around a Pu-239 particle imbedded in the palm of a machinist. 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 . . . .[8]

Considering the above observations, it would be surprising indeed if a physician would not suggest surgical intervention in a case where a patient had a few such imbedded particles. We feel that this lesion alone should cause one to be very cautious in estimating the hazard of hot particles.

That such lesions can develop in lung tissue is supported by the observations of Richmond, et al., on the lesions induced in experiments wherein hot particles were introduced into blood vessels of the lungs of rats:

> Such a lesion with collagenous degeneration and subsequent liquefaction, due to the large local dose of radiation at a high dose rate, has been reported by Lushbaugh et al., whose description of a plutonium lesion found in the dermis is very similar to that observed for plutonium in the lung.[9]

7/ Lushbaugh, C. C. and J. Langham, "A dermal lesion from implanted plutonium," Archives of Dermatology 86, October 1962, pp. 121-124.

8/ Ibid., p. 462.

9/ Richmond, C. R., et al., "Biological response to small discrete highly radioactive sources," Health Physics 18, 1970, p. 406.

Richmond and co-workers continued these experiments with hamsters and the following appears in their latest progress report (Particular attention is drawn to the last sentence):

> Most of the animals placed on study early in the program have reached the end of their normal life span without developing significant pulmonary lesions. During the past few months, we have observed some histological changes in the lungs of very long-term animals (15-20 months). In these animals, an extension of bronchiolar epithelium into the alveolar ducts and alveoli has occurred. In some cases, the alveoli are lined with cubiodal or columnar epithelial cells (Fig. 1). This lesion has been observed almost entirely in the higher activity levels (levels 4-6) and in animals given relatively small numbers of spheres (2000-6000). An interesting recent observation has been given larger numbers of spheres of approximately 60,000. This group of animals has been exposed only about 6 months. A consistent observation of this lesion after drastically different induction times could lead to speculation that the amount of tissue irradiated is an important element in timing of the tumorigenic response. There has been no increase in frank tumors observed within the past year; however, the epithelial changes described above could be considered as precursors of peripheral adenomas.[10]

The particle activity in these hamster experiments was considerably lower than that associated with the excised palmar lesion and the lesions in the rat experiments. The particle activity from the excised palmar lesion was 5 nCi and those in the rats experiment were 40 nCi and greater. The level 4 particles in the hamster experiment contained only 4.3 pCi and level 6 contained 60 pCi. The initial lesions observed surrounding these lower activity particles were called granulomas measuring

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10/ Richmond, C. R. and Sullivan, E. M., (eds.), Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division for 1973, Los Alamos Scientific Laboratory Report LA-5633-PR, May 1974, p.7.

200-500μ in diameter (about the same size as the excised palmar lesion).[11]

It is of importance to compare the description of the lesion in the hamsters wherein there is an extension of the bronchiolar epithelium into the alveoli. This is a suggested mechanism for the histogenesis of bronchiolo-alveolar carcinomas.[12] Moreover, the description of the hamster lesions indicated that, in some cases, the alveoli are lined with cubiodal or columnar epithelial cells. Such lining cells are a histological feature of bronchiolo-alveolar carcinoma.[13] We see no reason for being complacent about these lesions.

These experiments strongly support the proposal that a single particle embedded in tissue is capable of eliciting a carcinogenic response. The killing of cells and the development of a lesion surrounding the particle is the suggested mechanism of carcinogenesis (an injury mediated mechanism). It appears reasonable to propose that the mechanism is similar to that involved in the experiments of Brues et al, wherein sarcomas developed in the fibrous capsule that forms adjacent to a film of plastic and other inert materials, several months after they were implanted subcutaneous in rodents.[14]

11/ Richmond, C. R. and Voelz, G. L.,(eds.), Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division for 1971, Los Alamos Scientific Laboratory Report, LA-4923-PR, April 1972, p. 31.

12/ Evans, Winston R., Histological Appearance of Tumors, Second Edition, Williams and Wilkins Company, Baltimore, Maryland, 1966, pp. 1112-1113.

13/ Ibid, p. 1111.

14/ Brues, A. M., Auerbach, H., De Roche, G.M., and Brube, D., "Mechanisms of carcinogenesis," Argonne National Laboratory, Biological and Medical Research Division Annual Report for 1967, ANL-7409, 1967, pp. 151-155.

The association of lung tumor with peripherally situated scars is discussed in cancer textbooks:

> It is known, for example, that scars in lung tissue marking injuries received years before increase susceptibility of the involved cells to cancer development.[15]

It is reasonable to propose that these lesions disrupt the local tissue architecture and thereby interfere with the normal biochemical and physical communication between the cells that control processes such as contact inhibition which are responsible for maintaining tissue stability. They thus create an area with an increased cancer risk.

While we have here stressed the formation of the lesion surrounding the hot particle, it is important to recognize that many of the cells on the periphery of the lesion are the progeny of cells that received radiation damage during the formation of the lesion. This is implied in the Lushbaugh and Langham quotation on page 5 above. This added effect of radiation damage will be of particular importance for reactor fuels. In this case, the plutonium will be contaminated with beta emitting isotopes that, because of the longer range of beta particles in tissue, will subject the cells surrounding the lesion to appreciable radiation dosage.

Although no tumors appeared in association with the microspheres in the animal experiments, the description of the lesions is suggestive of an incipient tumorogenic response. Richmond, et al, state that they could be considered as precursors of peripheral adenomas and their description is consistent with that of developing

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15/ Cowdry, E. V., Etiology and Prevention Of Cancer In Man, Appleton-Century-Crofts, New York, N.Y., 1968, p. 137.

bronchiolo-alveolar carcinoma. It is reasonable to propose that the induction period for a frank tumor by this mechanism is longer than the life span of rats and hamsters. We submit that the lesions observed around these particles are sufficient to indicate that radiation protection standards should limit the exposure of human lungs to very few hot particles.

B. Quantitative Aspects

The hot particle hypothesis as presented above contains two quantitative parameters. The first is the risk of cancer associated with a particle produced lesion and the second is the particle activity that constitutes a hot particle capable of producing such a lesion. We shall discuss each in turn.

1. Cancer risk per particle produced lesion

In our Hot Particle Report we assumed a cancer risk of 1/2,000 per particle produced lesion. This value was derived from the tumor risk per atrophied hair follicle in the experiments of Albert, *et al.* (see page 4). To our knowledge this is the only biological data that quantitatively relates the radiation induced disruption of a tissue mass to cancer production. As we indicated in our Hot Particle Report, this risk estimate is not necessarily conservative. One could argue that the descriptions of the particle produced lesion cited above suggest a greater risk. We can see no justification for assignment of a lower risk. While we have been criticized for using rat skin data to estimate the risk in human lungs, we have not seen any suggestion for a better approach that is based upon available biological data.

2. The hot particle activity

In our original Hot Particle Report we selected 1,000 rem/year as the local tissue dose for setting the minimum activity for a hot particle. The 1,000 rem was derived from the experiments of Albert, et al, and Laskin, et al., wherein 1,000 rem was the lowest dosage associated with a carcinogenic response. The one year was based upon the apparent epithelial cell turnover time in the lung. This method of defining the minimum activity for a hot particle carried considerable uncertainty and was so criticized.

Since the publication of the Hot Particle Report, three reports have appeared which present experimental data that allow a more direct determination of the minimum particle activity without resorting to tissue dosage or turnover time. We shall discuss these reports beginning with the one that suggests the largest limiting particle activity and ending with the smallest.

a. Richmond and Sullivan.[16] This report is the latest progress report on the microsphere experiments with hamsters at LASL. As the quotation on page 6 above indicates, lesions were observed almost entirely in the activity levels 4 and above. The particles in level 4 contained 4.3 pCi/particle. It is also indicated that lesions were observed in association with particles from level 3 (0.9 pCi/particle). However, this occurred in animals given 60,000 spheres and the lesions may have been associated with clumping on aggregates of particles.

16/ Richmond, E. R. and Sullivan, E. M., op. cit.

This experiment thus suggests a range for the limiting activity of 0.9 - 4.3 pCi/particle with the lower limit somewhat tentative.

These experiments, at this time, represent the only direct observation of particle produced lesion and serve to establish the upper limit for the minimum particle activity. Had the life span of the animals been longer, it is quite possible that lesionswould have developed around particles of lower activity. Thus, the minimum particle activity is most likely below 4.3 pCi/particles. This 4.3 pCi represents a 60 fold increase in the minimum particle activity relative to the 0.07 pCi (based on 1000 rem/year to beal tissue) adopted in the Hot Particle Report.

Nevertheless, a 60 fold increase in activity requires only a 4 fold increase in particle diameter--for Pu-239, a change from 0.6 μ to 2.4 μ; for Pu-238, a change from 0.09 μ to 0.36 μ and for high burn-up nuclear fuel, a change from 0.4 μ to 1.6 μ. These particles are still in the range that permits deposition in the lower respiratory zone. In other words, even when using this upper limit value, the nuclear industry has a potential hot particle problem.

b. McInroy, et al.[17] This report presents a particle size analysis of plutonium particles in a tracheobronchial lymph node of a Los Alamos plutonium worker. Another study of human respiratory exposure to plutonium relates to 25 workers exposed to plutonium at Los Alamos during the Manhattan Project.[18] The

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17/ McInroy, James F., et al., "Studies of Plutonium in Human Tracheobronchial Lymph Nodes," Los Alamos Scientific Laboratory Preprint, LA-UR-741454, 1974.

18/ Hemplemann, L. H., et al, "Manhattan Project Plutonium Workers; A Twenty-Seven Year Follow-Up Study of Selected Cases," Health Physics, Vol. 25, Nov. 1973, pp. 461-479.

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." If these 25 workers combined retained a total of 2,000 hot particles then the chance of none of them developing lung cancer would be about 0.3 (assuming a tumor risk per particle of 1/2,000). Thus, the particle size distribution given by McInroy, et al., can be used to obtain a limiting particle size that would correspond to some 2,000 hot particles retained in the 25 workers.

Healy, et al., estimates that the initial burden in these workers was about 10 μCi.[19] Table I presents the particle size distribution given by McInroy, et al., wherein the incremental activity in a size range was determined by multiplying the incremental activity fraction by the total activity (10^7 pCi). The particle number was then obtained by dividing the incremental activity by the activity per particle.

Inspection of Table I indicates that for these workers to contain only 2,000 particles a minimum activity somewhat larger than 0.8 pCi/particle is required. There is considerable uncertainty attached to this estimate (see discussion in Letter to Mr. Robert B. Minoque attached as Appendix A to this submission). For one thing the autoradiographic sizing technique tends to overestimate the large particle fraction and hence,

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19/ Healy, I. W., et al., "A Review of the Natural Resources Defense Council Petition Concerning Limits for Insoluble Alpha Emitters," Los Alamos Scientific Laboratory Report, LA-5810-MS, Nov. 1974, p.15.

TABLE I

Estimated Particle Size Distribution For
The Manhattan Projects Workers
(Assumes a total lung burden of 10^7 pCi for the 25 workers)

Diameter Particle μ	Incremental Activity Fraction	Incremental Activity	pCi/particle	Number of particles
<0.1	0.12	1.2×10^6	3×10^{-4}	4×10^9
0.1 - 0.3	0.58	5.8×10^6	9×10^{-3}	6.4×10^8
0.3 - 0.5	0.23	2.3×10^6	4.1×10^{-2}	5.6×10^7
0.5 - 0.7	0.056	5.6×10^5	1.1×10^{-1}	5×10^6
0.7 - 0.9	0.011	1.1×10^5	2.4×10^{-1}	4.7×10^5
0.9 - 1.1	0.002	2×10^4	4.3×10^{-1}	4.7×10^4
1.1 - 1.3	0.0009	9×10^3	7×10^{-1}	1.3×10^4
>1.3	0.0001	1×10^3	>1.0	$<10^3$

the limiting activity. Another is that this lymph node particle size distribution may not adequately represent the lung burden of the individual from which it was obtained. In this regard, the exposure of this Los Alamos worker may not be representative of the 25 Manhattan Workers. An examination of the corresponding lung tissue is underway and this may be quite helpful. (See letter from McInroy to Cochran attached as Appendix B to this submission). Finally, assuming it is inappropriate to apply this distribution to the Manhattan Workers and instead applying it only to the individual from which it was obtained (see Appendix A) leads to a minimum activity to constitute a hot particle of 0.14 pCi/particle.

c. Rocky Flats Fire.[20] The approach used above can also be applied to the individuals contaminated during the October 1965 , fire at Rocky Flats. This will again give an upper estimate of the minimum activity since, as we discussed in the Hot Particle Report, lung cancer may develop in these individuals over the next 15 or so years.

Mann and Kirchner report that the MMD for the particles in this incident was 0.32 μ with a standard deviation of 1.83.[21] The data they present indicates that the combined lung burden of 25 exposed workers was 1.2×10^6 pCi.[22] Table II was constructed using these data and the same approach as used above for the Manhattan Workers.

Inspection of Table II indicates that for the Rocky Flats Workers to contain only 2,000 hot particles, the minimum activity to constitute a hot particle would have to be some 1.6 pCi/particle. If, however, the minimum particle activity were only 1 pCi/particle, Table II would suggest that around 3 lung cancers could be anticipated in the next 15 or so years (using a risk per particle of 1/2,000).

20/ 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.

21/ Ibid, p. 881.

22/ Ibid, p. 880. (When a range in lung burdens was given, we used the midpoint)

TABLE II

Estimated Particle Size Distribution For
The Rocky Flats Workers
(Uses a total lung burden of 1.2×10^6 pCi)

Diameter μ	Incremental Activity Fraction	Incremental Activity (pCi)	pCi/Particle	Number of Particles
0.6 - 0.7	0.05	6.0×10^4	0.09	6.7×10^5
0.7 - 0.8	0.033	4.0×10^4	0.14	2.9×10^5
0.8 - 0.9	0.022	2.6×10^4	0.20	1.3×10^5
0.9 - 1.0	0.017	2.0×10^4	0.28	7.1×10^4
1.0 - 1.2	0.014	1.7×10^4	0.44	3.9×10^4
1.2 - 1.4	0.007	8.4×10^3	0.72	1.2×10^4
1.4 - 1.6	0.004	4.8×10^3	1.15	4.2×10^3
1.6 - 1.8	0.0016	1.9×10^3	1.62	1.2×10^3
1.8 - 2.0	0.001	1.2×10^3	2.24	5.4×10^2

This possibility cannot be ruled out at the present time.

d. Sanders and Dagle.[23] This report presents preliminary results of a continuation of experiments wherein Sanders induced a large incidence of lung cancer in rats following exposure to low doses of soluble Pu-238. Of particular interest in these new experiments are three exposure groups involving insoluble particles in which no lung cancers have appeared. One of these groups was exposed to $^{238}PuO_2$ and we shall analyze it because it will be the most critical with respect to particle size and activity.

There were 60 rats in this group who were exposed to an average of 160 pCi of $^{238}PuO_2$ as measured one day after inhalation of particles with a CMD ranging between 0.1 and 0.3 μ. The report indicates that 23 of the 60 rats have died so far with no evidence of lung cancer (571 days past exposure).

We shall use the midpoint of the CMD range. A CMD of 0.2 μ corresponds to a MMD of 0.3 μ. The distribution of particle sizes about the median was not given. We shall therefore arbitrarily assume the particle size distribution obtained at Rocky Flats (MMD = 0.32 μ, σ= 1.83). Table III presents the particle distribution on this basis for a total exposure of 9,600 pCi to the 60 rats.

Inspection of Table III suggests that we can draw no inferences from this experiment at this time. Above 0.1 μ there are only 5,000 particles leading to an expectation of only 2 cancers (assuming a risk of 1/2,000 per particle). If no cancers appear this experiment, this would only suggest a minimum particle activity of around 0.6 pCi/particle. We say

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23/ Sanders, C. L., and G. E. Dagle, "Studies of Pulmonary Carcinogenesis In Rodents Following Inhalation of Transuvanic Compounds, Pacific Northwest Laboratories, Biology Dept., Preprint.

TABLE III

Estimated Particle Size Distribution For Rats Exposed to $^{238}PuO_2$
(Total exposure for 60 rats = 9,600 pCi)

Particle Diameter u	Incremental Activity Fraction	Incremental Activity (pCi)	pCi/Particle	Number of Particles
<0.1	0.02	192	0.08	2,400
0.1 - 0.2	0.20	1,920	0.64	3,000
0.2 - 0.3	0.24	2,300	2.2	1,060
0.3 - 0.4	0.18	1,730	5.1	340
0.4 - 0.5	0.12	1,150	10	115
0.5 - 0.6	0.09	865	18	48
0.6 - 0.7	0.05	480	28	17

only suggests because with only 5,000 particles the chance of no cancers appearing would be 0.08 which is not generally considered statistically significant. Furthermore, as stated above, the particle size distribution is unknown and must be assumed somewhat arbitrarily.

Table IV presents a similar analysis for a group of 60 rats exposed to 12,000 pCi of $^{239}PuO_2$. Inspection of Table IV indicates that if no tumors appear in this group, it would be suggestive of a minimum particle activity of 0.14 pCi/particle (assuming a risk of 1/2,000 per particle).

It must be recognized that the above analysis is quite tentative not only because the particle size distribution is speculative, but also because more than half of the rats were still living when these interim results were reported. Moreover, as with the hamsters, the life span of the rats may compromise the induction period for hot particle mediated carcenogenesis.

Minimum hot particle activity. We are now in a position to summarize estimates of the minimum hot particle activity. As stated earlier, our initial definition of the minimum hot particle activity was based upon the dose to surrounding tissue which was quite uncertain. The experimental results above allow assessment of this parameter without resort to dose calculations. These observations and analysis lead to the following estimates of the minimum activities:

Minimum Activity pCi/particle	Experimental Basis
0.9 - 4.3	Observation of particle produced lesions
1.6	Rocky Flats Workers
0.8	Manhattan Workers
0.6	$^{238}PuO_2$ in rats
0.14	$^{239}PuO_2$ in rats and (in Appendix) from lymph node
0.07	1,000 rem/year

210

TABLE IV

Estimated Particle Size Distribution For Rats Exposed To $^{239}PuO_2$ (Total exposure For 60 rats = 12,000 pCi)

Particle Diameter μ	Incremental Activity Fraction	Incremental Activity (pCi)	pCi/particle	Number of Particles
0.6-0.7		600	0.09	6650
0.7-0.8		395	0.14	2820
0.8-0.9		263	0.20	1315
0.9-1.0		204	0.28	730
1.0-1.2		163	0.44	37
1.2-1.4		84	0.72	117
1.4-1.6		48	1.15	42

These activity values range over a factor of 60 but the diameter varies by only the cube route, or a factor of 4. Particle produced lesions which could be considered as precursors of peripheral adenomas were observed around the 4.3 pCi particles. Hence, it would be fortuitous if this value did not overestimate the minimum activity.

Until more experiment data becomes available we would choose a conservative approach to selecting the minimum activity. Consequently, we can see little justification for assuming a minimum activity greater than 0.6 pCi/particle and we believe it prudent to select a lower value as we have previously proposed.

III. The Sensitive Tissue

In his statements and questions during the December 10 and 11, 1974, hearings, Dr. Radford implied (and attempted to solicit concurence) that hot particles can only be expected to induce cancer in man in the more proximal bronchi because in man this is the sensitive tissue. We cannot agree with this and as the transcript (pages 2-262 to 2-268) indicates, Dr. Bair did not concur.

While the predominant lung tumor in man is bronchiogenic, bronchiolo-alveolar carcinomas also occur. It would appear that because of genetic factors, influenced by the prevalent carcinogens, the more proximal bronchi are the most sensitive tissue. Nevertheless, we submit that alpha-emitting hot particles represent a new and unique carcinogenic agent. As such, we see no a priori reason for doubting that, as in animals, bronchiolo-alveolar carcinoma will be induced in man by PuO_2 deposited in the peripheral regions of the lungs.

Along with Dr. Little, Dr. Radford has presented evidence demonstrating that Po-210 in cigarette smoke concentrates in the segmental bifurcation.[24] Dr. Edward A. Martell has proposed that the Po-210 is contained in insoluble particulates which accumulate at these bifurcations.[25] As a consequence the dose to the local tissue is several rem/year. This is suggested as the carcinogenic mechanisms related to cigarettes.

24/ Little, J. B. and Radford, E. P., Science, 155, 1967 pp. 606-607.

25/ Martell, Edward A., Nature, 249, May 17, 1974, pp. 215 - 217.

The Po-210 particles involved have 2 orders of magnitude less activity than hot particles. The mechanism involves continuous exposure at "low" dose rates while the hot particle hypothesis involves a significantly higher dose rate that is capable of producing a tissue disruptive lesion around the particle. Because of the particle size distribution, exposure to PuO_2 aerosols could involve both mechanisms. As a consequence, the risk could be larger than that estimated by each hypothesis independently.

IV. PuO_2 Exposure Standards

In our petition and Hot Particle Report, we concluded that, consistent with the whole body exposure standard of 5 rem/year, the alpha-emitting hot particle standard should be 2 particles in the human lung. Using the estimated minimum hot particle activity of 0.07 pCi, this resulted in the suggested reduction of the MPLB by 115,000. However, as we stated in our Hot Particle Report, this factor of 115,000 would apply only when it was not determined that the activity was not on hot particles. Using the particle size distribution determined for the Rocky Flats fire, and allowing only 2 particles above 0.07 pCi would still have required a reduction of the MPLB by a factor 16,000.

Table V presents the particle size distribution (using the Rocky Flats statistics) for high burnup Pu fuel that would be used in Pu recycle in LWR's or in the LMFBR. This table serves to illustrate the nature of the problem associated with hot particle exposure standards in the nuclear reactor industry. As we indicated above, we can see little justification for selecting a

TABLE V

Estimated Particle Size Distribution For High Burnup Pu Fuel (0.2 Ci/g) (Assuming a Lung Burden Of 16,000 pCi)

Particle Diameter μ	Incremental Activity Fraction	Incremental Activity (pCi)	pCi/particles	Number of Particles
0.6-0.7	0.05	800	0.32	2500
0.7-0.8	0.033	530	0.47	1100
0.8-0.9	0.022	350	.66	530
0.9-1.0	0.017	272	0.91	300
1.0-1.2	0.014	224	1.58	142
1.2-1.4	0.007	112	2.49	45
1.4-1.6	0.004	64	4.10	15
1.6-1.8	0.0016	26	5.30	5
1.8-2.0	0.0001	16	7.30	2

minimum hot particle activity greater than 0.6 pCi/particle. Inspection of Table V indicates that a 2 particle limit at 0.6 pCi/particle would still require a reduction of the MPLB by a factor approaching 2000.

A 1000 fold reduction would cause the MPLB for occupational exposure to be only 16 pCi and as such would be far below the limits of detectability. But that appears to be the situation with plutonium. Dr. Morgan, at the December 10th hearings, recommended a reduction in the whole body burden by about a factor of 400 based on other considerations; namely, exposure to the bone. It appears that commensurate with other radiation protection standards, if you can detect Pu in the human body, a significant overexposure has already occurred. This, we propose, is the conclusion to be drawn from the record of the EPA hearings in Washington, D.C. and Denver, Colorado.

Since acceptable levels of Pu in humans are below detectable levels, it is apparent that the exposure standards can be enforced only by enforcing strict compliance to design specifications and operational procedures that have the objective of zero release. We submit that compliance with adequate design specifications and operational procedures is the only way to effectively meet any exposure standard and we suspect that it was quite effective at the Army's bacteriological warfare laboratory where zero release was an objective.

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February 4, 1975

Appendix A

Mr. Robert B. Minogue
Acting Director
Standards Development
Nuclear Regulatory Commission
1717 H Street, N. W.
Washington, D. C.

Dear Mr. Minogue:

We are writing in response to a suggestion at the meeting of January 9, 1975, that it would be useful if we provided written comments on two issues discussed in "A Review of the Natural Resources Defense Council Petition Concerning Limits for Insoluble Alpha Emitters," J. W. Healy, C. R. Richmond and C. E. Anderson, LASL, LA-5810-MS, November, 1974. These issues to be addressed are:

(a) The discussion beginning on page 4, "B. Limitations on the Usefulness of Radiation Dose" with particular emphasis on the statement,

> "It is for these reasons that most scientists have refrained from using dose calculations, such as those given earlier, to arrive at conclusions as to the effect of radioactive particles but have preferred to depend upon experimental evidence which bears more directly on the actual conditions."

(b) The statement on page 15,

"In a recent study McInroy et al., 37 measured the distribution of plutonium particle size in a lymph node of a deceased worker by the autoradiographic technique. Although this individual was exposed at a later time than those discussed above, it is of interest that these estimates also indicated that 15% of the plutonium was in particles larger than 0.07 pCi."

We do not agree with the first part of the statement from page 4. Most scientists who have considered the particle problem have used dose calculations to arrive at the conclusion that particle irradiation is unique and that its consequences may be significantly different from uniform irradiation.

At the same time, we agree that it is preferable to use experimental data that bear directly on the problem when estimating the risk from particle irradiation. In fact, in responding to criticisms of our Hot Particle Report, such as WASH-1320, much of our effort was directed toward demonstrating that most of the cited experiments were not relevant to hot particles.

We would suggest that the most pertinent observations involve the lesion excised from the palm of a mechanic by Lushbaugh and Langham and the microsphere experiments conducted by Richmond, et al. These experiments strongly suggest that a single hot particle embedded in tissue is capable of eliciting a tumorgenic response. Richmond, et al, described the lesions induced in the lung of hamsters as precursors of peripheral adenomas. We submit that these observations alone are sufficient to indicate that every effort should be made to prevent such particles from being deposited in human lungs. They strongly suggest that a single hot particle represents a significant hazard and

that the radiation protection standards should certainly limit the exposures to very few particles.

This, however, leaves us with the problem of what constitutes a hot particle. In the subsequent discussion of the statement on page 15, we address this issue and define the hot particle from experimental data without the use of dose calculations.

With respect to the statement on page 15, the unpublished paper by McInroy, et al., reports new and potentially significant data that were not available when we wrote, "Radiation Standards for Hot Particles." Our analysis of these data and their implication with respect to the proposed hot particle standards is given below.

The following data were presented for Case 7-138, the metal fabrication worker who died of a crushed chest in 1973, twenty six years after his first exposure.

Lymph Node activity (12 nodes)

Mean concentration	770 ± 493 pCi/g Pu-239
	80 ± 43 pCi/g Am-241
Maximum concentration (node #11)	1800 pCi/g

Distribution of $^{239}PuO_2$ particles (in node #6):

Mass median diameter	MMD = 0.3 μm
Geometric std. deviation	σ_g = 1.6
Count median diameter	CMD = 0.2 μm

Based on these data (and McInroy, et al.'s, Table 5 and Figure 2) we find that 7 percent (as opposed to 15% reported by Healy, et al.) of the lymph node activity was estimated to be on hot particles. This represents a substantial number of hot particles by our definition (activity $\geq$0.07 pCi).

The measured distribution of $^{239}PuO_2$ particles size is given in McInroy, et al.'s Table 5. For particle diameters larger than 0.6 μm (corresponding to 0.07 pCi) the following data are presented:

Dia μm	Midpoint	Incremental Fraction	Activity pCi/particle	Activity pCi/node	Particles per node
0.6					
	0.7	0.056	0.11	37	340
0.8					
	0.9	0.011	0.24	7	29
1.0					
	1.1	0.002	0.43	1	2
1.2					

Total activity in sample (node #6) = 657 pCi.

Assuming the total mass of the tracheobronchial lymph nodes is 15 grams, the total number of hot particles (activity >0.07 pCi) in the lymph nodes is

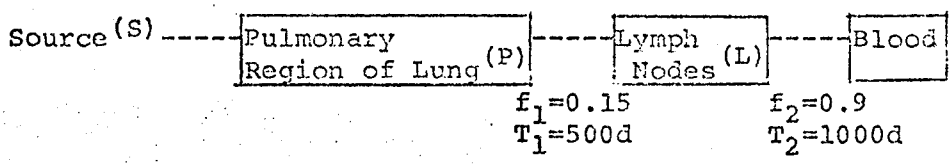
$$(15g)\left(770\ \frac{pCi}{g}\right)\left(\frac{340 + 29 + 2}{657\ pCi}\right) \text{ particles} = 6500 \text{ hot particles.}$$

This probably overstates the number of hot particles in the lymph nodes for the following reasons: (a) smaller particles tend to aggregate into larger particles in lymph tissue (See WASH-1320, pp. 10-12), (b) according to McInroy (private communication with TBC, Jan. 20, 1975) aggregates (particularly with respect to the larger particles) were observed and reported as single particles (the experimental design of the particle size measurements, because of aggregation, tended to maximize the estimate of large particles), and (c) because one is looking at a plane view, it is difficult using the audioradiographic technique to distinguish star track coming from two point sources at different depths but along the same line of view. Paul

Morrow pointed out to one of us (TBC private communication, Jan. 20, 1975) that the audiographic technique is not very reliable for particle sizes below about 0.5 μm to 1.0 μm because of the difficulty in distinguishing individual particles. In other words, aggregates of particles would appear as point sources below this size range. In addition, there is a sizable (64 percent) statistical uncertainty in the 770 pCi/g estimate, and the lymph nodes analyzed may not be representative of the total tracheobronchial lymph node mass.

It is possible to make a crude estimate of the lung burden based on the lymph node concentration, or burden at death. We have done this using the ICRP lung model (ICRP Publication 19, p.6) for lack of better data.

In our case the ICRP model is simplified to



where f and T are the regional fraction and biological half-life, respectively, for Class Y compounds.

The rate equations are

$$\frac{dP}{dt} = S(t) - \lambda_1 P \qquad \text{pulmonary region}$$

$$\frac{dL}{dt} = f_1\lambda_1 P - f_2\lambda_2 L \qquad \text{tracheobronchial lymph nodes}$$

where

$$\lambda_1 = \frac{0.693}{(500/365)} = 0.5 \qquad f_1 = 0.15$$

$$\lambda_2 = \frac{0.693}{(1000/365)} = 0.25 \qquad f_2 = 0.9$$

We have assumed the rate at deposition of activity in the pulmonary region, is constant throughout the 26 year exposure period to simplify the calculation, i.e. $S(t) = R$. This yields

$$P = \frac{R}{\lambda_1}(1-e^{-\lambda_1 t}) \ ,$$

$$L = \frac{f_1 R}{f_2 \lambda_2} \quad 1 + \frac{e^{-\lambda_1 t}}{\frac{1}{f_2 \lambda_2} - \lambda_1} - \frac{e^{-f_2 \lambda_2 t}}{\frac{1 - f_2 \lambda_2}{\lambda_1}}$$

For t = 26 yr, the time of death

$$L(26) = 12 \text{ nCi} \approx \frac{2}{3} R$$

$$R = 17 \text{ nCi/yr}$$
$$P(26) \approx 2R \approx 34 \text{ nCi}$$

There is considerable uncertainty in these values for a number of reasons reviewed on pp.5-9 of ICRP Publication 19. The parameters are for a class of compounds as opposed to PuO_2. Retention may be a strong function of particle size. The biological half-lives are not known within a factor of 5; $T_{1/2}$ = 500d may represent anything between 100d and 10,000d. The ICRP model parameters are not consistent with uranium miner exposure data. It should also be noted that the calculated pulmonary and lymph node burdens are higher than the 33 nCi of ^{239}Pu based on urine assay (McInroy, et al., p.3) and that the exposure was surely not uniform over the 26 year period, but probably highest "during the early years of laboratory operation (1945 - 1955) before improved industrial hygiene and health physics requirements reduced significantly the air levels of plutonium in the laboratories and the workers were provided with more efficient personal respiratory protection" (McInroy, et al., p.5.).

Nevertheless, assuming a pulmonary burden of 34 nCi and the particle size distribution in the lung is the same as the measured distribution in the lymph node, the number of particles greater than 0.07 pCi (0.6 μm dia) is

$$(34\ \text{nCi})\left(\frac{371\ \text{particles}}{657\ \text{pCi}}\right)(10^3\ \text{pCi/nCi}) = 20{,}000\ \text{hot particles}$$

The probability of cancer induction at a risk of 1/2000 per particle would be essentially unity.

Assuming a minimum activity to constitute a hot particle is 0.14 pCi, corresponding to a 0.8 μm diameter $^{239}PuO_2$ particle, the number of hot particles in the pulmonary region would be

$$\frac{(34)(31)}{657} \times 10^3 = 1600\ \text{particles.}$$

The tumor risk would be about 0.5.

There is an obvious need for a careful particle size analysis of the lung tissue available from Case 7-138, and a pathological examination to determine whether lesions are associated with the larger particles. Dr. McInroy has informed one of us that an examination of the lung tissue is underway.

It can be argued that it is premature to modify the proposed hot particle standard ["Radiation Standards for Hot Particles"] by shifting the minimum hot particle activity until the lung data is available. However, our original choice of the minimum hot particle activity carried considerable uncertainty (See "A Critique of the Biophysical Society's DRAFT Comments on "Radiation Standards for Hot Particles," pp.4-6). The choice of 1000 rem/year to the local tissue as the cut off defining a hot particle was based on the choice of (a) 1000 rem supported by the experiments by Albert, et al., and Laskin, et al., (b) one year as the tissue repair

time in the lung, and (c) the Geesaman lung model assuming the lung was inflated to 1/2 maximum.

The combined uncertainty associated with these assumptions is more than a factor of two. Had the lymph node data been available to us at the time we prepared our report we would have used these data in establishing the minimum (or critical) hot particle activity. We therefore propose increasing the minimum hot particle activity by a factor of two to 0.14 pCi. This new value should of course be re-examined as in light of any new data, particularly the Case 7-138 lung data when it becomes available.

One of the criticisms of our report raised by Dr. Gamertsfelder, and others, is the arbitrariness or uncertainty in the choice of 1000 rem/yr to the local tissue as the definition of the critical particle activity. We can avoid the use of dose or dose rate altogether in defining the critical particle activity by basing the cut off on the observations by Lushbaugh, et al., the hamster experiments of Richmond, et al., and the lymph node study by McInroy, et al. Lushbaugh, et al. reported a lesion in palmer tissue that developed around a particle containing 0.08 μg (5nCi) of Pu-239. Richmond, et al. observed lesions in the lungs of hamsters around particles containing 4.3 pCi. Moving down further in activity, we postulate on the basis of the study of McInroy, et al. that lesions probably did not occur around particles less than about 0.14 pCi, otherwise Case 7-138 probably should have developed cancer according to the hot particle hypothesis. On the basis of these data it seems logical to select a critical particle activity between 0.14 pCi/particle and 4.3 pCi/particle. We would suggest a value close to 0.14 pCi/particles to be conservative and because of the limitations of the Richmond, et al. experiments set forth on pp.25-28 of our critique of WASH-1320 namely, that had these experiments been performed with animals that have longer life spans, it is quite possible that lesions would have developed around particles of lower

activity. Notice we have avoided the use of dose altogether.

Finally, as we stated earlier, the observations of Lushbaugh and Langham along with those of Richmond, et al., strongly suggest that a single hot particle represents a significant hazard and that the standards should limit exposures to very few particles. Moreover, the direct observation by Richmond, et al. of lesions induced by particles containing 4.3 pCi clearly demonstrates that there is a hot particle problem associated with the nuclear industry. Particles with this activity are within the size range that can be deposited in the deep respiratory tissue. In other words, there is experimental evidence that bears directly on this problem and that evidence indicates the need for more restrictive standards when hot particles are involved.

If you wish to discuss these, or other issues further, don't hesitate to call us.

Sincerely,

Thomas B. Cochran
Arthur R. Tamplin

cc: Dr. William A. Mills
Dr. W. D. Rowe

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LOS ALAMOS SCIENTIFIC LABORATORY
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Appendix B

IN REPLY
REFER TO: H-5-75-165
MAIL STOP: 486

February 10, 1975

Thomas Cochran, Ph.D.
National Resources Defense Council
1710 Nth Street, N.W.
Washington, D. C. 20036

Dear Dr. Cochran:

In reference to our recent telephone conversation concerning the particle size distribution of PuO_2 in the tracheobronchial lymph nodes of a former employee of Los Alamos Scientific Laboratory ("Studies of Plutonium in Human Tracheobronchial Lymph Nodes", LA-UR-74-1454), I checked with the person that had counted the tracks associated with the "stars" in our autoradiographs about the possible presence of clusters of particles. He did not find many stars in which he was able to distinguish more than one center from which the tracks originated. However, it is my feeling that there is no way in which we could identify whether the tracks were formed from the decay of plutonium in a single particle or a cluster of small particles. The best we can say is that if a group of small particles was counted as a single particle, the size estimate was of this larger, composite diameter. This means that our estimate of the activity median diameter of 0.32 μm may be on the high side as counting any aggregate of particles as a single particle tends to maximize the estimate of the size distribution.

I am very interested in your calculation of the lung burden from the lymph node concentrations. My estimate of plutonium in the lung, based upon the average concentration of ^{239}Pu in eight transverse sections, taken from the superior lobe of the right lung, was 36 ± 19 nCi. The variance is quite large due to the variation in the distribution of the particles throughout the lung. The estimate, however, is remarkably close to your calculated value of 34 nCi. This may be fortuitous but I will be able to improve our estimation as we continue to analyze sections from this lung.

As regards our plans for continued study of this autopsy case, I have discussed with our pathologist the possibility of examining sections of lung tissue for lesions that could be associated with the presence of plutonium and, at the same time, attempt to measure the particle size distribution within the lung, using the same techniques used with the lymph nodes. We have decided to attempt this, although there are several serious problems that are evident, For example, our lung specimen was inflated with dry nitrogen, shortly after the autopsy, to a configuration approximating the natural shape it would have in the thoracic cavity. This was frozen in this form and has since been used in studies with our lung counter to compare the in vivo and in vitro measurements of plutonium and americium present The sectioning, mounting and staining of tissue that has been frozen presents some

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problems when attempting a histological examination due to the disruption of structure by freezing and to the dehydration that has occurred during storage

I will be happy to keep you informed as to our progress. If you have additional questions and/or suggestions, please write or call me at 505-667-4709.

Sincerely,

James F. McInroy

James F. McInroy
Tissue Section Leader
Industrial Hygiene Group

mlg

UNIVERSITY OF OREGON

INSTITUTE OF
MOLECULAR BIOLOGY

EUGENE, OREGON 97403
telephone (code 503) 686-5151

December 5, 1974

Dr. William Rowe
Office of Radiation Program
Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Rowe:

Enclosed please find a series of reports and documents which have been assembled in connection with a study of the radiation standards for "hot particles" problem which was undertaken by the Science and Technology Information and Advice Service (STAIS) of The Biophysical Society, which was undertaken at the request of the Center for Science in the Public Interest, as outlined in the covering memorandum. Further details on the origin of the study, and the procedures followed in carrying it out, are presented in the enclosed documents. I understand that the EPA is currently undertaking a review of this problem, and I send you the enclosures in the hope that they may be helpful to you in the course of this review.

Please let me know if we can help in any other way.

Sincerely yours,

Peter H. von Hippel

Peter H. von Hippel
Editor, STAIS and Past-President,
The Biophysical Society

PHvH:bm
Enclosures

228

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November 25, 1974

MEMORANDUM

To: Whom It May Concern

From: Peter H. von Hippel, Editor, The Biophysical Society Science and Technology Advice and Information Service; and Past-President, The Biophysical Society.

Subject: Enclosed STAIS Report on Radiation Protection Standards for Hot Particles of Plutonium and Other Actinides.

At the request of the Center for Science in the Public Interest, the STAIS group of the Biophysical Society undertook a study on the problem of Radiation Standards for Hot Particles. Information about the Biophysical Society's Science and Technology Advice and Information Service, as well as the procedures and ground rules under which we conduct studies, are attached.

As indicated in the enclosures, a committee of experts in various fields relevant to the problem under consideration was put together, and their efforts were coordinated by Drs. Jane and Richard Setlow of the Brookhaven National Laboratory. The committee members names and addresses are listed on an attached sheet. As indicated in the summary provided by Drs. Setlow, the committee did not actually meet, but each member was provided a copy of the Natural Resources Defense Council petition, and the Tamplin-Cochran report, which formed the basis for the request for a STAIS study by the Center for Science in the Public Interest. Each member of the committee was asked to evaluate the information contained in the petition and report, and the literature on which these documents were based, and to submit a written report to the Drs. Setlow on completion. Since each committee member went about this assignment in a different way, and from a somewhat different point-of-view, we have felt it appropriate to submit a summary of their findings, together with their individual reports, rather than trying to assemble an integrated report at this point. The committee members have specifically asked me to point out that their individual reports represent more individual "impressions" following perusal of the available literature (though by people expert in the field), than definitive studies. For this reason we would also

like to designate the present report as a draft version, and would welcome comments of other individuals knowledgeable in the field who might wish to comment upon, or differ with, some of the conclusions our experts have reached. Such comments should be sent to me (or Drs. Jane or Richard Setlow), and we will pass them on to the individual or individuals on the committee most competent to assess them. Such comments, together with the responses of the committee members, could then form a part of the final record of these deliberations.

In addition to sending this report to the requesting group, we have also sent copies to various other agencies and groups involved with current studies on this problem, including: the Atomic Energy Commission, the Environmental Protection Agency, the General Accounting Office, the Natural Resources Defense Council, and the National Academy of Sciences. We hope that these groups will find these statements useful in putting together their own reports on this problem.

In conclusion, I would like to add some personal comments which seem to me to be implicit in the attached reports. First, the main reason this body of experts was unable to reach a single definitive conclusion is, of course, that really adequate experimental data do not exist. Thus, all studies on this subject are of necessity somewhat subjective, since they must be based on rather difficult extrapolations of only marginally relevant experimental work. In this connection, we might wish that more directly relevant studies had been launched by the Atomic Energy Commission some years ago, so at this stage there would be better data to go on. Also, I think it is important to point out that the National Resources Defense Council has performed a very useful public service in calling our attention to the possible inadequacy of the present radiation protection standards, since, regardless of whether the present standards are ultimately found to be adequate or not, they had obviously not been formulated with the "hot particle" problem in mind. Our contribution to this has been to make our best unbiased attempt to estimate the validity of the present standards, based only on the relatively scant experimental information available. Based on these studies and others, the nation's policy makers will ultimately have to find the appropriate balance between health hazards to society, and the energy requirements of the nation.

THE BIOPHYSICAL SOCIETY

SCIENCE AND TECHNOLOGY ADVICE AND INFORMATION SERVICE

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The concept of such a service arose because many members of our Society have expressed a desire to utilize their scientific and technical training on a part-time basis to help with the various problems facing governmental agencies (at the federal, state and local levels) and public interest groups in formulating and implementing policy and programs which have scientific and technological components. To this end the Society has surveyed its membership and developed a Roster of interested members who are able and willing to participate.

Agencies or groups requiring such assistance are invited to write or call the Office of the Secretary of the Biophysical Society for further information, or to receive an "Advice Request Form". Specific questions, or requests for advice, can also be directed to any of the members of the STAIS Editorial Board.

The ground rules under which STAIS will operate are as follows:

"The basic purpose of the Science Advisory Service of the Biophysical Society is to contribute to the improvement of conditions of society. For example, a project in connection with which advice is solicited might be designed: to relieve suffering and prolong life; to improve the environment by reducing pollution of the air or water, or by protecting natural resources; or to help provide needed facilities for people which improve or maintain the quality of life." The Editorial Board of STAIS will retain the right to reject a request which, in its opinion and after suitable investigation, does not seem to meet these general criteria. It is expected that advice will <u>not</u> be provided in confidence, and both the designated advisory group and the Editorial Board will retain (and generally exercise) the option of making the results of investigations public if such results are of public concern. The Biophysical Society will take no official responsibility for the advice given, nor should that advice be construed as representing the position of the Biophysical Society on the question at issue. The only role the Society as such will play in STAIS is to maintain the Roster and the Editorial Board, and to put groups requesting advice in contact with the appropriate advisors. The Biophysical Society is a non-profit organization, and the only charges to the group seeking advice will be to cover costs.

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RADIATION STANDARDS FOR HOT PARTICLES

(Summary by Jane and Richard Setlow, Committee Coordinators)

Summary of the reports of five committee members asked by the Biophysical Society to evaluate the present radiation protection standards for hot particles of plutonium and other actinides.

The group has not met to discuss the problem since the individual members have perused the relevant literature. Each member wrote an independent evaluation and these have been summarized by the coordinators, R. B. Setlow and J. K. Setlow, as follows.

1. The problem raised by the Natural Resources Defense Council petition of what should be the maximum permissible lung burden (MPLB) of hot particles, is a valid and serious one. However, the call for a decrease in MPLB by 10^5 is exaggerated. More animal and epidemiological data are needed for a truly adequate estimate of what should be the radiation protection standard. A crucial piece of missing information concerns the distribution of particle sizes involved in the Manhattan district accident. Twenty-five individuals followed for almost 30 years have no lung cancer from 3 - 10 nCi of plutonium in the chest. Calculations in the Tamplin and Cochran report accompanying the petition indicated that the particles were too small to be effective. Other calculations resulted in the opposite conclusion. One of the reviewers suggested an experimental reenactment of this accident (without humans present) for the purpose of measuring particle size.

The lung burdens of 25 Rocky Flats workers exposed to plutonium fires range from one to ten times the present MPLB. No lung cancer has been detected in any of these individuals after nine years. Since there is evidence that the latent period for cancer induction after a large exposure may be as short as this, these data again suggest that the factor of 10^5 is too large.

2. The reviewers who looked into the quantitative aspects of the Tamplin-Cochran report all concluded that it contained exaggerations and lack of adequate reasoning (a,b,c). This report includes interpretation of the data of others, sometimes at variance with the authors' own interpretation. Two of the reviewers used existing published animal data and several biological models to estimate the probability of cancer induction in the human lung from hot particles. They conclude that the existing MPLB should probably be decreased by some factor between 40 and 10^4, but that this figure at present can only be tentative, because of the paucity of data. Another reviewer finds no reason to alter current standards.

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a) The single instance of a hand sarcoma following plutonium contamination is inadequate for a quantitative argument, especially since there was no evidence that the plutonium penetrated the skin.

b) The single instance of supposed precancerous changes in the neighborhood of a puncture wound involving plutonium, later excised, is also not suitable for a quantitative argument, especially since there was another similar but unexcised case in which no cancer developed in 30 years.

c) The use of the data of Albert et al on rat skin tumors induced by fast electrons to estimate the risk from hot particles seems unjustified on four grounds. (i) The rat data involved a single dose, whereas the lung irradiation being considered is chronic. (ii) Tamplin and Cochran do not cite data showing that nonuniform irradiation by beta and alpha particles is less effective than uniform radiation. (iii) Previous experiments cited by the Albert group showed no tumor production by 0.3 MeV electrons, external alpha particles and protons. (iv) The hair follicle seems to be the sensitive structure for radiation-induced cancer in the skin. No similar structure has been identified in the lung, nor is there any estimate of the probability of a hot particle being close to such a structure. (Albert, Burns and Heimbach, Rad. Res. 30: 514, 525, 590 [1967]).

August 27, 1974

M.L. Randolph: Appraisal of "Radiation Standards for Hot Particles",

Report of A.R. Tamplin and T.B. Cochran, February 14, 1974

A. OVERALL VIEWS

I have chosen to evaluate primarily the report "Radiation Standards for Hot Particles", Feb. 14, 1974, of Tamplin and Cochran, and the biological and theoretical evidence they directly refer to, rather than the more general problem of maximum permissible ^{239}Pu lung burden and the foundations on which it does and/or should rest because the more limited problem is easier to consider and seems the scientific basis on which to decide the immediate question of whether or not the Biophysical Society should support the petition of the Natural Resources Defense Council to AEC and EPA. Other arguments such as that of Myers (Health Physics 22 (1972) 905) might well influence a more thorough appraisal of the general problem.

This appraisal is organized as follows: A) Overall views B) Major technical considerations C) Minor (?) technical considerations D) Appendix. Tentative estimate of maximum permissible lung burdens.

My major conclusions regarding Tamplin and Cochran's report and Geesaman's work which furnishes the backbone for their report are:

1. The authors are to be commended for their approach to radiation hazards that standards should be based on specific, rather than overly broad, hazards; for the analysis of the dose from hot particles, and (perhaps) for their conservative approach that the crucial parameter for tumor induction by hot particles is the number of particles of damaging size in the lung rather than the gross dose. Unfortunately their work has not yet been published in the open literature (to my knowledge).

2. The quantitative arguments (as I understand them) that Tamplin and Cochran advance in support of their proposed reduction of maximum permissible lung burden by a factor of 10^5 at best warrant the Scottish verdict "Not proven". The main trouble seems to be loose interpretation of the biological results — e.g., use of Albert et al's (Rad. Res. 30 (1967) 515, 525 and 590) data on skin cancers produced by penetrating electrons in the face of ~~the authors'~~ Albert's comments that no skin tumors were observed with protons, alpha particles or low energy electrons. Eight sets of biological data adduced by Tamplin and Cochran are discussed under heading "B. Major technical considerations."

3. Data more directly applicable can be expected in about 10 years when the latent period for detection of cancers in the Rocky Flats workers is over and when results from the new low-level inhalation study with beagles are complete.

4. Re-enactment (without humans present!) of the exposure conditions for the Manhattan District workers with concurrent measurement of particle size distribution might promptly provide useful information.

5. I have been told that "professionals" — i.e., the ICRP and the European Radiation Research Society — have official committees looking into the hazards of hot particles. If so, I wonder how much effort we amateurs, except Hempelmann, should put forth.

6. Results of very tentative appraisals (D. Appendix of this report) of lung tumor risks based on data compiled by Bair suggest that the maximum permissible lung burden should be reduced by a factor of 40 to 10^4 instead of the factor of 10^5 suggested by Tamplin and Cochran.

Randolph

B. MAJOR TECHNICAL CONSIDERATIONS

As I understand it, Tamplin and Cochran's conclusions stem almost completely from their conviction that airborne 1 μm diameter particle of $^{239}PuO_2$ has a high probability of being deposited in the deep respiratory zone (DRZ) of a human lung and thereafter imposes a risk of 0.001 to 0.0001 that a tumor will be produced (within perhaps 20 years).

This conviction is founded primarily on three bases:

1) the work of Mann and Kirchner (Health Physics 13, 877 (1967)) on the percent of inhaled PuO_2 aerosol deposited in the pulmonary region;

2) D.P. Geesaman's theoretical analysis (UCRL-50387, 1968) from lung geometry, alpha particle range, etc., of energy deposition for such hot particles;

3) from considerations by Geesaman (UCRL-50387, Addendum, 1968) and by Tamplin and Cochran of seven biological "experiments".

The first two physical bases seem to depend on rather straightforward analyses. In the work of Mann and Kirchner the probability of deposition of aerosol is derived for the special case of PuO_2 in terms of particle size, whereas other more general reports (e.g., ICRP Task Group on Lung Dynamics, Health Physics 12 (1966) 173; Dix and Dobry, Health Physics 22, (1972) 569; and Craig et al, Health Physics 22 (1972) 845) are in terms of "aerodynamic diameters". In the first approximation of spherical particles the aerodynamic diameter $= \sqrt{(\text{particle density} \approx 11.5)}$ X (physical diameter). Taking this factor into account various results seem in reasonable agreement, although at very small diameters (smaller than of concern to us) Mann and Kirchner find less deposition than do others. Geesaman's ingenious approximate treatment of the otherwise intractable geometrical problem of dose from hot particles in the DRZ seems reasonable if his picture of the human lung structure is correct. (Although I am not an expert on this, my superficial checking revealed no discrepancies.) Some other lesser (?) details related to the physical dose calculations are given in Section C.

Of a large literature (e.g., Wright Langham compiled 1500 references on Pu toxicity) possibly pertinent to the problem, I have attempted to evaluate only 8 main instances mentioned by Tamplin and Cochran. Obviously, the best scientific evidence would be results from a large number of human lung exposures to chronic alpha particle irradiation under well known conditions. The 8 cases are:

1) (p. 22-26). The work of Albert, Burns and Heimbach (Rad. Res. 30 (1967) 515, 525 and 590) on skin tumors and atrophied hair follicles in rats caused by fast electron irradiation of up to 7 krads over 24 cm^2/rat. Up to 5 tumors/rat at 80 weeks post-irradiation were observed, and regardless of incident electron energy continuous dose response curves were found when the dose was taken as that at 0.27 mm depth*. Furthermore, they observed that the ratio of tumors/rat to atrophied hair follicles was nearly constant — 1/2000 to 1/4000. This ratio seems , but is not explicitly stated, to have been taken as a principal basis for Tamplin and Cochran's interpretation (p. 26) "when a critical architectural unit of a tissue (e.g., a hair follicle) is irradiated at a sufficiently high

*Incidentally, the shape of Albert et al's dose curve suggests that it might be fit by something of the form: tumors/rat $= (AD^n)\, e^{-\alpha D}$, representing a multi-hit ($n = 4$ to 6?) tumor generation and an exponential cell killing.

dosage, the chance of it becoming cancerous is approximately 10^{-3} to 10^{-4}." By a logic not clear to me, by page 36 (if not sooner) this seems to become "a risk of cancer equal to 1/2000 per hot particle". Specifically, I don't understand how the number of hair follicles damaged by large area electron irradiation relates to the volume irradiated by a hot particle and would think protagonists of these exposure limits should make this point clear. One way to estimate the hazard of a 1 micron hot particle would be assume tumor induction proportional to the volume irradiated. Thus from the electron experiment we have no more than (5 tumors)/(24 cm^2 X 0.07 cm average electron range) = 3 tumors/cm^3. For hot particles then we expect 3 (tumors/cm^3) X (65 x 10^{-6} gm/particle X 1 cm^3/gm) = 2 x 10^{-4} tumors/particle. This is a poor calculation (i.e., overestimate of tumors) because it assumes the maximum radiosensitivity at all skin depths and doses. Albert et al concluded that a zone of maximum sensitivity exists at a depth of around 270 μm depth. This is 6 times the range of ^{239}Pu alpha particles in tissue. Albert et al (Rad. Res. 30 (1967) 515) also mention that previous experiments with 0.3 MeV electrons (range perhaps 80 μm), with external alpha particles, and with protons with range of about 170 μm, did not produce tumors. Hence one wonders why Albert et al's data were used to estimate alpha particle hazards quantitatively.

2) (p. 25-26). Templin and Cochran cite experiments in which 16 krad from ^{32}P plaques induced an average of one skin tumor/animal. Again we have the problem of how to relate the effect of a relatively large area beta particle irradiation to that from a hot particle.

3) (p. 27-28). Puncture wound involving 0.08 μg or 5 nCi ^{239}Pu reported by Lushbaugh and J. Langham (reference obscure) who are quoted by Templin and Cochran as: "Although the lesion was minute, the changes in it were severe. Their similarity (italics added by MLR) 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 . . . ". Templin and Cochran rephrase this as: "In this case, less than 0.1 μ of ^{239}Pu produced (italics added by MLR) precancerous changes in human tissue"; then say at the time (about 1960?) there were (known?) less than 1000 puncture wounds and conclude " . . . this wound data would suggest that insoluble plutonium particles could offer a risk of cancer induction in man that is greater than 0.001 per particle." I would ignore this case for attempts at quantitation of lung cancers produced by a single hot particle because: (a) it is uncertain if even precancerous tissue was present; (b) statistics based on but one case are inherently poor; (c) this case involves skin rather than lung cancer and is not quite an external irradiation; and (d) this case involved 5 nCi of ^{239}Pu which is more than 10^4 times that of a standard hot particle.

4) (p. 27-28). Mr. Gleason's possible ^{239}Pu-induced skin cancer. Again the vagaries of the case (such as cited for example 3 and the lack of independent evidence of how much ^{239}Pu got where into the skin or flesh by what mechanism) make this unsuitable for quantitative appraisal.

5) (p. 30). Work of Larkin et al (J. Nat. Cancer Inst. 31 (1963) 219) on intrabronchial tumors in rats caused by exposure to beta particles from ^{106}Ru

is referred to by Tamplin and Cochran but there seems no quantitative use of these data to relate them to the risk associated with a single hot particle.

6) (p. 31-32). The work of Park, Bair and Busch (Health Physics 22 (1972) 803) on lung tumors in beagles produced by inhalation of 0.25 to 0.5μ median diameter $^{239}PuO_2$ particles is cited. Twenty of twenty-one dogs that survived more than 1600 days postexposure had pulmonary neoplasia. The lung burdens ranged from 0.2 to 3.3μCi. Tamplin and Cochran note that "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." As Tamplin and Cochran point out, lung burdens, smaller by orders of magnitude, might also give a high tumor incidence. On the other hand, one might assume a linear tumor to radioactivity incidence and say that since these exposures and tumor incidence both were roughly 3 orders of magnitude greater than the present maximum permissible lung burden and BEIR estimate of annual cancer risk from a 5 rem/year whole body exposure, these data support continued use of the present standards. These lines of reasoning are both merely speculative.

7) (p. 34-37). Rocky Flats fire. Twenty-five workers each received a $^{239}PuO_2$ lung burden which Tamplin and Cohcran calculate as more than 10^4 hot particles. By their analysis each worker should suffer one or more tumors eventually. Investigations during the 9 years since the incident reveal no detectable tumors. Tamplin and Cochran, without citing documentation, suggest that the latent period between exposure and development of cancer is much longer. (We should ask a radiation cancer expert - Hempelmann? - if this is correct.) If so, these exposures may provide valuable information in perhaps 10 years.

8) (p. 38-40) Twenty-five Manhattan Project workers with measurable body burdens of Pu whose health and lung burdens of Pu have been followed for 27 years (Hempelmann, Langham, Richmond and Voely, Health Physics 25 (1973) 461). No clear cases of radiation induced lung tumors were found. Fourteen of 21 workers checked in 1971-72 had chest (equivalent to lung?) burdens of 3-10 nCi, 7 had 7-10 nCi. If these exposures were from 1μm diameter $^{239}PuO_2$ particles, each person would have more than 10^4 hot particles, and by Tamplin and Cochran's estimates one would expect 5 or more tumors/person or a total of perhaps 100 tumors. Tamplin and Cochran argue that under the most frequent circumstances of contamination they estimate the Pu particle masses would be ≤ 0.01 that of a standard hot particle. Hence they discard this evidence.

Since this could be the most direct source of information on lung tumor induction by hot particles in humans that we shall have for a decade, I would think every effort should be made to use it. Should not someone (perhaps an AEC lab?) repeat the contamination experiments (sans human lungs!), measure the particle size distributions and from these estimate the probable numbers of various size particles (in pCi) in the lungs? (Particles of 5μm diameter might classify as hot particles.) Hot particles being a few percent of the total radioactivity or a fraction of a percent of the total number of particles would give an expectation of more than one lung tumor in this population under Tamplin and Cochran's hypothesis.

C. MINOR (?) TECHNICAL CONSIDERATIONS

(more or less in order of appearance in Tamplin and Cochran's report)

1. p. 7 For 1 μm diameter spherical $^{239}PuO_2$ particles, using $\rho = 11.5$ gm/cm^2 and $t_{\frac{1}{2}} = 89$ years, I get around 3.3 rather than 2.2 disintegrations/sec.

2. p. 11 I agree that AEC and/or EPA regulations should include and clearly delineate standards for both radiation workers and population at large. (I thought the regulations did cover both groups.)

3. p. 14. I would have thought that DF should like RBE, be defined as:

$$DF = \frac{\text{Dose of standard (uniform) radiation}}{\text{Dose of test (nonuniform) radiation needed to produce same effect}}$$

This is numerically equivalent to what Tamplin and Cochran give only for linear dose ~~xxx~~ response.

4. p. 16, lines 7-8 "more than 8 orders of magnitude". I get $(11 \times 10^3)/(3 \times 10^{-4}) = 4 \times 10^7$, or more than 7 orders of magnitude.

5. p. 17 line 1. I get $(16 \times 10^{-9})/(2.8 \times 10^{-13}) = 57{,}000$ instead of 53,000 particles.

6. p. 24-26. I would have thought one should compare the number of cells at risk with electron irradiation versus those at risk with hot particle irradiation.

7. p. 33. How reliable is Geesaman's statement that lung repair time is of the order of one year?

8. p. 33. Re: "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". At some dose level (perhaps above 5000 rem/yr) cancer risk may decrease (e.g. Albert et al's data) because of cell killing.

9. p. 48. I would think (a) inclusion of "of other radionuclide" at least superficially sounds good. (b) Addition of lung hot particle burden limit seems good if this really is a major problem. (c) Likewise establishing criteria for other cases of hot particle releases seems ok.

10. Nowhere do I see mention of (a) possibility that $^{239}PuO_2$ particles may "creep" ~~as~~ ^{210}Po is well known to do from recoil as alphas are emitted (for Pu this may be small because of much greater half-life); (b) possible importance of PuO_2 concentration in thoracic lymph nodes.

D. APPENDIX. TENTATIVE ESTIMATE OF MAXIMUM PERMISSIBLE LUNG BURDEN

Here we use Bair's (Adv. Radiation Biology 4, Lett, Adler and Zelle, Eds., Academic Press, 1974, p. 255, especially p. 305) compilation of experimental data to estimate the tumor risk associated with hot particle inhalation sufficient to give various lung doses to experimental animals. Replotting (roughly) Bair's compilation on log-log paper we find that all the experimental date lie below the line "C" and all but two points below the line "R". Equations for these lines in terms of the probability (T) of tumors and cumulative lung dose (D) are:

$$T_c = D/10^2, \quad \text{for } D \leq 100 \text{ rads}$$
$$= 1 \quad \text{for } D \geq 100 \text{ rads}$$
$$T_r = D/10^3 \quad \text{for } D \leq 10^3 \text{ radd}$$
$$= 1 \quad \text{for } D \geq 10^3 \text{ rads}$$

By this conservative or reasonable criteria the cumulative dose at T = 1/2000 is either 0.05 rads or 0.5 rads. Assuming that all these doses are accumulated over a 3 year period and that 1 hot particle ~~xxxxxx~~ gives a dose of 2.7×10^{-5} rads/year in a 1000 gram human lung (this value x (DF = 10) gives Templin and Cochran's value given in their Table III), we get 600 or 6000 particles versus Templin and Cochran's 2.

One might assume that the probability of a lung tumor is dependent only on the number of hot particles in the lung. The cumulative dose (D) for 0.28 pCi $^{239}PuO_2$ particles we calculate as:

$$D = (\text{number of hot particles}) \times \frac{(\text{energy, in ergs, deposited})}{(\text{hot particle} \times \text{years})} \times \frac{1}{(\text{mass of lung in gms})}$$

$$\times\ (\text{years of energy deposition}) \times \frac{(\text{rad-gm})}{(100\ \text{erg})} = N\,(2.7)Y\ /\ 100\ M$$

where N, Y, and M are the number of hot particles, years of exposure, and lung mass. We take Y ≈ 2 years for rodents and 10 years for dogs, and the lung masses as mice 0.5 gms, rats 3 gms, and dogs 100 gms. We then calculate N and replot in Fig. 2 the same data in terms of log tumor incidence versus log N. Linear extrapolation reveals that T = 0.0005 at around 25 hot particles, which suggests that the maximum permissible lung burden should be reduced by a factor of about 10^4. This very tentative conclusion is questionable on such grounds as: (a) why is sensitivity to ^{239}Pu (all points for which in Fig. 2 would appear at less than 10 particles) so different from ^{239}Pu? (b) numbers of hot particles should be calculated more precisely from real size estimates rather than assuming 1 μm diameters; (c) is it fair to extrapolate from mouse to rat to dog to man when the dog and rodent data seem to differ by an order of magnitude in number of particles at maximum effect?

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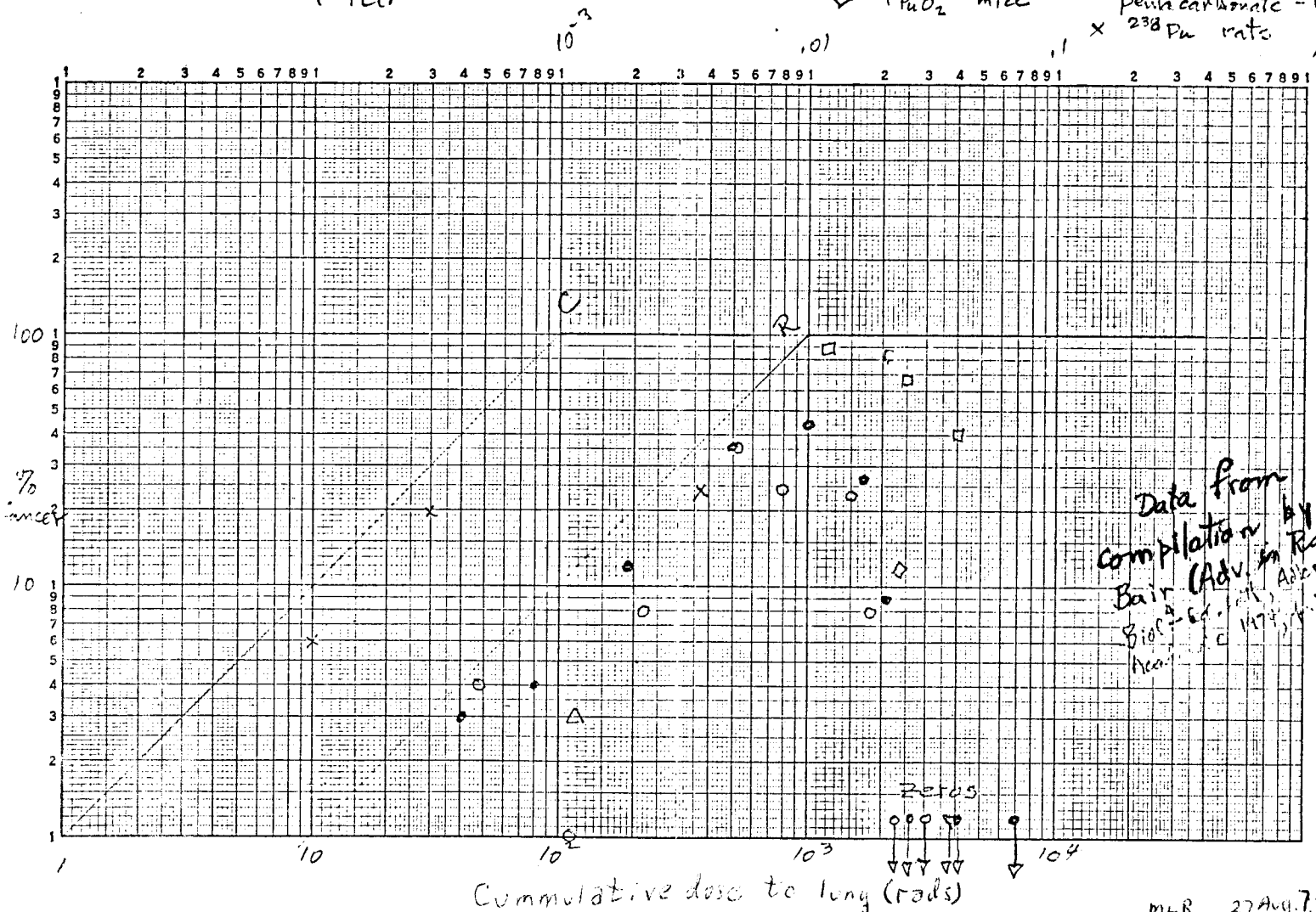
FIG. 1.

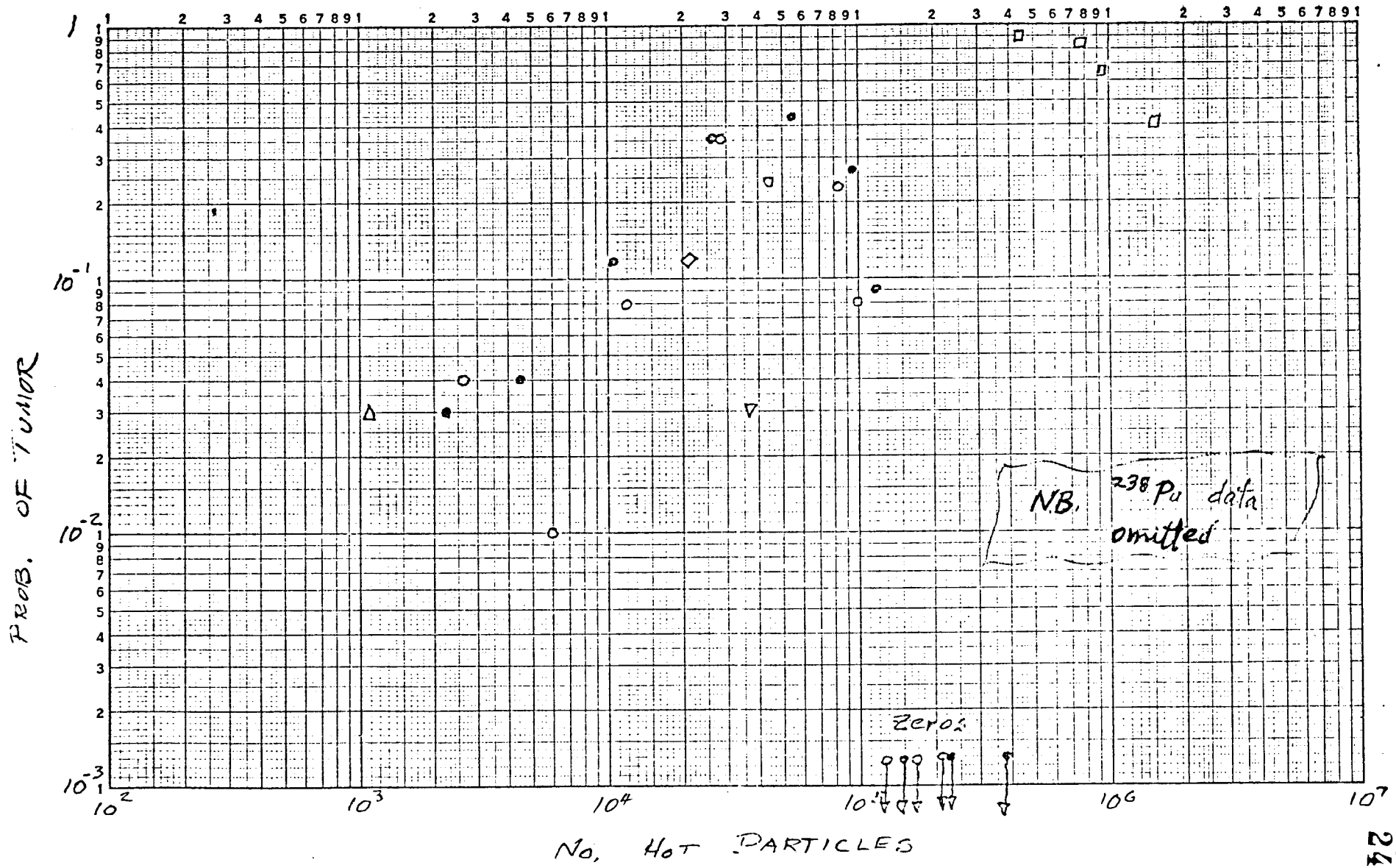
□ $^{239}PuO_2$ dogs
▽ $^{239}PuO_2$ mice
△ $^{239}PuO_2$ mice
◇ $^{239}PuO_2$ mice
○ ^{239}Pu citrate rats
● Ammonium ^{239}Pu pentacarbonate - rats
× ^{238}Pu rats



Data from compilation by Bair (Adv. in Radiat. Biol. Acad. Press 1964, p. 305)

MLR 27 Aug. 74

Randolph



MLR 27 Aug 74

(entitled "A radiobiological assessment of the spatial distribution of radiation dose from inhaled plutonium" (Wash-1320), 1994

August 18, 1974

Louis Hempelmann Response to the Tamplin-Cochran Report on Radiation Standards for Hot Particles (February 14, 1974)

The Tamplin-Cochran Report is filled with misleading information of the following type:

1. They imply in a quotation from Geesaman that Bair and his associates at Batelle have concluded that non-uniform irradiation of the lung is clearly more carcinogenic than the same total dose of radiation uniformly distributed throughout the lung. As a matter of fact, Bair is the senior author of an extensive AEC review (~~about to be published~~) that comes to the exact opposite conclusion.

2. They imply that Mr. Edward Gleason's synovial sarcoma of the hand was the result of accidental plutonium contamination by a leaking plutonium carboy but offer no evidence that the plutonium penetrated the skin. Plutonium could have reached the synovial sheath only by way of a deep penetrating wound which Mr. Gleason would have surely remembered. Thousands of plutonium workers have had contaminated wounds sometimes incompletely excised without developing cancer. Synovial sarcoma is a rare tumor but it has developed in many people who have had no plutonium exposure.

3. They quote the Lushbaugh-Langham paper as proof that a single plutonium particle containing 5 nCi of activity can cause cancer. The paper actually points out the similarity of the cellular reaction in the vicinity of the particle and precancerous changes in epidermal tissue and raises the question as to the ultimate fate of the lesion if it were not excised. One of the Los Alamos workers has had six nannocuries of plutonium embedded in the skin of his hand for 30 years without developing cancer.

Tamplin and Cochran make the following assumptions that I do not agree with or cannot understand:

1. They assume that the Albert data (1967) on skin cancer induced by exposure of large areas of rat skin (up to 24 cm^2) can be applied directly to small volumes of human lung irradiated by point sources. In the first place, the skin cancers are derived from atrophic hair follicles and there are no comparable architectural structures in human lung. The original data cannot even be applied quantitatively to mouse skin (Albert, 1972) or to other strains of rats (Albert, 1961), much less to human lung.

2. The derivation of the risk value for lung cancer of 10^{-3} - 10^{-4} per single "hot" particle is incomprehensible to me, even though I have read the passage at least ten times. It seems to me that they jump from a risk value for skin cancer in rats of 2-4 x 10^{-3} per atrophic hair follicle to a risk of lung cancer in man of 10^{-3} to 10^{-4} per hot particle. There is no justification of the assumption that both risk values are of the same order of magnitude.

3. The lung burdens of the 25 Rocky Flat workers most heavily exposed to the plutonium fires range from the presently accepted values for maximum permissible lung burden to ten times this value. These burdens are 115,000 to 1,500,000 times larger than the permissible lung burdens recommended by Tamplin and Cochran. It is well known that the higher the dose, the shorter the latent period for tumor induction. Since lung cancers have been reported in uranium miners after a latent period of less than ten years, we would certainly expect to find many lung tumors resulting from the nine year exposure of Rocky Flats personnel if the true MPLB were of the order of magnitude recommended by Tamplin and Cochran.

4. Tamplin and Cochran dismiss the data on Los Alamos plutonium workers with measurable lung burdens for 30 years as not pertinent to the "hot" particle problem. Assuming that all exposures were due to inhalation of droplets of a plutonium

solution involved in one chemical operation, they make a simple calculation to show that none of the inhaled droplets contained enough plutonium to be considered as hot particles (more than 0.07 pCi). Using a much more sophisticated calculation involving particle size distribution of plutonium particles collected under working conditions, Los Alamos scientists estimated that each person could have inhaled as many as 4×10^5 hot particles (E.C. Anderson, personal communication). If the Templin and Cochran risk value of 10^{-4} were correct, we would have expected 40 lung cancers per man. Instead no cases of cancer have occurred in the 30 years since the inhalation of the radioactivity.

There is good experimental evidence that non-uniform irradiation of rat skin by beta rays or of hamster lung by alpha rays or rat lung by beta rays is less carcinogenic than uniform irradiation with the same doses. Reference is made to the following experiments:

1. Irradiation of rat skin by the same dose of beta rays from point sources of different strength or from a uniform flat source (Passanneau et al, 1952).
2. Irradiation of hamster lung by alpha rays from $polonium^{210}$ in solution or absorbed on hematite particles (Grossman et al, 1971; Little et al, 1973).
3. Irradiation of hamster lung by alpha particles from $Pu^{239}O_2$ in zirconium oxide microspheres embedded in pulmonary capillaries (Richmond et al, 1970; E.C. Anderson , personal communication).
4. Irradiation of rat lung by beta rays from $Ba^{35}SO_4$ in a relatively uniform manner and from Sr^{90} embedded in glass beads (Cember and Watson, 1958 a and b).

The results of all these studies definitely contradict the simplistic models of Geesaman (1968) and Dean and Langham (1969), which assume that tumor induction can be calculated solely on the basis of cellular radiation exposure.

In conclusion, the question of hot particles and non-uniform radiation has been thoroughly considered on a number of occasions by committees of knowledgeable scientists of the ICRP, NRCP and NAS-NRC. On the basis of the evidence or assumptions presented in the Tamplin-Cochran Report, I see no reason to modify the current standards of radiation protection recommended by these committees.

Respectfully submitted

References not in Tamplin-Cochran Report:

Albert, R.E., Burns, F.J. and Hermback, R.P. Radiation Research 1967 30:590.
Albert, R.E., Newman, W. and Anshuler, B. Rad. Res. 1961, 15: 410.
Albert, R.E., Burns, F.J. and Bennett, P. J. Nat. Canc. Inst. 1972 49: 1131.
Anderson, E.C., Halland, L.M., Prine, J.R. and Richmond, C.R. To be published in the Proceedings of the Symposium on Experimental Respiratory Carcinogenesis and Bioassays held at the Battelle Seattle Research Center, June 23-26, 1974.
Cumber, H. and Watson, J.A. Amer. Indust. Hygiene 1958a, 19: 36.
Cumber, H. and Watson, J.S. AMA Arch. Induct. Health 1958b, 14: 230.
Grossman, B.N., Little, J.B. and O'Toole, W.F. Rad. Res. 47: 253 (abstract).
Little, J.B., Grossman, B.N. and O'Toole, W.F. In: Radiation Carcinogenesis (C.L. Sanders, J.E. Ballow and D.D. Mahlum, Eds.) Cont. 720509: 19 AEC Symposium Series no. 29 1973
Richmond, C.R., Langham, J. and Stone, R.S. Health Physics 1970, 18: 401.

Comments on "Radiation Standards for Hot Particles" by A.R. Tamplin and T.B. Cochran

(Comments by Andrew M. Rauth)

My general reaction in reading this report was that it was well written and informative. I would like to read the opinions of those workers whose data is cited to the final conclusion that the Maximum Permissable Lung Particle Burden (MPLPB) be set at 2 particles. My reservations are based on the fact that none of the data cited is the experimental work of the authors A.R. Tamplin or T.B. Cochran. Since this report is a synthesis and interpretation of the work of others, it is subject to a greater possibility of out of context statements than a single scientific presentation of the authors' own work. The following specific points struck me as important.

On page 13, the authors wish to calculate the dose equivalent (DE) for Pu^{239} which they say is obtained by multiplying the absorbed dose in rads by the modifying Quality Factor (QF) for Plutonium α particles of 10 and a Distribution Factor (DF) which is presently taken as 1 for the lung, but which the authors indicate should be 115,000. As far as I can see this choice of increase in DF is based primarily on two experimental pieces of information, the work of Albert and coworkers cited on pages 22-24 on electron irradiation of rat skin, and the work of Bair on $Pu^{239}O_2$ inhalation in Beagles, which together are interpreted to indicate one hot particle has a 1/1000 to 1/10,000 chance of causing cancer when trapped in the lung.

The authors make no comments on the Albert data on the facts that

1) this is a macroscopic tissue irradiation (24 cm^2) in a single acute dose.
2) Not only does the tumor incidence go up at 1,000 rads, but it also goes down at doses above 2,000 rads.

The insoluble hot particle problem the authors deal with is one of very small (100 μ diameter) radiation fields subject to chronic rather than acute irradiation.

In addition high radiation doses to microscopic tissue areas might cause cell sterilization as well as carcinogenic changes. In light of these problems their paraphrase of the "Geesaman hypothesis," on p. 26 that gives a chance of a cancerous change of 10^{-3} to 10^{-4} for critical volume high dose irradiation may or may not be valid. For example, radiation therapy patients receive large skin doses during fractionated radiation without skin tumors appearing.

Bair's work with Beagles and $Pu^{239}O_2$ inhalation is also of interest in this regard. His work (ref. 54, Park et al) is part of a whole series of papers appearing together in Health Physics on this problem. (22, pp.533-957) As the authors point out, the smallest burden (at death) in a dog showing lung cancer was 0.2 μCi which was estimated to be a particle burden of about 10^7 particles, a very large number in relation to the proposed limits in man. As indicated in this article (ref.54), studies were underway in 1972 on lower burdens down to .002 μCi which may in time give some dose dependence data. It is also of interest in this paper that a great deal of concern is expressed about dosage to draining lymph nodes as well as lung.

Obviously one can argue further pros and cons on much of the data cited. The conclusion I came to after such superficial considerations is,

1) The authors Tamplin and Cochran have delineated a potentially important radiation protection area.
2) Like many such controversial areas, the final decision on exposure standards should be based,in part,on the recommendations of people actually working in the field who have first hand knowledge of the data.
3) I could support the concern implicit in the report qualitatively but not quantitatively on the basis of the data presented. Obviously the least risk is no exposure, but this must be balanced out by practical benefits. No indication of the practicability of the proposed limits

is indicated.

4) A broader base of informed scientific support should accompany this report before its detailed recommendations should be submitted for serious consideration. For example, what do people whose papers appear in the Hanford Symposium (Health Physics 22, 533, 1972) say about such a proposal?

REVIEW OF REPORT ON RADIATION STANDARDS FOR HOT PARTICLES

By Arthur R. Tamplin and Thomas B. Cochran

(Comments by Arthur Cole)

The authors of the report present a valid statement that hot particle exposure guides should not be based on standards established for uniform radiation exposures. In spite of the fact that many specific assumptions and extrapolations do not appear to be justified, the data which they cite certainly suggests that a reduction in the present standards is indicated.

There are a number of specific points in the report that are of particular interest.

P. 26. The "critical architectural unit" is an attractive and simplifying hypothesis. However, little evidence is available to support it. For example, multiple insults at different sites can be invoked in carcinogenesis.

Pp. 27-28. The Lushbaugh study of one observed lesion in 1,000 puncture wounds provides no statistical basis for estimating a tumor induction probability.

P. 36. The Mann and Kirchner study summarized in Table V probably represents the most critical source of human data available, and an effort should be made to follow up these studies, including those personnel exposed at lower levels.

P. 41. Section VII. To say that the existing evidence "strongly suggests that a (single) hot particle ---- represents a (cancer) risk of 10^{-3} and 10^{-4}", is not justified. Present available data appears to suggest a possible range of between 10^{-3} and 10^{-6}.

I was curious to play with some numbers in a naive way to compare with the sort of numerology given in the report. For example, Albert's data

(Radiat. Res. 30:515) indicates that, at the optimum induction dose of about 2 krads, 5 tumors were induced per 24 cm^2 skin exposed to short range electrons. If we assume a sensitive (critical ?) tissue thickness of 0.2 mm., this gives 0.5 g. of tissue at risk. This comes to 1 tumor induced per 10^{-1} g. of tissue at risk (2 krad dose). A very general and poorly justified extrapolation of this result might be applied to lung aveoli exposure by an "ideal" hot particle that delivers an optimum dose to comparably sensitive cells. If the paticle were attached to the inner surface of an aveoli having an inner diameter of about 150 μm (filled with air), the total tissue mass exposed to alpha particles would be about 10^{-5} g., assuming a 50 μm. particle range in unit density. Thus, Albert's data suggests that; Aveoli tumors per ideal hot

$$\text{particle} = \frac{10^{-5} \text{ g. sensitive tissue}}{10^{-1} \text{ g. sensitive tissue per tumor}}$$

$$= 10^{-4}$$

Imposing a Quality Factor of from one to ten for tumor induction by high LET α-particles gives a range for tumors induced per ideal hot particle of 10^{-4} to 10^{-3}, which is in agreement with Geesaman's assumption. However, this probability applies to those hot particles which deliver optimal tumorogenic doses and which are ideally located within critical structures. Presumably, other doses or locations would yield smaller probabilities.

Blair's data is more directly applicable to the problem, although a different kind of extrapolation must be made. Essentially all the dogs developed (multicentric) tumors after exposures of 10^7 hot particles and greater. If we assume that a reasonably large probability (approaching unity) would still occur for only 10^6 hot particles per dog and also assume a linear induction response, then the probability for tumor induction per hot particle

would be in the order of 10^{-6}. On the basis of this experiment alone, one would like to limit hot particles per man to 10^{3} for occupational exposures and 10^{2} for general population exposures in order to safely restrict the chance for tumor induction to 10^{-3} and 10^{-4} in each category. This represents a reduction of about 50 fold in current standards.

I recently discussed the hot particle problem with Dr. Doris Dugas and Gerry Huth at UCLA and have enclosed a letter which they had submitted to Science in addition to Dr. Dugas' personal review of the Tamplin-Cochran report. I think these comments are useful and pertinent.

Final comments: It is obvious that more experimental work need be done. A dog experiment at lower burden levels would certainly be appropriate. Particle size effects should be reevaluated. The double trauma hypothesis of Huth and Dugas should be considered. Hopefully, the personnel exposed to the Rocky Flats accident will be monitored. In addition, a rather simple study of cell transformation in cell culture induced by hot particles attached to the culture vessel would be a useful adjunct to the animal studies.

CONCERNING THE REPORT "RADIATION STANDARDS FOR HOT PARTICLES"

A.R. Tamplin and T.B. Cochran, NRDC, February 14, 1974

(Comments by Doris J. Dugas)

General Comment: The basic intention of the report is a valid one, namely to re-evaluate the maximum permissible lung burden for the special case of inhaled insoluble alpha-emitting particles. However, the data presented by this report does not adequately support the quantitative conclusions. Specific reasons are given below.

Section V-A, p. 22: The Geesaman hypothesis generalizes too much from R.E. Albert's experimental data on rat skin. While it is true that the number of tumors and number of atrophied hairs were correlated, this does not necessarily mean that one was causative of the other. (We do not know what event caused the hair follicles to atrophy.) Furthermore, even if we accept the hair follicle as the "critical structure" and the ratio of number of tumors/number of atrophied hair follicles as a measure of tumor risk for rat skin, how can this be applied to areas of the body which are not hairy (such as the lung) and therefore have none of the so-called critical structures?

V-B, p. 26: The case of a man who had a puncture wound contaminated with .005 μCi of plutonium-239 is cited. Cytological changes suspicious of cancer were observed near the radioactive particle. Since there were less than 1000 other cases of contaminated wounds at the time, and apparently no others developed tumors, the authors deduce that the risk of cancer from plutonium contaminated wounds in man must be $\frac{1}{<1000}$, or something >.001/particle. Obviously, with only one case reported, there is little justification for establishing this as a statistical risk.

V-C, p. 29: The various animal experiments show that internal radioactivity can cause cancer, but do little to help quantify the risk per particle. From the authors' own figures, the risk calculated from the beagle dog data is some unknown amount greater than $1/10^7$. What they should be saying, I think, is that the risk/particle is one *if* the particle is located in the right place, and what needs to be determined is the probability of a particle being located in that place.

VI, p. 32: The statement that epithelial tissue repair time in the lung in one year was not really established by Phillip's experiment, because no damage was observed in one year. Yet this figure is a critical part of their

calculation of risk/particle.

VI-A, p. 42: The calculation of numbers of particles in plutonium workers from whole body counting seems unnecessary, especially after the statement that most particles will be located in the lymph nodes anyway. In aggregate, the particles may be delivering high doses to the nodes, and it would not matter if each particle "qualified as hot" individually in size or activity.

Table VI, p. 43: The calculation of risk/particle assumes three things:

1) That 1 cancer occurs per 1000 to 10,000 critical structures irradiated. This is taken from Albert's data on β-irradiated rat skin.

2) That 1 year is the repair time for lung epithelium. This is Geesaman's interpretation of Phillip's experiment.

3) That 1000 rem is the critical dose to tissue to induce cancer. This is from Albert's data.

Although the author's allow a factor of 10 uncertainty in the first assumption, I believe that there may be one or more orders of magnitude uncertainty in each of the assumptions, which when combined, give a tremendous uncertainty in the final calculation of permissible burden. The author's also

implicitly assume that each particle acts independently, which seems contrary to the evidence that most of the inhaled particles move to the tracheobronchial lymph nodes, forming an aggregate source.

In summary then, it seems to me that the author's quantitative result for the maximum permissible particle burden as calculated from this data is highly suspect. However, I would again emphasize that their basic tenet is sound, i.e. that current MPLB's which are based on average organ doses, are not valid for inhaled radioactive particles. Further investigations should be made to establish appropriate standards for such particles.

Richard P. Spencer COMMENTS ON STANDARDS FOR HOT PARTICLES

The crucial point is summarized on page 6 of the "Petition To Amend Radiation Protection Standards As They Apply To Hot Particles", in that "... the risk of cancer from a single hot particle in the lung should be considered equal to one chance in 2,000." The entire discussion comes down to whether non-uniform radiation to the lungs is more or less dangerous to the recipient than uniform radiation.

A. An intense local radiation dose would likely kill adjacent cells.

B. Slightly more peripheral cells, damaged by the radiation, could likely become carcinogenic.

A human experiment to look at this, has (unfortunately) been carried out. I am referring to the plutonium fire at Rocky Flats (October 15, 1965). Since it has been nearly 9 years since that accident, there might be a wealth of data on the individuals (and lung biopsies would be invaluable).

The mass of evidence, that the permissible hot particle load in the lungs should be revised downward is impressive. We urgently need any and all data on the individuals exposed at Rocky Flats in order to render a final view.

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IN REPLY
REFER TO: H

November 22, 1974

Dr. William A. Mills
Director, Criteria & Standards Division (HM-560)
Office of Radiation Programs, EPA
Waterside Mall, E-635
401 M Street, S. W.
Washington, D. C. 20460

Dear Dr. Mills:

In view of the hearings to be conducted by the Office of Radiation Programs of the Environmental Protection Agency to consider whether new guidelines or standards for transuranium elements are needed, I wanted to inform you of several items which will not be completed in time for consideration but which may be of interest. The first is an analysis of the report submitted by Drs. A. Tamplin and T. Cochran in support of the NRDC petition to lower limits for insoluble alpha emitting particles. This is now being printed and initial copies will be available about the first week in December. We will send you several copies as soon as they are available.

The second item is the publication of the symposium, "Plutonium - Health Implications for Man," which was held at Los Alamos at the end of May. We were very pleased to have the participation of individuals from EPA and believe that the papers and discussions at that meeting could be pertinent to your objectives. Unfortunately, the material will not go to the publishers until about the middle of December and it will be about five months later before it will appear in "Health Physics." If, in the meantime, there is any of the materi from that symposium that would be useful to you in your deliberations we would be happy to cooperate.

While we do not plan to present a statement at your hearings, we do plan to have a representative present. If he can be of any use to you, please call on him.

Sincerely yours,

J. W. Healy

J. W. Healy

JWH/ed

AN EQUAL OPPORTUNITY EMPLOYER

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IN REPLY
REFER TO: H

December 24, 1974

Dr. William A. Mills
Criteria and Standards Division
U. S. Environmental Protection Agency
401 M Street, S. W.
Washington, D. C. 20460

Dear Bill:

I am submitting the enclosed report, "A Proposed Interim Standard for Plutonium in Soils," LA-5483-MS, for inclusion in the record of your hearings on plutonium. I plan to continue work in this area and will be extremely interested in the conclusions of your hearing panel and the actions of EPA.

I am also enclosing a copy of LA-5810-MS, "A Review of the Natural Resources Defense Council Petition Concerning Limits for Insoluble Alpha Emitters," which Chet Richmond mentioned in his letter of December 17, 1974.

Sincerely yours,

J. W. Healy

J. W. Healy

JWH/ed
Enclosures: a/s

AN EQUAL OPPORTUNITY EMPLOYER

A PROPOSED INTERIM STANDARD FOR PLUTONIUM IN SOILS

by

J. W. Healy

ABSTRACT

Current standards for controlling health effects from plutonium in the body are discussed. Available information on possible sources of exposure of people living in an area where the soils are contaminated with plutonium is analyzed to arrive at estimates of intake. From these estimates, a recommended interim standard for the upper limit of concentration of plutonium in the soils in inhabited areas is derived. The recommendation is based upon conservative assumptions where information is lacking and further studies should result in revision. The subjects of resuspension, deposition velocity of particles and effectiveness of radioactive particulates in producing lung cancer are discussed in appendices.

I. INTRODUCTION

Plutonium has been utilized and processed in relatively large quantities (hundreds to thousands of kilograms total) in several different countries over the past three decades. It now can be found in small quantities in soils and oceans over the entire world as a result of widespread dissemination from nuclear weapons tests in the atmosphere and one burn-up of a space nuclear power generator containing ^{238}Pu. More localized distributions are found in the immediate vicinity of facilities used for processing plutonium, at the locale of several accidents involving weapons containing plutonium, and in remote areas which were used for safety tests with weapons. In each of these areas, some measure of potential hazard is necessary to enable adequate decisions as to future disposition of the area or any special considerations on habitation or land use restrictions. Thus, it is necessary to have some indication of the degree of hazard associated with various levels of plutonium so that such decisions can be adequately based.

The following study was commissioned by the Division of Operational Safety of the U. S. Atomic Energy Commission to provide an interim or provisional standard for plutonium in the soil to meet this need. It was recognized that the problem of establishing such a standard is very complex due to the many potential mechanisms of exposure from this source and that the data available for detailed definition of the problem are inadequate. However, the necessity of making decisions on acceptable levels requires that some guidance be made available for comparison with measurements made in potentially contaminated areas. To answer this need, it was decided to apply the information now available and to arrive at a standard which, while overly conservative from a hazard

standpoint, would give some guidance in making these decisions. At the same time, it was felt that such a study would permit assessment of the information available so that future research and development programs could be more effectively aimed at the areas of greatest uncertainty.

It is stressed that a conscious effort has been made to err on the conservative side in view of the many uncertainties. (In this case, the conservative side is defined as the over-estimation of exposure from the plutonium in the soils.) In addition, the depth of investigation and the conditions considered have been limited in a number of possibly important areas in an attempt to arrive at some guidance as soon as possible. For these reasons, it is urged that the numerical values derived herein be regarded as truly provisional and not be incorporated into rules and regulations which are difficult to change. It is anticipated that changes in the numbers, and perhaps the concepts, will be forthcoming from future work.

In the derivation of the numerical guidance, we have considered primarily considerations of health and hazard to man. In recent years there has been a tendency to derive such standards based upon the practicality of achievement rather than upon effects on health. While such standards have their rightful place in providing control of sources of pollution, there is a tendency to regard them as safety standards so that exceeding them becomes a matter of great concern. It is also of importance, even on the practicality basis, that an upper limit be clearly established, as based on safety, so that one can assure that the practical limits are, indeed, safe, and that the additional margin of safety attained by lower limits can be assessed in comparison to the costs in resources and manpower of achieving them. With this philosophy we have not provided two standards, one for control of sources on a continuing basis and one for the application of countermeasures in an area already contaminated, although such considerations are appropriate to any safety program and in the application of a standard such as the one derived here. Again, as better information becomes available and we reach a stage where intelligent and informed assessment of actual risks at various levels of plutonium in the soils are possible, such considerations will be included.

Supplemental information on several items not covered in detail in the literature are given in appendices. These include a model for calculating resuspension of particulates in Appendix A, a treatment of the deposition velocity for particles in Appendix B, and a discussion of the effectiveness of radioactive particles in producing lung cancer in Appendix C.

II. PLUTONIUM STANDARDS

A. Properties

Plutonium is not a simple material. It is a man-made element in which the isotopic composition, and thus, the radioactive properties, vary widely depending upon the history of its production and any subsequent neutron exposure as a reactor fuel or in a detonation. There is increasing evidence that the metabolic behavior and, in some situations, the gross chemical behavior may vary with specific activity of the isotope probably because of the influence of the energy emitted as radiation as well as mass effects. The isotopic compositions of several typical mixtures are given in Table I.

The compositions given in Table I are illustrative rather than definitive with wide variations possible, particularly in the materials used for power fuels. The low irradiation material is reasonably representative of that used in the weapons programs of the AEC which has utilized a large fraction of the plutonium produced in the past.

Since the isotopes of plutonium are primarily alpha emitters with little accompanying penetrating radiations, the hazard associated with plutonium is almost completely due to potential intake into the body. Plutonium-241 is a beta emitter but,

TABLE I

PLUTONIUM MIXTURES

Isotope	Principal[1] Emission	$T_{1/2}$ yrs	Specific Activity Ci/g	Low Irradiation wt %	Pu Recycle in LWR wt %	Heat[2] Source wt %
^{236}Pu	α	2.85	532	----	5×10^{-6}	10^{-4}
^{238}Pu	α	86.4	17.4	0.0115	2.9	80.3
^{239}Pu	α	24,390	0.0614	93.6	39.6	15.87
^{240}Pu	α	6,580	0.226	5.9	25.6	3.00
*^{241}Pu	β (0.02 MeV)	13.2	112	0.4	16.8	0.72
^{242}Pu	α	3.79×10^{5}	3.9×10^{-3}	0.013	15.0	----
^{244}Pu	α	7.6×10^{7}	1.9×10^{-5}	0.02	------	----
Specific activity (alpha) of mixture (Ci/g)				0.073	0.059	14
Specific activity with ^{241}Am ingrowth (Ci/g)				0.084	1.08	14

* Daughter is ^{241}Am, an alpha emitter, with half-life of 458 years and a specific activity of 3.24 Ci/g. This will reach a maximum from the ^{241}Pu in about 70 years with one gram of ^{241}Pu resulting in 2.91 Ci of ^{241}Am.

again, the energy of the beta particle is low enough that self-absorption and small penetration into the body makes the external dose insignificant. It is true that massive quantities of plutonium, as encountered in fuel fabrication plants or other facilities handling large quantities of plutonium, pose some problems in control of external exposures to workers, particularly as the quantities of isotopes of higher mass than 239 increase in heavily irradiated fuel materials. However, in the quantities expected in soils, these external radiations are of no significance in comparison to an internal uptake. Thus, our concern with the properties of plutonium is limited to those which will influence intake.

On an overview basis, plutonium is probably not as bad an actor in the environment as many other isotopes because of its relative insolubility. As a result, it is not taken up to any large extent in the ecosystems so that transfer by biological mechanisms is usually minimal particularly for plutonium in soils. Although there are measurements indicating some concentration in marine organisms,[3,4] none seem to indicate anything other than biological discrimination in plants and animals on contaminated soils. It must be noted, however, that experience in this regard is relatively limited and some mechanisms for biological uptake in terrestrial situations may occur, even if only in limited areas where the soil and biological conditions are proper. For example, the action of natural chelating agents in the soils may result in compounds which could be biologically more active. However, with the information now available, it appears that for purposes of this interim standard, the physical modes of transport and intake are of the most importance.

One further reservation on the behavior of a mixture of isotopes in the environment relates to the eventual buildup of ^{241}Am. This isotope begins to appear in significant quantities from ^{241}Pu mixtures within a few months to years. While the assumption is frequently made that all of the transuranic elements have similar metabolic behavior (as in ICRP 2),[5] this assumption was based primarily on the need for MPC's to be used for control purposes. The chemical properties of americium

are, indeed, different from those of plutonium particularly in the tendency of plutonium to produce insoluble polymers and one would expect the ecological behavior to be different. Some measurements have indicated a much higher uptake of ^{241}Am by plants[6] while others have shown the transfer from plasma to milk[7] to be higher than for plutonium. Since the contaminated areas now of interest have resulted from plutonium with low ^{241}Pu content, this has not been an important consideration. As information becomes available, the importance of this factor to the interim standard will be assessed.

B. Basic Limitations on Plutonium in Humans

As a basis for the potential harm to humans from the intake and deposition of plutonium we will use the current standards as recommended by the NCRP and ICRP. These were basically derived for occupational exposures and are applicable primarily to adults in reasonably good health. In application to populations they are reduced to allow for the lower risk which should be applicable to such groups and to provide a margin for children or ill individuals. A brief review of the origin of the occupational standards is given in this section to provide a basis for the application to population groups in the next section.

It will be noted that we have not based our studies on estimates of the risk to individuals in spite of the fact that this approach is advocated by many people. Such estimates, even for low LET radiation, require many assumptions and are based upon data which have a wide range of uncertainties. As a result, the estimates reflect more the individual assumptions and interpretations than they do the real risk. There is a wide difference between arriving at a value which the evidence indicates is "safe" without attempting to quantify this term and in providing a quantitative, numerical value for the risk. In the former case, the informed judgment of people who have studied the information available can be used. In the latter case, a mechanistic calculation is substituted with judgments on the assumptions compounding the uncertainties in the final number. It is true that value judgments as to "how safe is safe" are required for the non-numerical method but the general agreement among bodies as diverse as the NCRP,[8] the ICRP,[9] and the Federal Radiation Council[10] would indicate a remarkable similarity in such value judgments in spite of the differences in objectives and composition of these groups. In the case of alpha emitters, such as plutonium, we would also note that the uncertainty in the risk estimates may be greater than for low LET radiation because of the uncertain RBE to be applied and the apparent lack of repair of damage from these high LET radiations. (Note that the rem should not be applied in such risk estimates since this unit is defined for use in radiation protection and uses the Quality Factor which is arbitrarily assigned as based on a conservative estimate of all effects.)[11] The rem is intended for control of radiation exposures and not for estimates of risk.

1. Body Burden. The basic standard for plutonium absorbed into the body (i.e., outside of the lung or other site of initial deposition) is 0.04 μCi for occupational exposure. This value was derived by biological comparison of the late effects when injected into animals with those of radium for which a significant body of information on the effects in humans exists. A recent review of the derivation of this value and its application to obtaining maximum permissible concentrations was made by Langham and Healy.[12] The value resulted from the work of Brues[13] at the Argonne National Laboratory, in which known quantities of both plutonium and radium were injected into animals and the comparative late damage noted. As a result of these experiments, it was determined that the relative toxicity of plutonium is about 15 times that of radium-226 on the basis of equal injected doses (microcuries). In the rodents used, the retention of plutonium was about 75% while that

of radium was about 25%. On a retained basis, this would lead to the conclusion that plutonium is about five times as toxic per microcurie; however, a large part of the energy delivered from radium results from the radon daughters. In these animals the retention of the radon was about 15-20% as compared to about 50% for man. Thus, in man, the higher radon retention would lead to expectation of increased damage for the radium in comparison to the plutonium. The relative toxicity of the plutonium per microcurie retained would be expected to be lower by about a factor of two or about 2.5 times that of radon. Thus, for man, the maximum permissible body burden is 0.04 microcuries. However, on an energy delivered basis, the energy from the plutonium alpha particles is five times as toxic as that from radium since the total energy from the radium is about twice that of the plutonium in man. The increased effectiveness of the plutonium energy has been attributed to the fact that the plutonium is not as uniformly distributed through the bone matrix as radium (although radium is not uniformly distributed) tending to concentrate on the surfaces so that a smaller portion, and perhaps more sensitive portion, of the bone receives a higher insult. Similar experiments performed at the same time with ^{89}Sr gave results similar to these and the increased effectiveness of these two materials on an energy delivered basis has been generalized to the "dose distribution factor" of five which has been applied to all bone seekers except radium.

The value of 0.04 μCi was first derived at, and immediately following, the Chalk River Conference in 1949[14] and still remains as the primary standard for plutonium in the body. Additional studies with dogs at the University of Utah[15] have essentially confirmed the number although the Utah results indicate that the relative toxicity on an energy basis may be closer to ten than five and have demonstrated that other organs may have significant uptake and retention of plutonium depending upon the path of administration. In their experiments, the plutonium was administered intravenously in a citrate solution and the liver appeared to be a major site of deposition although the majority of late effects noted seemed to be primarily involved with bone. Studies of the effects of plutonium on animals continues and it is anticipated that some revision of the 0.04 μCi value may occur in the next few years, but a major change, for reasons of health effects, is not anticipated. (The qualification on health effects is necessary since there is a growing tendency to base such standards on practicality of attainment rather than potential damage. For example, the FRC recommendations[16] for the intake of ^{226}Ra and ^{90}Sr are based on their conclusion that operations can be carried out without exceeding the recommended intake. In application of these standards it is important to recognize the basis.) There are now some human data[17] based on exposures of 27 individuals in 1944 and 1945 (28 years). Estimates of the body burden by urine analysis are uncertain, but the latest analysis of the data indicates that 60-70% of the individuals have plutonium burdens at or above the 0.04 μCi level with the maximum individual perhaps 5-10 times this value. Followup medical examinations have shown no changes which could be attributed to this plutonium. While the sample is small and the time is relatively short in comparison to the life span of man, these data are encouraging in that they indicate no gross problem such as occurred with radium.

It should be noted that this derivation is based directly on biological evidence of damage and does not utilize the concept of radiation dose except indirectly in the comparison of energy delivered by the two materials. There has been an attempt to fit the derived value into the overall framework of dose calculations with the result that the original basis for the number and the meaning of the derived numbers is not always clear. For example, in their 1959 report on internal emitters,[5] the ICRP presented the concept as follows: "The effective

RBE dose delivered to the bone from internal or external radiation during any 13 week period averaged over the entire skeleton shall not exceed the average RBE dose to the skeleton due to a body burden of 0.1 μCi of ^{226}Ra (derived from a dose rate of 0.06 rad/week, an RBE of 10 and n = 1)." In this statement, n is the so-called dose distribution factor and corresponds to the value of five on an energy basis derived from the reasoning described above for plutonium. The dose rate from 0.1 μCi of ^{226}Ra retained in the body was obtained assuming that 99% of the radium in the body was in the bone, the mineralized portion of the bone weighing 7000 grams was the appropriate organ, 30% of the radon daughters were retained in the bone and a quality factor of ten was appropriate to describe the LET effects of the alpha particles. In this calculation, 0.04 μCi of plutonium in the body with 90% in the bone would deliver an average dose rate of 0.5 rads per year to the mineralized portion of the bone or, with a quality factor of ten and a dose distribution factor of five, about 25 rems per year which, within the accuracy of the estimate, is the same as the radium value of 30 rems per year.

2. Lung Burden. The basic limitation to the lung for workers is a dose equivalent rate of 15 rems per year as derived from the experience with external radiation exposure and the application of the critical organ concept first set forth by the NCRP.[18] This translates, for a 1000 gram lung, to a lung burden of 0.016 μCi of plutonium based on the average dose to the entire lung. However, in contrast to plutonium mobilized into the body which is retained with great tenacity, the lung has elimination mechanisms which serve to remove plutonium or other materials. As a result, the total dose delivered by a given deposit is limited by the time of retention of the material in the lung. In addition, the deposition of material in the lung is strongly affected by a number of factors, the most important of which is undoubtedly the effective particle size. The ultimate fate of the material deposited in the lung must be considered in relation to the radiation dose received by other parts of the body. These questions of intake and retention will be discussed in the next section of the report.

The applicability of a dose calculated on the basis of the average dose to the lung (i.e., total energy delivered divided by the total weight of the organ without regard to the distribution of the energy within the organ) is frequently questioned on the basis that the plutonium particulates will produce "hot spots" where the local radiation dose far exceeds the average. These high doses to limited volumes of tissue are, then, presumed to constitute a high risk. A recent review of the information available on this question[19] (reproduced here as Appendix C) indicates that the experimental data available, while not completely adequate for low activity particles, strongly supports the finding that the non-homogeneous distribution of dose is probably less hazardous than the uniform dose. Nearly all of the support for the increased effect of single hot particles arises from theoretical calculations of doses to individual cells with the cell response assumed from experiments on other types of cells in different configurations of dose distribution. The evidence for cancer induction from limited volume irradiation strongly indicates that a calculation of the dose on the average organ basis is conservative if the irradiation is from particulate sources. For this reason, we will use the average organ dose throughout for the lung.

C. Application to Population Groups and Individuals

It is generally recommended that exposure of population groups or individuals in the population be limited to values below those recommended for occupational workers. However, there are some differences in the recommendations of various groups as to the exact degree of reduction to apply. A brief review is given to aid in choosing the limits to be applied in this work.

The ICRP position as of 1965[9] recommended that the annual dose limits for members of the public be one-tenth of the corresponding annual occupational limit with the exception that the thyroid dose to children under the age of 16 be limited to 1.5 rems rather than the previously used 3.0 rems. The occupational limit listed for "bone" is given as 30 rems per year and for "all other organs" as 15 rems per year. Thus, the dose limit for plutonium in the body would be 0.004 μCi (assuming the rem is calculated as given earlier) and the maximum quantity in the lung would be 0.0016 μCi. For genetic exposure, they recommend a maximum of 5 rems in 30 years or an average for a population group of 0.17 rems per year. However, for the somatic dose of concern here, they state "--it is expected that the dose limits for individuals will ensure that the number of somatic injuries that could possibly occur in a population will remain at a low level." From this, it appears that they did not feel that a specific limit for groups, based on somatic effects, was necessary.

The current recommendations of the NCRP[8] provide dose limitations based on somatic considerations for individuals and for the average population dose. These are given as: "The dose limit for the critical organs (whole body) of an individual not occupationally exposed shall be 0.5 rem in any one year---" and "The dose equivalent to the critical organs (whole body) for the population of the United States as a whole from all sources of radiation other than natural radiation and radiation from the healing arts shall not exceed a yearly average of 0.17 rem (170 mrem) per person." This establishes the population group of interest as the entire population of the country with, presumably, averaging of the dose permitted over this group as a whole. Under these conditions, it is apparent that the dose to the individual is strongly limiting for any accidental or industrial situation where the sources of exposure are relatively limited in number. The definition of the 0.5 rem and its application to plutonium is not clear. If one takes it at face value using the definition of the rem as including the "dose distribution factor" of five derived from the comparative experiments with radium, then the maximum body burden for an individual becomes (0.5/30) x 0.04 = 0.0007 μCi or about one-sixth of the ICRP value for the individual. However, referring back to the statement on occupational exposure we find the words: "For the purposes of this recommendation, the critical organs are considered to be the gonads, the lens of the eye, and red bone marrow." and "--assessed in the gonads, lens of the eye or red bone marrow--." In paragraph 202, with respect to other organs, they indicate; "Detailed discussion is left to other NCRP reports." Thus, it appears that the current NCRP recommendations do not apply to the case of an organ such as the bone, where the damage of concern does not include the red bone marrow or other specifically designated critical organs. In the previous report on internal emitters,[22] the position was: "--The maximum permissible average body burden or radionuclides in persons outside of the controlled area and attributable to operations within the controlled area shall not exceed one-tenth of that for radiation workers." While it appears clear that the NCRP intends a lower average for the population than for the individual, the best interpretation of their recommendations at the moment seems to be 0.004 μCi in the body of an individual.

The Federal Radiation Council has considered the problem of population dose both with respect to external radiation[10] and the somatic dose from radium-226[16] specifically. For environmental contamination they point out that there may be conditions where the only data available may be related to average contamination or exposure levels. They then suggest the use of an arbitrary assumption that the majority of individuals do not vary from the average by a factor greater than three. From this, and their recommendations of 0.5 rem whole body radiation for individuals, they obtain a value

of 0.17 rem for yearly whole body exposure of average population groups. They also warned 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 dose within the population included. For radium, they rejected the use of the factor of ten between the occupational exposure limit and that for the individual in the population because of the differences in characteristics of the child, the longer time for carcinogenesis and the difference in distribution of the radium in the bone from an environmental accumulation over a number of years and the acute type of exposure from the worker exposures. They noted that the dose to the skeleton from all natural causes averaged between 0.1 and 0.15 rads per year while the quantities of radium and its daughters required to give comparable doses were about 0.003 to 0.005 μgm. They also compared the natural occurrence of radium in the skeleton which they quote to range from about 0.001 μgm to some two or three times this amount in most areas of the U.S. In considering the dose to the bone they state: "There is insufficient information on the relative biological effectiveness of the radiation from radium to attempt a realistic conversion of this dose in rads to the skeleton from radium and its decay products into rems." They, thus, specifically reject the conversion of the body burden into dose equivalent as a basis for deriving or expressing limits to the bone. In considering operations involving the release of radium to the environs, they feel that such operations can be carried out in such a manner that the average daily intake in an exposed population group will not exceed 20 pg. They also quote that data on the average intake and average body burden indicate that the quantity of radium in the adult skeleton does not exceed a value of about fifty times the daily intake. They then chose a value for the daily intake of 20 pg per day as the radiation protection guide with an alternate value for individuals in the general population of 0.003 μg in the adult skeleton. For a suitable sample of the exposed population, the average value was set at 0.001 μg in the adult skeleton. It can be seen that the factor of fifty for the body burden of the average individual as compared to the intake, when applied to the RPG of 20 pg/day intake, corresponds to the value for the average of the population group, while the value for the individual is three times this. Application of these values to the plutonium case, again selecting a factor of five for the dose distribution factor would indicate that one could permit only 0.0012 μCi in the body of the individual or about 0.0004 μCi in the adult skeletons of a suitable sample of the population. However, it is also noted that the value was selected on the basis of a finding that operations could be conducted with radium at this level but the same finding has not been made for plutonium. The direct application of this recommendation is therefore in doubt.

For the purposes of this document it appears appropriate to consider an upper limit for deposition in the body of an individual in the population of 0.004 μCi of plutonium and one-third of this value as applied to a suitable sample of the population as defined by the FRC. This will result in an average dose rate to the mineralized portion of the bone of 0.03 to 0.06 rads per year or, using a quality factor of ten for the alpha particles and a dose distribution factor of 5, a dose equivalent rate of 1.5 to 3 rems per year depending upon the fraction of the plutonium deposited in bone as compared to other organs. The lung dose will be based on a limit of 1.5 rems per year (0.15 rads/year) calculated on the basis of an average dose to the entire lung. As noted earlier, this method of calculation is believed to be conservative in control of actual damage.

III. UPTAKE AND RETENTION IN THE BODY

The application of the foregoing standards for the maximum quantity permissible in the body is usually done through "maximum permissible

concentrations" (MPC's) for air and water to be breathed or ingested. These are derived by considering the uptake and metabolic patterns of the isotope in the body. Such MPC's have been given primarily for occupational exposure and, for the values currently in use, the models used for describing the retention and elimination are outdated. For these reasons, we have chosen to review the current information and arrive at independent assessments of the proper intake levels appropriate to the population exposure rather than to rely on published MPC's. The reasons for this decision are discussed in this section of the report along with the derivation of values to be used.

A. Inhalation

The current MPC's recommended by the NCRP[20] and the ICRP[5] were calculated by the use of a simple lung model which dates conceptually back to the Chalk River Conference in 1949.[14] This model differentiates between "soluble" and "insoluble" materials without, however, any definition of the terms other than their assumed behavior in the body. For soluble materials, it was assumed that 25% of the material is retained in the lung and absorbed rapidly into the bloodstream from which it is deposited in other organs of the body, the remainder is eliminated by exhalation or ciliary action to the throat. For insoluble materials, it was assumed that 25% is exhaled with 50% deposited in the upper respiratory passages and subsequently eliminated by ciliary action and swallowed. The remaining 25% is deposited in the deep lung with one-half of this eliminated from the lung and swallowed within the first 24 hours. The remainder (12.5%) is retained in the lung with a half-life of 365 days (for plutonium) with this portion assumed to be taken up by body fluids. Thus, on this model, the inhalation of one microcurie of material will result in the deposition in the lung for long term retention about 0.125 μCi. In general, the components retained for shorter times are ignored in the dose calculations because of the relatively small dose which they will deliver over the period of elimination. In this model, a deposit of 0.016 μCi in the long term retention compartment will then deliver $15/\lambda$ or 15×1 year/0.693 = 22 rems over the period of elimination. However, the material taken up by body fluids remains to be accounted for. If one assumes that all of this material goes to the bloodstream and is later deposited according to the pattern for soluble material, then the uptake to the body becomes limiting and not the lung dose. On this basis, the MPC for insoluble material should be about twice that of the soluble since the uptake by the blood is considered to be only half of that of the soluble. In practice, the MPC for the soluble material is about 0.06 times that of the insoluble because the insoluble value was calculated based only on lung dose without consideration of this fraction taken up into the body.

However, it is known that not all of the material retained by the lung eventually passes into the bloodstream. Instead a major portion is taken up by the lymph nodes which drain the lungs. This has been demonstrated by autopsy on individuals[21,22] who have inhaled plutonium as well as by animal experiments.[23] In response to this, as well as to improved information on the overall deposition and retention of various materials, a Task Group working under the auspices of the ICRP[24] has described a more definitive lung model which provides in some detail the variation in retention in various parts of the lung with particle size and gives some indication of the fate of the materials deposited in various parts of the respiratory tract. Although this lung model has not, as yet, been adopted by the ICRP, and there apparently will be some changes when issued, it is useful for indicating the relative comparison between the older model used for calculating current MPC's and these more refined considerations. The model provides curves for estimating deposition in three regions of the respiratory tract depending upon the particle size.

It then provides three clearance classes depending upon the rate of pulmonary clearance: Class Y - those materials retained in the lung for long periods, perhaps years; Class W - those materials with intermediate retention on the order of weeks; and Class D - those materials rapidly cleared. Classes Y and D correspond to the "insoluble" and "soluble" materials considered in the earlier lung models. Although the Task Force, presumably because of the lack of detailed studies of the behavior of various compounds in the lung, implies that certain of the chemical compounds of plutonium may belong to Class D, the tendency of soluble compounds of plutonium to hydrolyze in body fluids and, in some forms, to produce colloidal polymers would indicate that even the more soluble compounds should be in Category W rather than D. This is at least, partially confirmed by the studies at Hanford using Beagle dogs in inhalation of the nitrate and the fluoride.[23] Here pulmonary retention times of 100 to 200 days were observed.

A summary comparison of the lung model used by the ICRP in deriving the present MPC's with the Task Force model for several particle sizes is given in Table II along with the MPC's evaluated for a worker exposed 168 hours per week. Although the Task Group chose a value for the half-life in the pulmonary region for the Class W plutonium of 38 days as based on early studies with nitrate, we have retained their general 90-day half-time for this class on the basis of the studies with dogs cited earlier. In general, there are no really striking differences apparent in this comparison, although the inclusion of the uptake in the body for the insoluble calculation eliminates the former wide discrepancy between the "soluble" and "insoluble" concept.

This discussion was presented to illustrate the uncertainties which exist in estimating the deposition and transfer of material from the lung. In general, it is concluded that the MPC for soluble compounds as calculated on the old lung model may be somewhat conservative in estimating the buildup of plutonium in the body. On the other hand, it does not fully account for the final site of deposition since both the injection experiments at Utah[15] and the inhalation experiments at Hanford[23] indicate that considerably less than 90% of the plutonium in the body is in the bone with the liver (and lymph nodes) as the major alternate sites of deposition. Since the effects on the bone still predominate in the Utah experiments, however, this partition means that less energy will be deposited in bone compared to that in the total body since the fractional bone deposition is smaller. While some concern may be felt for use of the insoluble MPC in some situations because of the lack of accounting for the movement into the body, the results from the newer lung model would indicate that the transfer from the lung to the blood may be on the order of a factor of three to ten lower than was considered on the older model so that overall buildup even at the higher MPC should not exceed the body burden limits. However, it is noted that considerable uncertainty exists with respect to the initial deposition in the lung because of the lack of data on particle sizes in the usual situation. This will be serious only in the very small particle sizes where the deposition will be increased. On the other hand, even for particles of 0.1 micron size, the pulmonary deposition is predicted by the new model to be only 50%, a factor of two higher than was used for the one micron particle. In view of the many other uncertainties, including the uncertainty in the dose limitation to the lung, such a factor is of little real significance, particularly when the conservative nature of the present MPC's is considered.

For application to the public, it is believed to be inappropriate to use two limits based on the "soluble" and "insoluble" concept without considering the interactions between the two. Possible values of the MPC for an individual in the population based on lung dose of 1.5 rems per year as

TABLE II

COMPARISON OF LUNG MODELS (INHALED BASIS)

Model	Pulmonary Deposition %	Long Term Retention %	Half-life da	To Blood %	To Lymph %	[a]MPC's Lung μCi/cc	[a]MPC's Body Burden μCi/cc
ICRP - "Soluble"	25	---	---	2.5	-----	-----	6×10^{-13}
ICRP - "Insoluble"	25	12.5	365	12.5 (?)	-----	1×10^{-11}	-----
Task Force - Class Y							
0.1 μm	50	30	500	3.34	[b]7.5	4×10^{-12}	4×10^{-12}
1 μm	25	15	500	2.0	[b]3.75	8×10^{-12}	6×10^{-12}
5 μm	12	7.5	500	1.61	[b]1.8	2×10^{-11}	7×10^{-12}
Task Force - Class W							
0.1 μm	50	30	90	10.9	[c]2.5	2×10^{-11}	1×10^{-12}
1 μm	25	15	90	8.8	[c]1.25	4×10^{-11}	1×10^{-12}
5 μm	12	7.5	90	10.7	[c]0.6	9×10^{-11}	1×10^{-12}

(a) For worker - 168 hour per week

(b) 10% of this transfers to blood with 500 da $T_{1/2}$ (included in blood)

(c) Transfers to blood with 90 da $T_{1/2}$ (included in blood)

recommended by the ICRP, 0.5 rems per year as recommended by the NCRP or a total deposition of 0.004 μCi in the body are given in Table III as adapted from Table II.

Since it appears unlikely that there would be significant airborne concentrations of the Class W compounds in pure form from resuspension and processes of agglomeration in the soil could result in relatively large average particle sizes, an MPC in air of 3×10^{-13} μCi/ml applicable to both classes would appear to be appropriately conservative.

TABLE III

INHALATION MPC'S FOR AN INDIVIDUAL IN THE POPULATION
μCi/ml

	Lung 0.5 rems/yr	Lung 1.5 rems/yr	Uptake in Body
Class Y			
0.1 μm	1×10^{-13}	4×10^{-13}	4×10^{-13}
1 μm	3×10^{-13}	8×10^{-13}	6×10^{-13}
5 μm	6×10^{-13}	2×10^{-12}	7×10^{-13}
Class W			
0.1 μm	7×10^{-13}	2×10^{-12}	1×10^{-13}
1 μm	1×10^{-12}	4×10^{-12}	1×10^{-13}
5 μm	3×10^{-12}	9×10^{-12}	1×10^{-13}

B. Absorption from GI Tract

Plutonium is only slightly absorbed from the GI tract when ingested so that intake with foods or other materials through this path is not usually considered to be a limiting method of exposure. In rats, chronic ingestion at low mass concentrations of the nitrate resulted in an average uptake of 0.003% of that fed with 90% of the small fraction which was absorbed deposited in the skeleton.[25] It was estimated with a 90% confidence level that the retention did not exceed 0.01% in 99% of the rats. A similar absorption of 0.002% was noted in pigs following feeding of pH2 nitrate solution.[26] The MPC in drinking water of the NCRP[20] and the ICRP[5] for so-called "soluble" plutonium is based on an uptake of 0.003%.

The uptake from the GI tract can be affected by the presence of complexing agents, the valence state of the plutonium and the age of the animal. The variation with valence state and the presence of citrate is shown in Table IV as obtained from Thompson's review.[27] Thompson also reported experiments by Carritt et al in which the absorption of nitrate in rats was increased from 0.01%

TABLE IV

ABSORPTION OF PLUTONIUM FROM SEVERAL SOLUTIONS FED INTRAGASTRICALLY TO RATS

Principal Anion	pH	Type of Solution Fed: Plutonium Valence State Identified % (III)	(IV)	(VI)	Plutonium Retained 4 Days after Single Feeding, %
Nitrate	1		68		0.28
Nitrate	1			100	1.9
Nitrate	2	90	10		0.006
Nitrate	2	7	93		0.005
Nitrate	2		96		0.0013
Nitrate	4		97		0.0017
Citrate	2		99		0.03
Citrate	2		96	4	0.29
Citrate	2		85	15	0.41

in the absence of citrate to 0.3% with 5% sodium citrate.

In one day old rats, the absorption of plutonium from a pH2 nitrate solution averaged 0.25%. This absorption dropped to 0.1% at 7 days of age, to 0.02% at 21 days and to the adult value of about 0.003% at 33 days of age.[27]

Although these uptakes are low in most normal situations, they cannot automatically be dismissed in all environmental situations. Romney et al,[28] for example, report data on the plutonium content of the lung, GI tract and bone of kangaroo rats and jackrabbits at the Nevada Test Site where they had been living in areas contaminated with plutonium. Data from the animals taken from the higher contamination areas are reproduced in Table V.

At first glance, the bone values appear to be high considering the low absorption of plutonium. However, the high GI tract contents indicate the possibility of ingestion of considerable amounts of soil so that a large quantity of plutonium is available for transfer. In the last column we have calculated the amount of plutonium which would be expected in the bone after one year considering that the GI tract contents represent one day's intake and 0.003% of this quantity is transferred to the bone each day. Even ignoring any absorption from the lung, it can be seen that, within the accuracy of the estimate, the apparently high bone values can

TABLE V

PLUTONIUM IN ORGANS OF ANIMALS LIVING IN CONTAMINATED AREAS

Station	Bone dis/min	GI Tract Contents dis/min	Lung dis/min	[a]Transferred to Bone per Year dis/min
Kangaroo Rats 1958				
11D	7.13	2052	11.40	22.5
13-2	4.30	1255	0.12	13.7
Kangaroo Rats 1966				
11D	47.05	1050	61.28	11.5
13-3	2.72	170	5.80	1.9
Jackrabbits 1958				
13-1	128.48	5.5×10^5	57.50	6000
13-2	11.68	3.2×10^4	0.36	350
13-3	1.75	5712	0.24	63
Jackrabbits 1966				
11D1	665.40	4.1×10^4	98.25	450
11D2	88.76	1.6×10^4	8.92	175
13-3	19.27	1360	1.92	15
13-5	2.34	781	0.10	9

[a]Assuming GI content measurement represents one day feeding and 0.003% per day transferred to bone.

be accounted for on this basis. It may also be noted that these values, even though significant, should be of little concern in a predator food chain because of the low uptake from the GI tract of the predator.

The effects of unabsorbed plutonium passing through the GI tract have been studied in acute administrations to rats.[27] A dose of 88 mCi/kg of nitrate caused death in the first day, apparently from effects other than radiation. Doses of 56 mCi/kg did not produce grossly evident damage. Oxide doses as high as 230 mCi/kg produced no gross evidence of damage while 155 mCi/kg produced transient histological changes in the cecum and colon which appeared three days post administration but not at six days. These data have indicated that the alpha radiations do not penetrate to the sensitive tissues of the GI tract with any efficiency and serve as the basis for the ICRP and NCRP assumption that only 1% of the alpha energy at the surface of the GI tract contents is effective in producing a dose to the GI tract.

The foregoing data would indicate that the 0.003% absorption from the GI tract chosen for calculation of the MPC's for occupational exposure is appropriate for this use. However, for the environmental exposure of the public in situations such as living in a contaminated area where exposure can be continuous, both the higher absorption by children and the possible effects of combination of ingestion along with foods containing various additives such as citrates, preservatives and even chelating agents must be considered. For the young rat, absorption above 0.1% was in the first week of life corresponding approximately to the age of the human baby when motility is low and the environment is relatively carefully controlled so that access to ingestion by routes other than foods is small. The high uptakes with citrate occur with high acidity and significant percentages of the plutonium in the +6 valence state both of which are unlikely to occur with any degree of regularity under normal conditions. Thus, it is concluded that an uptake about ten times larger than that used by the ICRP for occupational exposure and about one-tenth of the highest values noted for very young animals or citrate complexed plutonium would be reasonable and, at the same time, relatively conservative particularly for the relatively insoluble forms of plutonium expected to occur in the environment. This would, then, be an uptake of about 0.03% and would apply particularly to the most susceptible group, children between the ages of about one and ten years.

C. Skin Absorption

Although the intact skin serves as an excellent barrier against the passage of plutonium on its surface, a small rate of absorption through the skin can occur. Such rates are insignificant for most cases of sporadic, infrequent skin contamination but we must consider the possibility of long term accumulation from living in a contaminated area where a continued maintenance of some level of contamination on the skin can be assumed.

Data on the absorption of plutonium nitrate from 0.1 N acid solution on rat skin indicates absorption rates of $2 - 30 \times 10^{-5}$ percent per minute over periods of 15 minutes to one day.[29] When applied in a mixture of tributyl phosphate and carbon tetrachloride with traces of nitric acid, the initial rates were up to ten times higher, with indications that higher rates were maintained through at least five days. Human data are meager and may indicate somewhat lower absorption rates as could be expected from data on other materials with several species of animals as compared to humans.

In deriving skin contamination limits for control purposes, a rate of penetration of 10^{-5}% per minute was used for plutonium based upon an examination of available data.[29] This primarily relates to contamination resulting from solutions rather than the more insoluble particulates. However, the possible effects of agents such as lotions, detergents, and various household chemicals have not been examined to see if they could have a possible effect of increasing the penetration. One would expect the plutonium in soils or the environment to be initially in the form of insoluble oxide or firmly attached to other particles so that the skin absorption should be lower than for the solutions. In view of the uncertainty of possible effects of other agents, however, the absorption rate of 10^{-5}% per minute will be used as a conservative value.

If we again limit the intake by absorption to that which would result in a deposition of 0.004 μCi after 70 years (ignoring elimination) the rate of absorption is 0.35 dis/min per day or assuming a 10^{-5}% per minute rate of skin absorption, one could permit continuously over the 70-year period some 2400 dis/min on the body. The surface area of the body is about 1.85 m^2 for an average man, about 1.6 m^2 for an average woman and about

0.25 m^2 for the newborn.[30] Data are not available for the average quantity of dirt or soil carried on th the body. Treagar[31] indicates that about 1 mg/cm^2 of liquid is about as much as can be held on the human skin without forming a noticeable liquid pool. Since the skin is normally cleansed at intervals, particularly before bedtime, and it is protected over a major portion by clothing, an average quantity of environmental soil of about 0.1 mg/cm^2 is assumed to be continuously present. Note again, that the child, who is more likely to be somewhat soiled, has a smaller surface area and, thus, for the same deposit a smaller total quantity of dirt. Under these assumptions, the average man would have some 1.85 grams of dirt on his body which could contain about 1300 dis/min per gram.

This calculation assumes the dirt on the body to contain the same concentration of plutonium as the soil in the environs. Since one would expect the smaller soil particles to be preferentially deposited on the body, a mechanism for concentration or depletion of the plutonium in the soil on the body depending upon the relative particle size does exist. Normally, however, one would expect the smaller plutonium particles to be attached to soil particles, particularly after a residence in the environment of some significant period of time so that this possibility of concentration may not be as significant as it would seem, particularly with the inherent conservatism of the calculation.

The possibility of by-passing the skin barrier by deposition in an injury or damaged skin also exists. The mechanism is of particular concern in plant operations where concentrated quantities of plutonium are handled and significant amounts, in relation to the maximum permissible body burden, can be introduced into a single wound. However, at the low concentrations expected in soils at an acceptable level, the amount of plutonium associated with the soils is very small. Data on absorption through cuts indicates that uptake may be 10-100 times that noted through intact skin.[32] Thus, for this mechanism of uptake into the body to be equally effective compared to skin absorption, some 1-10% of the body must be continually abraded and contaminated to these levels. Probably of more significance in this case is the reduction of conservatism in the number derived.

IV. INTAKE IN CONTAMINATED AREAS

The problem of estimating the intake of plutonium by a heteorogeneous group of people visiting or living in a contaminated area is exceedingly complex and provides the major source of uncertainty in the derivation of a standard. Past investigations[33,34] have used a simplified concept of the resuspension factor to provide estimates of the air concentrations and the resulting inhalation. Intake by ingestion, absorption or through ecological chains was shown to be negligible in comparison to the inhalation. While it appears that the general concepts of these prior investigations are reasonable, a more detailed study of the various methods whereby air concentrations or ingestion can occur is needed to assure that the generalized concept of the resuspension factor, for example, covers all of the cases.

It is noted at this point that the mechanisms of intake to be discussed are primarily physical in nature rather than biological as can occur in an ecological chain leading to concentration in one or more links. While the evidence is not complete that biological accumulation may not be important in some situations, particularly as the plutonium in the soil ages and is possibly recycled through biological systems, it now appears that plant uptake or uptake in higher animals is low enough that the physical methods of direct contamination will be of greatest interest in this problem. This complicates the study because of the large number of possible direct contamination transfer systems, marked variability with different situations and the lack of firm experimental data, all of which limit our ability to quantify and rank these mechanisms in order of importance.

A. Mechanisms of Intake

The intake of plutonium from the soils can be by a varied series of pathways, either direct or indirect, which are dependent upon the nature of the contaminated area, the nature and distribution of the contaminant and the actions of individuals in the area. We have not attempted to formalize these pathways at the present time since they need considerable additional definition and data to quantify them. As will be seen, however, there are a few generalizations which can be used to approximate the hazard in such situations.

If one considers the situation occurring in an area where soils are contaminated and families are living, it is immediately apparent that a relatively complex description is needed. We can start with the ambient air concentrations which will result from wind pickup. This will depend upon the type of terrain and vegetative cover, the wind speeds and directions with respect to the contaminated area, the penetration of the particles into any shelter plus other variables as discussed in Appendix A. This type of exposure will be relatively constant in time and, given certain of the variables, can be generally evaluated for the average concentration. Other perturbations in the exposure conditions are both more localized and intermittent depending upon certain actions at the time. For example, mechanical disturbance of the soils by such simple actions as walking or digging can produce localized air concentrations. These, in turn, can result in contamination of the body or clothing from which additional plutonium intake can occur by ingestion, absorption through the skin, or inhalation as a result of localized actions (i. e., taking a dress or shirt off over the head). Further, such a mechanism can result in transfer of contamination to other areas, such as the home or a vehicle, where the nature of the surroundings is such that more intimate and prolonged contact could result in significant intake. A probably more important variation of the same mechanism is that of children at play in the area. This is because of their generally more active nature and more intimate contact with soils during such activities. The presence of pets in many homes provides another mechanism for transfer of contamination into the home with possible intake by individuals. Of particular interest here is the localized concentration for inhalation which could occur by fondling or hugging the pet.

Aside from living in the area there is the question of working. Agricultural pursuits (including home gardening) involve considerable effort directly with the soils and disturbance of the soils by mechanical and animal activities. It is possible that just this type of disturbance may result in mixing of the contaminant in the soil making it less available or causing redistribution over a wider area. Again the possibility of transfer to houses or vehicles with more intimate contact and exposure of other people exists. Other types of out door work, such as construction, is usually for a limited period of time and, while soil disturbance is large, it usually results in a high degree of mixing and, frequently, burying some portion of the contaminant in an inaccessible location.

It will be noted that we have concerned ourselves with areas in which people are living. While it is appropriate to consider the possibility of different standards for areas with only occasional visitation, the data available on contamination transfer and the long-term behavior of the plutonium are not now adequate to provide an assessment which would be applicable to conditions some years after the contaminating event when habitation of the area is possible.

Much of the effort on these mechanisms of exposure for this interim standard has been devoted to the question of resuspension and inhalation since this still seems to be the predominant mode for taking plutonium into the body. However, future studies will attempt to better define and quantify these other possibilities, and in particular the transfer mechanisms, in order to remove uncertainties

and to, perhaps, permit a range of values applicable to different situations.

B. Ingestion

Primary methods of ingestion of plutonium from the soils are considered to be casual ingestion by transfer from the hands (or other parts of the body) to the mouth or by contamination of food crops grown in the area. There is a definite possibility of deliberate ingestion of the soils by young children.

Data on the quantities likely to be ingested in this manner are not available but, for the casual ingestion, it would appear that one gram per day would be a high estimate with 0.1 gram per day a more likely value. If we limit the total intake by this mechanism so that the body burden at the end of 70 years is 0.004 μCi with an uptake of 0.03%, the 0.1 gram per day ingestion would lead to a soil concentration of 5×10^{-3} μCi/g or 11,000 dis/min per gram.

The deliberate ingestion of soil by children is limited to a relatively short period of time, say one year, and is intermittent over this period. If we assume an average of one gram per day ingested with the limitation on accumulation during this one year at 1/70th of the maximum permitted body burden, the soil concentration should not exceed 5×10^{-4} μCi/g or about 1100 dis/min per g.

In the above analysis, we have lumped several individual pathways of exposure into our value of 0.1 g of soil (or the plutonium contained therein) ingested per day. These include the intake with foods, casual ingestion, and intake with water which may have become contaminated from runoff from the contaminated area. Data for individual assessment of each of these mechanisms are not adequate to trace, in any detail, the intake from each of these. However, for terrestrial environments, it is noted that the root uptake of plutonium by plants is low[35,36] so that this should not comprise a major source of plutonium to humans. Data on the transfer to muscle of animals[26] or to milk[37] again indicate a low uptake from the GI tract and translocation to these sources of food. The rate of transfer to streams or lakes by normal erosion processes will vary with the terrain and climactic conditions, but the insoluble nature of most plutonium compounds and the tendency of solubilized material to undergo ion exchange means that the great majority of the material washed into bodies of water will end up in the sediments. This may well produce a mechanism of greater significance for concentration in the biological chain than for the terrestrial ecosystems since, as was noted earlier, there is evidence of concentration of plutonium in certain marine organisms. Probably of greater significance, however, is the direct contamination of plants used as food by man and animals by the direct physical mechanisms in the environs such as resuspension and impaction on the plant surface.

These potential sources of exposure all require additional study and definition before one can arrive at a final standard. However, it is believed that the assumptions made in this section are reasonable for the present, particularly when it is realized that other mechanisms of exposure, such as inhalation, appear to be more limiting by an order of magnitude.

While these are crude estimates, it is believed that they are conservative in that the chemical forms of plutonium expected in the soils (usually oxides or polymeric forms of the hydroxides) are insoluble and the uptake from the GI tract would be expected to be considerably lower than the 0.03% chosen. It may be noted at this point that the fraction of the soil involved in the casual ingestion will be heavily weighted toward the small particle fraction since these smaller particles are more likely to stick to the clothing, skin or food crops than the larger particles. In the specification of the final standard, this particle size effect must be considered.

C. Skin Absorption

In Section III C, a value of 1300 dis/min per gram or about 6×10^{-4} μCi/g was derived as a limiting concentration in the soils for the possibility of skin absorption. Again, it is believed that this value is conservative because of the relatively high absorption rate chosen, particularly for the compounds expected in the soils. It is deliberately conservative, however, in view of the uncertainty of the influence on the absorption rate of the various lotions, makeups, soaps and other chemical materials used on the skin.

As was noted, the smaller particle fraction in the soils (or of the contaminant) is again of particular interest since this fraction will stick to the skin.

D. Inhalation

In order to be inhaled, the particles must become airborne and arrive at the vicinity of the nostrils. Usually, this requires energy from an external source to dislodge them from their resting place and to keep them suspended in the air for a time period sufficient for inhalation. (Although one can visualize a direct transfer to the air stream entering the nostrils by "sniffing" or inhaling vigorously with the nostrils close to a contaminated object.) For inhalation and retention of the particles in the respiratory tract, the particle size must be relatively small, usually considered as less than 10 μm aerodynamic diameter. Larger particles will deposit in the upper respiratory tract and be eliminated from the body in a matter of hours to days through the GI tract. Because of the low absorption from the GI tract and the protective layer of mucous between the contents and the GI tract wall, this fraction is of little or no concern for the alpha radiations from plutonium. The fraction of the particles retained in the respiratory tract increases as the particle size decreases with the best estimate of this factor as given by the ICRP Task Force on Lung Dynamics.[24] This factor has been discussed and considered in the revised MPC to be used for this study in Section III A.

The need for considering particle size of the contaminant in the soil and in its transfer to the air is of considerable importance in all of the inhalation transfers. Particles of the contaminant which are larger than the "respirable" size in the soils are of little concern from a potential inhalation hazard standpoint unless reasonably efficient mechanisms for breaking the particles into smaller sizes are available. Thus, in the following considerations, primary emphasis is placed on the smaller particles and mechanisms for movement which affect the larger particles, such as saltation or surface creep, are considered to be of secondary importance.

In the transfer of particles to the atmosphere or to surfaces, the distribution of the contaminant through the soil is an important factor. One can visualize, for illustration, two theoretical limiting conditions. The first condition prevails for an indeterminate period of time following an initial deposition when the material is spread over the surfaces of the ground and other objects in a thin layer. As time passes, the erosive effects of the wind or runoff and the removal of the material from the surfaces of plants by washing, growth and decay, or from other surfaces by winds or rains, leads to the condition where the contaminant is mostly in the soils and is distributed through a layer extending to a depth dependent upon the time since deposition, the nature of the soils, the influence of physical factors acting on the soil (such as freezing, thawing, rainfall leaching, or wind or mechanical movements resulting in mixing) and even the biological factors such as microbial action, burrowing animals, etc. This is further complicated by the fact that plants and other surfaces will intercept resuspended materials, usually diluted by the accompanying soils, and these items will serve as sources for further

resuspension. However, it can be seen that the vulnerability of the material on the surface is much higher in the condition representing an initial deposit since all of the material is in a position to be affected by winds or other disturbance while in the latter condition, a portion of the material has penetrated into the soil and its availability depends upon the depth of penetration of any disturbance. In addition, its availability may also be affected by any chemical or physical reaction, such as binding of contaminant particles to soil particles, which may have occurred. In the final limiting condition, the contaminant will be essentially uniform throughout the soil profile and will behave much as the other constituents of the soil in producing airborne dust when disturbed.

The above considerations apply directly to the airborne accident case when the deposition occurs in a short period of time so that penetration into the soil and binding to the soil particles does not occur during the period of deposition. In the industrial situation of continuous, low level, airborne releases, the deposition continues over a period of time so that these mechanisms are continuously at work and only the material deposited recently is in the upper layer of highest susceptibility and undiluted with soil particles. Another situation of interest in the industrial area would be that in which the contaminant is carried in a liquid, such as the buildup of materials on sediments from low level effluents, or the situation in an area where higher level wastes are percolated through the ground to remove the contaminants by adsorption on soils. In these cases, the penetration of the contaminant into the soil layers is much greater so that, even after drying, the contaminant is diluted to a large extent with soil and the plutonium is associated with the normal particle sizes in the soil although there may be a strong tendency for association with the smaller particles because of the nature of the ion exchange process. Another special case is the area used for burial of solid wastes. Here the immediate problem is controlled by covering the contaminated material with a thick layer of clean dirt and excluding the area from use. Concern with such practices stems from the possibility of later use of the area with digging into the material, from later erosion bringing the material to the surface, or from translocation by leaching. Here, again, the effects of time and physical and biological processes will result in a much more uniform mixing of the material (particularly if the contaminated objects are biodegradable) than when they were buried. If one could, for example, postulate complete mixing then the appropriate limits could be based upon the total contaminant and the total volume of the burial area.

1. Estimate from Dust Loading. The normal dust loading of the atmosphere results, at least in part, from the resuspension of soil particles from the earth's surface to the air. Thus, the quantity of such material normally found in a given region can be considered to be a crude index of the resuspendability of the surface materials for some indeterminate distance upwind. (Note that in a dust storm the material in the air at a given location could have originated miles upwind as, for example, from a large area of plowed fields, so that the dust load must be regarded as an index to the average condition over a large area.) If we assume that the plutonium contamination is uniformly mixed with the soil particles so that the same mechanisms which result in the resuspension of the soils are equally effective in causing resuspension of the plutonium, then a limiting concentration in that part of the soil layer which is resuspended can be estimated from the standards for particulates and for radioactivity. We have earlier concluded that an MPC of 3×10^{-13} μCi/cc seemed appropriate for the exposure of an individual in the population when applied to materials most likely to be encountered. The Federal Secondary Standard for particulates in the air is expressed as a geometric mean (mean of the logarithms of the concentrations) of 60μg/m³.[38]

The geometric mean is smaller than the arithmetic mean by a factor depending upon the geometric standard deviation of the measurements. Since the average exposure (and thus, presumably the average amount inhaled and retained) depends upon the arithmetic mean, it is necessary to convert this standard. Equations for this purpose are given by Drinker and Hatch.[39] Experience with most airborne contaminants indicate that the most likely geometric standard deviation is about two. For this value, the arithmetic mean concentration corresponding to the standard is 76 $\mu g/m^3$. However as the standard deviation increases, the mean increases rapidly, being 116 $\mu g/m^3$ for $\sigma_g = 3$, 152 $\mu g/m^3$ for $\sigma_g = 4$, and 219 $\mu g/m^3$ for $\sigma_g = 5$. We will arbitrarily, and somewhat conservatively, consider a geometric standard deviation of three to be appropriate with a mean concentration of 120 $\mu g/m^3$ to correspond to the standard. Thus, the concentration in airborne dust, when the particulates and the plutonium both reach their appropriate standard, would be 2.5×10^{-3} $\mu Ci/g$ or about 5500 dis/min per gram of dust.

The dust in the atmosphere will result primarily from the lower particle size fraction of the soils so that the comparison here is for the smaller particles rather than the total soil. It is conceivable that the contaminant may have a particle size distribution sufficiently different from that of the soil that some fractionation will occur during the pickup and subsequent dispersion of the dust and soil. An excellent illustration of this possibility would occur during the early resuspension of a contaminant initially deposited on the surface following an accident since it is not distributed into the soil profile and could be preferentially injected into the air. In the longer time period, however, mixing with the soil will minimize this factor.

Although the uncertainty of possible fractionation exists, it is believed that the value derived by this reasoning is conservative in that the dust loading at a given location will be as the result of the pickup over a wide area, possibly much larger than most areas of contamination and it is assumed that the dust loading is continually at the standard. In practice, the dust standard may be exceeded for periods of time but the average loading should be lower, particularly for the time spent inside the buildings, and the concern, in terms of plutonium accumulation, is with the average concentration over long periods of time. It is also believed that this value is probably the least uncertain at this time since it does not involve calculations based on uncertain and poorly defined mechanisms.

2. General Resuspension. General Resuspension consists of those mechanisms which result in a relatively uniform concentration over a reasonable area. It includes wind pickup and mechanical disturbances far enough upwind that the concentration in the air is more dependent on the dispersive mechanisms of the general atmosphere than on the very local source. It is distinguished here from the localized concentrations resulting from mechanical disturbance or other means of producing concentrations in a small volume in order to permit use of different estimating techniques. A discussion of general resuspension, the equations used and current data available is given in Appendix A supplemented by Appendix B.

As can be seen from a review of Appendix A, the information available on the resuspension rate under various conditions of atmospheric stability and ground cover is very meager and is confined almost to several experiments with ZnS particles which were freshly deposited. For the wind pickup, a value of K, the resuspension rate, divided by the wind speed squared of about 2×10^{-8} appears to be reasonable for a level, poorly vegetated plain with unstable atmospheric conditions. Higher values are appropriate for localized areas upwind where disturbances, such as vehicle traffic or construction occur, but these are generally short-lived either in duration or, if continuous, will result in rapid depletion of the contaminant or mixing through the soil profile.

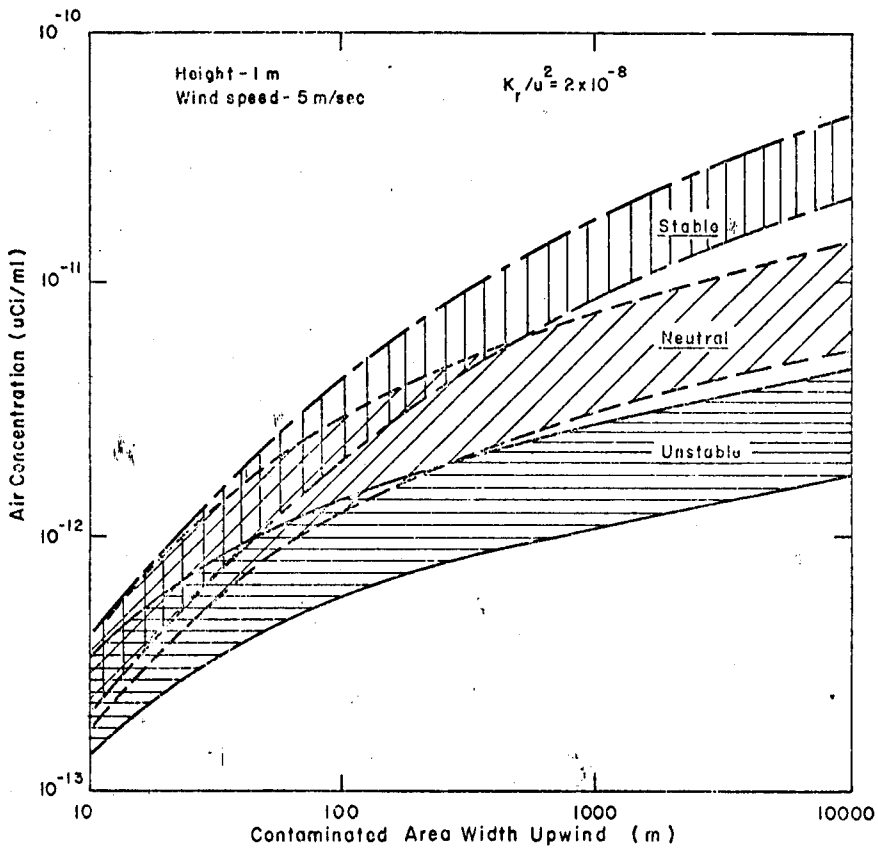

Fig. 1. Air concentrations at maximum point downwind from an infinite crosswind area uniformly contaminated to 1 μCi/m^3.

As would be expected, the concentration at a given location is highly dependent upon its location with respect to the contaminated areas and the wind directions. Thus, a precise definition of the expected air concentration could only be given for a particular location if the contamination pattern and meteorology were well defined. Some parametric calculations on the importance of this factor are in progress but are not completed to the point where they would be particularly useful in this study. However, in view of the variations possible, we have used a situation where the location of interest is at the edge of a uniformly contaminated area which extends to infinity in the crosswind direction. The wind is blowing directly over the plain toward the sampler. The results for this calculation, using the freshly deposited value of K/u^2 for zinc sulphide particles of 2×10^{-8} are given in Fig. 1. Since the wind pickup is assumed to increase as the square of the wind speed and the dispersion as the first power of the wind speed, the concentration downwind should increase directly with the wind speed. The value of 5 m/s used in the calculation is a reasonable average for many locations. The bands in Fig. 1 result from calculations using various values of deposition velocities, depending upon particle size and vegetative cover in the area.

In assessing Fig. 1, there are several uncertainties and factors of conservatism that should be borne in mind. The applicability of this calculation

to the stable case is particularly questionable since wind speeds are generally lower in this condition. The use of the same pickup rate for this stable condition as for the unstable case would seem to overestimate the concentration since one would expect the pickup to be lower because of the decreased wind turbulence. The effect of aging on the pickup rate is uncertain. While others have assumed a decrease in the air concentration[33,34] with a half-life of 35 days, examination of the data available (Appendix A) does not substantiate the continued decay at this rate. There is no doubt that the resuspension rate will decrease with time, probably rapidly at first and then at a decreasing rate. Calculations on the air concentrations at one area in Nevada (GMX) are given in Appendix A. A few measurements in this area[40] indicate the resuspension rates to be significantly lower than those calculated. This is some 20 years or so after the deposit was laid down. An additional uncertainty is in the effect of particle size on the pickup rate. The pickup constant was derived primarily from data using ZnS particles. Data are not available to assess possible changes in the rate with particle size. The question of wind variability has not been seriously considered since the formalized type of calculation used for Fig. 1 tends to average the concentration over a wide angle due to the assumption of an infinite extent in the crosswind direction. This, in turn, tends to maximize the estimate of the air concentration in the real case.

Although the uncertainties are large in this type of estimate, it would appear that a uniform concentration (or average over a large area) of about 0.1 $\mu Ci/m^3$ for material freshly deposited from the atmosphere would be reasonably conservative in meeting the average MPC. One would expect the air concentration to decrease with time. It is believed that a decrease of a factor of ten would not be unreasonable over the first year.

3. Resuspension Factor. The resuspension factor approach has been widely used for estimating air concentrations from surface deposits. It does tend to give average values for the particular conditions under which it is measured and a reasonable amount of information is available for several different conditions. We tend to believe that it is more useful for describing localized concentrations resulting from various types of disturbances but, in view of the paucity of other data, its use for this problem is discussed below.

The resuspension factor is defined as the ratio of the air concentration to the quantity of material per unit area on the ground. If the air concentration is given in quantity per m^3 and the unit area on the ground is in m^2, the resuspension factor will have units of m^{-1}. Outdoor measurements of this factor have been made in arid or semiarid country following safety tests of nuclear weapons with a few studies in other areas using relatively small plots seeded with known levels of radioactive materials. Stewart[41] has concluded that a representative value for quiescent conditions outdoors is about $10^{-6} m^{-1}$ while in areas of moderate activity the value may increase to $10^{-5} m^{-1}$. A review of values accumulated from the literature by Mishima[42] indicates values ranging from 8×10^{-10} to $3 \times 10^{-4} m^{-1}$ under conditions of no mechanical disturbance and from 1.5×10^{-6} to $5 \times 10^{-4} m^{-1}$ as measured under conditions of vehicular or pedestrian traffic or in areas with people working. It is noted that the minimum values under quiescent conditions occurred from one test using ^{91}Y. If these values were excluded, the range without mechanical disturbance is reduced to 8×10^{-8} to $3 \times 10^{-4} m^{-1}$. Langham,[34] in assessing limits for a weapons accident, uses a value of $10^{-6} m^{-1}$ with, however, an exponential decrease with time with a half-life of 35 days, thereby implying a value of 7×10^{-10} one year after deposit and $5 \times 10^{-13} m^{-1}$ two years after deposit. Kathern[33] assumes a value of $10^{-4} m^{-1}$ decreasing with a half-life of 45 days. The primary evidence cited for the

decrease with time is a series of air samples taken over a period of 20 weeks in an area contaminated by plutonium following a weapons safety test. We do not believe this magnitude of decrease to be appropriate, as is discussed in Appendix A, but do believe that some decrease will occur over the first year following a deposition.

It is noted that the resuspension factor is sensitive to the methods used for estimating the quantity of plutonium on the ground, to the location of the sampler with respect to the contaminated area and to the meteorological conditions at the time of the measurement. Thus, if measurements are made under quiescent conditions, as far as mechanical disturbance is concerned, the wind speed and the depth of the plutonium in the soil would appear to be important factors for the particular locality. Measurement of the surface contamination by an alpha meter will detect to depths of a few milligrams per square centimeter (perhaps a few hundreths of a millimeter) and may underestimate the contamination. Sampling of the area to a depth of two inches will include soils which will not be affected by the surface disturbance and, if contaminated to the full depth, will result in an overestimation of the surface quantity available for resuspension. While the measured factor will indicate the probable air concentration under identical conditions of plutonium distribution, these variables plus others discussed earlier make the extrapolation of the values to different areas or different types or patterns of contamination extremely uncertain.

However, if we use the value of $10^{-6} m^{-1}$ as recommended by Stewart as an overall average value for quiescent conditions, we find that a quantity of $0.3 \mu Ci/m^2$ will result in an air concentration of $3 \times 10^{-13} \mu Ci/cc$. Although mechanical disturbance of the surface will result in higher concentrations, the time period over which such disturbances will occur is usually fairly short, continued disturbance over a long period of time will result in depletion of the resuspendable material and the values are still within the uncertainty of the estimate of the overall factor.

4. Mechanical Disturbance. Mechanical disturbance of the soils by actions ranging from walking across an area to heavy vehicular traffic or even excavation of the area can result in increased dust loading downwind and, presumably, increased resuspension of contaminants contained in the soil. Such actions can also hasten the "aging" process by mixing the material in the upper layer of the soil and diluting the contaminant particles with soil particles. In extreme cases, such as the presence of heavy vehicular traffic over a given area for an extended period of time, the expected increase in resuspension rate will result in depletion of the contaminant from the particular area.

Quantitative evaluation of the potential effect of such mechanical disturbance requires a quantitative relation between the degree of disturbance for a particular area and the resuspension, a reasonable description of the disturbance expected in the contaminated area and the relation between these and the location of people. As was noted earlier, Stewart has concluded that an increase by a factor of ten for the resuspension factor for an area of moderate activity is reasonable. In Mishima's tabulation, the lower end of the range of resuspension factors is increased by about three orders of magnitude while the upper end is increased by a factor of two when areas of vehicular or pedestrian traffic are compared to undisturbed areas. However, in this tabulation it is difficult to account for degree of disturbance or for the frequencies with which measurements were taken under each condition. In a rough analysis of data obtained by Mork on air concentrations downwind from a vehicle driven across a contaminated area of the Nevada desert, (see Appendix A), it was concluded that resuspension rates up to one hundred times those caused by the winds could occur. It is noted that mechanical disturbance is a mechanism

whereby material from the ground can become airborne during conditions of maximum atmospheric stability and minimum wind speed, thereby resulting in minimum dilution downwind. At the same time, this mechanism results in the dislodging of large numbers of soil particles so that a dilution of the contaminant particles with these soil particles occurs, making the dust calculation more appropriate to this condition than the more conventional resuspension assessment.

From the data available, it appears that mechanical disturbance can result in increased air concentrations downwind over those to be expected solely from wind actions. However, if the disturbance is over a short period of time, the contribution to the average concentration will be well within the uncertainty in knowledge of the wind effect. For more intense disturbances or longer duration, the effect of mixing in the soils and/or depletion of the source will, again, minimize the contribution to the long term average. This is not to minimize the possible importance of such a factor in certain situations but, rather, it would appear that the uncertainties in the knowledge of resuspension and changes of resuspension with time will incorporate the variations due to such disturbances in most situations.

5. Personal Contamination. A possible mechanism of intake by inhalation is contamination of the skin or clothing while working or playing in a contaminated area, followed by resuspension of the material directly from the surface of the skin or clothing into the breathing zone or transfer of the contamination into the home with subsequent exposure of those living there.

Data on the transfer of contamination from the ground to the skin or clothing are very sparse so that any direct calculation of the resulting intake will produce results of limited value. However, it has been estimated from data available in the literature[29] that the inhalation from contaminated clothing or skin during normal activities could be equivalent to the inhalation of the contamination from about one cm^2 per hour. If we consider the inhalation rate to be $20\ m^3$ per day, the MPC of $3 \times 10^{-13}\ \mu Ci/ml$ would permit the inhalation of about $6 \times 10^{-3}\ \mu Ci/day$. If the inhalation from clothing continues over the full 24 hours at the above rate and the clothing is continuously contaminated to the same level throughout the day, then the allowable skin or clothing contamination would be about $2.5 \times 10^{-7}\ \mu Ci/cm^2$ or about 0.5 to 0.6 dis/min per cm^2.* If we consider an average of one mg/cm^2 (or about 20 grams total on an adult male) to be a reasonable value for the soil transferred to clothing and skin, the concentration would be on the order of 500-600 dis/min per gram. (Note that the $1\ mg/cm^2$ here includes both clothing and skin and not just skin as was used earlier.) Again, the considerations of particle size and mixing of the contaminant with the soil discussed earlier are pertinent to this evaluation. It should also be noted that the rate of intake will vary widely depending upon clothing changes, bathing, etc., and may well be lower at night because of the decreased physical activity.

The possible problems encountered from the movement of plutonium into the home on the clothing of workers was also examined in Reference 29. Here it was assumed that 30% of the material brought into the home was transferred to the home area and that it remained in resuspendable form with a half-life of one week. Resuspension rates of 5×10^{-4} per hour were used as representative of the activity in the house with two air changes per hour. Under these conditions and using an MPC for air of $2 \times 10^{-14}\ \mu Ci/ml$ it was concluded that $0.01\ \mu Ci$ could be brought in per day without exceeding the maximum permissible limits. For the revised MPC of $3 \times 10^{-13}\ \mu Ci/ml$ used for this study,

*This value for the allowable surface contamination is higher than that given in Reference 29 because of the reexamination of the appropriate MPC in this study.

TABLE VI

PROBABILITY OF INHALATION OF PARTICLES

Action	Probability of Inhaling One Particle	Grams of Soil Contaminated to 10^3 dis/min per gram
Changing Tire	3×10^{-5}	400
Sweeping Bus	5×10^{-4}	30
Sweeping Car	6×10^{-4}	20
Driving Car for One Hour		
No Ventilation	6×10^{-7}	20,000
High Ventilation	2×10^{-6}	7,000
	$*2 \times 10^{-5}$	700

*Measured immediately after placing powder on floorboards. Other value represents the mean of two additional determinations.

it is concluded that this rate could be 0.1 to 0.2 μCi per day. As a comparative figure, if the soil contamination were 1000 dis/min per gram, this would require bringing in about 200 to 400 grams per day. While this is not a physical impossibility, particularly in muddy weather, this rate seems somewhat high for most conditions.

Schwendiman[43] has measured the probability of inhalation of particles under several conditions associated with automotive transport and cars, using ZnS particles of about 2 μm median diameter. A summary of these probabilities and the quantity of soil which must be present at a contamination level of 1000 dis/min per gram to cause the inhalation of 6×10^{-6} μCi (the amount which could be inhaled in one day at the MPC of 3×10^{-13} μCi/ml) during the given action are listed in Table VI.

These values, while for a time shorter than the full 24 hours per day, represent measured conditions in confined areas with relatively severe activity. As such, they provide some indication that the previous values estimated for the home are not unreasonable.

V. A PROPOSED INTERIM STANDARD

In the preceeding discussions, we have touched on several points which are of importance in considering the conversion of estimates of exposure to a standard for soils. To some extent these factors are interrelated and involve the questions of distribution in the soil profile, units of measurement and sizes of the particles of concern.

Previous recommendations for soil limits have been expressed in units of quantity of plutonium per unit area (i.e., μCi/m² or μg/m²) because the primary mechanism of exposure was considered to be resuspension in the atmosphere and inhalation. However, this method of designation has led to uncertainties in interpretation since the layer of soil involved and of interest was presumably that associated with the resuspension factor applied and this was not defined in the studies. Thus, there was, in these recommendations, no clear guidance as to the depth in the soil profile to which the limit should apply and varying sampling and measurement depths have been used in different studies. In the assessment of exposures in this paper, we have used both the concentration in the soil and the quantity per unit area depending upon the type of estimate made. The two methods of expressing the limit can be interrelated if such factors as the thickness of the soil profile of interest and the soil density can be defined. Thus, either method can be used as a primary unit as long as the information to permit conversion to the other is provided.

The concentration in the soil is preferred in this study because many of the potential mechanisms of exposure are more directly related to this quantity and the common methods of measurement, sampling and analysis, provide answers directly in concentration units. Even with direct measurements of external radiation, such as with the FIDLER, the quantity per unit area is applicable only when the material is in a thin layer on the surface. For a uniform depth profile with a large thickness compared to the effective range of the photons, the reading with this type of instrument is proportional to the concentration in the soil. We have, therefore, chosen to express the standard in units of plutonium concentration but, also, including a specification of the thickness of

the soil layer to be considered. This, of course, has the advantage of relating the standard to the measurements to be made in a contaminated area.

The selection of an appropriate layer must consider the mechanisms of exposure and their relative importance. For dust loading of the atmosphere and resuspension, the appropriate thickness will depend upon the type of disturbance which causes the input to the atmosphere. Similar considerations also apply to the transfers to the body since the material available for transfer must be that to which the individual is exposed in the soil layer.

We have tended to consider the material at the very surface of the ground to be limiting in the sense that it is more available for transfer or for resuspending. The definition of the "very surface of the ground" is difficult since it can change with conditions such as wind speed, turbulence or degree of mechanical disturbance. Further, the sampling and measurement of a thin layer on the ground surface is difficult even on bare ground and next to impossible in heavily vegetated areas (such as a lawn). However, in heavily vegetated areas the access of people to the soils is limited so that somewhat different considerations will apply to exposure from the soils. In view of the potential importance of wind pickup and the lack of information on the thickness of the layer actually involved in this phenomenon, we have arbitrarily chosen a layer on the order of one mm thick to serve as a standard for the low vegetated areas. The wording "on the order of" is deliberately chosen to indicate that the actual thickness cannot be specified closely because of the impossibility, in most cases, of sampling a precisely defined layer with any degree of precision. It is suggested that a reasonable interpretation of this term would be a shallow scraping of the surface layer taking into account the many imperfections and various sizes of small objects encountered in such a scraping. The use of a measured area and weighing of the sample will permit an estimate of the average thickness. For vegetated areas, where the surface is not as readily available a thickness on the order of 5 mm would seem to be appropriate. Since the specification of this thickness provides an averaging thickness over which the plutonium in the soil is measured, such a specification would permit the averaging of a thinner layer over the full depth and would permit a total of up to five times as much expressed as quantity per unit area in the vegetated area as in the barren area. The decreased exposure to people due to the smaller access to these soils and to the decreased pickup by the winds would appear to more than compensate for this.

For layers deeper in the soil profile, a thicker layer would again appear to be appropriate since exposure would result only by mechanisms which either remove the upper layers or mix the soils to a significant depth. Thus, for the soils beneath the surface, averaging over a one centimeter depth would seem to meet the intent of the limitation.

Note that in the above discussion, the limit on concentration in the soil remains constant regardless of the thickness of the layer with those mechanisms of exposure resulting from direct contact or transfer of the soil and plutonium to the body not affected. The main purpose of specifying the layer is to provide an appropriate thickness for averaging and controlling this thickness so that averaging over deeper depths will not result in samples meeting the limit but still presenting a high level at the surface for the resuspension mechanisms.

The second parameter of interest is the particle size of the contaminant. For inhalation, particles larger than about ten μm aerodynamic diameter will have a very low probability of retention in the lung and the solubility of plutonium is such that particles deposited in the upper respiratory tract will not be of significance in adding to the body burden before they are eliminated. It is noted that for plutonium oxide particles, a ten μm

aerodynamic diameter corresponds to an actual particle size of about 3 μm for spherical particles due to the density effect. In the earlier discussions it was noted that even for mechanisms involving transfer to the body, fractionation toward the smaller sizes will occur both in the dislodging of the particles from their resting place and in the consideration of the retention on the body. There is, therefore, good reason for believing that the smaller particles are of predominant importance in all mechanisms of exposure and some recognition should be given to this in the formulation of the standard.

If the overwhelming and only consideration in exposure were inhalation one could confidently use an upper limit for the size to be considered on the order of 5-10 μm based on the possibility of attachment of plutonium to particles of low density. For pure oxide particles, the size limit could be even lower. There is, however, the problem of the uncertainty in the estimates of the other mechanisms and the possibility that they may assume some importance for the somewhat larger particles if the controlling size were based only on inhalation. In addition, the possible problems of aggregation with breakup under the disturbance which transfers the material to the air must be considered. We have, therefore, chosen an arbitrary limit of particle sizes for these considerations of less than 100 μm to represent the fraction of the soil of concern. This corresponds to a screen in the Tyler series of 150 mesh (actually this is 105 μm). It is recognized that normal screening will not break up some of the aggregates which could later be broken up and serve as a source of exposure, but the choice of the 100 μm size should provide sufficient conservatism that such errors will not be important.

However, for considering the state of the contaminated area over long periods of time, one must also consider the possibility that breakup of the particles in the normal processes of soil formation will occur and will serve as a source of smaller particles continuously feeding to the fraction of interest. Although it is believed that redistribution mechanisms over the time periods of interest for soil formation will predominate in determing the soil concentrations, it is proposed that the total concentration in all particle sizes be limited to an arbitrary value of twice the concentration in the fraction below 100 μm. Since the times for soil formation from the matrix material range from decades to tens of decades, this limitation should be extremely conservative.

With this background on the application of the numbers, we are now ready to review the estimates from the exposure mechanisms to arrive at a value for the plutonium concentration in the defined layers and fractions. The estimates of soil concentration obtained earlier are summarized in Table VII for this purpose.

In assessing these values and considering the degree of conservatism relative to each, it was concluded that a value on the order of 500 d/m per gram or about 2×10^{-4} μCi per g would be appropriate. The resuspension values for fresh deposits are somewhat lower than this, but, for the long term exposure, it is expected that the values will increase by a factor of ten or more. Further, the estimates were deliberately made for an unrealistic type of area in which it would be expected that the calculations would lead to a high air concentration.

TABLE VII

ESTIMATES OF LIMITING SOIL CONCENTRATIONS FOR SEVERAL MECHANISMS OF EXPOSURE

Mechanism	Soil Concentration μCi/g	D/M per g	μCi/m²
Ingestion			
Casual	5×10^{-3}	11,000	-
Deliberate	5×10^{-4}	1,100	-
Skin Absorption	6×10^{-4}	1,300	-
Inhalation			
Dust Loading	2.5×10^{-3}	5,500	-
General Resuspension			
Fresh Deposit	*6×10^{-5}	*130	0.1
Aged Deposit	*6×10^{-4}	*1,300	1
Resuspension Factor	2×10^{-4}	*330	0.3
Clothing	3×10^{-4}	600	-

*Based on 1 mm thickness of soil with a density of 1.6 g/cm²

TABLE VIII

RECOMMENDED INTERIM STANDARDS FOR PLUTONIUM IN SOILS

	In <100 μm particle Size Fraction			Total**		
	D/M per g	μCi/g	μCi/m²	D/M per g	μCi/g	μCi/m²
Top 0.1 cm*	500	2×10^{-4}	0.4	1000	4×10^{-4}	0.8
Any one cm layer	500	2×10^{-4}	4	10000	4×10^{-4}	8

* For bare soil or areas with sparse vegetation. Where area is reasonably well vegetated (greater than 50% of the area is covered with low vegetation) and a reasonable root mat exists to hold the soil, the concentration listed can be applied to a 0.5 cm layer which would permit up to 2 μCi/m² in this layer.

** With the provision that the fraction with particle sizes less than 100 μm is known not to exceed the limits given. If this is not known, the values for the <100 μm fraction should be applied to the total.

This standard for the concentration can now be combined with the previous discussion as to the limits of applicability to provide the final set of standards as given in Table VIII.

The resuspension mechanisms, which strongly influence the choice of the concentration value tend to average the pickup from wide areas so that the presence of small areas in the general vicinity which have higher concentrations are not of great importance. Since the other methods of more direct transfer from the soil give higher estimates for the limiting concentration and, in themselves, require consideration of occupancy factors and types of human activity in the contaminated area, it is tempting to specify that the above values are averages over large areas and that smaller localized depositions of several times these concentrations could be permitted. In view of the many uncertainties and the magnitude of the values, an allowance of this nature is not recommended for general use at this time. However, as additional data are obtained it is anticipated that the standard will be revised to include such a feature. In the meantime, it is possible that detailed investigation of a particular area may provide sufficient information for that area to permit the application of such a concept for that area. Such investigations aimed at a particular situation will always provide better answers than a general standard of this nature and such an approach to individual problems is entirely appropriate.

REFERENCES

1. C. M. Lederer, J. M. Hollander, and I. Perlman, "Table of Isotopes," Sixth Edition, John Wiley and Sons, Inc., New York, London, Sydney, 1967.

2. F. X. Hass and A. R. Campbell, "Table for the Ingrowth of Daughter Products from the Decay of Plutonium Isotopes Used in Heat Sources," USAEC Report MLM-1919 (Mound Laboratory) June 12, 1972.

3. K. C. Pillai, R. C. Smith, and T. R. Folsom, "Plutonium in the Marine Environment," Nature 203, 568 (1964).

4. K. M. Wong, V. F. Hodge, and T. F. Folsom, "Concentrations of Plutonium, Cobalt, and Silver Radionuclides in Selected Pacific Seaweeds," USAEC Document LA-4756 (Proceedings of Environmental Plutonium Symposium held at LASL, August 4-5, 1971) pp. 93-98, 1971.

5. International Commission on Radiological Protection, "Report of Committee II on Permissible Dose for Internal Radiation," Health Physics, 3, June 1960.

6. J. F. Cline, "Uptake of Am-241 and Pu-239 by Plants," USAEC Document BNWL-714 (Battelle, Pacific Northwest Laboratories), pp. 126-127 (1966).

7. R. O. McClellan, H. W. Casey, and L. K. Bustad, "Transfer of Some Transuranic Elements to Milk," Health Physics, 8, 689-694 (1962).

8. National Council on Radiation Protection and Measurement, "Basic Radiation Protection Criteria," NCRP Report No. 39, NCRP Publications, Washington D. C., January 15, 1971.

9. International Commission on Radiological Protection, "Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965)" ICRP Publ. 9, Pergamon Press, Oxford (1966).

10. Federal Radiation Council, "Background Material for the Development of Radiation Protection Standards," Report No. 1, U.S. Dept. of Commerce, May 13, 1960.

11. International Commission on Radiological Units and Measurements, "Dose Equivalent," Supplement to ICRU Report 19, ICRU Publications, September 1, 1973.

12. W.H. Langham and J.W. Healy, "Maximum Permissible Body Burdens and Concentrations of Plutonium: Biological Basis and History of Development," in Handbuch der exp. Pharmakolgie, Band XXXVI, J.N. Stannard, Ed. Springer Verlag, Berlin, Heidelberg, New York (In Press).

13. A.M. Brues, "Comparative Chronic Toxicities of Radium and Plutonium," Argonne Laboratory Quarterly Report (February-March and April 1951), ANL-4625, pp. 106-124.

14. G.E. McMurtrie (Secretary), "Permissible Doses Conference, held at Chalk River, Ontario (September 1949), Report RM-10 (May 1950).

15. T.F. Dougherty, "Annual Report of Work in Progress in the Internal Irradiation Program, 1971, USAEC Report COO-119-244 (University of Utah).

16. Federal Radiation Council, "Background Material for the Development of Radiation Protection Standards," Report No. 2, U.S. Dept. of Health, Education, and Welfare, Washington D.C., (September 1961).

17. L.H. Hempelmann, C.R. Richmond, and G.L. Voelz, "A Twenty-Seven Year Study of Selected Los Alamos Plutonium Workers," USAEC Document LA-5148-MS (Los Alamos Scientific Laboratory), January 1973.

18. National Committee on Radiation Protection, "Permissible Dose from External Sources of Ionizing Radiation," National Bureau of Standards HB 59, (September 24, 1954).

19. J.W. Healy, "Contamination Limits for Real and Personal Property," Progress Report January to June 1972, USAEC Document LA-MS (Los Alamos Scientific Laboratory) in preparation.

20. National Committee on Radiation Protection and Measurements, "Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water," NCRP Report 22, National Bureau of Standards Handbook 69, (June 5, 1959).

21. T.L. Shipman et al, "Acute Radiation Death Resulting from an Accidental Nuclear Critical Excursion," J. Occ. Med. 3, 146-192 (1963) Special Supplement.

22. E.E. Campbell et al, "Plutonium in Autopsy Tissue," USAEC Document, LA-4875 (Los Alamos Scientific Laboratory), January 1973.

23. W.J. Bair, "Plutonium Inhalation Studies," USAEC Document BNWL-1221 (Pacific Northwest Laboratories, Battelle Northwest) February 1970.

24. International Commission on Radiological Protection, Task Group on Lung Dynamics, "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract," Health Physics, 12, 173-207, February 1966.

25. J. Katz, H.A. Kornberg, and H.M. Parker, "Absorption of Plutonium Fed Chronically to Rats," Amer. J. Roentgenol. 73, 303-308, February 1955.

26. M.H. Weeks, et al, "Further Studies on the Gastrointestineal Absorption of Plutonium," Rad. Res. 4, 339, 1956.

27. R.C. Thompson, "Biological Factors," in: Plutonium Handbook, a Guide to the Technology, O.J. Wick, Ed. Gordon and Breach, Science Publishers, New York, London, Paris (1967).

28. E.M. Romney, H.M. Mork, and K.H. Larsen, "Persistence of Plutonium in Soils, Plants, and Small Mammals," Health Physics, 19, 487-491, 1970.

29. J.W. Healy, "Surface Contamination: Decision Levels," USAEC Document, LA-4558-MS (Los Alamos Scientific Laboratory) September 1971.

30. A.M. Laylee, "Anatomy and Physiology of the Skin," in: Radiation and Skin, E.T. Wray, Ed. AHSB (RP) R 39, 1-10, (1964).

31. R.T. Tregear, "Physical Functions of Skin," Academic Press, 1966.

32. W.D. Oakely and R.C. Thompson, "Further Studies on Percutaneous Absorption of Plutonium Solutions in Rats," in: Biology Research-Annual Report 1955, USAEC Document HW-41500, (Hanford Atomic Products Operation, General Electric Company) February 1956.

33. R. L. Kathern, "Towards Interim Acceptable Surface Contamination Levels for Environmental PuO_2," USAEC Document BNWL-SA-1510 (Battelle Pacific Northwest Laboratory) April 1968.

34. W. H. Langham, "Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads," USAEC Document UCRL-50639 (Lawrence Radiation Laboratory) April 1969.

35. D. O. Wilson and J. F. Cline, "Removal of Plutonium-239, Tungsten-185 and Lead-210 from Soils," Nature 209, 941-942, February 26, 1966.

36. J. R. Buchholz, W. H. Adams, C. W. Christenson, and E. B. Fowler, "Summary of a Study of the Uptake of Plutonium-239 by Alfalfa from Soils," USAEC Document LADC-12897 (Los Alamos Scientific Laboratory) 1971.

37. R. O. McClellan, H. W. Casey, and L. K. Bustad, "Transfer of Some Transuranic Elements to Milk," Health Physics 8, 689 (1962).

38. Environmental Protection Agency, "National Primary and Secondary Ambient Air Quality Standards," Federal Register 36, 21, January 30, 1971.

39. P. Drinker and T. Hatch, "Industrial Dust, Hygienic Significance, Measurement and Control," 2nd Ed., McGraw-Hill Book Company, Inc., New York, 1954.

40. P. Phelps, Lawrence Radiation Laboratory, private communication.

41. K. Steward, "The Resuspension of Particulate Materials from Surface Contamination," in: Surface Contamination, B. R. Fish, Ed., Pergamon Press, pp. 151-158 (1967).

42. J. Mishima, "A Review of Research on Plutonium Releases During Overheating and Fires," Hanford Atomic Products Operation Report HW-83668 (1964).

43. L. C. Schwendiman, "An Application of Fluorescent Pigment to the Measurement of Particle Inhalation Probabilities," USAEC Document HW-32292 (Hanford Atomic Products Operation, General Electric Co.) July 1, 1954.

APPENDIX A

PICKUP OF PARTICLES FROM THE GROUND AND DOWNWIND DISPERSION-GENERAL RESUSPENSION

Airborne concentrations resulting from particulate contaminants in the soil can be a possible mechanism of exposure of people and animals to the contaminant. Such airborne concentrations can be of two general types which are distinguished by their persistence and the nature of the investigations required to define their relative importance. The first type is the localized concentration where the material may be in high concentration in the breathing zone of one or a few people usually due to some mechanical disturbance of a contaminated soil or object. Such a localized concentration can result directly from the soils or by contamination of other objects which can, then, transfer the contamination to the localized breathing zone of an individual. In general, the magnitude of the concentration will be a function of the contamination level and characteristics of the contamination over a relatively small area. The second type of concentration is the more general, widespread concentration which results from the pickup of materials from the ground to the atmosphere with dispersion downwind over a large area and, possibly, involving many people. Such concentrations can result from either wind or mechanical disturbance of the soils and are a function of the contamination levels over a relatively wide area upwind for the wind disturbance and of the localized levels at the site of a disturbance for mechanical suspension. In this discussion, we are concerned with the second type of concentration which we will refer to as "general resuspension" as opposed to the local resuspension for the first type.

I. APPROACH

The general resuspension, along with the localized resuspension, has been described in terms of a resuspension factor which is defined as the ratio of the air concentration to the level of contamination on the ground at a given location.[1,2] A brief reflection on the upwind source, with a potentially large source area involved, will indicate the inapplicability of this concept to the problem of general resuspension. This is one of the chief reasons for distinguishing between the localized and general types of resuspension. Thus, the material in the air at the receptor may arise from the pickup many meters or even kilometers upwind. Further, the source will change with the wind direction or the specific area in which mechanical disturbance occurs.

In order to permit estimation of air concentrations resulting from such a contaminated area, a rough model based upon work done some years ago in estimating the importance of wind pickup and transport of larger particles with eventual impaction on the person[3,4] was revised. In this model, each element of the contaminated area is considered to be a source for airborne material with the source strength defined by the rate at which the contaminant enters the air with the specific disturbance considered. The concentration downwind is then estimated from the dispersion and deposition relations developed over the past years.

In general terms, if we consider a point source of material on the ground which is subject to resuspension, the concentration in the air at some distance downwind is given by Eq. (A-1).

$$\chi = K\Omega D'D'' \qquad \text{(A-1)}$$

In this equation, χ is the air concentration, Ω is the quantity of material on the ground in a position where it is subject to being injected into the atmosphere, K is the fraction of this material which is injected per unit time by the specific disturbance considered, D' is the dispersion which occurs

downwind as a result of turbulent diffusion, and D'' is the fraction of the material which is not deposited between the point of pickup and the receptor. In atmospheric dispersion terms, $K\Omega$ is the source term and the remainder of the equation is conventional.

The air concentration resulting from a contaminated area can then be evaluated by integrating the point source equation over the area taking into account the variations in contamination level.

$$\chi = \int_{x_1}^{x_2}\int_{y_1}^{y_2} K\Omega\, D'D''\, dy\, dx \qquad \text{(A-2)}$$

In the application of Eq. (A-2), the dispersion and deposition downwind can be evaluated from existing information resulting from micrometeorological studies, although the exact choice of parameters will affect the results and the choice is made somewhat difficult by the fact that the source is truly a ground level source. The distribution of contaminant on the ground can be measured by a number of possible techniques but, again, there is some difficulty in defining exactly the depth of importance and the other parameters, such as particle size, which will be appropriate. The primary value for which little data are available is K, the rate of resuspension under the particular conditions of interest. However, it may also be noted that Eq. (A-2) may be used in the study of the values of K in areas where the other parameters are known.

While Eq. (A-2) appears relatively simple, it describes a number of very complicated processes, many of which can be described only in semi-quantitative terms at this time. In several of the areas where existing information is adequate for other purposes, added accuracy may well be needed to permit a realistic and adequate description for this use. However, we have attempted to survey such information as is available and to apply it in a simplified fashion in order to both illustrate the application of the technique and to derive some feeling for the sensitivity of the result to some of the parameters.

II. DISPERSION AND DEPOSITION

A. Equations

The basic equations used for estimating the dispersion downwind and intervening deposition according to conventional models are well-known and have been documented elsewhere.[5] A brief presentation of these equations is included here for reference and for orientation of the user, particularly in those aspects having to do with the uncertainty.

The dispersion in the atmosphere from a continuous point source by turbulent diffusion is usually described as a Gaussian distribution of the material in the horizontal and vertical crosswind direction. Thus, the concentration at a point (x, y, z) with the origin at the source, x taken in the downwind direction and y and z the distances in the horizontal and vertical crosswind directions respectively is given by:

$$\chi_{(x,y,z)} = \frac{R\,(K\Omega)}{2\Pi\sigma_y\sigma_z\,\bar{u}}\exp\left[-\left(\frac{y^2}{2\sigma_y^2}+\frac{z^2}{2\sigma_z^2}\right)\right] \qquad \text{(A-3)}$$

In Eq. (A-3), u is the average wind speed, R is a reflection factor to account for the presence of the ground and σ_y and σ_z are the lengths corresponding to the standard deviation of concentration in the y and z directions. The other symbols are as given earlier.

Deposition from such a plume will result in depletion of the material originally airborne thereby reducing the quantity available at the receptor. The evaluation of the deposition rate is usually accomplished by use of a deposition velocity, V_d, defined as the ratio of the rate of deposition on a given area to the air concentration at a reference height above the area.[6] The dimensions of such a ratio are those of velocity. The product of the deposition velocity and the concentration gives the absolute rate of deposition from the atmosphere at

a given location. The quantity depositing between the source and the receptor is, then:

$$\frac{\partial (K\Omega)}{\partial x} = \int_{-\infty}^{+\infty} \chi V_d \, dy \qquad \text{(A-4)}$$

$$D'' = \exp\left[- \frac{R V_d}{\sqrt{2\pi}\,\bar{u}} \int_0^x \frac{\partial x}{\sigma_z} \right] . \qquad \text{(A-5)}$$

In this method of accounting for deposition, a fraction of the material in the plume is assumed to deposit per unit of plume length and this fraction is removed from the plume. In essence, this correction factor reduces the source term to allow for the material which is lost. It is unsatisfactory in many ways since it implies a uniform depletion through the full height of the cloud and does not account for the concentration gradient which will exist in the profile above the ground because of the continual depletion at the ground surface. An alternate approach would be to account for the rate of change in the vertical cloud dimensions as expressed by the change in σ_z as a factor in bringing the material to the layer above the ground. However, this equation will be used in this model until further development of concepts can be made.

In order to apply these equations, relations between the values of σ_y, σ_z and the distance from the source must be used. A number of different methods of expressing these correlations have been derived by different individuals. In one of the earliest methods, Sutton[7] provides a relation between the standard deviation and distance using two additional parameters which are dependent upon the atmospheric stability and the turbulence. The standard deviation for both the horizontal and vertical growth increase downwind as a power of the distance with the power changing as the atmospheric stability changes. Pasquill[5] provides a set of curves for the growth of σ_y and σ_z based upon a classification of the stability and values of σ_θ, the standard deviation of the wind direction fluctuations which have been found to be reasonably characteristic of these conditions. Fuquay[8] uses the product of $\sigma_\theta \bar{u}$ where the σ_θ term is the same as in Pasquill's and the $\bar{u}$ is the average wind speed. In addition, Fuquay expresses the dispersion as a function of the time of travel rather than the distance. Other systems of classification are available but the above indicates some of the variations. While the experience of the author indicates that the system of Fuquay has much merit and gives about as good correlation as can be expected, it also has the disadvantage that the expressions are complicated, making integration difficult, and the information available in most situations to evaluate $\sigma_\theta \bar{u}$ is meager. The Pasquill scheme, with the data expressed as curves, again makes it difficult to perform integrations, such as those given in Eq. (A-5) In view of the uncertainties in many of the other parts of the problem, the Sutton method was chose for the expression of the equations with, however, the reservation that the actual values of the coefficients could be chosen to provide a fit to the other schemes. In this method of expression, the standard deviation of plume width or height is given in the general form:

$$\sigma = \frac{C x^{\frac{2-n}{2}}}{\sqrt{2}} . \qquad \text{(A-6)}$$

The coefficient C can be different for horizontal or vertical growth. The value of n is dependent upon the stability of the atmosphere and controls the rate of growth of the plume. Using this method for expressing cloud dimensions, Eq. (A-3) for the point source corrected by Eq. (A-5) for the deposition becomes:

$$\chi = \frac{RQ}{u \Pi C_y C_z x^{(2-n)}} \exp\left[-\left(\frac{y^2}{C_y^2 x^{2-n}} + \frac{z^2}{C_z^2 x^{2-n}}\right)\right] \exp\left[-\frac{2RV_d}{\sqrt{\Pi}\, C_z n \bar{u}} x^{\frac{n}{2}}\right] \tag{A-7}$$

From the point source equation, the contribution from other configurations of the source can be evaluated by integration. For a complex deposition pattern which cannot be expressed as an equation, this integration must be performed by numerical techniques. However, there are several simple configurations for which analytical expressions can be derived, particularly with the Sutton method of expressing the plume growth. These are given below for the convenient, dimensionless parameter $\chi\bar{u}/K\Omega$.

Infinite Line Source Upwind

$$\frac{\chi\bar{u}}{K\Omega'} = \frac{R}{\sqrt{\Pi}\, C_z \bar{u} x^{\frac{2-n}{2}}} \exp\left[-\left(\frac{z^2}{C_z^2 x^{2-n}} + \frac{2RV_d}{\sqrt{\Pi}\, C_z n\bar{u}} x^{\frac{n}{2}}\right)\right] \tag{A-8}$$

Ω' - Source contamination per unit length of line.

Gaussian Line Source Upwind

(Sampler directly downwind from peak concentration, Ω_p. Material along line distributed with standard deviation of A meters.)

$$\frac{\chi\bar{u}}{K\Omega'_p} = \frac{\sqrt{2}\, RA}{\sqrt{\Pi}\, C_z x^{\frac{2-n}{2}} \bar{u}\sqrt{C_y^2 x^{2-n} + 2A^2}} \exp\left[-\frac{z^2}{C_z^2 x^{2-n}} + \frac{2RV_d}{\sqrt{\Pi}\, C_z n\bar{u}} x^{\frac{n}{2}}\right] \tag{A-9}$$

Uniform Area Source - Infinite in Y

(Receptor at ground level, x_1 distance to nearest boundary; x_2 distance to further boundary of contaminated area.)

$$\frac{\chi\bar{u}}{K\Omega''} = \frac{\bar{u}}{V_d}\left[\exp\left(-\frac{2RV_d}{\sqrt{\Pi}\, C_z n\bar{u}} x_1^{\frac{n}{2}}\right) - \exp\left(-\frac{2RV_d}{\sqrt{\Pi}\, C_z n\bar{u}} x_2^{\frac{n}{2}}\right)\right] \tag{A-10}$$

$$\frac{\chi\bar{u}}{K\Omega''} = \frac{\bar{u}}{V_d}\left(D_1'' - D_2''\right) \tag{A-10a}$$

Ω'' - source contamination per unit area.

Equation (A-10a) is somewhat misleading in that it provides the concentration at the ground surface rather than at some height above the ground. In this situation, the small area immediately upwind contributes strongly to the final answer while, in practice, the material from this area may contribute only slightly to a receptor at some height because the growth in the vertical height of the plume may be low enough in this distance so that the material from the ground does not have a chance to reach the receptor elevation. For an elevated source, a numerical integration is needed for the initial distance where $\exp(-z^2/C_z^2 x^{2-n})$ is less than one with application of Eq. (A-10a) beyond this distance.

B. Choice of Parameters

The dispersion parameters to be used in the foregoing equations can be obtained from the correlations of past experiments on turbulent diffusion.[5] These correlations are not completely satisfactory in a number of respects but they do represent a body of experience which can be applied without repeating all of the experimental work under the specific conditions of interest in this problem. However, we do emphasize the following limitations on these data. In the correlations, the data are stratified into arbitrary classifications of stability while the atmosphere in its variations acts as a continuum. Thus, some restraint is placed on the description of the variability by the categorization. Of probably greater importance, there is no agreed-upon method of defining stability for the purposes of classification so that different investigators will use different parameters (or different variations of the same parameter) in describing the classes. This also gives rise to a subjective interpretation of the meaning of the classes for experimenters working in different areas or for individuals applying the data to different areas. For example the term "strong inversion" can well have a different meaning to an individual in a flat desert country where very strong inversions can occur or to an individual in an area where temperatures are moderate with cloud cover a large portion of the time. In the following work, we will see instances where different parameters are used to describe the degree of stability for different parameters to be used in the equations. As will be noted, there is no assurance that the judgments made on these two different methods of expressing degree of stability represent the same condition of the atmosphere. While this factor is troublesome from the standpoint of logic and, to some extent scientific accuracy, this method of classifying the data is probably about as good as can be done without running into an overwhelming mass of detail and the results are undoubtedly adequate considering the remainder of the unresolved uncertainties that occur elsewhere in the problem. A more serious problem would seem to arise from the uncertain dependence of these parameters on the time of sampling or the time of interest at the receptor. Particularly for the value of σ_y the fluctuations in wind direction will increase as the time of sampling increases making the value of the cloud spread dependent upon the time. Sutton recognized this problem in his early work and specified his parameters for a relatively short sampling period.[7] Many of the differences between the present correlations and those of Sutton are undoubtedly due to the fact that most of the samples incorporated in these experiments were taken for periods of 30-60 minutes. The importance of this factor lies in the fact that the selection of a given parameter for the dispersion also implies a given fluctuation of wind direction and averaging of the downwind plume over these fluctuations which, in turn, implies a given time of sampling under the turbulent conditions existing. Such considerations are of greatest importance when attempts are made to derive values of the pickup rate from air concentration measurements around a known source of contamination on the ground. Related to both of these problems is the uncertainty of the growth of the plume in the vertical, particularly in the stable condition. While the argument can be made that wind pickup should not be of great importance under stable conditions because of generally low wind speeds and turbulence, this has not been demonstrated and the problem of dispersion from mechanical disturbances occurring under these conditions still exists. As an illustration of this problem, there are data for very stable conditions which indicate the vertical growth to be considerably lower than is predicted by any of the models normally used. The value of σ_z is of particular importance to these calculations since the deposition between the source and the receptor is strongly dependent upon this parameter and the importance of the long term average

concentration at a given elevation means that the primary dispersion mechanism over the long period of time is due to the vertical growth (i. e., the horizontal dispersion in the plume is averaged out by the changes in wind direction so that the value of σ_y is of interest only for the short sampling period.)

In view of the above considerations, it would seem that experiments designed to measure the pickup from the ground should provide a direct method of measuring the dispersion parameters and their growth during the time of sampling. This could be, for example, a smoke plume or other tracer material which would give direct evidence on the actual conditions at the time. Alternately, one could use a line source of sufficient length so that the value of σ_y is not important and concentrate on the vertical dispersion, perhaps by measuring a profile with height. This is not to say that the conventional measures of stability and wind fluctuation should be disregarded. Rather, these methods should be used to supplement the more conventional meteorological data.

For calculations in this paper, we have chosen a set of parameters reasonably representative of unstable, neutral and stable conditions. These are given in Table A-I.

The values of σ_y and σ_z resulting from these choices are compared with those of Pasquill in Figs. A-1 and A-2.

The selection of an appropriate deposition velocity is difficult because of the lack of an organized set of information on this subject. In order to provide a method of choosing the deposition velocity in relation to the particle size and the differing atmospheric conditions, a rough model to describe the

TABLE A-I

SUTTON PARAMETERS USED

	Atmospheric Condition		
	Unstable	Neutral	Stable
n	0.2	0.25	0.5
C_y	0.45	0.2	0.3
C_z	0.3	0.1	0.07

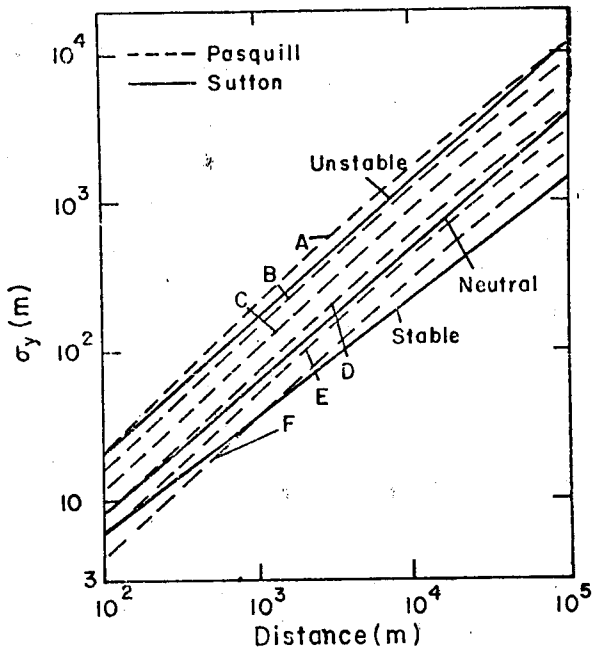

Fig. A-1. Comparison of σ_y for Pasquill's curves and Sutton using parameters of Table A-I.

deposition velocity was derived as based upon current data. This model is described in detail in Appendix B. This work predicts that the deposition velocity will vary directly with the wind speed for the particle sizes of interest and will also be dependent upon the deposition surface as measured by the surface roughness parameter z_o. It is in the choice of this value that one of the main difficulties occurs in selecting parameters which are consistent for a given stability class since the stability classification basis for the deposition velocity is the Richardson's number which can be only indirectly related to the stability used to describe the Sutton parameters chosen.

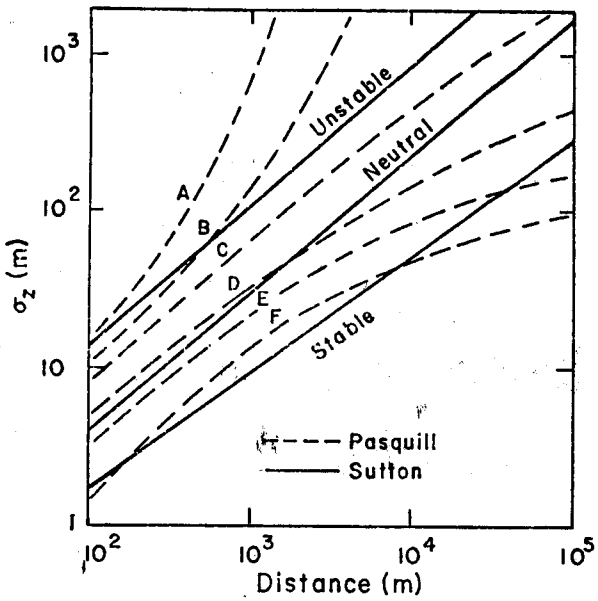

Fig. A-2. Comparison of σ_z for Pasquill's curves and Sutton using parameters of Table A-I.

In the development of the deposition velocity model, a constant rate of transfer of the particles through the boundary layer to the ground was considered due to the turbulent transfer across this boundary. The primary effect of particle size (in sizes less than those where the gravitational forces predominate) was considered to be in the retention once the particles were brought to the ground. From the data available, it appears that particles above about 1.5 μm will be strongly retained with the retention dropping off with particle size. For this reason, the reflection factor in the dispersion equations was written as (2-f) where f is the fraction of the material reaching the ground which is retained. Thus, for a material completely retained, the reflection factor becomes 1 or the concentration is simply due to the direct transport from the source.

The parameters for the deposition and reflection which have been chosen are given in Table A-II.

TABLE A-II

DEPOSITION PARAMETERS USED

	Atmospheric Condition					
	Unstable		Neutral		Stable	
z_o (cm)	0.1	2.3	0.1	2.3	0.1	2.3
>1.5 μm Particulates						
(2-f)	1	1	1	1	1	1
V_d/u	0.0093	0.017	0.0028	0.0080	0.0046	0.0029
<0.1 μm Particulates						
(2-f)	1.97	1.97	1.97	1.97	1.97	1.97
V_d/u	0.0003	0.0005	0.00008	0.0002	0.00001	0.00009

III. RESUSPENSION RATE MEASUREMENTS

The key to the application of a model such as this is, now, the definition of the rate of resuspension under the conditions of interest. Unfortunately, the information available from experiments which included the necessary meteorological data as well as the measured distribution of activity on the ground and the air concentrations resulting are extremely limited and cover only a few of the many terrain and soil possibilities of interest. However, in this section we will discuss the data available which bear on this question in order to arrive at the best possible answer at this time and to provide some illustration of the application of the method.

A. General

The movement of surface grains under the action of winds has been studied for desert sands by Bagnold[9] and for agricultural fields by Chepil[10] with only a few of Chepil's many papers referenced here. This work has outlined the mechanisms involved in producing soil erosion and has described the influence of a number of important factors. A brief outline of some of these concepts which may be important to resuspension on the scale of interest here is given below with no attempt to make this an exhaustive treatment.

Three chief methods of movement of soils are given as surface creep, saltation and suspension. In surface creep, the grains move along the surface either by the direct forces transmitted to them by the winds or by the impact of other grains.

In saltation, the grains rise into the air for a limited distance during which distance they gain momentum in the direction of the wind and fall back to the ground along a diagonal path to the horizontal. In suspension, the grains, once raised from the ground, are small enough so that the turbulence of the air stream keeps them suspended and will move them to higher altitudes by turbulent diffusion. Surface creep occurs at the ground surface while grains in saltation seldom rise more than a few feet above the ground. While these latter phenomena are of importance in spreading an initially contaminated area and in possibly eroding the size of the grains so that they become capable of suspension, the resuspension of concern in this paper is primarily associated with the fraction which these authors have categorized as the suspended fraction since this will be composed, at least partially, of grains with particle size in the inhalable range.

The soil grains in the medium size range of about 0.1 to about 0.5 mm are the ones affected by saltation with grains 1 mm or greater in diameter too large to be moved even in surface creep by ordinary erosive winds. Chepil[10] points out that the erosive action of the winds is primarily due to the saltation process since the bounding particles pick up energy from the winds during the period which they are airborne and this energy is transmitted to the soil particles upon impact to move the larger ones by surface creep, to provide energy to saltate more particles or to dislodge the smaller ones to permit the wind to carry them away in suspension. He has also observed a threshold velocity in the wind speed which will result in soil movement. This threshold velocity is least for soil particles of about 0.1 to 0.15 μm in diameter and increases for both smaller and larger particles. At this minimum, the threshold velocity is about 8 to 9 miles per hour at 6 feet above the ground. For the smaller particles, the higher threshold velocity is attributed to the smooth character of the surface attained and the nature of the turbulence in the air above the surface. However, for mixtures containing both erosive particles and fine particles the threshold wind velocity can be much lower than for the fine particles alone. Chepil also points out that the threshold velocity is not affected by surface roughness features such as ridges.

This work on the erosive properties of winds provides considerable insight into the forces and problems encountered in the resuspension problem. However, the complete applicability of the concepts and measurements to the problem at hand is questionable since the observations are necessarily of a gross nature because of the interest in the movement of large quantities of soil. Thus, considerable interest in the erosion work is attached to the condition where the mass of soil carried by the wind is sufficient to change the wind profile close to the ground because of the added momentum of the soil carried. In some of the data for mass flow over loamy soils, the suspension flow varies from about 30 mg/cm^2 at ground level to about 3 mg/cm^2 at 24 inches above the ground with wind speeds of 13 to 30 mph at a height of 12 inches. These flows convert to concentrations of tenths of grams to grams per cubic meter. This is not to say that such conditions are not of concern in resuspension work but that the mechanisms involved in resuspending the small particles of interest may be different from those observed in the gross erosion studies and the frequency of occurrence of the heavy loads in most areas is relatively small unless there is widespread disturbance of the soil surface, as in many agricultural practices.

There is a series of observations on the effect of soil condition and terrain which would seem to have some relevance to the resuspension studies. It is indicated that particles less than 0.005 mm (5 μm) do not exist, as such, in ordinary soils since they become aggregated into larger particles. However, it is also noted that large quantities of non-erosive soil are converted to erosive material by abrasion caused by the moving soil grains. Thus,

one would expect some breakup of the aggregates formed by the soil and a contaminant by a similar mechanism or by mechanical disturbances. If a surface has been undisturbed for some time, the initiation of erosive movement can require a higher velocity than for succeeding wind storms due to the formation of a surface crust which is broken by the erosion caused by the first high wind. When soil is carried by saltation, it can be sorted into dunes. This process can increase the susceptibility of the soil to later pickup and decrease the threshold velocity. Such increase in susceptibility may be of particular importance when the succeeding wind comes from a different direction and can, therefore, pick up material previously deposited in an eddy behind an obstruction. A rain storm may have an effect in increasing the threshold velocity but it has been observed that such effects will not persist after the rain since a few grains in saltation will break the surface crust.

It is obvious that no one resuspension rate will be applicable to all conditions. A listing of variables which would be expected to influence the results would include:

1. Particle size distribution in the soils
2. Particle size distribution of the contaminant
3. Distribution of contaminant through the soil profile
4. Moisture content of the soil
5. Chemical composition of the soil (cementing and compacting)
6. Type and magnitude of vegetative cover
7. Obstacles to airflow and turbulence inducers

Since all of these factors can also change with time, in particular the moisture content, vegetative cover, distribution of the contaminant in the soil profile and the particle size of the contaminant through aggregation, a thorough understanding of the mechanisms of resuspension will require characterization of many variables. Since powerful numerical techniques are now available for studying hydrodynamic problems such as this, a theoretical program to provide some insight into the importance of such variables would seem to be of great value. In addition to providing this insight, such a study could provide valuable guidance in defining the types of measurements to be required in the field.

One factor not considered above, nor in this calculational model, is that of surface redistribution through runoff of water or movement through the actions of the winds. There is no doubt that this is of considerable importance since such redistribution will affect not only the area covered but the redeposition will be in places where the susceptibility to resuspension may differ from that in the original position. However, the complexities of this problem are beyond the scope of this treatment and study of this will be deferred to a later date.

Item 3, the distribution through the soil profile, is of considerable importance in interpreting field results. For wind pickup, for example, the material which is deeply buried will not be in a position where the wind forces can act on it and should, therefore, not be included in any estimate of the source term. Thus, samples taken to a depth of several inches can be misleading if a significant part of the contaminant occurs below the surface but is included in the measurement of the inventory and is interpreted as part of the contamination of concern. It can be predicted that the critical thickness of the contaminated layer of interest will vary with the degree of disturbance which causes the material to become airborne. However, for most cases, the prediction of this thickness is not now possible. For pickup by winds we can speculate that this thickness may vary with the wind speed due, in part, to the increased size and energy of the particles carried in saltation or surface creep with higher wind speeds and their consequent ability to disturb a deeper layer of soil. Note that if this is true, the dependence of air

concentration with wind speed will have a term not usually considered if the contamination is deeper than the immediate surface. (i.e., the source term will change due to the availability of material buried in the ground at greater depths.) Thus, in measurements of resuspension, as well as in applying measured rates to other areas, it is of great importance to specify clearly the depth of burial of the contaminant and to take into account differences in the depth. Again, some theoretical studies using the numerical, hydrodynamic techniques now available could possibly be of use in better delineating this problem and some of its ramifications.

Another factor which can contribute uncertainty to the final result is the question of particle size of the contaminant (and soil) and its influence on the rate of resuspension. The work of Chepil, discussed earlier, indicates a definite dependency of the erosion rate on particle size distribution of the soils with uniform, relatively small particles requiring higher wind speeds to dislodge than heterogeneous mixtures of several hundred μm particles. Again, the mechanisms by which the particles are transferred to the air are of importance but not well defined quantitatively. For non-vegetated areas, the mechanical transfer of energy from particles in saltation or surface creep would seem to be the primary sources of such energy. Here, the energy available would seem to increase as the square of the wind speed but it is not clear that this energy would be transferred to the soil particles in such a manner that particles of all sizes would be dislodged in proportion to the energy. In other words, it is not now known whether all particle sizes will contribute to the source term in proportion to their fraction in the soil at all wind speeds or whether one would expect changes in the fraction of different sizes airborne as the wind speed changes. (Note that there will be a change in the upper end of the particle size spectrum, with the number of larger particles increasing with wind speed, simply because the energy available will dislodge larger particles at the higher speeds and the increased turbulence will result in longer times of residence in the atmosphere. The effect on the smaller, inhalable sizes, however, is not clear.)

B. Rate of Resuspension - Direct Experiments

The bulk of the information available in the literature on the resuspension factor is not adequately documented with meteorological conditions and extent of the contaminated area to permit derivation of the rate of resuspension from the measurements of the air concentrations although, as will be seen, some estimate of the order of magnitude under the conditions at the time may be derived.

In the earlier papers on resuspension,[3,4] a rate of resuspension was derived from experimental measurements downwind from a source of zinc sulphide particles spread on the ground. In this paper, an attempt was made to account for particle size by inclusion in the pickup coefficient of a term combining the particle density and area exposed to the wind, a refinement presently not believed to be completely applicable until more data on the effect of particle size are available. For this reason, the pickup coefficient in the earlier paper is not the same as the one used in Eq. (A-1). However, a conversion can be made by comparison of the equations. This technique was used since the pickup coefficient in the earlier paper was calculated using the actual wind direction in relation to the position of the sample in order to make a correction for an off-center plume and these data are no longer available. From this comparison, the MMD of the zinc sulphide particles used (7μm) and the density of ZnS (4.1 g/cc), the conversion from the coefficient in the earlier work (K') to the coefficient used in this paper (K) becomes:

$$K = \frac{K' \bar{u}^2}{\rho d} = 0.035\, K' \bar{u}^2 \qquad \text{(A-11)}$$

TABLE A-III

RATE OF WIND PICKUP OF ZnS PARTICLES

First experiment - sandy soil, sparse desert grass, and clumps of sagebrush 0.5 to 1 meter high.

$\bar{u}$ m/sec	K $Sec^{-1} \times 10^9$	$K/\bar{u}^2$ $Sec/m^2 \times 10^9$	$\bar{u}$ m/sec	K $Sec^{-1} \times 10^9$	$K/\bar{u}^2$ $Sec/m^2 \times 10^9$
2.7	90	9.5	1.8	60	17
3.1	140	15	2.7	150	20
2.7	50	6.7	2.2	60	13
0.9	130	16	3.6	160	13
2.7	70	9.5	1.8	26	8.1
2.7	40	18	1.3	40	24
1.8	10	3.9	1.3	40	24

Second experiment - prepared courses.

Course	$\bar{u}$ m/sec	K $Sec^{-1} \times 10^9$	$K/\bar{u}^2$ $Sec/m^2 \times 10^9$	Remarks
Control	5.8	120	3.5	
	10	2450	25	
	8.1	70	1.1	Damp
	6.7	310	6.7	Wet
	8.2	940	14	Wet then dry
Furrowed	5.8	350	11	
	10	700	7	
	8.1	920	14	Damp
	6.7	140	3.2	Wet
	8.2	240	3.5	Wet then dry
Rock	5.8	350	11	
	10	3500	35	
	8.1	230	3.5	Damp
	6.7	470	11	Wet
	8.2	470	7	Wet then dry
Snow fence	5.8	47	1.4	
	10	350	3.5	
	8.1	140	2.1	Damp
	6.7	310	7	Wet
	8.2	240	3.5	Wet then dry

Values of the rate of pickup for these particles as obtained from the conversion are given in Table A-III. The value of K/u^2 is included since the total energy available in the wind varies as the square of the wind speed although there are other factors, such as wind profile and turbulence which will also affect the results.

These results are representative of the particular type of particles used as a tracer and represent the pickup a relatively short period of time after the deposition has occurred. No significant change was seen in the rate of resuspension in the one week period over which measurements were made in the first experiment. It is of interest to note that positive concentrations were measured at low wind speeds, on the order of one m/s or less, or lower than the minimum threshold velocity given by Chepil. This may be due to the existence of gusts with speeds much above the average but cannot be attributed to pickup at an earlier time when winds were stronger because of the short distances involved in the experiment.

Several other experiments under field conditions were examined briefly to provide an order of magnitude estimate of the results. In most of these experiments, the published literature is inadequate to permit full evaluation since required details are not given. They are reviewed, however, with assumptions made as to the missing data.

Wilson et al[11] report on an air sampling program associated with the contamination of an area following a safety test with a nuclear device containing plutonium at the Nevada Test Site. Data on the detailed contamination patterns are not given although it is noted that samplers were located approximately northeast of ground zero at distances of about 7500 feet (at the $10 \mu g/m^2$ contour), 2500 feet at the $100 \mu g/m^2$ contour and at about 750 feet at the $1000 \mu g/m^2$ contour. It was further noted that the winds blew generally from the south during the period of the experiment (about 60% of the time) and that this wind direction missed the highest contamination areas. Because of the wide variation in the air concentrations measured, only the median concentration of the three samplers at each of the 10 and $100 \mu g/m^2$ isopleth and the two samplers at the $1000 \mu g/m^2$ isopleth are given. These were read from a plot in the reference. Sampling started about 23 days after the contamination pattern was established and continued for 20 weeks.

It was assumed, for rough estimation purposes, that the average concentration level in the area over which the wind blew was three times the level at the location of the sampler and the average distance of contamination over which the wind blew before reaching the sampler was equal to the distance from the sampler to ground zero. The average wind speed was taken to be two meters per second with neutral conditions. Since particle size analysis showed the resuspended materials to have

TABLE A-IV

ROUGH ESTIMATES OF PICKUP RATE FROM NEVADA STUDY

10 μg/m²			100 μg/m²			1000 μg/m²		
*dis/min	K x 10⁹ Sec⁻¹	K/ū² x 10⁹ m²/sec	*dis/min	K x 10⁹ Sec⁻¹	K/ū² x 10⁹ m²/sec	*dis/min	K x 10⁹ Sec⁻¹	K/ū² x 10⁹ m²/sec
630	60	20	1200	10	3	8000	9	2
---	--	--	680	7	2	1200	1	0.3
330	30	8	250	3	0.6	1200	1	0.3
330	30	8	1200	10	3	1800	2	0.5
140	10	3	130	1	0.3	230	0.3	0.06
31	3	0.7	620	6	2	950	1	0.3
55	5	1	290	3	0.8	2500	3	0.7
220	20	5	95	1	0.2	1100	1	0.3
90	8	2	120	1	0.3	2500	3	0.7
150	10	4	600	6	2	1100	1	0.3
82	8	2	230	2	0.6	1000	1	0.3
35	3	0.8	310	3	0.8	2100	2	0.6
110	10	3	70	0.7	0.2	850	0.9	0.2
68	6	2	35	0.4	0.09	140	0.2	0.04
58	5	1	150	2	0.4	650	0.7	0.2
42	4	1	200	2	0.5	490	0.5	0.1
65	6	2	23	0.2	0.06	180	0.2	0.05
39	4	0.9	130	1	0.3	200	0.2	0.06
52	5	1	85	0.9	0.2	100	0.1	0.03
22	2	0.5	95	1	0.2	180	0.2	0.05
av	10	3		3	0.8		1	0.4

*Quantity collected in one week at a flow rate of 17 liters per minute.

an average mass median diameter of 1.5 to 2.5 μm, the deposition velocity was assumed to be that corresponding to the turbulent transfer velocity. The values of the expected air concentrations for a unit deposition level were calculated from Eq. (A-10) and were corrected for the assumed two m/sec wind speed. These results are given in Table A-IV for the successive weeks of sampling.

The uncertainties in these results due to the necessary assumptions are obvious, but it is of interest that they are generally not greatly different from those measured with the zinc sulphide particles. It is not surprising that they are lower since the zinc sulphide data were obtained with short period air samples with the wind blowing from the source to the sampler while these values represent a week's sample with no correction for the fraction of time that the wind blew across the contaminated area. Further, there is no correction for periods of higher wind velocity nor for differences in stability of the atmosphere which would be expected from day to night. Soil sampling at the location of the samplers indicated the nominal isopleths to be high by a factor of about four at the 10 μg/m² location, 2.5 at the 100 μg/m² location and 2 at the 1000 μg/m² location. Use of these soil values for the calculation would increase the resuspension factors estimated by these factors. The generally lower resuspension rates for the higher contamination areas could be due to a number of different causes including a greater sensitivity in these areas to a misestimate of the effective path length of the wind over the contaminated areas and the fraction of the time that the wind blows over this path; shorter crosswind dimensions so that the area is not really infinite in the y direction as is assumed in Eq. (A-10);

differences in particle sizes of the contaminant with larger particles in the more heavily contaminated area close to ground zero; or failure of the contamination to reach the five foot height of the samplers in the shorter, more contaminated areas.

Mork[12] reports on an experiment at the Nevada Test Site in which a vehicle was driven back and forth across a stretch of ground contaminated with plutonium for one hour while air samples were taken at two points 20 feet from the vehicle path and at two points 100 feet from the vehicle path. The experiment was conducted in a region between two sampling points designated as number 62 and 63 which, in turn, were reported to have contamination levels of 6.21×10^5 dis/min per square foot and 6.63×10^5 dis/min per square foot. The vehicle path was 1320 feet long with the samplers located about one-third of the distance from each end. The experiment was conducted twice. The first in the daytime was from 1138 to 1238 and the second (labeled as at "night") was from 1725 to 1825. Since it is doubtful that the nighttime inversion would have set in by this time in the evening, both sets of data were evaluated considering the atmosphere to be unstable.

Neither the wind direction or the wind speed were given. It was, therefore, assumed that the wind was blowing across the vehicle path toward the samplers. Gummed paper deposition collectors were included which permitted a rough estimation of the deposition velocity to this surface by comparison with the air concentration. The results of this experiment as calculated under these assumptions are given in Table A-V.

The deposition velocities would indicate that larger particles were involved in most cases. The relatively lower values of resuspension in the second experiment may indicate that the course had undergone depletion or that some other factor in the conditions had changed. The value of K/u^2 is not estimated for these data since the source term is assumed to be more dependent upon the mechanical disturbance than on the energy transmitted by the winds. Although comparison with the values of K from the other experiments is difficult, it does appear that resuspension rates up to 100 times those from the natural winds can occur from this type of mechanical disturbance in this type of terrain.

TABLE A-V

MECHANICAL DISTURBANCE RESUSPENSION

	Location			
	20'		100'	
	West	East	West	East
1138-1238				
V_d (m/sec)	0.5	0.004	0.9	0.02
K (sec^{-1})	$160 \times 10^{-9}\bar{u}$	$9000 \times 10^{-9}\bar{u}$	$3000 \times 10^{-9}\bar{u}$	$2600 \times 10^{-9}\bar{u}$
1725-1825				
V_d (m/sec)	0.3	2	---	0.4
K (sec^{-1})	$130 \times 10^{-9}\bar{u}$	$150 \times 10^{-9}\bar{u}$	---	$180 \times 10^{-9}\bar{u}$

In a more recent paper by Sehmel[13] the particle resuspension due to moving vehicles on an asphalt road was measured by use of ZnS particles with a mass median diameter of about 5 μm. These particles were distributed uniformly over a length of 100 feet on one lane of an asphalt highway and cars and trucks were driven at different speeds either on the adjacent lane (by-pass) or through the contaminated lane. Runs were made while the winds were perpendicular to the highway and the fraction resuspended per pass was evaluated from air samples and deposition samples downwind. For purposes of this discussion, the values for the resuspension per pass were converted to fraction resuspended per second from consideration of the wind speed of the vehicle and the 100 foot path length. This was done to enable comparison with previously derived resuspension rates. The data from this work are given in Table A-VI.

As is pointed out by Sehmel, the relative constancy of the ratio of the rate of pickup to the square of the vehicle speed indicates the primary mechanism of pickup is due to the turbulence induced by the passage of the vehicle. Again, the data indicate increased pickup rates, in this case by as much as three or four orders of magnitude over those measured from ZnS in the soils and

TABLE A-VI

RESUSPENSION RATES OF ZnS PARTICLES FROM ASPHALT ROAD BY VEHICLES

Time Since Deposition (days)	Vehicle	Vehicle Path	Vehicle Speed (m/sec)	Fraction Resuspended per pass	Fraction Resuspended per sec (K)	$\frac{K}{(\text{Vehicle Speed})^2}$
0	Car	By-pass	2.2	4.8×10^{-5}	3.5×10^{-6}	7.0×10^{-7}
			6.7	2.8×10^{-4}	6.2×10^{-5}	1.4×10^{-6}
			13.4	7.7×10^{-4}	3.4×10^{-4}	1.9×10^{-6}
			22.4	1.1×10^{-3}	8.1×10^{-4}	1.6×10^{-6}
		Through	2.2	1.9×10^{-4}	1.4×10^{-5}	2.8×10^{-6}
			13.4	6.9×10^{-3}	3.0×10^{-3}	1.7×10^{-5}
			22.4	1.1×10^{-2}	8.0×10^{-3}	1.6×10^{-5}
	*Truck	Through	2.2	2.5×10^{-3}	1.8×10^{-4}	3.7×10^{-5}
			22.4	6.7×10^{-3}	4.9×10^{-3}	9.8×10^{-6}
4	*Truck	By-pass	6.7	1.2×10^{-5}	2.6×10^{-6}	5.9×10^{-8}
			6.7	4.8×10^{-6}	1.1×10^{-6}	2.3×10^{-8}
			13.4	8.6×10^{-5}	3.8×10^{-5}	2.1×10^{-7}
			13.4	8.2×10^{-5}	3.6×10^{-5}	2.0×10^{-7}
		Through	2.2	1.3×10^{-5}	9.5×10^{-7}	1.9×10^{-5}
			6.7	5.2×10^{-4}	1.1×10^{-4}	2.5×10^{-6}
			6.7	2.1×10^{-4}	4.6×10^{-5}	1.0×10^{-6}
			13.4	1.0×10^{-3}	4.4×10^{-4}	2.5×10^{-6}
			22.4	2.3×10^{-3}	1.0×10^{-3}	2.0×10^{-6}
5	Car	Through	13.4	5.7×10^{-5}	2.5×10^{-5}	1.4×10^{-7}
30	Car	Through	13.4	5.5×10^{-6}	2.4×10^{-6}	1.4×10^{-8}
			22.4	2.6×10^{-5}	1.9×10^{-5}	3.8×10^{-8}

*3/4-Ton Pickup Truck.

natural winds. The data taken at later times, however, indicate a relatively rapid decrease whether due to fixation of the particles or prior removal is uncertain. Sehmel does provide a rough calculation, however, which indicates that the depletion from such a surface with any significant traffic would be rapid.

C. Changes With Time

It is to be expected that the susceptibility of a contaminant to resuspension will change with time due to factors such as agglomeration with the soil particles; possible chemical changes of the contaminant; migration of the particles downward in the surface through action of rainfall, alternate freezing and thawing; and redistribution, perhaps into areas protected from the winds. For materials deposited in an area with high mechanical disturbance, such as a highway, the latter factor will be of great importance in moving the material to an area where the disturbance is lower.

Wilson, et al[11] investigated the resuspension of plutonium in an area at the Nevada Test Site which was contaminated during a safety test in April 1957. Samples were taken with impactors at a height of five feet above the ground starting about one month after the area was contaminated and continuing for twenty weeks. Three samplers were placed at the nominal $10 \mu g/m^2$ contour (~7500 feet from GZ), three at the $100 \mu g/m^2$ contour (~2500 feet) and two at the $1000 \mu g/m^2$ contour (~750 feet). The samples were pooled to give weekly estimates of the concentrations at these locations. The samplers were located so that the winds blew directly over the most contaminated area only about 10% of the time during the period of sampling. It was noted that: "The sampling data are too erratic to establish half-times for the 'decay' of air concentration beyond a very crude estimate." These estimates were obtained by plotting the median of the stations

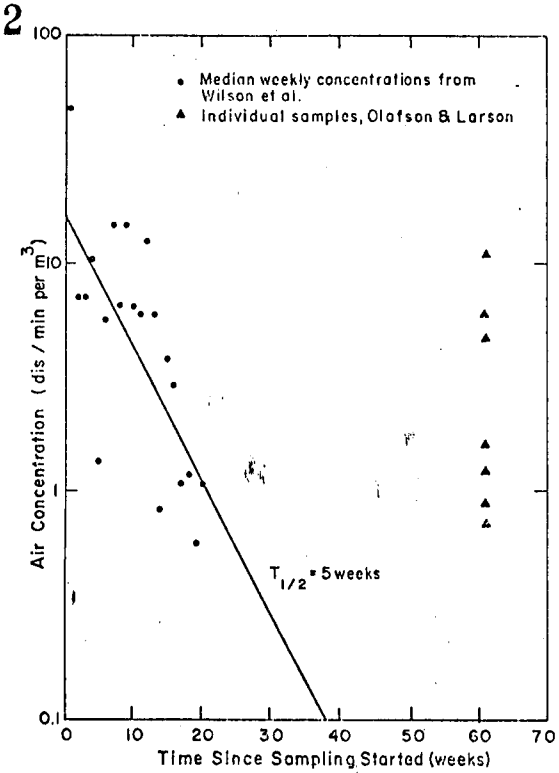

Fig. A-3. "Decay" of Air Concentrations - 1000 μg/m³.

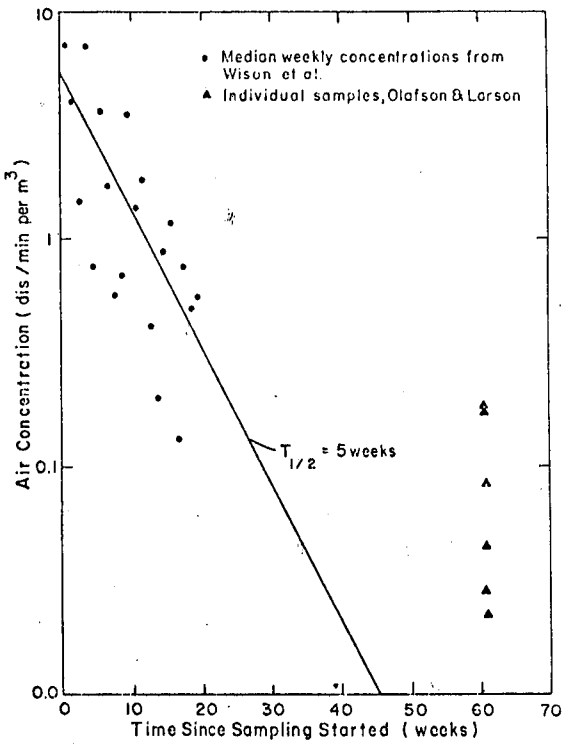

Fig. A-4. "Decay" of Air Concentrations - 100 μg/m³.

on a given isopleth. This yielded an estimate of five weeks for the half-time of concentration decay.

During the summer of 1958, studies of the air concentrations were conducted at the same site.[14] The two high level locations for the location of the samplers were quoted as being at "... essentially the same location as the nominal 100 and 1000 μgm locations of Wilson et al." The data for these two locations for the median of the weekly samples from Wilson and for the individual samples reported by Olafson and Larson are plotted in Figs. A-3 and A-4. While the use of the week-long sample and the median value of several samplers tends to reduce the absolute concentration due to wind fluctuations over this time and also tends to reduce the statistical spread, the concentrations reported 40 weeks after the first series raise some questions as to the long term applicability of the 35-week half-life. It is noted that there are other factors which could produce a reduction in the air concentrations such as a seasonal shifting of the winds resulting in a lower contaminated area upwind or a seasonal change in wind speed. Both of these variables could result in a regular decrease in air concentration with time if the change in the winds occurred in a systematic manner.

In one other experiment, Mork[12] reports data taken three feet above the ground in October 1956 and in July 1958 at Station 61 at the Nevada Test Station. These samples were in an area contaminated with plutonium during December of 1955 and January of 1956. In a seven day period in 1956 series the concentrations averaged about 2×10^{-8} μg/m³ with a range of about 4×10^{-9} to 6×10^{-8} μg/m³. In a 20 day period in the 1958 series, the concentrations averaged 2×10^{-7} μg/m³ with a range

in the values from 0 to 7×10^{-7} $\mu g/m^3$. While, again the wind directions and speeds may well have differed during these two periods, there is no indication of a measurable decrease with time over a period of some twenty months or about 85 weeks.

As was indicated, one would expect a change with time, but it is believed that the data now available are not adequate to permit the assignment of a particular decay rate, particularly for areas of different characteristics from the Nevada Site. In particular, it is believed that the use of the 35-day half-life is inappropriate since this would indicate that the concentrations drop rapidly and, for long term occupancy considerations, result in exposures estimated only for the initial period of occupancy. A more reasonable estimate to describe this phenomenon would be to consider a drop by a factor of ten over the first year or two with the conditions stabilized thereafter to give relatively little decrease.

In view of the information available on the initial concentrations at specified locations at the Nevada Test Site, it would appear to be reasonable to mount a campaign to resample these locations over a period of time and to attempt to reconstruct the meteorological conditions for the initial samples. While the deposits have been disturbed by the various activities in these areas, such a series of samples could give some indication of the long term decrease.

D. Dust Concentrations

The concentrations of natural dust in the atmosphere arise, at least in many areas, from resuspension of soil grains under natural or mechanical disturbance mechanisms. As such, data on these concentrations can be used to give at least gross indications of the importance of resuspension in various terrains and locations. Again, such information must be interpreted with restraint, considering the differences which may exist between the deposited contaminant and the natural soil particles with little real data to interpret the effect of particle size or the effect of depth in the soil profile on the rate of resuspension. In addition, calculations indicate that dust in the atmosphere may well have originated a considerable distance upwind and could have been resuspended under completely different meteorological conditions.

One set of data collected by Hilst[15] at the meteorological facility at Hanford has been examined. Here the number of dust particles per cubic foot were measured in five size ranges by use of cascade impactors. In one experiment, measurements were made at five heights ranging from 1.25 feet to 400 feet while in the others, the measurements were made at three intervals from 0.9 feet to 41.3 feet. These observations show that, in general, the dust concentrations, expressed as mass per unit volume, decreased rapidly with height. However, the mass median diameter of the particles also decreased so that the change in concentration of the smaller particles with height was much less pronounced and, in several cases was not detectable.

The fractional rate of pickup from the ground cannot be determined from these data because of the lack of information on the source material. However, the source term, itself, can be determined and compared to the wind speed. These data are given in Table A-VII. Again, the ratio of the rate of pickup, as determined from these data assuming unstable conditions and a source upwind approaching infinity, to the square of the wind speed appears to be relatively constant.

E. An Example of Application

As a part of the effort of the Nevada Applied Ecology Group to define potential problems with plutonium contamination on the Nevada Test Site, an extensive effort is being made to measure contamination patterns, resuspension and redistribution, and the ecological behavior of the plutonium. We have chosen the GMX area for analysis because

TABLE A-VII

DUST CONCENTRATIONS AND ESTIMATED PICKUP RATES

				0.9 - 5 μm $V_D/\bar{u} = 0.013$		5 - 20 μm $V_D/\bar{u} = 0.013$			20 - 60 μm $V_D = 0.2$ m/sec			60 - 240 μm $V_D = 1$ m/sec		
Date	Height Ft	$\bar{u}$ m/S	χ #/m³	KΩ $Sec^{-1}m^{-2}$	KΩ/$\bar{u}^2$ $Sec^{-1}m^{-3}$	χ #/m³	KΩ $Sec^{-1}m^{-2}$	KΩ/$\bar{u}^2$ $Sec^{-1}m^{-3}$	χ #/m²	KΩ $Sec^{-1}m^{-2}$	KΩ/$\bar{u}^2$ $Sec^{-1}m^{-3}$	χ #/m²	KΩ $Sec^{-1}m^{-2}$	KΩ/$\bar{u}^2$ $Sec^{-1}m^{-3}$
8/11 p.m.	1.25	6.3	2.3×10^5	37,000	950	91,000	15,000	380	26,000	6600	170	2500	2500	64
	50.	11	1.8×10^5	30,000	770	55,000	9,000	230	9,700	2400	62	320	320	8
	100.	12	1.2×10^5	20,000	500	40,000	6,600	170	6,500	1600	42	110	110	3
	200.	12	1.4×10^5	22,000	570	41,000	6,700	170	5,200	1300	33	35	35	0.9
	400.	14	8.0×10^4	13,000	340	29,000	4,800	120	3,100	780	20	70	70	2
5/4 a.m.	0.9	1.3	7.8×10^4	2,700	1500	12,000	440	240	1,300	260	150	70	70	40
	14.7	1.8	5.3×10^4	1,900	1000	6,700	240	130	210	40	20	10	10	6
	41.3	1.8	5.6×10^4	2,000	1100	6,400	220	130	280	60	30	30	30	20
5/4 p.m.	0.9	1.3	7.8×10^4	2,700	1500	21,000	740	400	2,000	400	220	50	50	28
	14.7	1.8	6.4×10^4	2,200	1200	6,400	220	130	280	60	30	10	11	6
	41.3	2.2	5.6×10^4	2,000	1100	6,400	220	130	320	60	40	20	21	10
*5/27 a.m.	0.9	2.7	3.1×10^4	2,200	300	5,300	370	50	670	140	20	35	35	5
	14.7	3.6	2.0×10^4	1,400	200	3,900	270	40	320	70	9	35	35	5
	41.3	4.5	2.5×10^4	1,800	250	3,900	270	40	460	90	13	35	35	5
5/27 p.m.	0.9	2.2	5.6×10^4	3,600	620	24,000	1,500	260	2,700	550	96	70	70	10
	14.7	3.1	4.9×10^4	3,100	540	11,000	700	120	850	170	30	70	70	10
	41.3	3.6	4.2×10^4	2,700	470	9,500	600	110	460	90	16	35	35	6
10/5 p.m.	0.9	2.7	1.8×10^5	12,000	1700	26,000	1,900	260	2,900	600	80	280	280	40
	14.7	5.8	1.2×10^5	8,700	1200	8,600	600	80	1,200	250	40	70	70	10
	41.3	8.0	1.8×10^5	12,000	1700	8,100	570	80	1,100	220	30	70	70	10

*Ground surface damp.

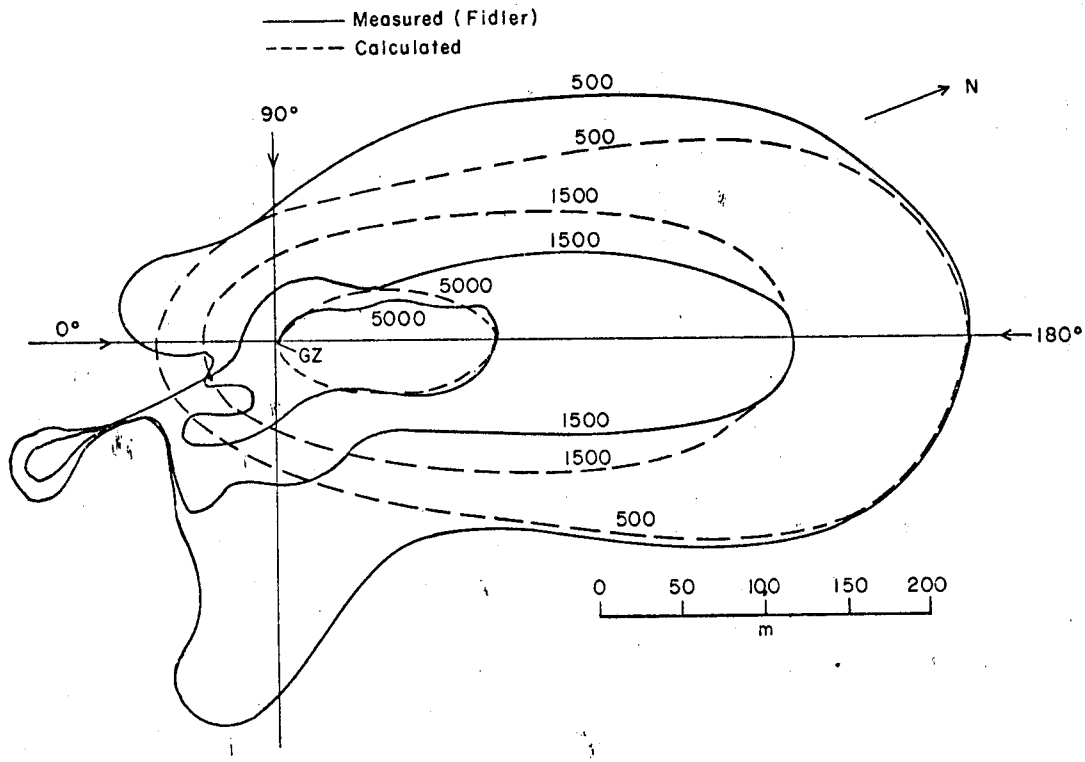

Fig. A-5. GMX Area.

initial resuspension measurements have been planned there and some preliminary data on ground deposition are available. The pattern of deposition has been measured by FIDLER surveys using the 60 kev photons from the ^{241}Am associated with the plutonium in June 1971.[16] This pattern is given by the solid isopleths in Fig. A-5. If we assume that the pattern is Gaussian in the cross-pattern direction with the standard deviation increasing exponentially with distance from ground zero (GZ) and the centerline deposition as given in Fig. A-6, the isopleths shown by the dotted lines in Fig. A-5 are obtained. These isopleths are considered to be sufficiently representative of the pattern to be usable in the calculation of expected air concentrations or the derivation of resuspension rates from measured air concentrations. Although it would be desirable to have an analytical relationship between the position on the pattern and the peak deposition at the centerline, none was found and the relation in Fig. A-6 was used. For the change in standard deviation of pattern width with distance from GZ, the following relation was used:

$$\sigma_y = 37.3 \exp(0.00335a) \qquad \text{(A-12)}$$

where σ_y is the standard deviation in meters at a distance of a meter from GZ.

Calculations were performed for locations on the centerline of the pattern to take advantage of the symmetry so produced. The basic approach was to calculate the expected concentration from Gaussian line sources at various distances from the station with the standard deviation of the pattern at that distance according to Eq. (A-12). Wind

directions were varied at 22.5 deg intervals from 0 deg (wind directly up-pattern) to 180 deg (wind directly down-pattern). These directions are indicated on Fig. A-5. The total concentration was then obtained from these values by weighting according to the centerline concentration from Fig. A-6 and multiplying by the interval represented between the successive line sources. Since the calculations were performed on a Wang 600 programmable calculator it was necessary to limit the number of upwind line sources considered to a total of 50 per calculation. The concentration resulting from a Gaussian line source with a standard deviation of A meters, a centerline deposition of Ω Ci/m^2 and a wind direction of θ to the pattern centerline can be obtained from:

$$\frac{\chi u}{K \Omega p} = \frac{(2-f)}{\pi C_y C_z} \sum_{y=-4A}^{y=4A} \frac{\Delta}{(a \cos\theta + y \sin\theta)^{2-n}} \exp\left[- \left\{ \frac{1}{(a \cos\theta + y \sin\theta)^{2-n}} \left(\frac{(a \sin\theta - y \cos\theta)^2}{C_y^2} + \frac{z^2}{C_z^2} \right) + \frac{2(2-f)Vd}{\sqrt{\pi}\, C_z n \bar{u}} (a \cos\theta + y \cos\theta)^{\frac{n}{2}} + \frac{y^2}{2A^2} \right\} \right] \quad \text{(A-13)}$$

In Eq. (A-13), the angle θ is measured between the perpendicular to the line source and the wind direction. In practice, calculations were made separately for the up-pattern wind direction (0-90°) and the down-pattern (90-180°) switching the sign of the coordinate system so that a was always positive.

Values of the pickup and dispersion parameter were calculated for stations located 150 m up-pattern from GZ; at GZ; and at 75 m, 200 m, and 400 m down-pattern from GZ. The values were then normalized to a value of 1 Ci/m^2 at the peak deposition point, 6400 c/m on the FIDLER. These results are given in Figs. A-7 and A-8.

Since the deposition pattern was measured with the FIDLER, a relation between this measurement and the quantity of plutonium in the soils in a position to be picked up by the winds is needed. Eberhardt and Gilbert[16] have provided a statistical summary of the data on soil analyses in this area including some preliminary correlations between the FIDLER readings and the soil analyses. In this study, FIDLER readings were made at given locations followed by sampling in three 5-in. circles to a depth of three centimeters. The number of samples taken was limited and no correlation was found for those measurements in the lower two isopleths. However, in the >5000 c/m isopleth, six samples showed an apparent correlation with the FIDLER reading. These data indicated about 0.3 dis/min of plutonium per gram of soil for each count per minute on the FIDLER. While this correlation is rough and the authors warn against applying it to the lower contamination levels, in this preliminary study we will accept it with the reservation that as more data become available the correlation (as well as the shape of the pattern) should be revised.

There is, however, no information on the change in plutonium concentration with depth in the soil profile or on the microdistribution of plutonium in the area measured by the FIDLER. Some rough calculations indicate that the FIDLER sensitivity for ^{241}Am decreases to about 50% of the surface value for a plane source buried 8 mm and to about 10% for a plane source at a depth of 2.5 cm. The soil sampling procedure averages the total quantity of plutonium over the sampling depth of 3 cm. Thus, while the correlation would indicate that there would be about 7×10^{-9} Ci/m^2 (assuming a soil density of 1.6 g/cm^3) per c/m on the FIDLER, the actual fraction of this which is effective in

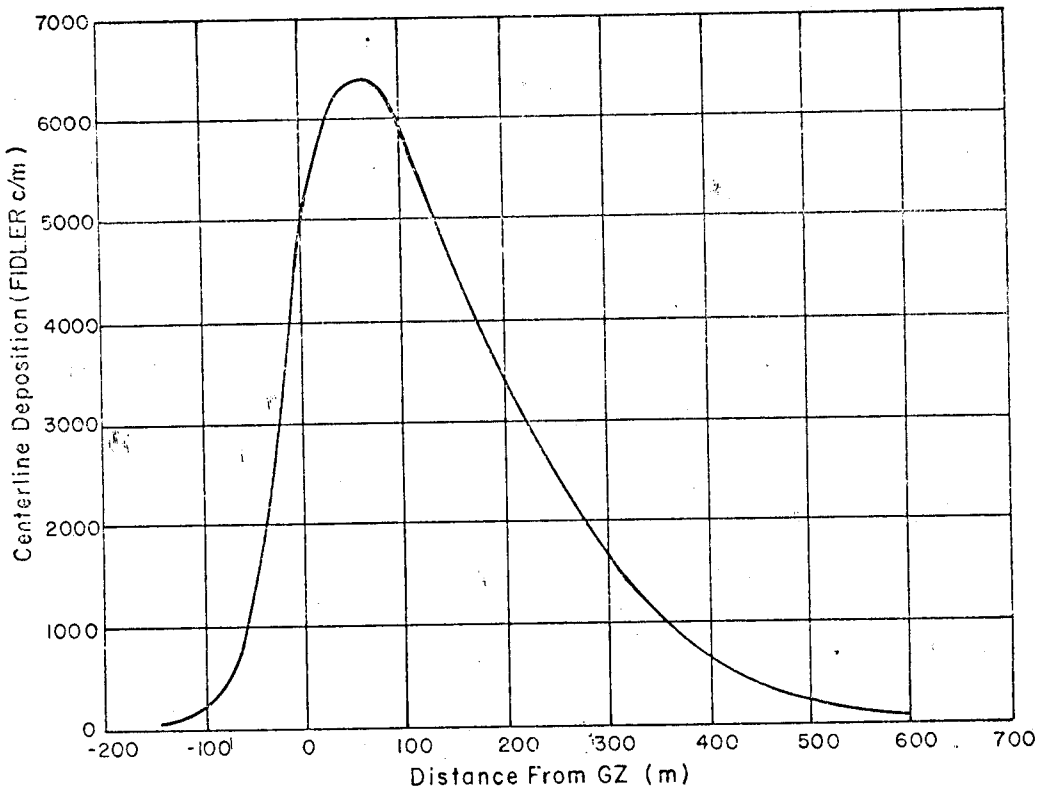

Fig. A-6. Centerline Deposition Pattern in GMX Area.

producing air concentrations is not known. For these preliminary calculations, we will use the above value but remember the reservations quoted.

Several air samples have been obtained in this area during tests of the Lawrence Livermore's high volume air sampler.[17] The sampler was located about 250 feet (76 m) north of GZ. Five samples were taken in April of 1972: two for periods of 17-19 hours and three for periods of 4-5 hours. The resuspension rates for the three shorter samples were estimated from Fig. A-7 assuming the peak deposition on the pattern to be 6400 c/m on the FIDLER or 4.3×10^{-5} Ci/m^2. These data are given in Table A-VIII.

For these calculations, it was assumed that unstable atmospheric conditions existed. This seems appropriate for the middle of the day at this time of year. For the longer samples, which were taken overnight, there was considerable variation in both wind direction and wind speeds with low wind speeds occurring during the middle of the night. In addition some of the data on wind speeds are missing. An attempt to approximate the value of K/u^2 was made by using the hourly recorded values of wind speed and direction. These results, while very crude indicated values on the order of 10^{-13} to 10^{-14} with the lower value increasing to about 10^{-13} if it were assumed that pickup occurred only during the unstable periods with higher wind speeds.

These resuspension rates have considerable uncertainty, particularly with respect to the definition of the surface deposit. If, for example, it is considered that the top millimeter of the soil is

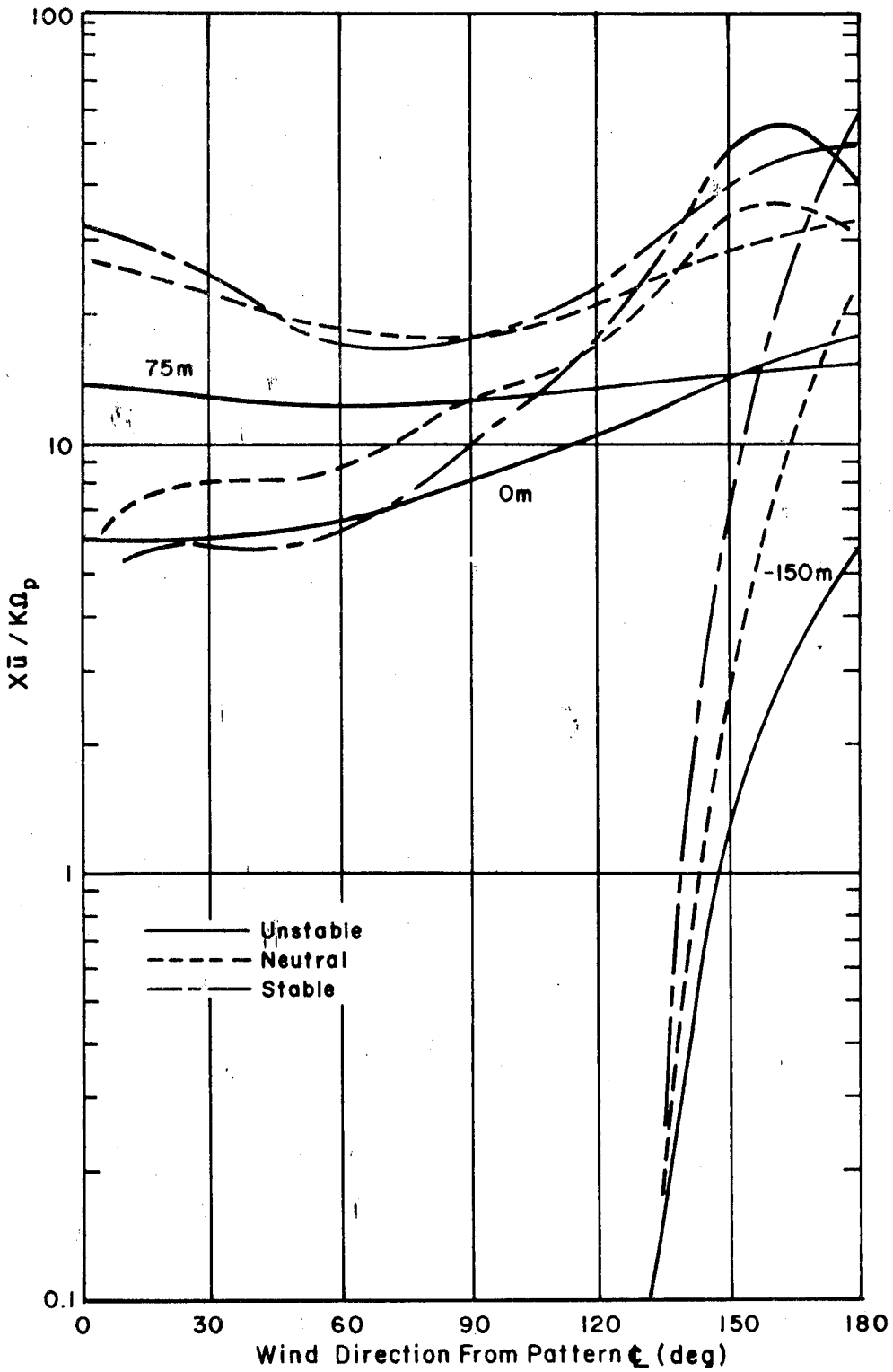

Fig. A-7. Concentration Integrals for Different Wind Directions with Respect to Pattern Centerline.

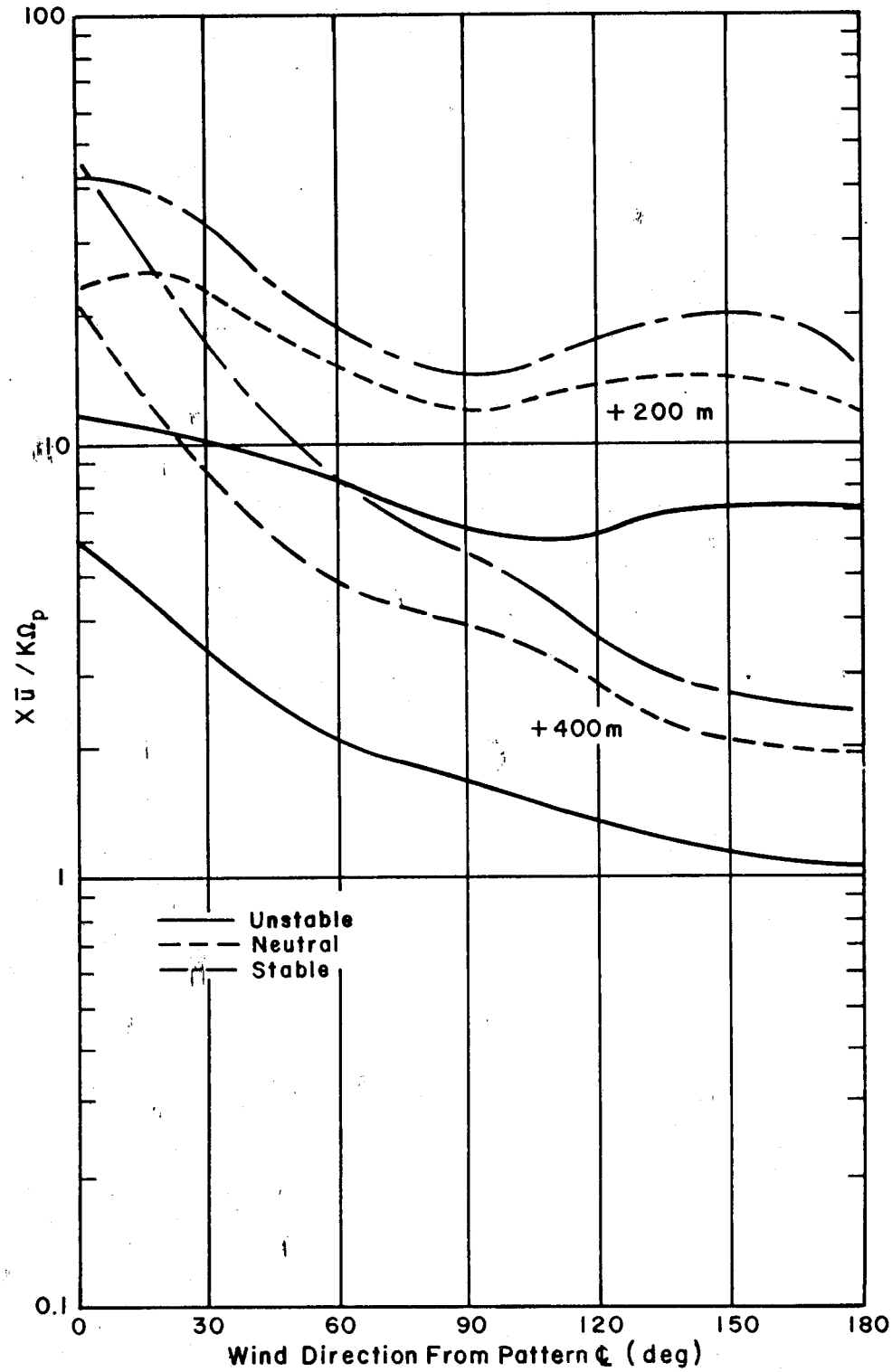

Fig. A-8. Concentration Integrals for Different Directions with Respect to Pattern Centerline.

TABLE A-VIII

ESTIMATED RESUSPENSION RATES FOR THE GMX AREA

Time of Sample	Measured Concentration (Ci/m^3)	Wind Direction True (deg)	Wind Direction *Pattern (deg)	Wind Speed (m/s)	$\frac{\chi u}{K\Omega_p}$	K	K/u^2
4/19/72 1200-1600	3.5×10^{-16}	350	155	3	15	1×10^{-12}	2×10^{-13}
4/27/72 1100-1600	1.4×10^{-14}	218	18	4	14	1×10^{-10}	5×10^{-12}
4/20/72 0930-1400	1.5×10^{-15}	220	20	5	14	1×10^{-11}	5×10^{-13}

*Pattern centerline is 20 deg east of true north. Value given here is for application to the directions used in the calculation.

the layer of importance and the plutonium is distributed uniformly through the three centimeter sampling thickness, then the effective surface deposit is only 1/30th of that used above and the resuspension rates will be increased by a factor of 30. In addition, the sampling period is relatively long in comparison to that believed appropriate to the dispersion coefficients used in the integrations and these may underestimate the actual cloud spread, again resulting in some increase in the resuspension rates. Even considering these factors, however, the value of K/u^2 appears to be considerably lower than the results quoted earlier for fresh deposits. Additional studies in the area will be needed to fully explain the results and the relatively large variation in K/u^2 from these few samples, but it can be postulated that at least a part of the reason for the lower values may be due to aging of the deposit and redistribution by particle size.

Data are not available on the influence of mechanical disturbance in this area on the resuspension rate. It is noted, however, that the calculation of the average concentration from the full area involves the derivation of values for a line source at various distances upwind with the wind in different directions. Studies of the influence of mechanical disturbance by people or animals walking across the area or a vehicle driving across could be made by sampling during such periods of disturbance. Such results would be extremely valuable in assessing the relative importance of wind pickup and such mechanical disturbance. Similarly no attempt has been made, as yet, to evaluate the long term average concentration taking into account the shifts in wind direction, speed and atmospheric stability. It is believed, however, that the above method, in conjunction with the appropriate meteorological measurements would result in a reasonable estimate.

The above example emphasizes the need for adequate meteorological support in providing the dispersion and deposition parameters to be applied during experiments to assess the pickup rate. Ideally, such measurements should be adequate to permit a more accurate estimate of the dispersion coefficients than has been used here and the actual equations used should be modified to apply the most accurate estimate of the dispersion.

REFERENCES

1. W. H. Langham, "Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads," USAEC Document UCRL 50639 (Lawrence Radiation Laboratory) April 1969.

2. R. L. Kathren, "Towards Interim Acceptable Surface Contamination Levels for Environmental PuO_2," USAEC Report BNWL-SA-1510 (Pacific Northwest Laboratory) April 1968.

3. J. W. Healy, "A Preliminary Estimate of Wind Pickup and Impaction of Particles," USAEC Document HW-35542 (Hanford Atomic Products Operation) March 1, 1955.

4. J. W. Healy and J. J. Fuquay, "Wind Pickup of Radioactive Particles from the Ground," 2nd UN Geneva Conference P/391 USA Pergamon Press, London.

5. D. H. Slade, Ed., "Meteorology and Atomic Energy," 1968. TID-24190, U.S. Atomic Energy Commission, Division of Technical Information, July 1968.

6. A. C. Chamberlain, "Aspects of Travel and Deposition of Aerosol and Vapor Clouds," Document AERE-HP/R 1261 (Harwell) 1953.

7. O. G. Sutton, "Micrometeorology," McGraw-Hill (1953).

8. J. J. Fuquay, Private Communication, 1957.

9. R. A. Bagnold, "The Physics of Blown Sands and Desert Dunes," Methuen and Co., Ltd., London, 1954.

10. a. W. D. Chepil, "Dynamics of Wind Erosion: I. Nature of Movement of Soil by Wind," Soil Science 60 (1945), pp. 305-320.

b. _____, "Dynamics of Wind Erosion: II. Initiation of Soil Movement," Soil Science 60 (1945), pp. 397-411.

c. _____, "Dynamics of Wind Erosion: III. The Transport Capacity of the Wind," SoiL Science 60 (1945), pp. 475-480.

d. _____, "Conversion of Relative Field Erodibility to Annual Soil Loss by Wind," Soil Science Society of America Proceedings 24 (1960), pp. 143-145.

11. R. H. Wilson, R. G. Thomas, and J. N. Stannard, "Biomedical and Aerosol Studies Associated with a Field Release of Plutonium," USAEC Document WT-1511 (University of Rochester Atomic Energy Project) November 1960.

12. H. M. Mork, "Redistribution of Plutonium in the Environs of the Nevada Test Site," USAEC Document UCLA-12-590 (UCLA Laboratory of Nuclear Medicine and Radiation Biology) August 1970.

13. G. A. Sehmel, "Particle Resuspension from an Asphalt Road Caused by Car and Truck Traffic," Atmospheric Environment 7, 3, 291-309, March 1973.

14. J. H. Olafson and K. R. Larson, "Plutonium, Its Biology and Environmental Persistence," USAEC Document UCLA 501 (UCLA Laboratory of Nuclear Medicine and Radiation Biology) December 1961.

15. G. R. Hilst, "Some Observations of Particle Distribution with Height in the Lower Atmosphere," Unpublished document.

16. L. L. Eberhardt and R. O. Gilbert, "Statistical Analysis of Soil Plutonium Studies, Nevada Test Site," Pacific Northwest Laboratories Report BNWL-B-217, September 1972.

17. P. L. Phelps, Private Communication.

APPENDIX B

THE VELOCITY OF DEPOSITION

The concentration downwind from a source of airborne material depends upon the amount of material removed from the atmosphere by natural processes in the region between the source and the receptor as well as upon the source strength and the atmospheric mixing processes. The removal rate varies with the physical nature of the airborne material, the state of the atmosphere and the nature of the terrain involved. Two basic removal mechanisms are washout or rainout, during periods of precipitation, and dry deposition at other times. In this treatment we will be concerned with the dry deposition phenomena since the purpose of the study is to estimate the amount remaining airborne rather than the amount deposited. Information on the washout processes[1,2] can be adapted to estimate the effects of these processes on the air concentrations during periods of precipitation.

Early experience with the effluents from a radiochemical separations plant at Hanford, as well as experiments by Chamberlain indicated that ^{131}I in vapor form deposits strongly from the atmosphere onto surfaces.[3,4] Later experiments by Megaw and Chadwick[5] using solid fission products generated by arcing an irradiated wire showed that the deposition rate of these fine particles was considerably lower than that for iodine vapor. The close-in fallout from nuclear detonations consists of large particles which have a terminal velocity sufficiently great that they will not remain suspended for any length of time but will settle from whatever height they reach in the initial cloud meanwhile being carried by the winds.

This information indicates that there must be at least three separate considerations in the dry deposition of such material from the air: (1) gravity settling for large particles; (2) transfer from the air to the ground by the turbulent eddies in the atmosphere for small particles which remain suspended for considerable periods of time and for gases or vapors; and (3) retention of the receiving surface once the material is brought into contact. Thus, both the fine particles and the iodine vapor should be brought to the ground at about the same rate by the turbulence of the atmosphere, but the iodine, being in a chemically reactive form is apparently retained at the ground surface better than the small particles. Such considerations permit separation of the problem into several parts depending upon the physical nature of the atmospheric contaminant.

It has been customary to express the rate of removal of a given material by the ratio of the rate at which it deposits to the concentration in the atmosphere at the point of concern. Thus:

$$\frac{\text{curies per m}^2\text{ per sec}}{\text{curies per m}^3} = \text{m/sec}\,. \qquad \text{(B-1)}$$

This ratio, which can be measured directly, is referred to as the velocity of deposition since it has the units of velocity. It is the purpose of this appendix to explore the various factors which can influence the velocity of deposition for particles and to derive a simplified model incorporating the important variables so that some indication of the variation expected with these parameters can be derived. The model is not exhaustive in its treatment of the various theories and information available since the intent is to provide an overall picture which is commensurate with our knowledge of the applications, particularly in regard to the influence of this variable on the air concentrations resulting from resuspension of particles from the ground.

I. GRAVITATIONAL SETTLING

The settling of larger particles under the influence of gravity has been studied for many years. This rate of settling is characterized by a terminal

velocity in which the force exerted by gravity is exactly balanced by the aerodynamic drag from the passage through the air. As the particle size decreases, the terminal velocity decreases to a point where the turbulent eddies in the atmosphere exert sufficient force to overcome the gravitational forces and the particle remains suspended. For our purposes a large particle can be defined as one in which the terminal velocity predominates over the atmospheric turbulence and the deposition velocity is essentially equal to the terminal velocity. Note that this is not a definition of a particular particle size since the eddy forces will depend upon the degree of turbulence in the atmosphere at a particular time. Thus, one would expect larger particles to be suspended in an unstable atmosphere than in a stable one.

The simplest particle is a sphere and the terminal velocity of such spheres are given by Stokes' Law[6,7] for particles up to 50-100 μm in diameter. Above this value the drag coefficient increases so that the terminal velocity is smaller than would be calculated from Stokes' Law. However, for simplicity and since we are primarily interested in particles smaller than this, we will apply Stokes' Law throughout. This gives the settling velocity as a function of particle size and density as:

$$V_g = \frac{2 g r^2 \rho}{9 \varkappa} \qquad \text{(B-2)}$$

Here V_g is the terminal velocity in cm/sec, g is the acceleration due to gravity in cm^2/sec, r is the particle radius in cm, ρ is the particle density in gm/cm^3 and ϰ is the viscosity of the air in poises. Actually, the density term should reflect the density difference between the particle and the air, but the air density can be neglected with respect to the density of most particles of interest. It may be noted that the viscosity of air varies from 171 micropoises at 0 deg C to 190 micropoises at 40 deg C so that a minor effect will occur because of temperature. The terminal velocity for several sizes of spherical particles with a unit density evaluated at 45 deg latitude and sea level with a temperature of 18 deg C are given in Table B-I.

TABLE B-I

TERMINAL VELOCITY OF UNIT DENSITY SPHERES IN AIR

Particle Size Radius μm	Particle Size Diam μm	Velocity cm/sec	Particle Size Radius μm	Particle Size Diam μm	Velocity cm/sec
0.05	0.1	3.0×10^{-6}	5	10	0.30
0.1	0.2	1.2×10^{-4}	10	20	1.2
0.2	0.4	4.8×10^{-4}	20	40	4.8
0.5	1.0	3.0×10^{-3}	50	100	30
1.0	2.0	1.2×10^{-2}	100	200	120
2.0	4.0	4.8×10^{-2}			

From Stokes' Law, the terminal velocity of a given diameter of particle will vary directly as the density. Thus, particles from a material with density ten will have terminal velocities ten times those given in the table. Since the velocity also varies as the square of the particle radius, the sizes of particles having the same terminal velocity will vary as the square root of the densities. That is, a one μm particle of a material with a density of nine will have the same terminal velocity as a three μm particle of unit density. Thus, the behavior of the higher density particle in situations where the behavior is dependent upon the aerodynamic properties will be similar to a larger particle of the lower density material.

The situation with spherical particles is highly idealized for most real-life situations. Instead, the particles may be highly irregular in shape so that it is difficult to even characterize them according to any one dimension. The drag characteristics of these irregular particles also vary from that of the sphere so that even particles of the same mass will have different terminal velocities depending upon their shape. The situation is further compounded by the fact that many particles of interest may be agglomerates of other particles frequently of particles of different composition and origin. For this reason, and the variation with density, an aerodynamic diameter is frequently used in particle work.

This may be defined as the diameter of a particle of unit density which has the same terminal velocity as the particle of interest. This diameter will be used throughout this paper unless a correction for density is specifically indicated. Such an aerodynamic diameter essentially defines the inertial behavior of the particle which is of importance in many problems, such as sampling by particle size where the separation is done by inertial means, consideration of impaction on a surface, or even deposition of the material in the respiratory tract. Rough conversion factors between spherical particles and particles of known and definable shapes can be found in the literature but are not considered here because of the preliminary nature of many of the data discussed herein and the resulting uncertainties from these causes.

II. TURBULENT TRANSFER

For the turbulent transfer to the ground, theoretical treatments have been published by Stewart,[8] Owen,[9] and Chamberlain.[10] Fuquay in unpublished work[11] has considered the transfer of mass across the boundary layer of the atmosphere to be equal to the transfer of momentum and has evaluated the transfer coefficient or velocity to be:

$$V_t = \frac{(u^*)^2}{u_z} \qquad \text{(B-3)}$$

Here, the transfer velocity is designated as V_t to indicate that it is the component due to the turbulent transfer, u^* is the friction velocity and u_z is the wind velocity at a reference height z. The friction velocity is the ratio of the shearing stress in the lower layers of the atmosphere to the density, with the shearing stress considered to be constant with height in the surface layers of concern. Chamberlain's expression for the resistance to transfer in the boundary layer[10] is the reciprocal of Eq. (B-3) and he has evaluated the velocity of deposition for submicron particles from a theoretical treatment by Owen to be $0.004u^*$. Markee[12] indicates that the deposition velocities for iodine with a one-meter reference height at the National Reactor Testing Station have shown an approximate linear relation with u^* where $V_d = 0.0121u^*$. Since, as will be seen, the friction velocity for a given atmospheric condition and surface is proportional to the wind velocity at a reference height, Eq. (B-3) reduces to the same form as these observations.

The value of u^* can be evaluated from the wind profile (change in wind velocity with height) and a parameter representing the nature of the surface. For a surface in which the irregularities are large enough so that a laminar layer submerging the irregularities cannot form the flow will be turbulent down to the surface.[13] Such a condition is called fully-rough flow and occurs for nearly all natural surfaces at moderate or high wind speeds. Sutton,[13] for example, indicates that for a wind speed of 5 m/sec at a height of two meters, only a surface such as smooth mud flats or a large sheet of ice would be aerodynamically smooth. A closely cut and well rolled lawn would be smooth for winds below 1 m/sec measured at two meters height but would be rough at higher wind speeds. Note that the wind must flow over a surface for some distance before the surface layer takes on the turbulence characteristics of that surface. This means that significant changes in the character of the turbulent layer occur with changes in terrain, with possible significant changes in the turbulence transfer velocity. Where artifical surfaces, such as paper of limited area, are used to sample deposition, it is probable that the transfer velocity is characteristic of the terrain immediately surrounding the sampler with the retention characteristics those of the sampling medium so that results from differing terrains or samplers may not compare.

The wind profile in a neutral atmosphere and its relation to the friction velocity has been studied more extensively and is better characterized than for stable or unstable atmospheres. For a neutral atmosphere, the wind profile is logarithmic and in

fully rough flow can be described[14] as:

$$\frac{\bar{u}_z}{u^*} = \frac{1}{k} \ln \frac{z}{z_o} \qquad \text{(B-4)}$$

In Eq. (B-4), k is the Van Karmen constant with a value of about 0.4, $\bar{u}_z$ is the wind speed at a height z and z_o is a constant characteristic of the surface. This constant arises as a constant of integration in the derivation of Eq. (B-4) and represents the height at which the flow can be extrapolated to zero. It can be measured for a given surface from the wind profile in a neutral atmosphere and is reasonably independent of wind speed, although in situations where the surface changes with wind speed, as in the development of waves on a body of water or the bending of tall grasses in the wind, the value of z_o can either increase or decrease with wind speed. Deacon[15] has given typical values for various surfaces along with an estimate of the wind speeds above which fully rough flow can be expected so that the treatment of Eq. (B-4) is applicable. His plot is reproduced in Fig. 1. It may be noted that this treatment is not strictly applicable to surfaces with higher roughness features such as a forest.

With stable or unstable conditions, the logarithmic wind profile no longer holds. For these conditions, Deacon[15] indicates that the change in wind velocity with height (du/dz) provides a reasonable fit to a power function. From this, he derives

$$\frac{\bar{u}_z}{u^*} = \frac{1}{k(1-\beta)} \left[\left(\frac{z}{z_o}\right)^{1-\beta} - 1 \right] \qquad \text{(B-5)}$$

The symbols in Eq. (B-5) are the same as those in Eq. (B-4) with the addition of β which is the exponent in the derivative of the wind profile. Beta is greater than one for an unstable atmosphere, less than one for a stable atmosphere and one for a neutral atmosphere. It is assumed that z_o is characteristic of the terrain and is the same in all stabilities so that a measure of this constant under neutral conditions will permit application to Eq. (B-5). The validity of this assumption has not been definitely shown and, particularly in very stable atmospheres, the criterion for fully rough flow may not be met and the profile may differ from the power function.

By combining Eq. (B-3) with either Eq. (B-4) or Eq. (B-5) we can derive a functional relationship between V_t and these parameters.

$$\text{Neutral} \quad V_t = \left[\frac{k}{\ln\left(\frac{z}{z_o}\right)} \right]^2 \bar{u}_z \qquad \text{(B-6)}$$

$$\text{Other} \quad V_t = \left[\frac{k(1-\beta)}{\left(\frac{z}{z_o}\right)^{1-\beta} - 1} \right]^2 \bar{u}_z \qquad \text{(B-7)}$$

The above derivation is not intended to be complete for all surfaces and some corrections have been omitted in the interest of simplicity. It is intended to indicate the functional form of this transfer with the meteorological variables under most conditions of interest in the field. If we accept the assumption that the transfer of mass is equal to the transfer of momentum in this situation then the transfer velocity will be directly proportional to the wind speed and should vary with the stability of the atmosphere, being greater for the unstable condition and smaller for the stable condition. This conclusion is of some importance since it indicates that the amount of material deposited from the atmosphere is independent of the wind speed for a given stability. This is because the concentration from a point source decreases inversely with the wind speed while the deposition increases directly as the wind speed so that the two terms cancel. It is also of interest that the ratio of the turbulent transfer velocity to the wind speed is equal to the "drag coefficient" as given by Priestley[14] or one half of the drag coefficient as given by Deacon.[15]

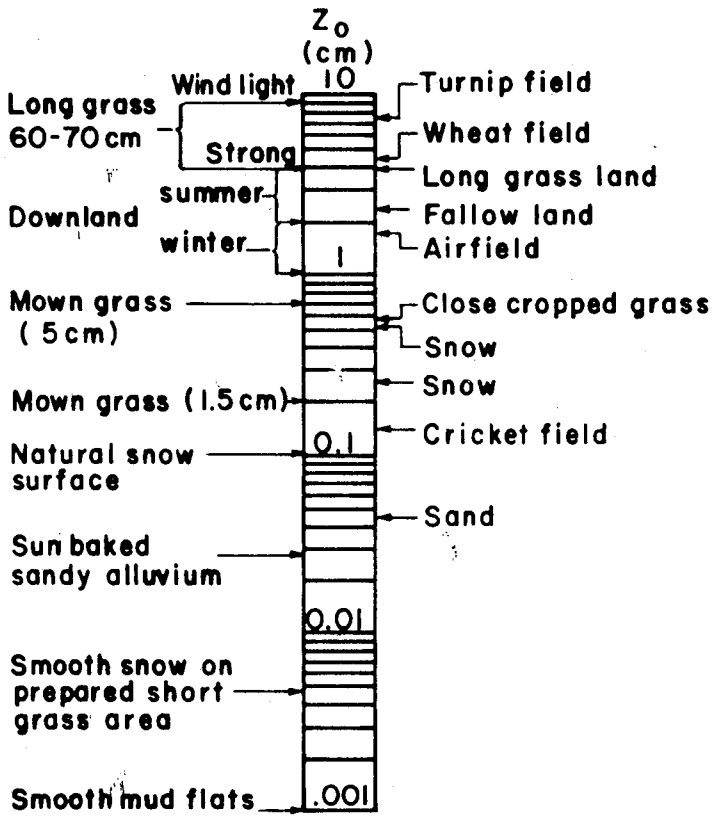

Fig. B-1. The Roughness Parameter of Various Surfaces (After Deacon.)[15]

We can obtain an estimate of the magnitude of the turbulent transfer velocity for various surfaces and the change with stability of the atmosphere by using values of z_o and β as given in the literature. The values of z_o chosen were those given by Sutton[13] as representative but to be used as general guides only. They may be compared with those of Deacon as given in Fig. B-1. Deacon[15] has plotted values of β as a function of stability (expressed as the Richardson's number) from measurements made over a short grass surface (z_o = 0.27 cm) and from observations over snow (z_o = 0.25 cm). We will assume, for purposes of illustration, that the value of β is independent of z_o and use these data to estimate the ratio of V_t to $\bar{u}$ as measured at a height of two meters ($\bar{u}_2$). (This is equivalent to calculating the transfer velocity for a wind speed of one meter per second at the reference height.) These values, along with the values of z_o are given in Table B-II.

It may be noted that the nature of the surface is more important in determining this transfer in the stable case than in the unstable. Thus, for high values of z_o, both the stable and unstable case are within a factor of two of the neutral case while for the low values of z_o the stable case transfer is lower by about a factor of twenty with the unstable case transfer higher by only a factor of six.

The variation in the turbulent transfer velocity is shown as a function of the stability expressed as the Richardson's number in Fig. B-2 for several of the values of z_o from Table B-II. The values of β for this plot were again taken from Deacon's plot and the Richardson's number is that for the layer of air between 0.5 and 4 meters.

TABLE B-II

CALCULATED VALUES OF $V_t/\bar{u}_2$

Surface	z_o (cm)	Stable $R_i = 0.08$ $\beta^i = 0.79$	Neutral $R_i = 0$ $\beta^i = 1$	Unstable $R_i = -0.2$ $\beta^i = 0.18$
Very smooth	0.001	0.000049	0.0011	0.0066
Grass up to 1 cm	0.1	0.00046	0.0028	0.0093
Thin grass up to 10 cm	0.7	0.0014	0.0050	0.013
Thick grass up to 10 cm	2.3	0.0029	0.0080	0.017
Thin grass up to 50 cm	5	0.0052	0.012	0.022
Thick grass up to 50 cm	9	0.0084	0.017	0.028

III. EXPERIMENTAL DATA

Data taken on an adequately controlled basis to permit checking of these concepts are scarce. In many cases the particle size or physical nature of the contaminant is not known while in others the wind speed or other meteorological variables are not given. Perhaps, the most common is the use of an isolated small area of a collection material either at ground level or at some arbitrary distance above the ground. The meaning of these results in terms of the local deposition is not known since the area is usually not large enough to establish the full turbulent layer over the test surface. For such surfaces on the ground, the final result is probably a mixture of the characteristic ground surface in the area and the retention characteristics of the test specimen.

A compilation of some of the data available on deposition are given in Table B-III separated according to stability. A brief discussion of the data identified by the letter in the source column of the table is given below.

A. These results come from experiments by Chamberlain and Chadwick and Megaw and Chadwick.[10] Elemental iodine was dissolved in CCl_4 and sprayed into the air. Measurements of the air concentration at several heights and of the deposited material were made across arcs at several distances downwind. The reference wind speed given in the table is at a height of two meters. It was assumed for the purposes of the tabulation that the measurements on a sunny day were in unstable conditions while those on a cloudy day or at dusk were in neutral conditions. Data are also given for the friction velocity. From this, the estimated turbulence transfer was calculated for a wind speed at the reference height of two

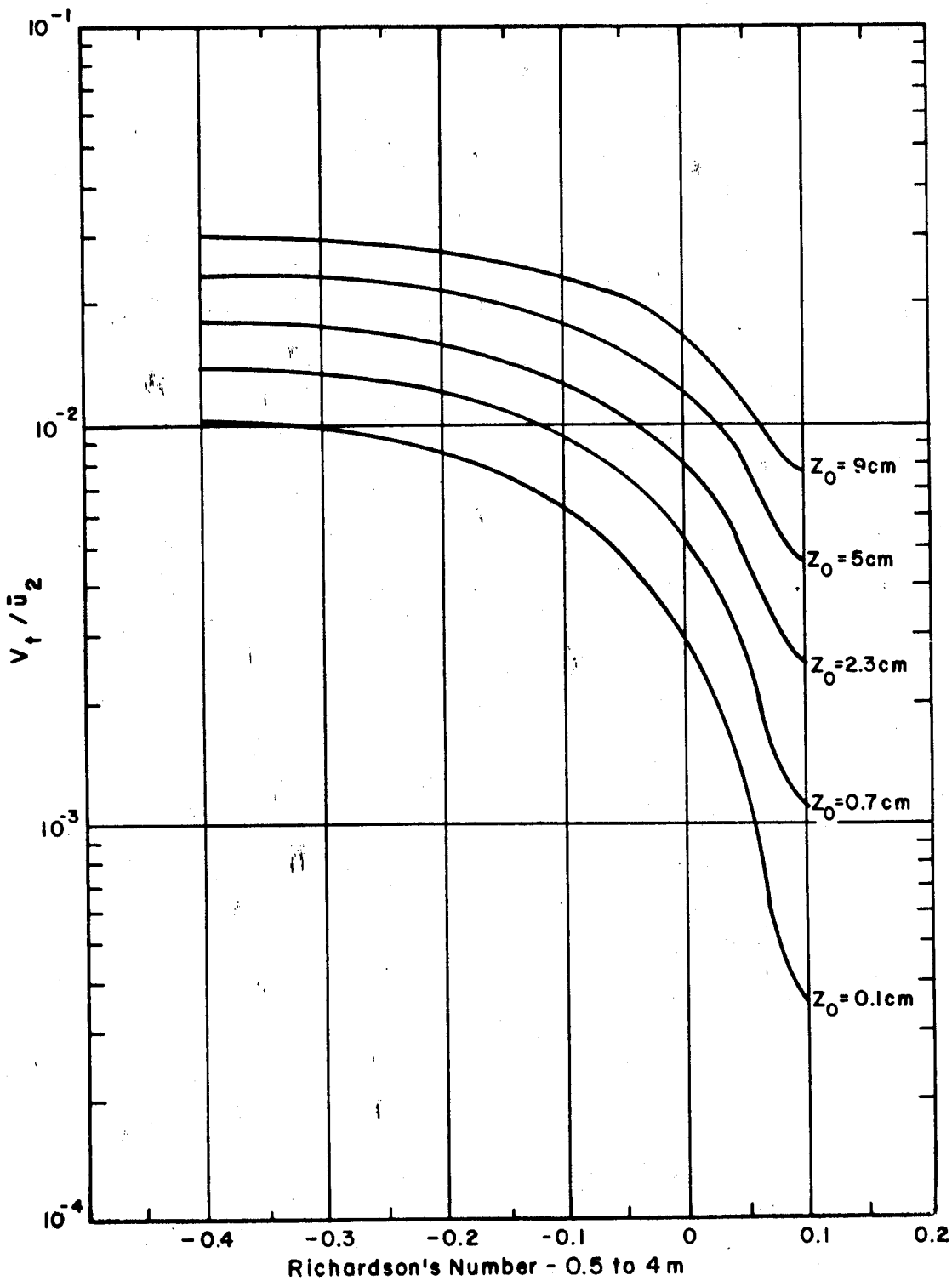

Fig. B-2. Change in Turbulent Transfer with Stability.

TABLE B-III

MEASUREMENTS OF DEPOSITION VELOCITY

Material	Source	Surface	$(V_d/\bar{u}) \times 10^2$
Stable Atmosphere			
ZnS tracer ~1 μm MMD	C	Desert	0.077, 0.88
ZnS tracer	G	Desert	0.85, 0.55, 0.78
Fission Products from Melted Fuel Element			
^{131}I	E	Sticky paper - 1 m	0.38
		Water	0.36
		Sand	0.12
^{137}Cs		Sticky paper - 1 m	0.018, 0.043, 0.029
^{103}Ru		Sticky paper - 1 m	0.64
		Water	0.86
		Sand	0.24
Zr-Nb		Sticky paper - 1 m	0.44
		Water	0.86
		Sand	0.98
Ce		Sticky paper - 1 m	0.14
^{131}I	F	Sticky paper - grd.	0.054, 0.10, 0.059
			0.14, 0.16, 0.13, 0.22
		Sticky paper - 1 m	0.094, 0.052, 0.17,
			0.16, 0.32
		Rye grass	0.42, 0.81
^{137}Cs		Soil	0.0091
^{103}Ru		Sticky paper - grd.	0.046, 0.14, 0.13,
			0.20, 0.078, 0.078
		Sticky paper - 1 m	0.11, 0.22, 0.02, 0.15
		Grass	0.12, 0.31
		Soil	0.029
Te		Sticky paper - grd.	0.15
Unstable Atmosphere			
^{131}I vapor	A	Grass	0.37, 0.35, 0.91, 0.76
		Dandelion leaf	0.25, 0.30
		Paper leaf	0.39, 0.37
		Paper - Petri dish	0.12, 0.17, 0.15
ZnS tracer ~1 μm MMD	C	Desert	1.33, 0.94, 0.87
			1.1, 1.1
Fission Products Arc	B	Grass & substrate	0.069, 0.049
		Filter paper	0.024, 0.018, 0.012
Neutral Atmosphere			
^{131}I vapor	A	Grass	0.60, 1.03, 0.28
		Dandelion leaf	0.30, 0.078
		Paper leaf	0.61, 0.16
		Paper - Petri dish	0.16, 0.18
^{131}I - melted fuel element	E	Sticky paper - 1m	0.16, 0.12
		Water	0.26, 0.34
	F	Sticky paper - 1m	0.13
		Sticky paper - grd.	0.16
		Rye grass	0.81

TABLE B-III (Continued)

Material	Source	Surface	$(V_d/\bar{u}) \times 10^2$
Rn daughters	D	Flat surface	0.01 - 0.02
ZnS tracer ~1 μm MMD	C	Desert	0.69, 0.47, 0.62, 0.56
Fission Products Arc	B	Grass & substrate Filter paper	0.023 0.023, 0.042
Fission Products from Melted Fuel Element			
^{137}Cs	E	Sticky paper - 1 m Water Sand	0.009, 0.017, 0.027, 0.055 0.01, 0.029 0.012, 0.009, 0.055
^{103}Ru		Sticky paper - 1 m Water Sand	0.20 0.25, 0.30 0.055, 0.063
Zr-Nb		Sticky paper - 1 m	0.12 0.71 0.18
Ce	F	Sticky paper - grd.	0.22
Te		Sticky paper - grd.	0.20

meters from Eq. (B-3). These values are compared to the measured deposition below.

Run No.	Calculated V_t/u	Measured V_d/u
1	0.0085	0.0037
2	0.0065	0.0060
3	0.0087	0.0035
4	0.0087	0.0091
5	0.0083	0.0103
6	0.0076	0.0074
7	0.0045	0.0028

B. Megaw and Chadwick[5] produced a fume of fission products by an arc between an irradiated wire and an electrode. Deposition was measured downwind along with the air concentration. Particles were probably submicron in size. Chamberlain[10] reports that cascade impactor samplers would indicate a particle size of 0.2 μm or less if the density of the particles was that of uranium oxide. It was noted that the deposition velocity of strontium from this experiment seemed to be less than that of the other solid fission products.

C. Islitzer and Dumbauld, as reported in Ref. 16, computed the deposition velocity for fluorescent tracer particles of one μm MMD from tracer material balance measurement at the National Reactor Test Station in Idaho over level terrain sparsely covered with sagebrush. They noted, in particular, a marked variation in the deposition velocity with stability. Measurements of the deposition velocity on flat, sand covered plates 0.1 m^2 in area were also made. In unstable conditions, these measurements indicated deposition velocities over an order of magnitude smaller than those found by the depletion technique.

D. Chamberlain[10] quotes Bullas as measuring the deposition of radon decay products onto flat surfaces. It was estimated that over 95% of the decay products would be attached to nuclei which Wilkening found to be about 0.02 μm median diameter. Bullas found the deposition velocity to depend on the wind velocity. For purposes of Table B-II it was assumed that the deposition quoted with "fresh" winds occurred in neutral atmospheres with wind speeds of about 5 meters per second. It is noted that in "calm" weather values of the deposition velocity were as low as 0.005 to 0.01 cm/sec. Measurements were also made by Bullas of the deposition

of fission products in long range fallout with deposition velocities ranging from 0.063 to 0.16 cm/sec. Similar observations by Stewart quoted in Ref. 10 gave a mean velocity of 0.07 cm/sec for the fission products. Booker is reported to have measured the ^{95}Zr component of long range fallout on filter paper with an average deposition velocity of 0.1 cm/sec. When he repeated the experiment indoors, the deposition velocity was 0.007 cm/sec although the atmospheric concentration was 80% of that outdoors. These values are not included in the table since there is no indication of the wind speed or stability.

E. A field experiment at the National Reactor Testing Station in Idaho was conducted for the Aircraft Nuclear Propulsion program.[17] Irradiated uranium fuel elements were melted in a furnace at ground level and the resulting fission products were measured downwind to a distance of about 3.2 km. Andersen samplers indicated that the bulk of the material penetrated to the backup filter. Particle size estimates for zirconium-niobium were 1 to 5 μm and for cerium 50% from 1 to 5 μm and 50% less than one μm. All others were estimated to be less than one μm. The majority of the deposition measurements were made on 13" x 13" sticky paper mounted on a support an unspecified distance (presumably about 1 meter) above the ground. Water trays with an area of 135 in.^{2} and sand trays with an area of 161 in.^{2} were placed at ground level and around sagebrush. The deposition velocity at each measurement arc was computed for the ration of the areas under the deposition profile to the area under the air concentration profile. This technique of fitting a Gaussian curve by area strongly weights the points in the middle of the profile and essentially ignores the points at the tail of the curve. If there were diffusion patterns leading to different deposition rates at the center and the tails of the curves, this technique would provide an estimate primarily of the centerline deposition. It may be noted that the points quoted in this test for unstable conditions are included in the neutral section of Table B-II. This is because the wind profiles for these tests were logarithmic and the Richardson's numbers estimated from the data available in the report were close to zero indicating a reasonably neutral atmosphere.

F. The second series of tests for the ANP project was made at the Dugway Proving Ground in Utah.[18] This is a very flat region with little vegetation or surface roughness to induce turbulence. Techniques were similar to the previous test except that the sticky paper was primarily used on the ground and patches of rye grass 8" x 6" at ground level were used for some of the tests. Most of the data were taken for stable atmospheres although one test had a small temperature differential between 4 and 16 m and was considered as neutral. It was concluded that there was some indication of a change in deposition velocity with wind speed but no change with stability. The data are variable, however, and the range in stability was not great. This report also indicated that the sticky paper used for the bulk of the measurements changes retention efficiency with the humidity of the atmosphere, thereby adding another variable to the measurements.

G. Simpson[19] has reported detailed measurements of the plume depletion and horizontal and vertical profiles of concentration following the ground level release of a zinc sulfide tracer (MMD ~2.7 μm) at Hanford. The deposition velocities were calculated from the estimated values of the exchange coefficient and the vertical concentration gradient. These runs were made under very stable conditions with Richardson's numbers at 1.5 to 3 m ranging from 0.046 to 0.223. He also reports values for the friction velocity which lead to estimates of the ratio of turbulent transfer velocity to wind velocity from 4.9 to 22 cm/sec per meter/sec.

The data from Tables B-II and B-III are compared for the neutral atmosphere in Fig. B-3. The terminal velocities for spherical particles with a density of four are included for comparison. Note that particles with a different shape or irregular

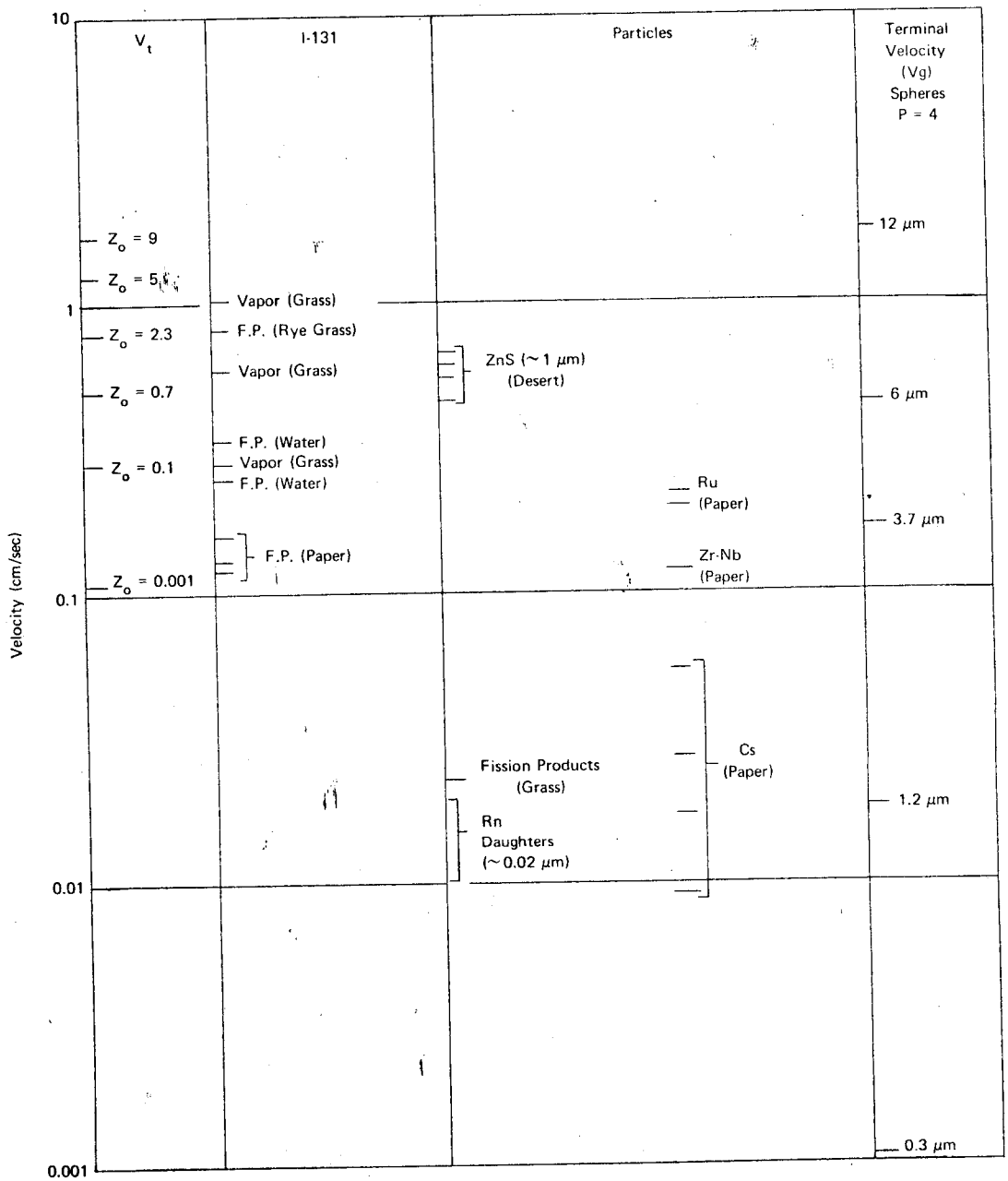

Fig. B-3. Transfer Velocities - Neutral Atmosphere.
$\bar{u}$ = 1 m/s

particles will have a lower terminal velocity.

IV. RETENTION ON SURFACES

The turbulent transfer velocity places an upper limit on the movement of the smaller particles through the boundary layer. If the material passing downward is retained on the surface with 100% efficiency, then the overall velocity of deposition must equal the turbulent transfer velocity. If, however, the particle rebounds from the surface or does not contact it because of inertial effects, then the retention can be less than 100% and the overall velocity of deposition will be smaller than the turbulent transfer velocity.

The iodine data over grass indicates that the measured deposition velocity for the vapor is close to that predicted by the turbulent transfer mechanism. The iodine from the fission product release may be somewhat low in its deposition on paper, but the form of the iodine, the retention characteristics of the sticky paper and its elevated position introduce uncertainties. The tracer material with a median diameter of about one μm similarly seems to have a high efficiency of deposition. It is of interest to note that the value of z_o, as obtained from two of the logarithmic wind profiles in the first series of release tests at NRTS, was about 1 cm, a value in close agreement with the deposition velocity measured for the same general area. Also, the measured deposition velocity for this material is much greater than the terminal velocity for gravitational settling indicating the importance of the turbulent transfer.

On the other hand, the data for radon daughters and fission products produced by an electric arc indicate deposition velocities much lower than would be indicated by turbulent transfer but still higher than would be predicted for submicron particles in gravitational settling. A similar pattern is shown for cesium in the ANP tests although the ruthenium and, perhaps, the zirconium-niobium velocities appear to be higher. These data lead to the conclusion that the finer particles, while transferred according to the theory, do not remain on the surface. Thus, the measured deposition velocity is lower than would be predicted. On the other hand, materials such as iodine vapor and μm-sized particles appear to be held with relatively high efficiency particularly on surfaces such as grass.

Possible mechanisms for retention on surfaces are varied and undoubtedly differ with the size and nature of the particle. We can speculate that absorption, adsorption, electrostatic effects, inertial effects and others may all be of importance under given conditions. In order to investigate the possible effects of one of these mechanisms, the inertial forces, a crude model was established and impaction efficiency estimated under several conditions. It is emphasized that these calculations are intentionally naive and are not intended to represent reality, but simply to illustrate one of the possible mechanisms.

Studies of the efficiency of impaction of small particles carried by an air stream have been made on a theoretical basis by Langmuir[7] and on an experimental basis by several investigators.[20,22] Figure B-4 presents the efficiency of impaction on a cylinder predicted by Langmuir with the results of several experiments for comparison. This efficiency is defined as the ratio of the quantity of material collected on the unit projected area of a cylinder to the quantity passing through the unit area normal to the direction of the flow. In Fig. B-4 this efficiency is correlated to the dimensionless parameter $V_g u/gC$ where V_g is the terminal velocity of the particle in free fall through the atmosphere, u is the velocity of the air stream, g is the acceleration due to gravity and C is the diameter of the cylinder upon which the particles are impacting.

It is noted that the experimental data are in reasonable agreement with the Langmuir prediction except for those points below the cutoff value of the parameter. The experimental values were obtained

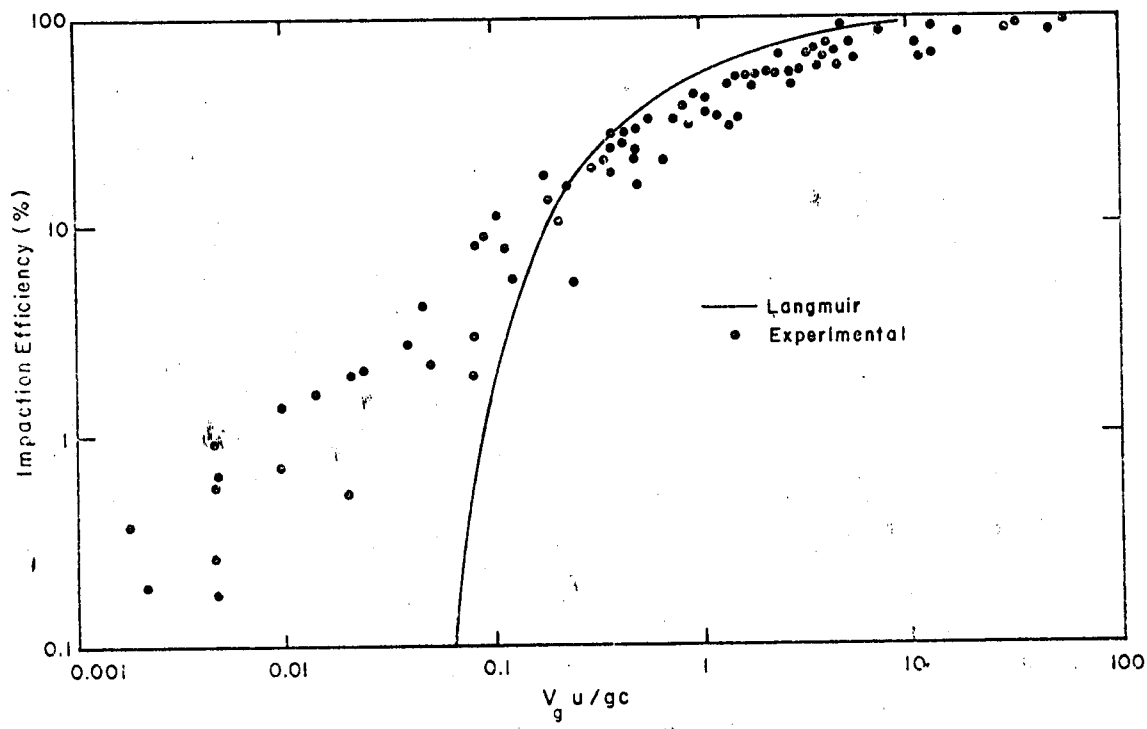

Fig. B-4. Efficiency of Impaction of Particles on Cylinders.

with aerosols of finite size distribution which means that there were particles much larger than the mean values used, and the measured collection at these small sizes was undoubtedly due to the presence of the larger particles. For the further calculations, the theoretical predictions of Langmuir will be used so that the values obtained will be characteristic of a uniform sized aerosol.

For these calculations, particles of density four were chosen since this corresponds to the zinc sulfide fluorescent tracer commonly used in meteorological experimentation. Figure B-5 presents the impaction efficiency for various values of u/C and particle sizes for spherical particles of density four. From these curves the impaction efficiency for various airstream speeds of diameters of impaction cylinders can be obtained. For example, for a one millimeter diameter cylinder, the curve of u/C = 10^4 gives the efficiencies for a ten meter/second airstream, while the curve of u/C = 10^3 gives the efficiencies for a one meter/second airstream. Conversely, for a ten meter/second airstream the curve of u/C = 10^4 represents the efficiency of impaction on a one mm cylinder while the curve of u/C = 10^3 represents the efficiency of impaction on a ten mm cylinder.

In order to illustrate the effects of this aerodynamic behavior of particles, a simple calculational model was used in which the particles are brought to the ground by atmospheric turbulence. Upon reaching the ground, they are carried past obstructions in the form of cylinders of several diameters where they are impacted according to the efficiencies given. Only the mechanism of impaction on cylinders was considered at this point so that any additional mechanisms such as electrostatic

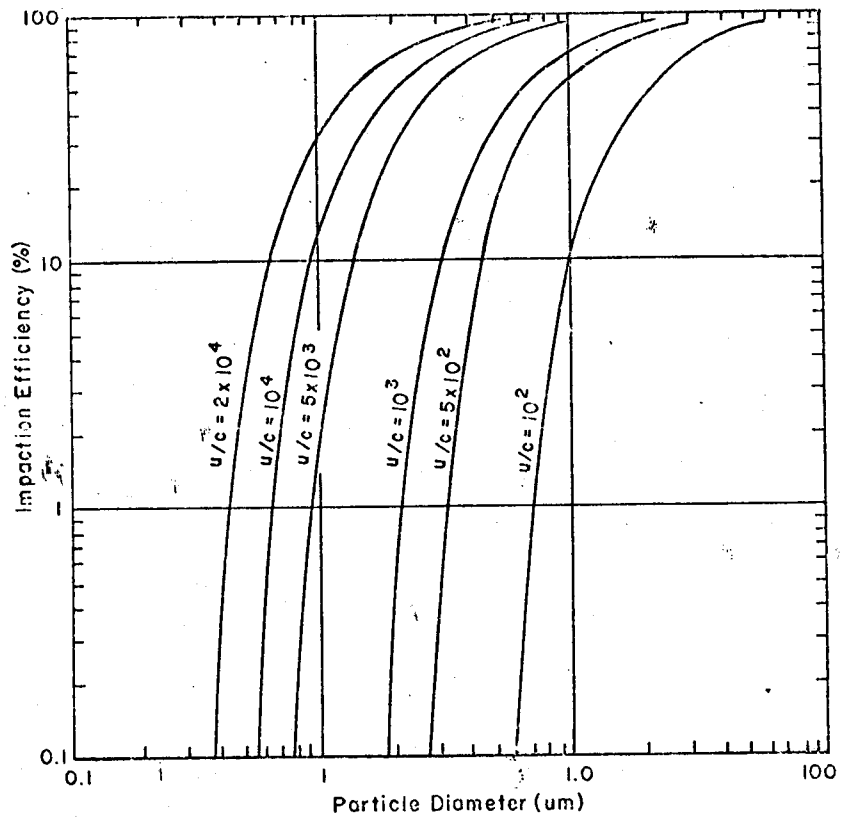

Fig. B-5. Impaction Efficiencies for Spheres of Density 4 g/cm^3

effects; impaction on surfaces other than cylinders; or, for the very small particles, diffusion to the interceptor surface will change the picture given.

It should be noted that two wind speeds are of importance in these calculations of the deposition velocity. The first is the wind speed at the boundary layer which determines the rate of mixing through the boundary layer or the limiting value of the deposition velocity. Thus, from the measurements available, the mixing phenomena can account for a deposition velocity of about 2.5 cm/sec in neutral conditions with a wind speed of 5 meters/second. At one meter/second the deposition velocity due to mixing should be about 0.5 cm/second. The other wind speed of importance is the speed of the airstream past the impacting surfaces. This is undoubtedly lower than the wind speed at the boundary layer due to surface friction effects and will probably vary with the nature of the surface (short grass, long grass, bushes, trees, etc.). In the present calculations, the speed of the airstream at the interceptor is taken to be the same as the wind speed since detailed information to choose a better value is not available.

The definition of the impaction efficiencies for Fig. B-4 must be considered in the model since they represent a fraction of the particles that are in the streamline intercepting the cylinder so that only those particles so exposed are represented. In other words, no correction is made for the relative areas of the interceptor surfaces and the total flow area. Two situations are calculated. The first uses the efficiencies directly from Fig. B-5. This assumes that the intercepting cylinders are so placed that each particle is on a streamline headed toward an interceptor once during the passage. The second

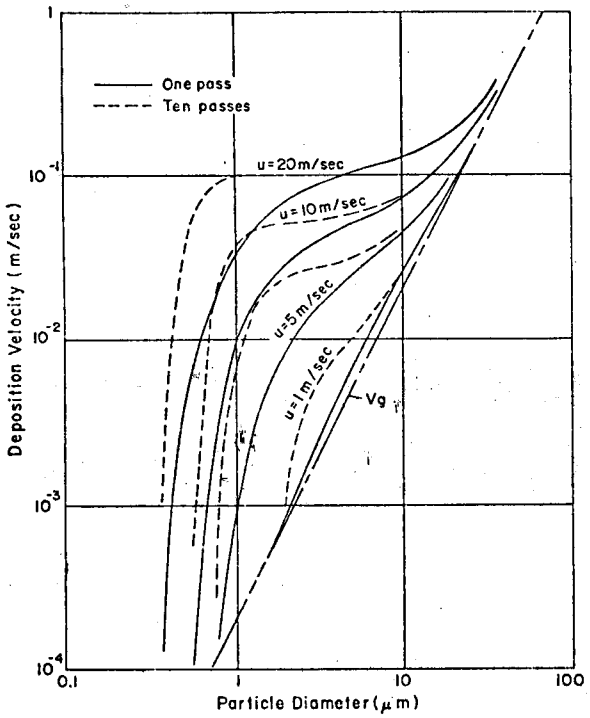

Fig. B-6. Calculated Deposition Velocity for Spheres of Density 4 Impinging on 1 mm Diameter Cylinders in a Neutral Atmosphere.

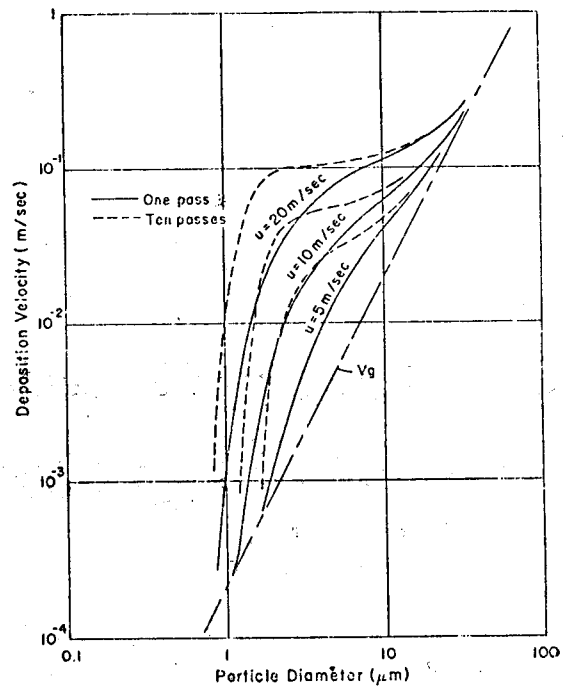

Fig. B-7. Calculated Deposition Velocity for Spheres of Density 4 Impinging on 5 mm Diameter Cylinders in a Neutral Atmosphere.

assumes a passage through a number of interceptors so that each particle is exposed to the chance of capture ten times.

The values of the deposition velocity in this idealized situation were computed from Eq. (B-8).

$$V_d = V_g + f\left(\frac{V_t}{u}\right)u \qquad \text{(B-8)}$$

Where V_d is the velocity of deposition, V_g is the terminal settling velocity of the particle, f is the efficiency of impaction, (V_t/u) is the limiting velocity of deposition from mixing across the boundary layer and u is the wind speed. Equation (B-8) is based on a simple additive process between the two mechanisms considered. It is doubtful that this is valid at low particle sizes where the energy which can be imparted to the particle by eddies in the atmosphere is much greater than the energy which can be dissipated in an equivalent time period by the simple settling of the particle.

In the case of the "multiple pass" over the interceptors, f was evaluated by:

$$f = 1 - e^{-f'n} \qquad \text{(B-9)}$$

Where f is the fraction remaining after n passes each with an efficiency of f' in removing material.

Figure B-6 presents the velocity of deposition calculated for both the "single-pass" and the "ten-pass" cases for impaction on one millimeter cylinders and wind speeds of 20 m/sec, 10 m/sec, 5 m/sec and 1 m/sec. It was assumed that the atmosphere had neutral stability so that (V_t/u) was 5×10^{-3}.

Figure B-7 presents the same calculations for impaction on five mm diameter cylinders.

V. DEPOSITION VELOCITY MODEL

From these considerations we can obtain a qualitative picture of the variation in deposition velocity with particle size. With large particles, the predominant mechanism will be gravitational settling. As the particles become smaller, they will become more likely to remain suspended by the turbulent air motions and, at some size, the velocity of settling becomes small in comparison to the turbulent transfer across the boundary layer. This transition size is a function of the stability of the atmosphere in determining the turbulence, the wind velocity, and the nature of the surface. From Fig. B-3 the transition size for a spherical particle with a density of 4 in a neutral atmosphere with a grass surface and a wind speed of 1 m/sec, would be about 6 to 10 μm. For shapes other than spherical, the transition size would be larger. Since the gravitational settling is not affected by the wind speed while the turbulent transfer velocity changes proportionately, the transition point occurs at larger particle sizes at higher wind speeds. In the previous example, but at a wind speed of 10 m/sec, the transition point would come at a particle size of 20 to 30 μm. Once the particles are brought to the ground, the probability of retention on the surface is undoubtedly a function of the particle size and the wind speed, although definitive data are not available on this. Variations in the retention with wind speed could account for some of the variability in the measured deposition velocity. One would expect that the larger particles would have greater retention by inertial impaction which would appear to be the primary force responsible for these particles. As is indicated by the data in Table B-III, the retention of zinc sulfide particles of one or a few μm appears to be high. Thus, the possibility of a plateau in the curve of measured deposition velocity versus particle size at the value of the turbulent transfer velocity seems probable. The length of this plateau and the particle size at which a significant decrease in retention occurs will probably depend upon the stability and the wind speed, but would seem to extend down to one micron or slightly less. Below this value the retention will decrease, although not as rapidly as the consideration of inertial forces would indicate, since new mechanisms, such as electrostatic attraction, will come into play for the very small particles.

Selection of numbers for this qualitative picture is difficult and uncertain because of the lack of detailed data. Using as a reference the turbulent transfer velocity for z_o = 2.3 cm, one can calculate the efficiency of retention for the measured deposition velocities in Table B-III. For the one μm MMD tracer particles at Idaho Falls, the efficiency of retention is 73% for neutral conditions, 63% for unstable conditions, and 28% for stable conditions. For the radon daughters on a flat plate the efficiency is about 2%. For the fission products produced by the arc and deposited on grass, the efficiency is about 3% in neutral conditions and 3.5% in unstable conditions. The deposition of Cs on the paper, sand, and water in the ANP tests varied from 2.5 to 3.4% in neutral conditions and from 3 to 10% in stable conditions. While the absolute magnitude of these numbers can vary with the assumption of z_o, the values are consistent with a rapid decrease in retention efficiency in the range of 0.5 to 1 or 2 microns with a relatively constant retention efficiency of about 1 to 5% at 0.1 micron. The other values in the ANP tests were not used here because of the indications of large particle size or the possible chemical reactivity of the ruthenium.

The information available obviously does not permit a detailed functional relationship between the particle size and efficiency of retention particularly when differences due to changes in stability, wind speed, and nature of the surface are included. For purposes of estimation, we will assume that the fission products produced by the electric arc in the experiments of Megaw and Chadwick[11] are about 0.1 μm with a retention of about 3% and the one μm tracer particles of Islitzer[22] have a retention of

about 70% with a linear relation between. Below 0.1 μm the efficiency is assumed to remain about 3% as based on the radon daughter deposition on the flat plate. The linear relationship was chosen as the simplest to represent the meager data, although it is probable that the actual relation is sigmoid with the steepest drop in retention somewhere between 0.1 and 1 μm. The linear relation can be approximately represented by $f = 0.74d - 0.04$ where f is the fractional retention and d is the particle size in μm. Extrapolation to 100% retention would indicate this to occur with particle sizes of about 1.4 μm, which is not in disagreement with the high retention implied by Simpson's data with 2 to 3 μm particles in stable atmospheres. At the upper end of the spectrum of particle sizes, it is assumed that the deposition velocity remains constant at the value for the turbulent transfer until $(V_t/\bar{u})\ \bar{u} = V_g$.

The particle density has not been included in the above considerations, again because of the lack of data on its influence on retention. Most of the experiments in Table B-III were run with particles of density ranging from about 3 to, perhaps, 10g/cm³ so that the retentions chosen may represent reasonably realistic particles of concern.

From this crude model of retention and the turbulent transfer velocities of Table B-II, it is possible to approximate the deposition velocities for various particle sizes and limited types of terrain. Some of these approximations are given in Table B-IV.

It is again emphasized that the fractional retention values are particularly uncertain so that these deposition velocities must have wide limits of uncertainty until appropriate experimental data and study permit better estimates.

TABLE B-IV

APPROXIMATIONS OF THE RATIO OF DEPOSITION VELOCITY TO WIND SPEED AT ONE METER HEIGHT

Particle Size	f	Short Grass $z_o = 0.1$ cm	Thick Grass 10 cm $z_o = 2.3$ cm	Thick Grass 50 cm $z_o = 9$ cm
		Neutral, Ri = 0		
>1.5 μm	1.0	0.0028	0.0080	0.017
~1 μm	0.7	0.0020	0.0056	0.012
~0.5 μm	0.3	0.00074	0.0024	0.0051
<0.1 μm	0.03	0.00007	0.0002	0.0005
		Unstable, Ri = -0.02		
>1.5 μm	1.0	0.0093	0.017	0.028
~1 μm	0.7	0.0065	0.012	0.020
~0.5 μm	0.3	0.0028	0.0051	0.0084
<0.1 μm	0.03	0.0003	0.0005	0.0008
		Stable, Ri = 0.08		
>1.5 μm	1.0	0.00046	0.0029	0.0084
~1 μm	0.7	0.00032	0.0020	0.0059
~0.5 μm	0.3	0.00014	0.0009	0.0025
<0.1 μm	0.03	0.00001	0.00009	0.0003

REFERENCES

1. R. J. Engelmann, "The Calculation of Precipitation Scavenging," USAEC Report BNWL-77 (Battelle Northwest Laboratory) July 1965.

2. A. C. Chamberlain, "Aspects of Travel and Deposition of Aerosol and Vapor Clouds," AERE-HP/R 1261. (Harwell) 1953.

3. J. W. Healy, B. V. Anderson, H. V. Clukey, and J. K. Soldat, Proc. 2nd Int. Conf. on Peaceful Uses of Atomic Energy, 18, 309 (1958).

4. A. C. Chamberlain, "Experiments on the Deposition of Iodine-131 Vapor onto Surfaces from an Air Stream," AERE-HP-R 1082 (Harwell) January 3, 1953.

5. W. J. Megaw and R. C. Chadwick, "Some Field Experiments on the Release and Deposition of Fission Products and Thoria," AERE-HP-M-114 (Harwell) December 1956, (Official Use Only).

6. P. Drinker and T. Hatch, "Industrial Dust, Hygienic Significance, Measurement and Control," 2nd Ed., McGraw-Hill Book Company, Inc., New York, 1954.

7. C. E. Lapple, et al, "Fluid and Particle Mechanics," University of Delaware, Newark, Delaware, March 1951.

8. K. Stewart, "The Resuspension of Particulate Materials from Surface Contamination, in Surface Contamination," B. R. Fish, Ed. Pergamon Press, 1967, pp. 151-158.

9. P. R. Owen, "Dust Deposition from a Turbulent Airstream, in Aerodynamic Capture of Particles," E. G. Richardson, Ed. Pergamon Press, New York, 1960, pp. 8-25.

10. A. C. Chamberlain, "Aspects of the Deposition of Radioactive and Other Gases and Particles, in Aerodynamic Capture of Particles," E. G. Richardson, Ed., Pergamon Press, New York, 1960, pp. 63-88.

11. J. J. Fuquay, Private Communications, 1957.

12. E. H. Markee, Jr., "A Parametric Study of Gaseous Plume Depletion by Around Surface Adsorption," Proceedings of the USAEC Meteorological Information Meeting, September 11-14, 1967, C. A. Mawson, Ed. AECL-2787.

13. O. A. Sutton, "Micrometeorology," McGraw-Hill Book Company, Inc., New York, 1953.

14. C. H. B. Priestley, "Turbulent Transfer in the Lower Atmosphere," The University of Chicago Press, 1959.

15. E. L. Deacon, "Vertical Diffusion on the Lowest Layers of the Atmosphere," Quarterly Journal Ray. Meteorol. Soc. 75: 89-103, 1949.

16. F. A. Gifford, Jr., and D. H. Pack, "Surface Deposition of Airborne Material," Nucl. Safety, 3, 4, 26-80, 1962.

17. U. S. Air Force Nuclear Research Facility, "Fission Products Field Release Test I," Doc. NARF-59-321, September 1959.

18. U. S. Air Force Nuclear Research Facility, "Fission Products Field Release Test II," Doc. NARF-60-10T, September 1960.

19. C. L. Simpson, "Some Measurements of the Deposition of Matter and its Relation to Diffusion from a Continuous Point Source in a Stable Atmosphere," USAEC Document HW-69292 Rev., April 1961.

20. H. D. Landahl and R. G. Herrman, J. Colloid Sc., 4: 103, 1949.

21. W. E. Ranz and J. B. Wong, Industrial and Engineering Chemistry, 44, 6, 1371-1381, June 1952.

22. G. Asset and D. Pury, Arch. of Industrial Hygiene and Occupational Medicine, 9, 4, 273-283, April 1954.

APPENDIX C

PARTICULATE LUNG DOSE EFFECTS

The following discussion appeared originally in a progress report for this study. It is reproduced here because of its importance to the subject of plutonium standards.

Current standards for limiting lung dose from internal emitters are based upon a calculation of the average dose delivered to the lung by assuming that the radiation absorption is uniform throughout the mass of the tissue. It is known that this condition does not exist for most "insoluble" radioisotopes which provide focal spots of high level radiation close to the particle decreasing with distance in a pattern dependent upon the type and energy of the radiation. Thus, a one μCi ^{32}P particle which, if the energy were averaged over the 1000 gram lung of the standard man would deliver a dose rate of 0.035 rads/day or a total dose of 0.75 rads, will deliver a dose rate of about 80,000 rads per day or a total dose approaching 2,000,000 rads to the tissue at 100 μm distance. Richmond, et al., report an alpha particle dose rate of 10 rads/hour at the surface of a 180 μm ^{238}Pu particle. The photon dose rate at the surface is about 500 rads/hour. Dose and dose rates drop off rapidly with distance from the particle so that the total volume of tissue involved is small. Table I illustrates the same point for the maximum permissible lung burden of plutonium if this lung burden is divided into uniform particles of various sizes.

From these illustrations, the physical conditions of such irradiation are vastly different from the uniform distribution and the particulate exposure results in a relatively small number of cells irradiated to widely differing doses. While one would expect differences in the outcome of irradiation of an organ by these two modes, it is not clear on a priori basis which would be the most damaging. For acute effects occurring after high levels, limiting the volume of tissue can greatly ameliorate the outcome. However, data are not available to indicate whether a similar situation exists for the late effects.

It is clear that this problem is a subclass of a more general problem in arriving at radiation protection standards--

TABLE I

RELATION BETWEEN PARTICLE DIAMETER, PARTICLE NUMBER, DISINTEGRATION RATE AND NUMBER OF CELLS IRRADIATED FOR A LUNG BURDEN OF 0.016 μCi $^{239}PuO_2$

Diameter (μm)	Number of Particles	Disintegration Rate ($d \cdot week^{-1} \cdot particle^{-1}$)	Number of Cells Irradiated[a]
0.01	5.4×10^{10}	6.7×10^{-3}	1.4×10^{13}
0.1	5.4×10^{7}	6.7	1.4×10^{10}
1.0	5.4×10^{4}	6.7×10^{3}	1.5×10^{7}

[a] For each particle size the number of cells exposed within a 40 μm alpha particle range is estimated. The total number of cells irradiated becomes the product of the number of cells irradiated per particle and the number of particles. A cell volume of 10^3 μm^3 is assumed.

that of nonhomogeneous dose in any organ. In order to focus more clearly on the important question, we have considered the current limits for uniform radiation to be acceptable and have, then, asked whether there is any evidence which indicates that the nonuniform radiation to an organ, such as occurs in extreme form in the particle problem, is more or less damaging than the homogeneous radiation. Thus, the focus is on the relative, and not the absolute effect. Further, since we assumed that no clear-cut information was available, we went to the literature to see if even a tentative conclusion could be made as to whether the preponderance of the evidence indicated which assumption should be made.

A. Review of the Literature - J. Furchner

A large number of papers and reports on radiation dose to the lung and subsequent damage was reviewed. One problem with much of the work reviewed, and particularly that having to do with individual implanted sources, was the lack of consistency in the dosimetry. Thus, some groups expressed the dose as the average to the lung while others calculated the dose at some reference distance from the source. While the original intent of this study was to attempt recalculation of the doses on a common basis, this was made difficult in some cases by the lack of data in the published article, the uncertainty of location of the source and the lack of time to complete a job of this magnitude, particularly when the initial appraisal indicated that the results would be uncertain.

For each paper of interest, or potential interest, to the present study a brief abstract emphasizing the actual data presented was prepared. Although such abstracts are of primary usefulness to those who are familar with the original article, they are presented below as orientation to the data available. In each case, comments by the abstractor are presented in parentheses.

1. IMPLANTED SOURCES

^{60}Co Warren and Gates 1960[2]

Mice 4-6 weeks old. ^{60}Co wire implanted through chest wall by trochar. Wire dimension: 2 mm by 0.5 mm. Radioactivity 170-250 μCi. Among 190 mice that survived 97 days (time of appearance of first lung cancer) 20 had carcinoma of the lung or bronchus. The last treated mouse died 315 days after implantation. The doses ranged from 90,000 to 460,000 rep. (Trauma to the lung is unavoidable; compare with Richmond et al. 1970).[8]

^{60}Co Warren and Gates 1968[3]

Mice, rats, hamsters, guinea pigs and rabbits. ^{60}Co wire, 2 mm long by 0.5 mm were implanted by trochar through the chest wall. The activity ranged from 70-636 μCi. Most sources were between 150-200 μCi.

Treatment Group (Species)	Number of Animals	Median Duration of Exposure (days)		Mean Total Dose (R x 10^3)		Malignant Cancer Incidence (%)	
		Lung	Esophagus[a]	Lung	Esophagus	Lung	Esophagus
Mice	286	180	188	262	145	20	15
Rats	20	204	202	353	121	75	30
Hamsters	25	495	368	443	178	8	28
Guinea Pigs	20	416	363	510	454	25	30
Rabbits	12	427	299	909	250	42	25

[a]Data do not include tumors at other sites or animals with no tumors. (As animal size increases the carcinogenic dose increases.)

^{106}Ru Laskin et al. 1963[4]

Rats. Hollow platinum cylinder plated with ^{106}Ru implanted in bronchus. 5 mm long, 1.2 mm diameter, wall thickness 0.2 mm. Hooks on cylinder maintained position in bronchus after implantation by trochar and tracheotomy.

Treatment Group µCi on implant	Number of Animals	Median Survival Time (Days)	Median Time to Cancer (Days)	Number Surviving 143 days[a]	Number with Lung Cancer[b]
0.008	37	340	430	27	2
0.057	56	310	400	39	6
0.59	57	320	380	40	15
5.0	67	225	325	42	25
13.6	48	190	315	29	20
Pt. control	60	330	---	--	--
Ru Control	22	320	---	--	--

[a]Two rats had cancer before this time.
[b]Squamous cell carcinoma.

The authors rearranged the groups on the basis of calculated doses for those animals surviving 143 days.

Average Dose (Rads)[a]	Number of Animals	Squamous Cell Carcinomas Numbers	%
740	6	0	0
3,400	41	3	7.3[b]
36,000	41	9	21.9
460,000	57	33	57.9
1,600,000	32	21	65.6

[a]Dose calculated at "target" tissue taken as basal layer of the epithelium of the bronchus in which pellet was implanted - 100 µm from pellet surface.

[b] One tumor at 1400 rads. (There is no mention of the incidence of respiratory infections or causes of earlier deaths. The considerable trauma associated with implantation may be a factor).

^{106}Ru, ^{32}P Laskin et al. 1964[5]

Rats. Hollow platinum cylinder plated with ^{106}Ru implanted in bronchus. 5 mm long, 1.2 mm diameter, wall thickness, 0.2 mm. Hooks on cylinder maintained position in bronchus after implantation by trochar and tracheotomy. A single dose level of ^{106}Ru (5 µCi) was given. The animals were autopsied after spontaneous death and serial sacrifice. Phosphorus-32 pellets were also implanted in rats by this technique.

^{106}Ru

Mean Time of Death Sacrificed A	Spontaneous B	Number of Animals A	B	Mean Dose[a] (Rads x 10^5) A	B	Cancer Incidence (%) A	B
122	129	10	9	4.2	3.2	0	0
166	158	13	18	4.3	4.5	15.4	11.1
198	193	10	10	5.2	5.2	30.0	20.0
212	233	10	10	5.2	5.5	60.0	50.0
225	300	16	13	5.4	6.9	81.3	84.6
247	347	10	11	6.3	8.0	90.0	81.8
282	378	9	9	6.5	8.6	88.9	88.9
357	424	8	9	7.7	9.2	100.0	100.0

(No cancers before 158 days. Only 4 animals in 31 had lung cancers after doses of 4 x 10^5 Rads over 160 days. Again no mention of chronic respiratory infection is made).

^{32}P

Treatment Group	Number of Animals	Lung Dose[a] (Rads)	Lung Cancer %
20.0 µCi	18	4×10^5	56
2.0 µCi	15	4×10^4	33
0.2 µCi	15	4×10^3	0

(The ^{32}P dose response agrees with the ^{106}Ru data from Laskin et al. 1963, better than does the ^{106}Ru data in this paper).
[a] See note on Laskin et al. 1963 for meaning of dose calculation.

^{106}Ru Divertie, Titus and Shorter 1967[6]

Rats 150-200 g. Silicone rubber pegs (2.5 mm x 1 mm) impregnated with 50µm ceramic spheres containing ^{106}Ru were inserted into a bronchus via tracheotomy. Twelve control animals had inert pegs inserted into a bronchus. Thirteen of 16 rats receiving radioactive pegs had squamous cell carcinomas. None were found in controls. No doses are given.

ANIMALS WITH SQUAMOUS CELL CARCINOMA

Duration of Exposure (wks)	18	19	20	21	23	25	30	34	34	35	36	36	38
µCi Dose Inserted	15.6	14.0	10.0	13.4	13.4	13.4	11.3	8.1	14.8	12.6	13.4	12.6	14.1
Recovered	11.3	10.1	7.5	10.0	9.6	8.1	7.6	6.0	9.4	7.6	8.1	7.6	8.3

In two of the three experimentals no pegs were recovered. Pneumonitis was usually found distal to the pegs.

^{90}Sr Altmann, Hunstein and Stutz 1961[7]

Rats. Plexiglass capsules containing ^{90}Sr were sewed to the underside of the diaphragm. The activity range was 27-62 µCi with most values between 39 and 48 µCi.

Treatment Group Exposure Time (Months)	Number of Animals	Lung Tumors Number Carcinoma	Sarcoma	Adenoma
0 - 3	28	0	0	0
3 - 6	21	2	0	0
6 - 9	48	8	0	0
9 - 12	36	30	1	0
over 12	31	26	1	1

The last two groups also had nonpulmonary tumors. (There is no clue to dose data given).

$^{238}PuO_2$ Richmond, Langham and Stone 1970[8]

Rats, male, 325 g. Injection of PuO_2 spheres via femoral vein. Spheres 122-200 μm in diameter. The spheres are trapped in the capillary network of the lung. The animals were sacrificed serially and examined histologically. Note this method involves no surgical trauma to the lung.

Time of Sacrifice (Days)	1	3	7	14	21	30	60	90	120	152	180	211
No. of Animals	3	2	2	2	2	6	10	5	9	6	7	6

The surface dose rates were 10^8 and 10^3 rad/hr for the alpha and gamma radiations respectively. A sphere of cellular debris and collagenous tissue surrounded the spheres. A footnote, added in proof, states that animals at 600 days postinjection show histological changes qualitatively similar to those seen at 90, 120, and 211 days. No tumors were found.

^{90}Sr Cember and Watson 1958[9]

Rats, male, 286 g. Glass beads with incorporated ^{90}Sr were implanted with a hypodermic needle through the chest wall. The beads were 320± 110 μm in diameter and contained from 1.09 to 59.3 μCi. Dose data was given only for tumor bearing rats. The mortality data for the inert Sr and saline control groups was much the same as that for the experimentals and was due to injection trauma.

Dose Rate (Rad/day)	Exposure Time (Days)	Total Dose (Rads x 10^4)	Tumor
160	561	9.0	Lymphosacoma
160	487	7.8	Squamous cell carcinoma
220	561	12.0	Squamous cell carcinoma
277	169	4.7	Lymphosarcoma
277	545	15.0	Lymphoma
440	581	26.0	Squamous cell carcinoma
660	333	22.0	Squamous cell carcinoma

The first death occurred at 131 days and the last at 575 days post injection. (4 of 23 rats had squamous cell carcinomas at doses of $\sim 10^5$ Rads).

2. INHALATION OR INTRATRACHEAL INJECTION (Beta Emitters)

^{35}S Cember et al. 1955[10]

Rats, female, 125-200 g. A single intratracheal injection of $Ba^{35}SO_4$ particles (1.45 μm ± 0.40 μm).

Treatment Group	Number of Animals	Dose to Lung (rep)
4.5 μCi	23	58
45 μCi	21	3,200
4,500 μCi	38	24,000
Controls	25	-

Rats were killed serially in a 9 month period. No tumors were found in any group. (The calculated doses were delivered almost entirely during the first month). Chronic and acute inflamation were common.

^{35}S Cember and Watson 1958[11]

Rats, female, 244 g. Intratracheal insufflation of 375 μCi of $Ba^{35}SO_4$ once a week for 10 consecutive weeks. Particle size 1.45 μm ± 0.40 μm. Dose estimated to be between 12,000 and 20,000 Rads.

Treatment Group	Number of Animals	Number Surviving 10 weeks	Number Dead at 500 days	Squamous Cell Carcinoma
Colony control	14	--	2	0
Inert $BaSO_4$	24	17	10	0
3,750 μCi	24	16	24	2

The tumors were found in rats that died at 312 and 319 days.

^{144}Ce Cember et al. 1959[12]

Rats, male, 279 g. $^{144}CeF_3$ by intratracheal injection. Particle size 1.0 μm, Std dev 1.4.

Treatment Group	Number of Animals	Number of Survivors[a]	Days to First Tumor	Lung Dose (Rads)	Number with Squamous Cell Carcinoma
Colony control	20	--	--	0	0
Inert CeF_3	29	--	--	0	0
5 μCi	27	10	178	2,400	1
15 μCi	23	21	48	5,100	1
25 μCi	28	19	93	10,700	7
50 μCi	15	6	83	21,000	4

[a]To observation of first tumor. Severe, acute pneumonia appeared in the two high dose Groups within several days. The first tumor appeared in 48 days. (Mortality of the inert CeF_3 group was not given nor was the duration of the experiment).

^{144}Ce Cember 1963[13]

Rats, male, 283 g. $^{144}CeF_3$ by oral, intratracheal injection. Particle size 1.0 μm std dev 1.4.

Treatment Group	Number of Animals	Days to First Tumor Death	Lung Dose[a] (Rads)	Primary Lung Tumors	Squamous Cell Carcinoma	Undifferentiated Carcinoma	Adeno-Carcinoma	Lymphoma
Inert CeF_3	29	--	--	0	0	0	0	0
0.5 μCi	41	528	600	8	3	3	3	1
1.0 μCi	44	367	1,100	6	2	1	3	0
2.0 μCi	34	620	2,500	4	2	1	1	2
4.0 μCi	42	381	4,400	14	11	1	2	0

[a]Dose at death with first tumor. Earliest tumor at 361 days. (Experiment lasted at least 1,033 days. No mention of chronic pulmonary disease was made).

^{144}Ce Cember and Stemmer 1964[14]

Rats, male, 258 g. $^{144}CeCl_3$ solution given by intratracheal injection of 0.15 ml by mouth.

Treatment Group	Number of Animals	Number of Survivors[a]	Days to First Lung Tumor	Lung Dose (Rads)[b]	Number of Primary Lung Tumors
Inert $CeCl_3$	21	21	--	--	-
10 µCi	68	61	306	14,000	9
15 µCi	55	52	197	19,600	31
30 µCi	58	37	70	25,000	27

[a]Two month survivors
[b]Dose at death with first tumor

(Dosimetry differs somewhat from Cember et al. 1959, where the dose rate for 5 µCi $^{144}CeF_3$ is given as 59.6 rads/day in a 1.5 g lung. Here the dose rate is given as 28 rads/day for 1 µCi/g. Cember considers all the data for ^{144}Ce given in Cember et al.1959,[12] Cember 1963,[13] and Cember and Stemmer 1964[14] in Cember 1964.[15,16]

^{144}Ce Cember 1964[15,16]

Values given here are estimated from Fig. 39, Cember 1946a[15] and from Fig. 5, Cember 1964b.[16]

1964a Dose (Rads)	1964a Tumor Frequency (%)	1964b Dose (Rads)	1964b Tumor Frequency (%)
660	2.2	650	1.5
1,300	4.0	1,200	2.5
2,500	5.6	4,500	5.0
5,500	9.5	14,000	10.0
15,500	12.5	20,000	12.5
26,000	22.2	41,000	22.5
43,500	29.8	49,500	25.0

^{144}Ce Hahn et al. 1973.[17]

Beagles. By inhalation of ^{144}Ce fused in clay particles. ~1.4-2.7 A.M.A.D., g std dev 1.5-2.3. To date 15 of 126 beagles are dead of fibrosis and pneumonitis at 143-410 days; and 5 are dead of pulmonary neoplasia at 750-1,318 days.

Initial Lung Burden µCi/kg	Initial Lung Burden Total µCi	Time To Death (Days)	Dose to Lung At Death (Rads)[a]	Lung Pathology
26	230	765	27,000	Hemangiosarcoma
27	190	1,185	23,000	Hemangiosarcoma
34	330	1,318	36,000	Hemangiosarcoma
35	380	916	34,000	Hemangiosarcoma + bronchiolo-carcinoma
46	470	750	48,000	Hemangiosarcoma + fibrosarcoma
33	320	193		Pneumonitis +fibrosis,no tumors
41	330	185		" " "
51	440	410		" " "
53	440	279		" " "
56	520	273		" " "
65	590	234		" " "
66	470	246		" " "
66	540	257		" " "
68	600	186		" " "
96	740	189		" " "
120	890	171		" " "
180	2,000	182		" " "
190	1,500	173		" " "
190	1,700	181		" " "
210	1,700	143		" " "

[a]Calculated by Hahn et al. (It appears that more than 700 days must elapse before tumors are found).

^{144}Ce Kurshakova and Ivanov 1962[18]

Twenty rabbits 2.5 - 3.0 kg were injected with 25 μCi of ^{144}CeF by piercing the anterior wall of the trachea through the skin. The particle size was 0.025 μm. One rabbit died of bronchopneumonia on the 3rd day. Half of the rabbits died between the 60th and 238th days of sclerosis, bronchiectasis, etc. Tumors were found in 6 of the animals surviving to 238 days. The last tumor was found at 327 days. The doses to the lungs at 238 and 327 days were 51.4 and 68.9 kilorads respectively. There were 5 bronchogenic and alveolar lung cancers and one squamous cell carcinoma of the esophagus.

^{106}Ru Temple et al. 1960[19]

Mice, female. $^{106}RuO_2$ in Tween-80 was injected intratracheally.

Treatment Group	Number of Animals	Days After Administration	Adenomas %	Number of Malignant Tumors	Dose (Rads)
Colony Control	28	403-470	78	0	0
Inert RuO_2	21	335-500	9	0	0
3.0 μCi	23	350	82	1 Bronchiolar carcinoma	9,000
1.93 μCi	11	369-422	90	1 Bronchiolar carcinoma	4,000
0.15 μCi	10	340	--	1 lympho sarcoma	300

(The natural incidence of adenomas is a factor of unknown importance to radiation carcinogenesis).

$^{152-154}Eu$ Berke and Deitch 1970[20]

Rats, female, 180-200 g. Rats were made to inhale aerosols of radio-europium chloride for 7 h/day, 5 days/wk for 6 months. The particles were characterized only as "submicronic". The lung dose varied with time and was as much as 6 x 10^4 rads at 720 days. No animals were free of pulmonary pathology. Severe chronic inflammatory changes and lung abscesses were present in the majority of the animals. There was a complete absence of pulmonary neoplasia.

^{24}Na, ^{32}P, ^{59}Fe, ^{198}Au Kochetkova et al. 1959[21]

Rats. These isotopes were given by intratracheal injection. Particle size unspecified.

Treatment Group	μCi	Number of Animals	Beta Dose (Rads) First Day	Beta Dose (Rads) Total	Metaplasia of Bronchial Epithelium Number	Metaplasia of Bronchial Epithelium Months	Lung Cancer Number	Lung Cancer Months
$^{59}Fe_2O_3$	1-27	52	10-20	500-5000	17	6-9	8	6-9
$Cr^{32}PO_4$	40-100	76	300-700	1300-15000	20	2-12	11	6.5-18
^{198}Au	100-150	30	1400-2100	5400-8000	4	1-3	3	2.5-12

100-200 μCi of $^{24}NaCl$ in single and multiple doses produced no tumors.

^{32}P Kochetkova and Avrunina 1957[22]

Rats - Intratracheal injection of $Cr^{32}PO_4$. No particle size specified.

Treatment Group (µCi injected)	Number of Animals	Mean Lung Burden (µCi)	Lung Dose (Rep) 24 h	Lung Dose (Rep) Total	Life Span (Days)	Pathology
270	10	180	2,900	19,000-46,000	3-32	Metaplasia
100	42	81.5	830	10,000-18,000	19-65	Metaplasia
70	34	54	670	8,000-16,000	15-395	3 Squamous Cell Cancers
40	10	38	350	4,500- 7,400	60-451	3 Squamous Cell Cancers

There were no tumors after single and multiple injections of $^{24}NaCl$ (200-1,900 µCi). Of 25 rats that received 320 µCi of radiogold all died in 2.5 months. Three of these rats had squamous cell cancer. The doses were in 9,000-9,700 rep range.

3. INHALATION OR INTRATRACHEAL INJECTION (Alpha)

^{239}Pu Temple et al. 1960[19]

Mice (BAF) PuO_2 suspended in Tween-80 or Pluronics for injection. Particle size 0.6-0.06µm mean 0.5 µm.

Treatment Group	Number of Animals	Days After Administration	Dose (Rads)	Lung Tumor
Colony Control	28	400	---	22 Adenomas
0.16 µCi	41	100	4,000	1 Bronchiolar carcinoma
0.06 µCi	17	400	2,300	2 Squamous cell carcinomas
0.003 µCi	21	500	115	1 Fibro sarcoma

At 400 days 78% of the colony controls had adenomas. The fibrosarcoma at the 0.003 level was considered non-radiogenic (the use of surface active agents as vehicles for the particles is a factor of unknown importance).

^{210}Po Yuile, et al. 1967[23]

Rats, male, exposed once to an aerosol of ^{210}Po as the chloride, particle size: geometric mean 0.098µm, geometric std dev 1.81.

Treatment Group	Number of Animals	Number of Deaths	Age Range At End (wks)	Lung Dose[a] (Rads)	Primary Lung Tumors	Squamous Cell Carcinomas
NaCl Control	147	88	87-100	0	0	0
0.15 µCi	119	119	106	538	22	17
0.05 µCi	129	98	95-100	202	15	5
0.02 µCi	132	71	89-91	71	4	1

Dose accumulated at 280 days - little increase thereafter. The aerosol was a NaCl solution acidified to a pH of 1. Pulmonary infection was endemic in the colony and an epidemic of acute pneumonia occurred during the second year. The experiment was terminated when the last high-dose animal died at the 96th week.

^{239}Pu Wager et al. 1955[24]

Mice (BAF) female - Intratracheal injection with Tween 80. Particle size 0.05 to 0.6 µm. Of 10 mice that received 0.06 µCi of $^{239}PuO_2$, 3 had squamous cell carcinoma at 1 year post-injection.

^{210}Po Scott and Thomas 1957[25]

Rats. Intratracheal injection of ^{210}Po nitrate solution. Experiment terminated at 15 months when there were 5 survivors.

Treatment Group	Number of Animals	Squamous Cell Carcinomas	Time to Tumor (weeks)
10 μCi/kg	15	0	----
5 μCi/kg	15	2	5, 15

All animals exhibited varying degrees of murine pneumonia.

^{210}Po Little et al. 1970[26]

Syrian golden hamsters. ^{210}Po adsorbed on 3 mg of Fe_2O_3 particles (98% < 0.75 μm): suspended in saline given in 15 consecutive weekly intratracheal injections.

Treatment Group	Number of Animals	Number of Dead Animals	Current Week	Tumor Bearing Animals No.	%	First Tumor	Total Dose at 2 yr (Rads)
Control	63	52	93	0	0	----	----
Fe_2O_3 only	32	30	93	0	0	----	----
0.2 μCi/wk	35	35	60	32	91	15th wk	4,500
0.01 μCi/wk	34	21	59	10	30	40th wk	225

The number of animals consists of the survivors of the 15-week treatment period which were autopsied (60 animals/group at start). The doses given are maxima-carcinogenic doses which are less than 225 rads.

^{210}Po Grossman et al. 1971[27]

A later report on Little et al. 1970[26] gives the incidence of bronchogenic tumors as 91% and 43% in the high and low dose groups respectively.
Syrian golden hamsters were given intratracheal injections twice weekly for 7 weeks.
The doses were given in two separate intratracheal instillations (a and b in table below).

Treatment Group a	Treatment Group b	47 wk Survivors %	Tumors at 27 wk
3 mg Fe_2O_3	0.2 μCi in saline	2	17
Saline	0.2 μCi	6	9
Saline	0.2 μCi on 3.0 mg Fe_2O_3	28	7
Saline	0.2 μCi on 0.3 mg Fe_2O_3	32	3

^{210}Po alone is said to be homogenously distributed in the lung.

^{238}U Leach et al. 1970[28]

Monkeys, dogs, and rats were exposed to UO_2 dust (M.M.D. 1.03 μm, g std dev 2.40) 5 mg/m^3 for 6 h/day, 5 days/wk. The rats, after an exposure of 1 year, showed no pathological changes in the lung apart from pigmented macrophages in the alveoli and bronchi. In dogs there were no pathological changes in the lung after 5 years of exposure and estimated radiation doses of 400 rads. Monkeys responded with a patchy hyaline fibrosis that first appeared at 3.6 years after a dose of 500 rads. No tumors were reported in any animals at the end of the 5 year exposure. Despite doses to the tracheobronchial lymph nodes of dogs and monkeys that were on the order of 10^4 rads no pathology other than an occasional necrosis and fibrosis were reported.

^{239}Pu Antonchenko et al. 1969[29]

Rats, 140-160 g, were exposed to an aerosol (90% 0.7-1.9μm, median diameter 1 μm) of Pu citrate or ammonium plutonium pentacarbonate. (pH 5 and 8, respectively) for 20 min.

Treatment Group	Number of Animals	Average Life Days	Average Life Dose (rads)	Lung Pathology (%) Carci-noma	Ade-noma	Adenoma-like Structures	Epithelial Metaplasia
Controls	248	673	0	---	---	----	---
Citrate							
1.028 μCi[a]	23	64	3,820	---	---	----	9.1
0.803 μCi	12	69	3,090	---	---	----	8.3
0.511 μCi	94	124	2,370	2.2	---	8.9	73.4
Carbonate							
1.460 μCi	12	77	7,320	---	---	----	9.09
0.774 μCi	23	78	3,900	---	---	4.4	13.0
0.455 μCi	69	139	2,780	4.6	3.08	12.0	61.6

[a]Initial deposition. (Apparently the short survival time in the higher dose groups precluded the development of the characteristic pathology).

^{239}Pu Buldakov et al.[30]

Rats. Inhalation of soluble Pu compounds: citrate and ammonium pentacarbonate.

	Treatment Group (μCi deposited)	Number of Animals	Mean Survival (Days)	Lung Dose (Rads)	Lung Tumors %
Citrate	0.008	157	635	47	5
	0.02	124	585	117	2.5
	0.04	203	545	234	8.4
	0.08	31	546	467	35.5
	0.15	105	464	852	23.8
	0.25	113	416	1,390	23.0
	0.36	39	221	1.740	7.7
	0.51	90	124	2,370	0
	0.80	12	63	3,090	0
	1.03	20	64	3,820	0
Ammonium-Plutonium-Penta-Carbonate	0.004	48	571	41	4.2
	0.007	101	571	80	5.0
	0.017	91	584	186	13.2
	0.045	126	582	497	36.4
	0.15	83	484	1,065	42.7
	0.25	126	361	1,615	26.4
	0.35	22	247	2,140	9.0
	0.46	65	139	2,780	0
	0.77	23	78	3,900	0
	1.46	11	77	7,320	0

The tumors were squamous cell carcinomas, adenocarcinomas and hemangiomos. Note the tumor incidence at low doses and the absence of tumors at high doses.

^{239}Pu Clark et al. 1964[31]

Dogs inhaled particles (0.5-0.65 μm). At 855 days 28 were dead. There was one lung tumor at 150 days. Six more died between 855 days and 1,446 days, of these, four had bronchiolo-alveolar tumors. The estimated doses were between 9,000 and 23,000 rads, resulting from burdens of 0.6 to 19 μCi.

^{239}Pu Park et al. 1967[32]

Dogs inhaled particles (0.5-0.65 μm) (continued from above). Of 25 dogs dying or sacrificed between 850 and 2,270 days, 12 had primary pulmonary tumors. The estimated doses to the tumor bearing animals ranged between 3,100 and 13,600 rads, resulting from terminal lung burdens of 0.5-2.7 μCi.

^{239}Pu Park et al. 1972[33]

Dogs inhaled PuO_2 particles (0.5-0.65 μm) (continued from above). Of 65 dogs exposed, 62 are dead, and 24 had pulmonary neoplasia. Between 55 and 1,600 days, 36 died of pulmonary insufficiencies (edema, fibrosis, hyperplasia etc.) Twenty of 21 dogs surviving 1,600 days had lung tumors. Estimated initial lung burdens were 0.2 to 3.3 μCi. At 11 years the average dose to the lungs of tumor-bearing animals was in the 2,000-12,000 rads range.

^{238}Pu Park et al. 1970[34]

Twelve dogs inhaled $^{238}PuO_2$ particles: CMD 0.05 μm GSD 1.9.

Terminal Burden (μCi)	Lung Burden (% Terminal Burden)	Survival Time (Days)
261	92	27
167	94	30
168	93	35
112	92	56
74	91	56
140	94	61
84[a]	90	70
88[a]	90	76
58	91	77
44[a]	91	94
17[a]	80	125
25	77	180

[a]animals with lung tumors: bronchiolo-alveolar carcinoma. Dose range, all animals, 8,000 to 26,000 rads. Almost total necrosis of tracheobronchial, mediastinal, and sternal lymph nodes.

^{238}Pu C. L. Sanders 1973[35]

Rats, female, were exposed to an aerosol of crushed $^{238}PuO_2$ microspheres: CMD 0.02, GSD 2.1. The material was considered soluble (72% ultrafilterable). Life-time study (>1,000 days).

Treatment Group	Number of Animals	Lung Dose[a] (Rads)	Lung Tumors %	Median Life Span (days)
Control	92	0	1.1	825
5 nCi[b]	30	9	6.6	∿ 650
18 nCi[b]	30	32	23.3	675
230 nCi[b]	30	375	25.0	550

[a] mean dose in 2 years
[b] mean initial lung deposition

Lung tumor incidence in the 5 nCi group was not significantly different from lung tumor incidence in the control group. Of the 19 pulmonary tumors found, 14 were bronchiolo-alveolar carcinomas, 2 were mixed carcinomas and there was one epidermoid carcinoma, one undifferentiated carcinoma and one lymphosarcoma. The author concludes: " - - - that spreading the Pu dose in the lung, as compared to concentrating in PuO_2 particles, is more carcinogenic due to the greater number of epithelial cells 'hit' by alpha emissions from Pu".

^{241}Am Thomas et al. 1972[36]

Dogs were exposed to an aerosol (AMAD 0.9 μm, GSD 1.5).

Lung Burden (μCi) Initial	At Sacrifice	Days to Sacrifice	Lung Dose (Rads)	Lung Pathology
31	2.3	127	3,000	Inflammation
21	1.0	256	3,200	Fibrosis
26	0.71	512	3,800	Fibrosis and mineralization
23	0.38	1,022	5,300	Fibrosis and mineralization

Doses to lung were delivered early; more than 90% of the ^{241}Am had left the lung by 127 days. The highest doses were delivered to the tracheobronchial lymph nodes (3,500-17,400 Rads), but the chief pathologies were fibrosis in the medullary areas and depletion of lymphoid elements.

^{237}Np Levdik et al. 1971[37]

Rats were injected intratracheally with nitrate and oxalate solutions of ^{237}Np (pH 2-3 and 5, respectively).

Treatment Group (μCi/kg)	Number of Animals	Average Life Span (Days)	Dose (Rads)	Lung Tumor Incidence (%) Malignant	Benigh
Control	274	700	0	3.65	0.36
Nitrate					
0.017	50	660	5	16.0	2.0
0.083	44	684	28	20.5	4.65
0.41	48	685	138	12.68	8.35
2.0	49	505	2,500	14.28	2.1
Oxalate					
0.017	85	645	27	10.6	0.0
0.083	89	661	134	9.0	3.36
0.41	89	649	671	28.0	5.6
2.0	81	453	3,220	37.45	3.75

Part of the increased carcinogenicity is attributed to the chemical toxicity of Neptunium.

4. EXTERNAL IRRADIATION

X-ray Koletsky and Gustafson 1955[38]

220 kV, 15 ma, filters: 1.0 mm Al, 0.5 mm Cu, 60 R/min.

Rats, male, 200 g were exposed to a single total dose of 660 R of whole-body radiation. The 123 rats that survived 6 months or more were autopsied at death.

Treatment Group (Time of Death)	Number of Deaths Irrad.	Con.	Number With Tumors Irrad.	Con.
6-12 months	46	3	7	1
12-18 months	47	6	32	0
18-24 months	29	14	29	2
over 24 months	1	13	1	5

One rat had primary carcinoma of the lung, an undifferentiated carcinoma in the lower left lobe. The right lung had an adenocarcinoma. The time of appearance is not given.

X-ray Cember et al. 1956[39]

100 Kv, 4 ma, filters: 1 mm Al. 61.8 R/min.

Rats, female, 270 g; equal doses on 5 consecutive days. Only the thoracic region was exposed. The rats were rotated 4 times during each exposure.

Treatment Group	Number of Animals	First Death (wks)	Median Lethal Time	Last Death	Tumors
Control	20	No losses	---	---	---
5,750	20	6	12 months	6 Sac.@ 15 months	2 lymphoma
11,500	20	3	166 days	11 months	1 lymphoma
17,250	15	3	37 days	6 months	1 lymphoma

The primary loci of the tumors is uncertain because of metastases. Broncho-pneumonia was the most common finding.

X-ray Maisin et al. 1958[40]

250 kV, filters: 1.0 mm Al, 0.25 mm Cu. 90 R/min.

Rats, 145-165 g were exposed to single doses of whole or partial body irradiation. Only animals alive at 6 months post exposure were considered. These survivors were autopsied at death.

Treatment Group	Number Autopsied	Total Number of Cancers	Leuco-Sarcomas	Epithelio-mas	Sarcomas	Epithelio-Sarcomas
Control	460	13	10	2	1	---
300-2,000 R	1,237	100	24	52	21	3
2,000 R left lung	10	5	--	3	2	---

Only one rat (cervico-sternal shield - 600 R) receiving less than 2,000 R had a pulmonary cancer (at 15 months post exposure). Of the 5 cancers in the 2,000 R group, which had the entire body, save the left lung region, shielded, 3 were bronchial epitheliomas appearing at 11, 15, and 23 months. There were 2 sarcomas of the left thoracic wall. No other pulmonary pathologies were reported.

X-ray Castanera et al. 1968[41]

200 kV, 15 ma, filters: 1.0 mm Al, 0.5 mm Cu. 27 R/min.

Rats, male, free of epidemic respiratory infection were exposed to single, whole-body irradiation with x-rays or fast neutrons (12 Mev H^+ on Be).

Treatment Group (Rads)	Number of Animals	Time of Last Death (Days)	Adenomas (%)	Adenocar-carcinomas (%)
X-ray				
430	88	700-800	16	3
680	107	600-700	11	4
Neutrons				
230	41	500-600	17	7
320	73	500-600	10	6
Control	129	800-900	1	0

Tumor frequency was estimated from chart 5. Operable tumors were removed surgically. There were no other primary pulmonary tumors.

Castanera et al. 1971[42]

Male rats were given a single whole-body exposure to fast neutrons (12 Mev H^+ on Be). The rats were free of epidemic respiratory infections.

Treatment Group Age (months)	Dose (Rads)	Number of Animals	Median Survival Time (Days)	Primary Lung Tumors Benign	Malignant
1	215	79	433	13	3
1	0	40	699	5	0
3	230	41	436	17	7
3	0	41	601	0	0
21	215	53	167	8	2
21	0	24	158	4	0

All tumors were bronchiolar in origin. Multiple tumors were found in other organs.

X-ray DeVilliers and Gross 1966[43]

135 kV, 4 ma, filters: 2.43 mm Al. ∿100 R/min.

Male Syrian golden hamsters and male rats were exposed to 5 equal doses of x-rays delivered on 5 consecutive days. A collimated beam was directed at the chest region. Four portals were varied through 90° per day. Hamsters received 4,000 R, rats 3,570 R.

	Time of Sacrifice (Months)	Number of Animals	Tumors Adenomas	Malignant
Rats	4	12	1	---
	8	11	---	---
	12	12	2	1 Adenocarcinoma
	24	12	2	2 - Reticulum cell sarcoma Squamous cell carcinoma

	Post Irradiation Time (weeks)											
Hamsters	6	7	10	11	12	13	14	15	16	17	18	19
Spontaneous Deaths	1	1	2	3	3	13	6	4	2	2	2	3
Squamous Cell Cancer	-	-	-	-	-	2	-	2	1	-	-	-

Fifty-seven hamsters were irradiated, all died spontaneously. Only those dying between 6 and 20 weeks (42) are listed. Pulmonary cancers were not found later than 3 1/2 months, although 4 at 6 months and at 12 months and 13 at 24 months, whereas 7 of the eight tumors found in rats were found at 12 or 24 months.

X-ray Gross et al. 1969[44]

3,000 R, 110 kV, 6.4 ma, filters: 1.83 mm Al. 75 R/min and 4,000 R, 110 kV, 8.6 ma, filters: 1.83 mm Al. 100 R/min.

Rats and hamsters were exposed to a collimated beam of x-rays directed at the chest region. The animals were rotated axially at 7 rpm during exposure which was given in 5 equal doses on 5 consecutive days. Eight weeks after exposure some animals were treated with dimethyl benzanthracene (DMBA) and/or jewelers rouge (Fe_2O_3).

Treatment Group (Rats)	Number of Animals	9 Month Survivors	Adeno-carcinoma	Squamous Cell Carcinoma	Fibro-sarcoma	Undiffer-entiated
4,000 R						
DMBA + Fe_2O_3	40	37	15	3	1	1
Fe_2O_3	40	29	7	0	0	1
Radiation only	43	42	18	4	1	1
3,000 R						
DMBA + Fe_2O_3	40	39	14	2	1	0
Fe_2O_3	40	39	16	0	0	0

Data for the appropriate controls are not tabulated. In the unirradiated rats Fe_2O_3 with or without DMBA caused no tumors. DMBA did not increase the prevalence of cancer in irradiated rats.

Treatment Group (Hamsters)	Number of Animals	2.5 Months Survivors	Number of Cancers	Time To Tumor (Months)
4,000 R				
DMBA + Fe_2O_3	40	37	2	2.5, 7.5
Fe_2O_3	38	36	0	---
Radiation only	46	46	1	11
3,000 R				
DMBA + Fe_2O_3	38	38	0	---
Fe_2O_3	38	37	1	14
Radiation only	8	8	0	---
Control				
DMBA + Fe_2O_3	40	35	2	9.7, 18.5
Fe_2O_3	20	19	0	---
No treatment	10	10	0	---

The first appearance of a cancer was the base for the selected survival times (column 3). Only frank malignancies are included, microlesions were not considered. Chronic bronchitis was prevalent. (Note the decreased incidence of cancer among hamsters compared with that of DeVilliers and Gross[43] 1966).

B. Discussion - J. W. Healy

Prior to World War II and the Manhattan Project, radiation exposure limits had been derived for X-rays or radium gamma rays, for radon in the air and for radium as an internal emitter.* The external limits were based on the radiation field to which the individual was exposed with little or no consideration of the distribution of radiation through the body or of the exposure of specific organs.** During the Manhattan Project, the need for considering radiations other than X- or gamma, the presence of varying energies of radiations and the availability of a wide variety of radioactive chemical species resulted in the extrapolation of these limits to the new conditions through the derivation of new concepts (such as the rem) and an increased sophistication in dosimetry as applied to individual organs.

Following the war, considerable attention was given to formalizing these concepts in a manner which could be used by those responsible for guiding radiation protection practices in the vastly increased uses of radiation and radioactive materials resulting from nuclear energy. This work was carried out by the NCRP in consultation with foreign scientists through conferences and informal discussions. In 1954, the NCRP subcommittee on Permissible Internal Emitters published their report[45] that first expounded on the critical organ concept which has served as the basis for the majority of the internal emitter limitations.* Here the critical organs were defined on the basis of experience with external radiation. The skin was chosen as one organ because of the production of skin cancers, usually on the hands from the greater exposure which they received. The increased incidence of leukemia in radiologists led to the designation of the blood-forming organs as one of the more important critical organs, while cataracts produced by high LET radiations resulted in the lens of the eye receiving special designation. Since leukemia was the primary outcome from whole body radiation in the experience available, it was considered "- - - safe to assume at present that the blood-forming organs constitute the most critical organs".** Exposure to the more deeply seated organs was then limited to that of the blood-forming organs.

In the 1954 NCRP document, the limits for the blood-forming organs and other organs were established at 0.3 rems per week (if received every week this would be essentially 15 rems per year). This is the limitation used by the Internal Dose Committee[46] in obtaining their values for organs other than bone. In 1957, the NCRP again revised their recommendations for

*For an excellent review of the information available on the effects of internal radiation on humans at the time of World War II, the reader is referred to "The Tolerance Dose" MDDC 1100 by S. T. Cantril and H. M. Parker.

**In this statement we are referring to the official limitations adopted by the NCRP and the ICRP. Individuals did concern themselves with these matters in reviewing the data available and in applying the limits.

*Subcommittee 2 on Permissible Internal Dose published its report in 1953[46] listing MPC's and maximum permissible body burdens based on the critical organ concept. The dose limitations were those given in the later report of the external dose subcommittee and seem to reflect the NCRP decisions arrived at in the later report.

**Genetic considerations are not pertinent to this review but they were not ignored. "From the point of view of genetic damage manifestable in future generations, the gonads, of course, constitute the critical tissues 'par excellence'." However, the contribution of occupational exposures to the dose to the population as a whole was not considered limiting.

***Bone limits were based upon a biological comparison with radium.

workers to lower the radiation doses to the whole body, head and trunk, active blood-forming organs and gonads to an average of 5 rems per year over the working years beyond age 18.[47] However, the recommended limits for internal organs other than thyroid, skin, and gonads remained at 15 rems per year. In the same document the NCRP recommended levels of one-tenth of those for workers for individuals outside of the controlled area. The latest report of the NCRP[48] continues the use of 15 rems per year for organs other than red bone marrow, skin, and gonads for occupational workers, but recommends a limitation of 0.5 rems per year to individual organs for members of the general public.

Thus, it can be seen that the current limitation of 15 rems per year for the lung of workers can be traced to the original critical organ concept and the dose limitations derived from early experience with external radiation. The recent lowering of the recommended limit for the lung of individual members of the public by the NCRP is by a factor of three and is expressly indicated as being "- - - based primarily on the desire for numerical simplicity in the standards and not on an established biomedical need." At the same time, the 1971 NCRP recommendations include a concept of "significant volume" over which the dose should be averaged. The implication being that any redistribution of a given dose within this volume would not significantly affect the outcome. The 1971 NCRP report continues, "It is usually assumed that the 'significant volume' should be of the order of one cubic centimeter. This will be grossly conservative under most circumstances, and in special estimations, use of a larger volume is justified."*

Although the original decision to use the average dose to the lung (or other organs) was made in the early period of the derivation of dose limitations, it should not be inferred that those bodies responsible for such recommendations have ignored the subject. In the Chalk River Tri-Partite Conference with scientists from the U.S., U.K. and Canada,[49] the statement is made: "In relation to the possible pathological effects of radioactive particulates in the lungs, Dr. Hamilton pointed out that the cells in the immediate neighborhood of a dust particle containing 1 or 2% of plutonium would be subjected to a dose of about 400 r/day. The general opinion which emerged from the discussion was that the carcinogenic effect per unit volume is probably considerably less for the irradiation of small masses of tissue than for large." The ICRP has addressed this general question of non-uniform dose periodically, usually by special groups commissioned by the ICRP to study the question. In its Publication 9 (1966),[50] the ICRP stated:

"In the case of non-homogeneous distribution of absorbed dose in the lung, an estimate of the Dose Equivalent to the whole lung, determined merely by the product of QF and the mean absorbed dose, may be greatly in error, but our full understanding of this problem must await further experimental evidence. In the meantime there is no clear evidence to show whether,

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*The foregoing review has been greatly shortened to indicate the salient points in the derivation of the current lung limitations. At the same time, it has focused on the NCRP recommendations because of their importance in the early days when the present limits were first derived. The ICRP recommendations differ in detail but follow the same general pattern. The reader with interest in this subject is urged to review these documents for further detail.

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." In Publication 14 (1969)[51] prepared by two Task Groups of ICRP Committee 1, the irradiation from radioactive particles was considered specifically. Here, it is stated: "The problems of high local concentration of dose are at their most severe with radioactive particulate material in the tissue, especially with alpha emitters. Here the local dose can reach very high values even though the mean tissue dose may be very low. Certainly it cannot be assumed that linearity of dose and effect will hold at these high doses and dose rates. On the other hand, there may be a great deal of cell death, and particularly with alpha emission, with its short and well-defined range, the number of affected but viable cells may be small compared with the number of killed cells. However, this ratio will depend on the size and activity of the particles, the extent to which they aggregate, and their movement within the tissue, and the movement of the cells past them.

"On the basis of general considerations and some experimental data and clinical experience the Task Group were of the opinion that, for late effects, the same radiation energy absorption might well be less effective when distributed as a series of "hot spots" than when uniformly distributed. Thus, with particulate radioactive sources within a tissue, a mean tissue dose would probably introduce a factor of safety. However, a severe practical problem has now been recognized in connection with the inhalation of plutonium particulates, and is now being considered in detail by a Task Group of Committee 1 of ICRP."

The Task groups also considered the problem of translocation of plutonium to lymph tissue and concluded:

""In the meantime, the Task Group are of the opinion that any immediate change in the dose limit for plutonium on the basis of risk of lymphoid tissue is not warranted."

The potential outcome of an inhalation of radioactive materials can be changed by a number of factors. If, for example, the material is readily translocated from the lung to other organs, the eventual damage to these other organs may well appear earlier than, and overwhelm any lung damage.* Thus, in considering lung dose we are focusing primarily on those materials which will be retained in the lung for reasonably long periods of time. If the quantity in the lung is large enough, death will result at early times due to pulmonary insufficiency resulting from an adema or destruction of functional living tissue. In practice, we are interested in low dose effects which will occur late in life and carcinogenesis would seem to present the end point of greatest interest. Life shortening has been noted in many experiments, particularly at higher levels, and is used as a criterion of damage. The statistical uncertainties in most experiments occasioned by the limited numbers of animals and the variation in death times make this a relatively nonsensitive indicator, even though the argument can be made that a finding of no significant life shortening is of importance since a death is a death, regardless of whether it is caused by a heart attack or a cancer. However, in many experiments in which life-shortening was not significant, the incidence of cancer at the end of life was significant, indicating that radiation effects did occur. As a result, the present studies focused primarily on cancer incidence as being the appropriate end point.

*Of particular interest in this respect is the recent work at Battelle Northwest[52] which indicates that certain forms of $^{238}PuO_2$ are rapidly translocated from the lung to the bone when inhaled resulting in the production of bone tumors.

In most of the experiments there appears to be a relation between the radiation dose and the time of occurrence of malignancies in animals: In general, the higher the dose (or in case of internal emitters, the dose rate) the shorter the time required for cancer production. This phenomenon is frequently used to invoke the possibility of an "effective threshold" since the time required to permit cancer formation following a low dose will be so great that it exceeds the normal life span even if the induction follows a linear relation with dose. However, in interpreting data, it must be borne in mind that the opposite phenomenon will occur when the dose or dose rate becomes too high. That is, the animal will die from other causes before there is time to induce cancer. This was seen in the results from the dogs at Hanford[31,33,34] where the early deaths were due to pulmonary insufficiency with cancers eventually appearing only in the animals with lower lung burdens and which had lived most of their life span. Thus, if radiation dose is used as a primary parameter in investigating incidence, it is important not only that the animals live out their normal life span so that the full cancer incidence develops, but that the total dose is not so high that deaths occur from other causes before the cancer can develop. These conflicting trends in causes of death can result in an apparent optimal dose for the production of malignancies. However, even at this optimal dose, the full expression of the malignancies possible per unit dose at lower values will not occur.

Akin to this concept is that of "overkill" of single cells close to the particle. In the case discussed above, the production of early death by causes other than cancer can be regarded as a result of "wasted radiation" in interpretations based upon the narrow concept of carcinogenesis as an end point.* From this standpoint, doses which lead to death before cancer appears can be considered to be overkill of the organism since the full expression of the carcinogenic effects is not attained. For a single particle in the lung (or other tissue) the dose rates at close approaches to the particle can be high enough so that even a relatively limited time of residence in the tissue will result in the death of cells within a given radius depending upon the activity of the particle and the type of radiation. Such cells will not be able to later reproduce and, regardless of the degree of damage, will not lead to cancer.** From this standpoint, therefore, one would expect that particles which lead to such overkill would be less hazardous than uniform radiation to the overall organ since not all of the radiation is used in attaining the final end point, cancer. In fact, such a concept would lead immediately to the conclusion that the larger the particle (in terms of activity) the less effective it would be in producing cancer since the dose rates close to the particle would increase as the activity increased thereby leading to a greater fraction of radiation wasted on dead cells. One clear cut experiment possibly showing this effect was done by Passonneau[1,53] using Sr-90 beads on rat skin. Here the same amount of activity was used for the same area of skin but the activity was distributed either as a uniform flat plate, in 50 beads, in 20 beads or in 10 beads. The results given in Table II indicate clearly a decrease in the tumor production efficiency as the activity was

*We have already mentioned that this is an appropriate end-point for consideration of dose limitation since it appears to be the latest effect in time to occur even when other effects are relatively ineffective in shortening the life span.

**However, the presence of dead cells, cellular products or fibrosis may be required before a cellular transformation can express itself as a cancer. This is an interesting possibility which needs more study.

TABLE II

TUMOR PRODUCTION IN RAT SKIN

UPON EXPOSURE TO FLAT PLATE AND POINT SOURCES

Source	Activity	No. of Animals	No. of Tumors	Tumors per μc	Relative Efficiency
Flat Plate 1000	28.6 μc/cm^2	71	89	4.94×10^{-4}	1.59
Flat Plate 1500	42.9 μc/cm^2	73			
50 beads	30 μc/bead	58	27	3.10×10^{-4}	1.00
20 beads	75 μc/bead	77	24	2.08×10^{-4}	0.671
10 beads	150 μc/bead	74	16	1.44×10^{-4}	0.464

subdivided into more active particles. Gamertsfelder, in an analysis of these data,[1] assumed a mid-lethal dose for cells of either 4635 or 9300 rads and a probability of tumor production increasing as the nth power of the dose to the cell. He then calculated the ratio of the number of tumors expected relative to those produced by the 30 bead configuration. The range of the experimental data is not great enough to permit distinguishing between the curves represented by different values of n but within this limited range, the calculations fit the observed trend. It is of interest to note that these calculations indicate a maximum in the relative efficiency of tumor production if n is greater than 1 while if n is equal to one, the curve approaches an asymtote as the activity per particle gets smaller. The value of this asymtote for the assumed median lethal dose of 4650 rads is 3.2 and for 9300 rads is 2.42. Since the condition where the activity per particle becomes very small is essentially that of a uniform, plane source, the comparison between this value and the value of 2.4 noted in the experiment (corrected linearly from the 1000 μCi flat plate source data) may be of significance. A somewhat similar calculation by Langham and Dean[54] but on an absolute basis, to predict the probability of tumor production from various sizes of plutonium particles, used data derived by Albert[55] on the production of tumors in rat skin versus dose to the cell. The results of this calculation show a very high probability of tumor production from most particle sizes. However, as the authors indicate, the paper was published to illustrate the method rather than to provide results. The results of this work can be questioned on many grounds including the use of the data on tumors in rat skin for lung tissue, the finding of Albert that the sensitive cells are at the base of the follicle in the rat skin and the fact that the assumed efficiency of production of lung cancer per cell does not conform to the experience with humans in the production of lung tumors from external radiations.

The results of wasted radiation in the production of lung fibrosis at high levels of administration of radioisotopes or the induction of other causes of death before cancer can develop raises the question of the possible effects of such wasted radiation in the particle case. Richmond, et al.[8] investigated the effects of Pu-238

dioxide particles lodged in the lung vascular following IV injection. These particles averaged about 180 μm in diameter and gave average dose rates to the entire lung of about 3.5 rems per hour with the dose rate in the vicinity of the particle on the order of 10^9 rads per hour. The longest exposure until sacrifice was a group of 6 rats which lived to 600 days. Examination of the lung following these exposures indicated the presence of a microleison with complete degeneration of the cells close to the particle. However, the evidence indicated that this was not simply a stable type of scar tissue but rather that the lesion was in a dynamic state in which the collagen was renewed constantly with subsequent liquification. Within this time period there was no indication of effects which would be deleterious to the animal's overall well being. It is noteworthy that the energy delivered to the lung, if averaged over the full lung would be on the order of 2,000,000 rads,* well in excess of those doses which have been shown to produce deaths in relatively short times when more uniformly distributed and considerably above the doses required to produce lung cancers.

One of the uncertainties with such an analysis of overkill of cells is, of course, the possibility of movement of the particles within the lung tissue so that the number of cells at risk becomes much greater and the doses delivered become smaller. In the experiment of Richmond, et al.[8] quoted above, the particles were relatively firmly held in the blood vessels and, therefore, were not representative of particles actually deposited in the alevoli. Movement of such particles is known to occur through ejection with mucus and movement by the cilia and by engulfment by macrophages. Thus, quantitative estimates of the degree of overkill of cells and the fraction of radiation wasted would be uncertain since such movement is difficult to model. However, it would seem that such arguments would be of more interest in the actual quantitative sense than in the conceptual sense. If the particles are large enough so that very high dose rates are encountered in the near vicinity, there still, will be a degree of overkill and wasted radiation although it may be considerably lower than would be estimated by the static model.

Additional uncertainty is added by the possible reactions of the cells located at the periphery of the zone of destruction caused by the radiation. This would involve cells receiving radiation doses ranging from just sublethal to essentially zero. If there is attempted repopulation of the volumes of destruction, this could result in rapid proliferation of these cells which have already been damaged. This situation would appear to be the most serious contender for the production of cancer and also one which would be the most difficult to investigate experimentally without an understanding of the basic mechanism of cancer production and the response of individual cells to these conditions in an otherwise normal environment and surrounded by otherwise normal cells. Information on this possibility is limited, but some indication that it is not a predominant problem can be obtained from the experiments of Passonneau[53] and Richmond[8] which did involve just such conditions in several types of tissue.

The outstanding example of increased carcinogenity of a deposited radioactive material due to localization and nonuniform dose distribution is plutonium in bone.

*Richmond, et al.[8] indicates that Halley has estimated the average dose to a human lung for the same size of particle to be 3.5 rems per hour. Using an RBE of 10 for alpha particles and considering the rat lung to be on the order of 1/500th the mass of the human lung, the dose in 600 days becomes:

$$\frac{3.5}{10} \times 500 \times 24 \times 600 = 2{,}500{,}000 \text{ rads.}$$

Here, the classical work of Brues[56] led to the conclusion that plutonium is about five times as effective for the same energy deposition as is radium, which is, in itself, nonuniformly distributed. Studies of the comparative deposition in bone of these two isotopes have indicated that the radium, being chemically similar to calcium, tends to deposit in the mineralized portions of the bone and eventually is distributed through the bone mineral by remodeling or is covered by new layers of calcified materials. By contrast, the plutonium is deposited on the bone surface in locations where it is adjacent to the regenerative cells and, in remodeling of the bone tends to redeposit on these surfaces. Thus, this represents the case of a very nonhomogeneous organ where the comparative isotope (radium), while not uniformly distributed, is more uniformly distributed than the plutonium. Further, the plutonium is preferentially deposited in the vicinity of the regenerative cells which are presumably more sensitive to the induction of cancer than the mineralized bone. This situation would seem to represent a localization of the radiation dose at cells which present a more sensitive target and therefore, eliminates some of the wasted radiation which occurs with radium in the mineralized portion of the bone. In essence, the bone can be regarded as composed of three regions of differing criticality: the marrow, the proliferating cells on the bone surfaces and the mineralized portion which has minimal metabolic activity and serves primarily as a structural supporting member for the body. In this case, the sensitive tissues are the marrow and the regenerating cells with the regenerating cells of most interest for plutonium as the average dose to the marrow from the poorly penetrating radiations from plutonium is comparatively low. Again, however, some significant dose rates to the marrow on a localized basis can be calculated. These are to a small fraction of the marrow falling within a few tens of micrometers of the deposited plutonium. The fact that leukemia is a relatively rare outcome in experimental animals given plutonium may serve as an indicator that irradiation of a small portion of an organ (the marrow) to a high dose is not particularly troublesome as long as the average dose is low.

A similar situation may, of course, occur in any organ as a number of different cell types can be present in the same organ and any mechanism which results in preferential irradiation of the more sensitive cell types could, theoretically lead to the same type of result. The high incidence of lung tumors in uranium miners from radon in mine atmospheres is attributed to the deposition of the particulate daughters of radon on the bronchi, particularly at points of division where the turbulence in the air stream produces increased impaction and deposition.

The estimation of the radiation dose to the assumed critical tissue, the bronchial epithelium, is complicated by the uncertainties in the areas of deposition and the thickness of the mucus layer which serves to absorb some of the energy of the radon daughters deposited on the surface. However, in a review of the dosimetry for the Federal Radiation Council[57] Parker considers, with important reservations, that one working level month corresponds to a dose to the bronchial epithelium of 2.8 rads. The working level for exposure to radon daughters is defined as any combination of radon daughters in one liter of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy. One working level month, then, is the total exposure resulting from working in such an atmosphere for 170 hours. If we assume that all of the alpha energy associated with the daughter products is released in the lung (i.e. all of the daughters are deposited and none are eliminated before

they decay) the average dose to a 1000 gram lung would be 0.44 rads. This is undoubtedly a maximum estimate since some of the daughters will be exhaled and a portion will be eliminated by ciliary action. However, much of the activity is associated with small particles which are deposited in the bronchi and lower pulmonary regions with relatively high efficiency. The linear velocity of particles moving up the bronchi is 0.25 to 1 cm/min while in the trachea rates can increase to 3 cm/min.[1] Because the longest half-life of the radon daughters of interest is 26.8 minutes, it would appear that a sizeable fraction of the material deposited in the bronchi would decay before elimination and that all of the material deposited below the ciliated region would contribute their full energy. If we apply this estimate of the average lung dose to the estimated exposures of the uranium miners in those exposure ranges where the incidence of lung cancer is high, we find that the dose to the total lung calculated on an average organ basis is, indeed, significant and in the range where animal data would indicate such an outcome to be expected.* Since there is uncertainty about the actual significance of the increase in lung cancer at the lower exposure levels, we will not discuss this phase. However, the dose levels corresponding to the exposure ranges used in the epidemiological study[57,58] assuming an average dose to the lung of 0.44 rads per WLM are listed in Table III.

*In order to permit a rapid appraisal of the data presented in the abstracts on the incidence of lung cancer at various dose levels, Fig. 1 presents a crude plot of the data for the alpha emitters. No attempt was made in this plot to reevaluate the dose estimates or to correct for experiments in which the incidence was measured before the full life-span of the animals. The five points at the lowest doses were the results of the ^{237}Np and the ^{210}Po administrations. The human data are estimates of doses received by a group of 37 individuals exposed during work with plutonium and represent periods of time ranging from 4 to 24 years after exposure.[59]

TABLE III

AVERAGE LUNG DOSES CORRESPONDING TO LEVELS OF EXPOSURE USED IN THE URANIUM MINER EPIDEMIOLOGICAL STUDY

Exposure WLM	Average Lung Dose-Rads
< 120	< 53
120 - 359	53 - 158
360 - 839	158 - 370
840 - 1799	370 - 792
1800 - 3719	792 - 1636
> 3720	> 1636

An additional argument concerning the present bases for radiation protection standards should be included in this discussion. As a basis for dose limitations, it is normally assumed that the response to a given dose is proportional to the dose received and that there is no threshold. While there is considerable evidence to support the use of this assumption, there is also evidence that the dose rate is an important factor, at least for low LET radiations, with the response decreasing as the dose is protracted, presumably due to the repair of the damage in the intervening time before the full dose is accumulated.* Acceptance of this assumption would indicate that the result of a dose to a small portion of a given tissue would be the same

*We note that the same argument cannot be made for alpha emitters since current evidence indicates that the damage from high LET radiations is not repaired. Thus, the assumption of linearity with dose, regardless of dose rate, would seem to be more appropriate for these materials than for the gamma or x-rays. As an aside, we also note that the amount of repair for gamma radiations appears to be on the order of 90%. If we assume no repair for the alpha radiations, the late result (after repair is over) would be about ten times as great for the alpha radiations as for the gamma. This appears to be about the same as the commonly accepted RBE or Quality Factor for alpha radiations.

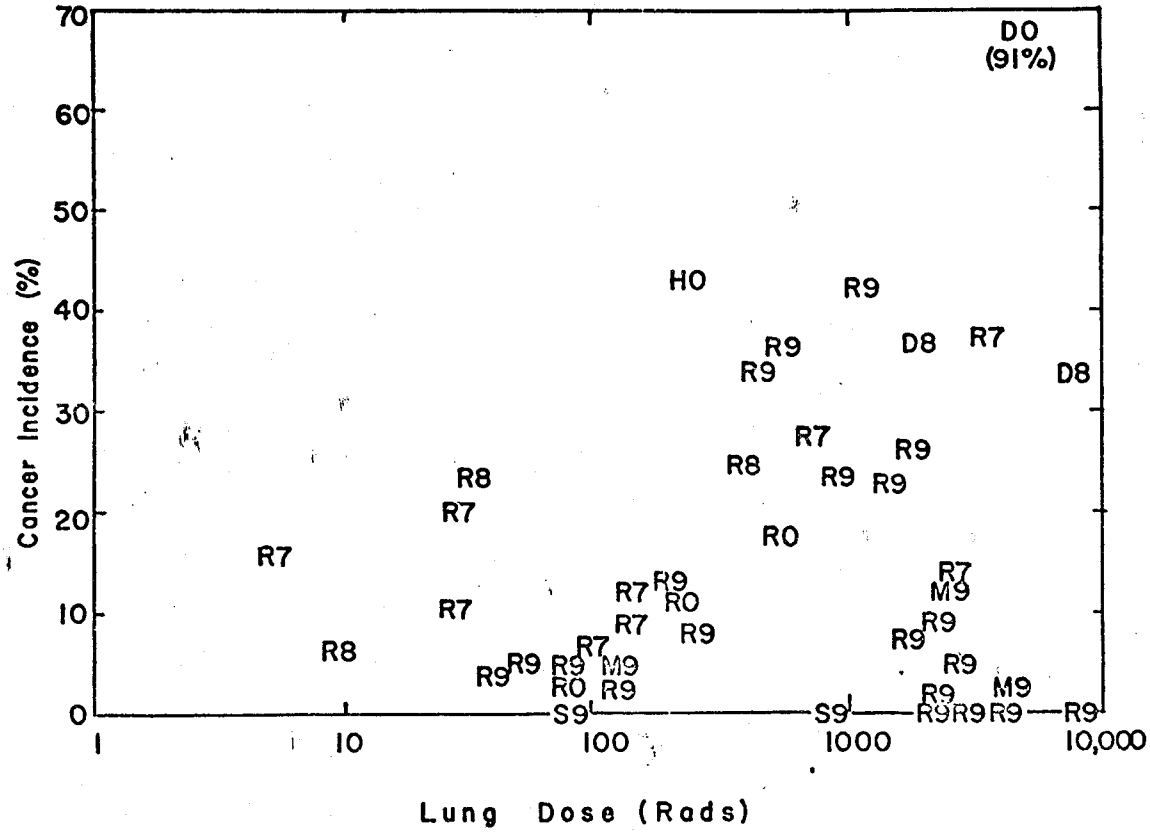

Fig. 1 A plot of the crude data for lung tumor incidence versus lung dose. Data have not been screened for length of exposure or accuracy of dose calculations. All doses are expressed as the average to the lung.
Legend: M-mice; R-rats; D-dogs; H-hamsters; S-humans.
0-^{210}Po; 7-^{237}Np; 8-^{238}Pu; 9-^{239}Pu.

as if the same amount of energy were distributed over the entire tissue. We have seen that this is not the case in some extreme situations such as in the overkill of cells close to a particle or the induction of more lethal effects at high dose rates. However, acceptance of the assumption of linearity at the more moderate conditions would lead to the conclusion that there should be no difference in outcome regardless of the distribution of the dose throughout the tissue, unless a critical portion of the organ is more sensitive. This would lead to the conclusion that nonuniform distribution of dose could have no greater effect than a uniform dose. Because this is based upon an assumption which is made in an effort to be conservative and is based upon effects at relatively low doses, we do not believe that this argument is very strong. However, a conclusion of nonlinearity of effect could have a major impact upon current radiations standard setting practices unless it is shown that such nonlinearity occurs only at very high cell doses.

No clear cut, overall picture of the relative effects of uniform versus focal dose can be drawn from the present data. It appears, from the $^{238}PuO_2$ microsphere data and the skin experiments with ^{90}Sr that, in the extreme situation of a single, very active particle, the focal radiation is considerably less damaging. Cember[15] concludes

that the focal source is less damaging for beta emitters than is the uniformly distributed source. The data of Grossman, et al.[27] for ^{210}Po on iron oxide particles indicates a seeming decrease in the tumor incidence as well as increased survival for the focal sources. Saunders,[35] as a result of his studies with soluble ^{238}Pu derived from crushed microspheres arrives at a conclusion that spreading the dose more uniformly results in an increased cancer incidence due to the greater number of epithelial cells involved. This conclusion was based on the observation of "- - a significant incidence of tumors in the lung and in other tissues at radiation doses that have not previously been shown to be carcinogenic in animals". In Figure 1, it is of interest to note that two of these data points are included in the five lowest dose points with the other points being the results of ^{237}Np administration. In both cases, significant numbers of tumors were also noted in locations other than the lung indicating a more general insult to the entire body.

Most of the support for particulates being more hazardous than a uniformly distributed material seems to arise from calculations based upon dose distribution around the particles and an assumed response of individual cells to this dose. In an overall appraisal of the information available, it does not appear that the majority of the data support the hypothesis that the particles are more hazardous than the uniform dose. A reasonable case can be made that they are less hazardous. The conclusion of this work to date, therefore, is that the preponderance of the evidence indicates that the use of an average lung dose is appropriate in limiting exposures and may well be conservative.

REFERENCES

1. Subcommittee on Inhalation Hazards, committee on Pathologic Effects of Atomic Radiation "Effects of Inhaled Radioactive Particles," Pulication 848, National Academy of Sciences-National Research Council, Washington, D.C. (1961).

2. O. Gates and S. Warren, "The Production of Bronchial Carcinomas in Mice," Am. J. Path. 36: 653 (1960).

3. S. Warren and O. Gates, "Cancers Induced in Different Species by Continuous Gamma Radiation," Arch. Environ. Health 17:697 (1968).

4. S. Laskin, M. Kuschner, N. Nelson, B. "Carcinoma of the Lung in Rats Exposed to the Beta Radiation of Intrabronchial Ruthenium-106 Pellets," J. Nat. Canc. Inst. 31:219 (1963).

5. S. Laskin, M. Kuschner, B. Altschuler and N. Nelson, "Tissue Reactions and Dose Relationships in Rats Following Intrapulmonary Beta Radiation," Health Physics 10:1229 (1964).

6. M. B. Divertie, J. L. Titus and R. G. Shorter, "A Simple Method for the Production of Experimental Squamous Cell Carcinoma of the Lung in Rats," Am. Rev. Resp. Dis. 96: 820 (1967).

7. H. W. Altmann, W. Hunstein, and E. Stutz, "Über Lungenveränderungen und Lungentumoren nach Bestrahlung mit Radioaktiven Strontium (^{90}Sr)," Beitr. Pathol. Anat. Allgem. Pathol. 124:145 (1961).

8. C. R. Richmond, J. Langham, and R. S. Stone, "Biological Response to Small Discrete Radioactive Sources - II. Marphogenesis of Microleisons in Rat Lungs from Intravenously Injected $^{238}PuO_2$ Microsphere," Health Physics 18:401 (1970).

9. H. Cember and J. A. Watson, "Carcinogenic Effects of Strontium-90 Beads Implanted in the Lungs of Rats," Am. Indust. Hyg. Assoc. J. 19:36 (1958).

10. H. Cember, T. F. Hatch, J. A. Watson, and T. Grucci, "Pulmonary Effects from Radioactive Barium Sulfate Dust," AMA Arch. Indust. Health 12:628 (1955).

11. H. Cember and J. A. Watson, "Bronchogenic Carcinoma from Radioactive Barium Sulfate," AMA Arch. Indust. Health 17:230 (1958).

12. H. Cember, J. A. Watson, and A. A. Apritzer, "Bronchiogenic Carcinoma from Radioactive Cerium Fluoride," AMA Arch. Indust. Health 19:14 (1959).

13. H. Cember, "Further Studies on Lung Cancer from $^{144}CeF_3$," Health Phys. 9:539 (1963).

14. H. Cember and K. Stemmer, "Lung Cancer from Radioactive Cerium Chloride," Health Phys. 10:43 (1964).

15. H. Cember, "Radiogenic Lung Cancer," Progress in Experimental Tumor Research v4:251 (1964).

16. H. Cember, "Empirical Establishment of Cancer-Associated Dose to the Lung from ^{144}Ce," Health Phys. 10:1177 (1964).

17. F. F. Hahn, S. A. Benjamin, B. B. Boecker, T. L. Chiffelle, C. H. Hobbs, R. K. Jones, R. O. McClellan, J. A. Pickrell, and H. C. Redman, "Primary Pulmonary Neoplasms in Beagle Dogs Exposed to Aerosols of ^{144}Ce in Fused-Clay Particles," J. Nat. Canc. Inst. 50:675 (1973).

18. N. N. Kurshakova and A. E. Ivanov, "A Model of Experimental Lung Cancer Caused by Intretracheal Introduction of Radioactive Cerium," Bull. Exptl. Biol. Med. 54:787 (1962).

19. L. A. Temple, S. Marks, and W. J. Bair, "Tumors in Mice After Pulmonary Deposition of Radioactive Particles," Int. J. Rad. Biol. 2:142 (1960).

20. H. L. Berke and D. Deitch, "Pathological Effects in the Rat After Repetitive Exposure to Europium 152-154," Inhalation Carcinogenesis Conf. 691001:429 (1970).

21. T. A. Kotchetkova, G. A. Avrunina, and N. D. Sagaidak-Chernyak, "Experimental Lung Cancer Induced with Radioactive Compounds ^{32}P, ^{198}Au, and ^{59}Fe," Acta. Unio. Int. Contra Canc. 19:684 (1963).

22. T. A. Kotchetkova and G. W. Avrunina, "Changes in Lungs and Other Organs After Intratracheal Dosage With Some Radioisotopes (^{24}Na, ^{32}P, ^{198}Au)," Arch.

23. C. L. Yuile, H. L. Berke, and T. Hull, "Lung Cancer Following Polonium-210 Inhalation in Rats," Rad. Res. 31:760 (1967).

24. R. W. Wager, N. L. Dockum, L. A. Temple, and D. H. Willard, "Toxicity of Radioactive Particles - Intratrachial Injection of Radioactive Suspensions," Hanford Works Annual Report, 1955, HW-41500:61 (1956).

25. J. K. Scott and R. Thomas, "Induction of Pulmonary Tumors Following Intratracheal Injection of Polonium-210," Univ. of Rochester Atomic Energy Quarterly Report Jul-Sept. 1957:30.

26. J. B. Little, B. N. Grossman, and W. F. O'Toole, "Respiratory Carcinogenesis in Hamsters Induced by Polonium-210 Alpha Radiation and Benzo-(α) Pyrene. Morphology of Experimental Respiratory Carcinogenesis," Conf. 700501:383 (1970).

27. B. N. Grossman, J. B. Little, and W. F. O'Toole, "Role of Carrier Particles in the Induction of Bronchial Cancer in Hamsters by ^{210}Po Alpha Particles," Rad. Res. 47:253 (1971).

28. L. J. Leach, E. A. Maynard, H. C. Hodge, J. K. Scott, C. L. Yuile, G. E. Sylvester, and H. B. Wilson, "A Five-year Inhalation Study With Natural Uranium Dioxide (UO_2) Dust I. Retention and Biologic Effect in the Monkey, Dog, and Rat," Health Phys. 18:599 (1970).

29. G. P. Antonchenko, N. A. Koshurnikova, and E. R. Lyubchansky, "Morphological Changes in the Lungs of Rats After Inhalation of Large Doses of Soluble Plutonium-239 Compounds," Radiobiology 9:97 (1969).

30. L. A. Buldokov, E. R. Lyunchansky, Yu. I. Maskalev, and A. P. Nifatov, "The Problems of Toxicology of Plutonium-239," Atom Publications Moscow (1969); also USAEC Document LF-TR-41 (1970).

31. W. J. Clarke, J. F. Park, J. L. Palotay, and W. J. Bair, "Bronchioloalveolar Tumors of the Canine Lung Following Inhalation of Plutonium Particles," Am. Rev. Resp. Dis. 90:963 (1964).

32. J. F. Park, W. J. Bair, W. J. Clarke, and E. B. Howard, "Chronic Effects of Inhaled Plutonium Dioxide in Dogs," Pacific Northwest Laboratories Annual Report 1966 BNWL-480:57 (1967).

33. J. F. Park, W. J. Bair, and R. H. Busch, "Progress in Beagle Dog Studies With Transuranic Elements at Battelle Northwest," Health Phys. 22:803 (1972).

34. J. F. Park, W. J. Bair, E. B. Howard, and W. J. Clarke, "Chronic Effects of Inhaled $^{239}PuO_2$ in Beagles," Pacific Northwest Laboratories Annual Report 1968 BNWL-1050:3.3 (1970).

35. C. L. Saunders, "Carcinogenicity of Inhaled Plutonium-238 from Crushed Microspheres," Pacific Northwest Laboratories Annual Report 1972, Part 1 BNWL-1750:28 (1973).

36. R. G. Thomas, R. O. McClellan, R. L. Thomas, T. L. Chiffelle, C. H. Hobbs, P. K. Jones, J. L. Manderly, and J. A. Pickrell, "Metabolism, Dosimetry, and Biological Effects of Inhaled ^{241}Am in Beagle Dogs," Health Phys. 22:863 (1972).

37. T. L. Levdik, V. K. Lambert, L. A. Buldakov, E. R. Lyubchansky, and V. M. Pesternikov, "Biological Effectiveness of Neptunium-237," Radiobiology 11:160 (1971).

38. S. Koletsky and G. E. Gustafson, "Whole-Body Radiation as a Carcinogenic Agent," Cancer Res. 15:100 (1955).

39. H. Cember, J. A. Watson, and T. Grucci, "Pulmonary Radiation Effects as a Function of Absorbed Energy Distribution," Am. Indust. Hyg. Assoc. Quart. 17:397 (1956).

40. J. Maisen, P. Maldague, A. Dunjic, Pham Hong-Que, and H. Maisen, "Carcinogenic Effect of a Single Dose of X-Rays in the Rat," Proceedings of the Second U.N. International Conference on the Peaceful Uses of Atomic Energy, Geneva (1958) Vol. 22:134.

41. T. J. Castanera, D. C. Jones, D. J. Kimeoldorf, and V. J. Rosen, "The Influence of Whole-Body Exposure to X-Rays or Neutrons on the Life-Span Distribution of Tumors Among Male Rats," Cancer Res. 28:170 (1968).

42. T. J. Castanera, D. C. Jones, D. J. Kimeldorf, and V. J. Rosen, "The Effects of Age at Exposure to a Sub-Lethal Dose of Fast Neutrons on Tumorigenesis in the Male Rat," Cancer Res. 31:1543 (1971).

43. A. J. DeVilliers and P. Gross, "Morphological Changes Induced in the Lungs of Hamsters and Rats by External Radiation (X-Rays)," Cancer 19:1399 (1966).

44. P. Gross, E. A. Pfitzer, J. Watson, R. deTreville, M. Kaschak, E. Tolker, and M. A. Babycek, "Experimental Carcinogenesis: Bronchial Intramural Adenocarcinomas in Rats from X-Ray Irradiation of the Chest," Cancer 23:1046 (1969).

45. National Committee on Radiation Protection, "Permissible Dose From External Sources of Ionizing Radiation," Natl. Bur. Stds. HB 59, Sept. 24, 1954.

46. National Committee on Radiation, "Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water," Natl. Bur. Stds. HB 52, Mar. 20, 1953.

47. National Committee on Radiation Protection, "Maximum Permissible Radiation Exposures to Man," NBS Tech. News Bul. 41, 17 (Feb. 1957).

48. National Council on Radiation Protection and Measurement, "Basic Radiation Protection Criteria," NCRP Report No. 39, NCRP Publication, Washington, D.C. Jan. 15, 1971.

49. G. E. McMurtrie (Secretary), "Permissible Doses Conference held at Chalk River, Ontario (Sept. 1949)," Report RM-10 (May 1950).

50. International Commission on Radiological Protection, "Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965)," ICRP Publ. 9. Pergamon Press, Oxford, 1966.

51. International Commission on Radiological Protection, "Radiosensitivity and Spatial Distribution of Dose, Reports Prepared by Two Task Groups of Committee 1 of the International Commission on Radiological Protection," ICRP Publ. 14 (Pergmon Prsss, Oxford, 1969.

52. J. E. Ballou, D. K. Craig, J. F. Park, H. A. Ragan, and C. L. Sanders, Pacific Northwest Laboratory Annual Report for 1972, Vol. 1, Part 1 BNWL-1750 Pt I, April 1973.

53. Passonneau, et al., "Carcinogenic Effects of Diffuse and Point-Source Beta Irradiation on Rat Skin: Final Summary," AEC Document ANL-4932, 1952.

54. P. N. Dean and W. H. Langham, "Tumorigenicity of Small Highly Radioactive Particles," Health Phys. 16:79-84 (1969).

55. R. E. Albert, F. J. Burns, and R. D. Heimbach, "The Effect of Penetration Depth of Electron Radiation on Skin Tumor Formation in the Rat," Rad. Res. 30:515-524 (1967).

56. A. M. Brues, "Comparative Chronic Toxicities of Radium and Plutonium," Argonne National Laboratory Quarterly Report (Fe., March and April 1951) ANL-4625, p. 106-124.

57. Federal Radiation Council, "Guidance for the Control of Radiation Hazards in Uranium Mining," Report No. 8 Revised, U.S. Government Printing Office, Washington, D.C., Sept. 1967.

58. F. E. Lundin, Jr., J. K, Wagoner, and V. E. Archer, "Radon Daughter Exposure and Respiratory Cancer Quantitative and Temporal Aspects," National Inst. for

Occ. Safety and Health and National Inst. of Environ. Health Sciences, Joint Monograph No. 1 (1971).

59. W. H. Langham, "Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads," Lawrence Radiation Laboratory, Document UCRL-50639, April 1969.

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A Review of the Natural Resources Defense Council Petition Concerning Limits for Insoluble Alpha Emitters

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A REVIEW OF THE NATURAL RESOURCES DEFENSE COUNCIL PETITION CONCERNING LIMITS FOR INSOLUBLE ALPHA EMITTERS

by

J. W. Healy, C. R. Richmond, and E. C. Anderson

The interpretations of the potential effects of insoluble alpha-emitting particles in the lung, as described in the document supporting the Natural Resources Defense Council petition of February 14, 1974, are reviewed in light of present evidence. It is concluded that the theories upon which the proposal is based are not in accord with the evidence and that the theories do not correctly predict the outcome of experiments actually using such particles.

I. INTRODUCTION

On February 14, 1974, the Natural Resources Defense Council (NRDC) submitted a petition to the U. S. Atomic Energy Commission (AEC) and the Environmental Protection Agency (EPA) requesting that they amend their standards as said standards apply to insoluble particles of plutonium and other alpha-emitting "hot particles."[1] (The terminology of "hot particles" is that of the NRDC and refers to particles which contain more than 0.07 pCi of insoluble alpha emitters.) In support of their petition, the NRDC included a report by Drs. Arthur R. Tamplin and Thomas B. Cochran which provides the basis for the proposal.[2]

The question of the possible biological effects from radioactive particles which can irradiate small quantities of tissue to large physical doses has been of interest to the scientific community and radiation protection groups for many years. In several studies involving large extrapolations of available data, an enhanced tumor production from numbers of such particles has been predicted.[3,4] However, the tenuous nature of the evidence and the indirect methods of arriving at the answer have, in general, prevented these predictions from gaining acceptance in the biomedical community, and the standards have continued to be based upon other evidence.

In view of the current interest in this question and the somewhat unusual procedure of submitting the proposal through legal channels rather than through scientific review, it was felt that an examination of the allegations and conclusions would be useful in informing those concerned as to the validity of the bases. This report, therefore, reviews in some detail the basis for the NRDC proposal and briefly indicates the experimental information available on the question.

II. THE CONTENTION

While it is difficult to condense the arguments of an author without running the risk of changing his meaning or emphasis, we will briefly summarize in this section, for the orientation of the reader, our understanding of this contention. However, it is urged that reference be made to the original document[2] to obtain their full viewpoint. It is our impression that the following are the key technical items upon which the petition is based.

1. The responsible standards-setting organizations, the International Commission on Radiological Protection (ICRP) and the National Council on Radiation Protection (NCRP), have given no guidance on the question of localized radiation dose resulting from an alpha-emitting particle.

2. In Tamplin and Cochran's words, the Geesaman hypothesis indicates that "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}." The Geesaman hypothesis was published in 1968 in a Lawrence Radiation Laboratory report[5] (now Lawrence Livermore Laboratory) but was never published in the open literature. In this theory, Geesaman relied upon a theoretical

investigation of the dose distribution around a particle in the lung and estimated sizes above which cell death would result in no cancer. In an addendum,[6] he used data on the induction of tumors in rat skin and the relation of these to atrophied hair follices as a result of radiation. Perhaps his conclusions can best be stated by quoting from the conclusions section of the addendum.[6]

"Summing up, intense radiation exposure of mammalian skin and lung tissue commonly results in cancers. Tissue injury and disturbance are a primary consequence of intense radiation insult, and are observed in association with carcinogenesis. Albert has exhibited a simple proportionality between skin carcinoma and atrophied hair follicles. No general description of precarcinogenic injury exists, but in a crude sense the available observations are compatible with the idea of an injury-mediated carcinogenesis. Cancer is a frequent instability of tissue. Since tissue is more than an aggregate of cells, and has a structural and functional unity of its own, it would not be surprising if some disrupted local integrity, a disturbed ordering, comprises a primary pathway of carcinogenesis. The induction of sarcomas with inert discs of Mylar, cellophane, Teflon, and Millipore is indicative that such a mechanism exists. Presumably mitotic sterilization is an important factor in any carcinogenesis mediated by radiation-induced tissue injury. The functional relation of this factor to the carcinogenic response may be quite different from a linearity in the surviving mitotic fraction.

"While regrettably unquantitative, the hypothesis of an injury-mediated carcinogenesis is suggestively descriptive. If the respiratory zone of the lung contains a structure analogous to the rat hair follicle, and if a radioactive particulate deposited in the respiratory zone has the capacity to disrupt one or more of these structures and create a precancerous lesion, then cancer risks of the order of 10^{-3} and 10^{-4} per particle can be expected for burdens much less than 10^{8} particles."

Again, however, the reader is urged to review the original document to obtain the full argument.

3. In deriving present limits for alpha emitters in the lung, Tamplin and Cochran indicate that no factor was included to account for the non-uniform distribution of radiation in the lung as is done in the ICRP and NCRP formulation of bone dosimetry. It was pointed out that such a distribution factor could be defined by:

$$DF = \frac{\text{number of cancers (non-uniform distribution)}}{\text{number of cancers (uniform distribution)}}$$

"Since direct experimental evidence are not available....,"[2] they chose to attempt a definition of this factor from the Geesaman hypothesis including the quantitative derivation of probability of cancer induction derived from rat skin hair follicles.

4. As regards human data, they discuss the case of a skin lesion from plutonium embedded in the epidermis; a purported case of synovial sarcoma due to contamination during handling of a carboy; the Los Alamos cases which date back to the Manhattan Project and are dismissed as not having received particles of sufficient activity; and a group of exposed Rocky Flats workers which are, again, dismissed on the grounds that the time since exposure has not been long enough for cancer to develop. In the first case, the statement of the pathologist that "their similarity to known precancerous epidermal cytological changes, of course, raised the question of the ultimate fate of such a lesion....."[7] seems to be interpreted as proof that cancer would have developed. In the second case, a series of circumstantial inferences is quoted to "prove" that the cancer was due to plutonium.

5. Since the Geesaman hypothesis,[6] as given in his earlier reports, seems to have no dependence of effect on radiation dose or amount of activity per particle but states that the effect is due to the number of particles, Tamplin and Cochran modify this hypothesis by establishing a critical particle size below which the effect will not be noted (i.e., a threshold?). Their basis is given by the following quotations:[2]

"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] ..... This suggests

that a particular level of tissue damage must occur before this unique carcinogenic response occurs. The experiments of 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. .....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."

Reference 55 in the above quotation is to Albert *et al.*;[8] reference 56 to Laskin *et al.*;[9] and reference 57 to Geesaman.[5]

6. From their definition of a "hot particle," Tamplin and Cochran derived values for occupational exposure by comparing the risk of lung cancer from dose rates of 15 rems/yr to the lung to assumed risks from particles of 1/1000, 1/2000, and 1/10 000 per particle. They then recommended as ".....a somewhat arbitrary compromise and ..... not the most conservative value....."[2] the use of a risk of 1/2000 per hot particle in determining the maximum permissible lung burden for insoluble alpha-emitting radionuclides in hot particles. From this they arrived at a value of 2 particles or 0.14 pCi for a reduction in the maximum permissible lung burden by a factor of 115 000.

For individual members of the public, a value of 0.2 hot particle, while recognizing the disparity in risk occasioned by a fractional number of particles per person, is recommended along with a value of 0.07 hot particle as the average lung burden for members of the public. Limiting values for soil contamination and accidents are also derived by similar considerations.

III. PARTICLES AND RADIATION DOSE

The origin of the NRDC proposal lies in the very non-uniform radiation dose to the tissue surrounding a radioactive particle. For this reason, we will initially provide some description of the nature of this non-uniformity and the application of the concept of radiation dose to biological problems.

A. The Radiation Dose around a Particle

The unique feature of a particulate source of radioactive material (particularly for an alpha emitter because of the short range of the alpha particle) is the rapid change in dose or dose rate as one moves away from the particle and the relatively small amount of tissue exposed to the dose. If one ignores the details of the Bragg curve, the dose in a uniform density tissue at reasonable distances from the particle follows the inverse square law for alpha particles. For the lung, the presence of the alveoli and air passages results in varying degrees of absorption, depending on the actual mass of tissue encountered, so that the inverse square relation is distorted by the varying absorption and the dose pattern may be non-symmetrical. Geesaman[5] has approximated this dose pattern by assuming a cubical lattice representing the air spaces in the human, while Anderson and Dean[10] have used micrographs and computer programs to calculate the pattern for the hamster.

The effect on the calculated dose of varying the volume over which energy deposition is averaged is shown in Fig. 1. (This is not the radial dose distribution, which extends only from the particle surface to the maximum alpha range and for which the

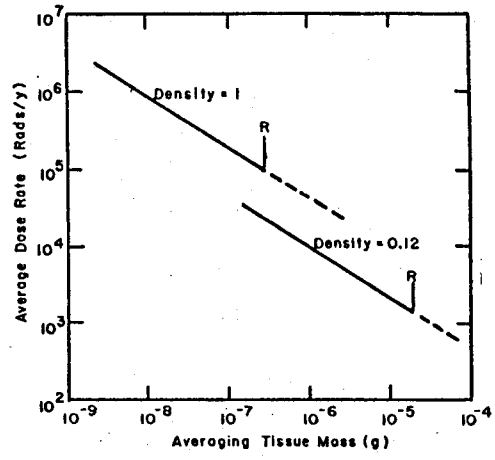

Fig. 1. Calculated dose rate averaged over different distances from a 0.28-pCi particle vs the tissue mass involved in averaging. R represents the range of the particle.

abscissa would be distance.) The calculations are for a particle of 0.28 pCi of ^{239}Pu in tissues of two different densities. It is assumed that the energy loss per unit path length is constant so that the alpha particle deposits energy uniformly along its path. The range in unit density tissue is taken as 40 μm,[11] with the range for other tissues scaled to the tissue density. The doses given are annual doses averaged over the volume of tissue given. The curve indicated as density = 1 is calculated for unit density tissue, and the curve for density = 0.12 is for a uniform tissue having a density of 0.12, corresponding to the average bulk density of Geesaman's lung model at half inflation.[5] No correction was made for the self-absorption in the particle, although this should be negligible for these small PuO_2 particles in comparison to the errors caused by other assumptions. The annual doses are given both in rads which can be converted to rems by the conventionally used quality factor of 10.[12] It must be emphasized that this conversion to rems is particularly uncertain for this case, since there are no data which can be used to assess the relative effects of alpha radiation and the reference radiation in this particular geometry of irradiation.

Figure 1 is intended to indicate the wide variation in dose which can be calculated by different assumptions of averaging volume. Even here we have minimized the dose to individual cells by plotting the average over the volume to the fraction of the range considered. The dose to an individual cell at differing distances varies even more than this average.

We have not considered in this calculation the photon dose from x rays or infrequent gamma rays from either ^{238}Pu or ^{239}Pu, since the focus of the discussion is on alpha-particle effects. It should be noted, however, that these photons are more penetrating and will result in lower doses at distances beyond the range of the alpha particles.

B. Limitations on the Usefulness of Radiation Dose

Calculations such as those given in the preceding section are interesting and have been made by various individuals for many years. The question remains as to their usefulness and meaning in assessing a biological problem.

The primary use of radiation dose, in practice, is as a physical parameter to be used in correlating and extrapolating experimental data on biological effects on an empirical basis. Thus, the present limits for radiation exposure are based upon observations of effects in humans for whom the dose has been estimated. There is no *a priori* basis for assigning an effect to a given dose, since our understanding of the basic mechanisms of radiation carcinogenesis and the influence of cellular interactions is completely inadequate. Thus, radiation doses are meaningful only when related to empirical data on the outcome. As a corollary, the further one extrapolates from the experimental conditions under which the dose-effect relationship is measured, the greater the uncertainty. Thus, predicting the behavior of the effects on individual cells or aggregates of cells in a functioning organ from *in vitro* studies in cell culture is a very wide extrapolation which ignores the very different environment of the cells in the organ and the potential interactions which occur among cells. (Such *in vitro* studies, however, are of great interest for other reasons, such as studies of the mechanisms of damage at the cellular level.) Similarly, extrapolating from the effects of a partial organ irradiation to a full organ (or vice versa) can lead to a misestimate. It is for these reasons that most scientists have refrained from using dose calculations, such as those given earlier, to arrive at conclusions as to the effect of radioactive particles but have preferred to depend upon experimental evidence which bears more directly on the actual conditions.

A further factor of importance in the use of physical dose as a correlating concept is the exact method of expression of dose. That is, if a correlation with effect is established using one method of calculating the dose, it is not valid to apply this correlation if another basis for dose calculation is chosen. As an illustration which, incidentally, seems pertinent to the problem at hand, Vaughan[13] indicates that 90% of the ionization along an alpha particle track formed in unit density tissue is contained in a cylinder of 0.01-μm radius with the axis of the cylinder along the track. For an alpha particle with 5.15-MeV initial energy, the range is about 40 μm. The average dose to this limited volume, therefore, is about 6×10^6 rads, with even higher average doses for smaller radii and at the peak of the Bragg curve. For a 1000-g

organ of unit density tissue, the current occupational limit of 1.5 rads (15 rems) per year, even assuming homogeneous distribution of the alpha tracks, means that about 0.25 mm^3 of tissue is irradiated to doses above 4×10^6 rads, or if a dose of 1000 rads were chosen, a volume of some 1500 mm^3. Since unit density tissue was chosen for this illustration, the results do not compare with those for a particle using the Geesaman model. However, it is clear that even a "homogeneous" distribution of alpha radiation through a body of tissue results in considerable non-uniformity in dose distribution. Further, for the example chosen, one could express the limits as 15 rems to the 1000 g of tissue or as a limitation on the volume of tissue exceeding a given dose. For example, no more than 0.3 mm^3 of tissue shall exceed 4×10^7 rems or no more than 1500 mm^3 shall exceed 10 000 rems. Although the latter methods of expression involve numbers that are frighteningly high in more normal context, all three methods define the same total energy deposition. However, note that it would be highly improper to apply the 15-rems value to the dose along the track just as it would be improper to apply the dose along the track to the dose arising from an assumption of uniform tissue distribution.

A specific point in the Tamplin-Cochran dissertation[2] is the use of the "distribution factor" (DF) in calculating the dose in rems for internal emitters and is supported by the fact that a DF of 5 is used in calculating the dose for bone. They then indicate that a DF should be applied to lung. However, it must be realized that a dose calculation was not used to arrive at the present maximum permissible body burden for plutonium.[14] Instead, a comparison of biological effects (primarily on bone) was made between plutonium and radium. On the basis of these data, it was deduced that plutonium in the body is 2.5 times as harmful as radium on a microcurie basis. Since the maximum permissible body burden for radium had been established from studies of humans as 0.1 μCi, the maximum permissible body burden for plutonium was set at 0.04 μCi.

The dose considerations quoted by Tamplin and Cochran arose in an attempt to use these experiments, and others with strontium, to provide a physical formulation of the results which could be used for extrapolation to other bone-seekers. For the purpose of such calculations, it was *assumed* that radium was uniformly distributed in bone. Further, it was assumed that 90%, or essentially all, of the plutonium in the body was in bone. Since the individual plutonium disintegration liberated about half of the alpha energy of one disintegration of radium with its accompanying daughter products, the foregoing damage ratio of 2.5 on a microcurie basis becomes 5 on an average energy-delivered (dose) basis.

The key to this comparison lies in the assumptions. We know, for example, that radium is *not* uniformly distributed in bone. In fact, if anything, it is more non-uniformly distributed than plutonium. However, the deposition sites are different from those of radium so that the plutonium affects a different, and more sensitive, portion of the bone. One could presumably eliminate the confusion caused by the distribution factor by redefining the critical organ to include only the sensitive portion of the bone and comparing the dose to this region from plutonium and radium. We also note in passing that the more recent examination of the distribution of plutonium in animals indicates that only about 40 to 50% of the plutonium is in the bone. If this were true in the comparison animals (as seems likely), then the actual distribution factor for bone calculations should be 10 rather than 5.

We have introduced this rather lengthy discussion on bone dose calculations to indicate, once again, the difficulty in applying dose calculations and concepts derived for one use to a different problem without full understanding of what was done. In the above case, the salient feature is that radium is non-uniformly deposited so that some sections of the bone receive doses orders of magnitude greater than others.[15] The distribution factor is not intended to indicate greater localized dose from plutonium but, rather, that the distribution in bone is different from that of radium on a gross basis.

C. Previous Guidance

An interesting point in the Tamplin-Cochran document is: "It is important to recognize that the ICRP has given no guidance with respect to non-uniform irradiation of the lung by insoluble alpha-emitters such as insoluble plutonium particles." They then quote one of many statements made by the ICRP[16] and other groups which indicate that there is no clear evidence as to whether the effect of the

non-homogeneous dose is greater or less than that of the homogeneous dose. They interpret this statement as: "In effect, the ICRP is saying that there is no guidance....." A quote from the NCRP follows concerning the significant volume of tissue which concludes: ".....For example, if a single particle of radioactive material fixed in either lung or lymph node might be carcinogenic, the averaging of dose either over the lung or even over one cubic centimeter may have little to do with this case."[12]

While we do not feel that it is useful to quote such bodies at length, there is evidence that the problem has been considered since the early days of the derivation of limits. One of the earlier statements arose from a Tri-Partite Conference in 1949[17] at which scientists from the United Kingdom, Canada, and the United States were arriving at the conclusions which were later applied by many of these same people in the ICRP and NCRP recommendations: "In relation to the possible pathological effects of radioactive particles in the lungs, Dr. Hamilton pointed out that the cells in the immediate neighborhood of a dust particle containing 1 or 2% of plutonium would be subjected to a dose of about 400 r/day. The general opinion which emerged from the discussion was that the carcinogenic effect per unit volume is probably considerably less for the irradiation of small masses of tissue than for large." This conclusion undoubtedly affected the practice of calculating dose as the average dose to an organ at that time and comprises definite guidance on the handling of such problems. However, the matter did not rest there, since several national and international groups continued investigation from that time to the present, with frequent statements as to the lack of definitive information.[16,18-21] However, in spite of the indications of periodic questioning and reviews, there has been no revision in the practices which they recommended of using the average organ dose as a basis for establishing standards.

From the above, it seems clear that the ICRP and the NCRP *did* furnish guidance on the pertinent dose to be used for standards-setting: the use of an average calculated dose to an organ, with full recognition of the non-uniform distribution of dose around the particle. In spite of numerous reviews of the question over the intervening years, they have reiterated this guidance by not changing it. It is difficult to support any claim of no guidance in view of this record on the part of bodies which have traditionally been in the forefront of recognizing potential problems (i.e., genetic effects) and providing generally conservative recommendations.

One recommendation of the NCRP[12] (while perhaps not completely applicable to the particle case as is shown by their example situation quoted earlier) is of interest when combined with Geesaman's estimate of a particle size above which cancer would not be expected due to cell death.[5] The NCRP statement is, "Simplifications in practice hinge largely on reporting a single representative protection dose for a limiting organ system even when the actual irradiation is grossly non-uniform. The representative dose is taken as the highest that can be obtained by averaging over a prescribed significant volume. The implication of this concept ..... is that any redistribution of a given dose within such a volume does not materially alter the radiation response. It is usually assumed that the 'significant volume' should be of the order of one cubic centimeter. This will be grossly conservative under most circumstances, and in special situations use of a larger volume is justified." It is not clear why the NCRP recommended a significant volume rather than a significant mass, since this results in averaging over a smaller mass in the lung than in other tissues due to the density difference.

However, if we calculate the dose over 1 cm^3 of lung tissue with an average density of 0.12 g/cm^3 for the "hot particle" of 0.07 pCi derived by Tamplin and Cochran,[2] we obtain a dose of only 0.055 rad or 0.55 rem per year. Thus, one would require an activity of 1.9 pCi to reach the limit of 15 rems per year for this single cubic centimeter of tissue (or an activity of 15 pCi for a single cubic centimeter of unit density tissue). Geesaman[5] quotes an activity for a 1-μm-diameter particle of ^{238}Pu as 60 pCi and arrives at a conclusion that ".....unless the source size, s, is smaller than or of the order of 0.25 μ the yearly flux will be lethal for all epithelial populations in the exposed volume. The source size condition will only be slightly less stringent for endothelial populations s < 0.35 μ." The implication of the above is that no cancer will

develop for particles larger than those described since the cells are killed. According to the constants used by Geesaman, a 0.25-μm particle would have an activity of about 2.5 pCi, which compares with the 2 pCi to give 15 rems to one cubic centimeter of tissue. Thus, if Tamplin and Cochran had chosen to use this available NCRP guidance along with the Geesaman study, their conclusions would have been considerably different.

IV. THE GEESAMAN HYPOTHESIS

The Geesaman hypothesis was published as a Lawrence Radiation Laboratory (now Lawrence Livermore Laboratory) report in February 1968,[5] with an addendum in October 1968[6] containing the quantitative estimates of cancer production. This work has never been published in the open scientific literature but remains an unreviewed and unrefereed study.

Since his conclusions seem to be based primarily upon the studies of follicular cancer produced in rat skin, we quote below those sections of the report in which he uses these data with his references and footnotes deleted.

"Albert's study of radiation-induced carcinoma in rat skin gives some quantitative description of a high-dose carcinogenic situation. Since such descriptions are rare, and since Albert's results have implications to risk analysis in general, his experiment is outlined here.

"A skin area of 24 cm^2 was exposed to electron radiation with various depths of maximum penetration. ..... In all cases the response scale at sufficiently high doses was large, ~1 to 5 tumors per rat at 80 weeks after exposure. It was noted by Albert that when the dose was normalized to a skin depth of 0.27 mm, the three response curves became continuous. Since this 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 observation that most of the tumors are similar to hair follicles, and that in the nonulcerogenic dose range the number of tumors per rat was in nearly constant ratio (1/2000 to 1/4000) with the number of atrophied hair follicles..... Thus the carcinogenesis in this experiment was remarkably correlated with the dose to and the 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 R, but not at doses of 2300 R. The reduction, however, was again consistent with the reduction in damage as characterized by atrophied hair follicles.

"For perspective it is valuable to relate these observations to cellular descriptions. Carcinogenesis in Albert's experiment is maximum in the neighborhood of 2000 R. It is well documented *in vitro* and to a lesser extent *in vivo* that the fraction of mitotically competent cells as measured by clonal formation decreases in a nearly exponential fashion with the dose. From these results a surviving mitotic fraction of approximately 10^{-5} would be expected in a population of germinal epithelial cells exposed to 2000 R. Even in this pre-ulcerative dose regime the cell population suffers severe mitotic injury. It is significant that Albert's dose response curves show no simple relationship with the surviving fraction of mitotically competent epithelial cells. There is certainly no exponential decrease of the response in the neighborhood of D_{37}, and, in fact, the tumorigenesis is maximum in a dose region where the population of mitotically competent cells should be initially depleted by about 5 orders of magnitude.

"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 particular skin structure."

The reasoning leading from this information, plus a discussion of other experiments with high doses and particle sources leading to the conclusion (quoted earlier) of a cancer risk of 10^{-3} to 10^{-4} per particle, is not given but is presumed to result from the correlation with atrophied hair follicles from Albert's experiments.

There is a similarity between this work and the theory propounded by Virchow in 1863 that the cause of cancer is chronic tissue damage.* This

*We are indebted to Dr. Roy E. Albert, New York University, for this line of reasoning.

theory was disproved by experiments which showed that cancer can be produced by very potent substances that vary widely in their capacity to cause cancer, whereas many agents which cause damage do not cause cancer. Thus, while there is a frequent association between tissue damage and cancer, there are types of cancer and types of damage for which no association exists.

There are several aspects of the data from the skin experiments used by Geesaman, as well as information published later from the same series of experiments, which should force some modification of the proposal but are not included in the Tamplin-Cochran document. These and their implications for the Geesaman hypothesis are reviewed below.

A. Type of Tumor

In a 1961 paper, Albert *et al.* first explored the tumors resulting from irradiation of rat skin with ^{91}Y beta rays.[22] Two strains of rats were used with the tumor types and frequencies as given in Table I. They indicate the Holtzman strain to be similar to the Sprague-Dawley strain, but the animals were considerably older (~ 40 weeks compared to ~ 20 weeks for the Sprague-Dawley).

A variety of tumor types were obtained. In Fig. 2, we have plotted the dose-incidence curve

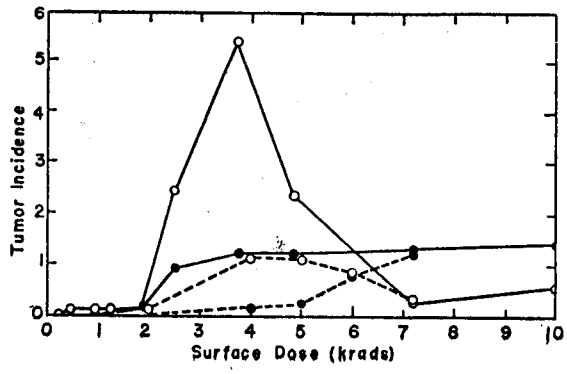

Fig. 2. Tumor incidence per animal vs surface dose of electrons: (——) Sprague-Dawley strain; (---) Holtzman strain; (-O-) adnexal tumors; and (-●-) other tumors.

for both strains for the predominant tumor type (follicle and sebaceous or "adnexal") and the sum of all other types. The incidences were corrected for the unidentified tumors by assuming these to arise in the same proportion as the identified ones. It is of interest to note the wide disparity between the response curves of the adnexal tumors and those of other types, as well as the disparity between the curves for the two strains (whether due to strain or age is not determined). Since the remainder of the experiments focused upon the adnexal tumors, with

TABLE I

TUMOR TYPES AND FREQUENCIES FROM IRRADIATION OF RAT SKIN

	Dose (rads)									
Sprague-Dawley Strain	10 000	7200	4870	3750	2500	1900	1225	950	470	230
Initial number of rats	12	12	13	14	15	10	15	23	25	24
Epidermoid carcinoma	9	11	6	5	5	1	0	0	0	0
Adnexal tumor	5	2	26	62	23	3	1	3	3	1
Connective tissue tumor	2	1	0	1	1	1	1	0	1	0
Squamous papilloma	0	0	6	6	3	0	0	0	0	0
Cysts	0	0	1	2	0	0	0	2	1	1
No pathologic examination	8	5	8	17	19	0	2	0	0	0
Total	24	19	47	93	51	5	4	5	5	2

Holtzman Strain	7200	6000	5000	4000	2000	1000	500
Initial number of rats	9	10	8	11	16	20	50
Epidermoid carcinoma	7	6	1	2	0	0	0
Adnexal tumor	2	7	6	11	2	0	1
Connective tissue tumor	0	0	0	0	0	0	1
Squamous papilloma	2	0	0	0	0	0	0
Cysts	0	0	0	0	0	0	0
No pathologic examination	3	4	3	3	0	0	1
Total	14	17	10	16	2	0	3

data on other types discarded, the information is aimed at a very specific tumor type even for the organ considered: rat skin.

B. Volume of Irradiated Tissue

As was discussed earlier, the extrapolation from one condition of irradiation or method of expressing dose to another must be done with great caution and a full understanding of the parameters involved. How, then, do the conditions of the rat skin experiments compare with those of the particle irradiation?

The particle doses typically involve tissue quantities of tens of micrograms (see Fig. 1). In the rat skin experiments, areas ranging from about 5 to 30 cm^2 were used with depths from about 0.4 to about 1.5 mm. Thus, the tissue volumes ranged from about 0.2 to about 5 cm^3 or, for unit density tissue, 0.2 to 5 g.[8,23-25] This is an extrapolation in tissue volume on the order of 10^3 to 10^5.

There are several observations in the rat skin experiments which are pertinent to the validity of extrapolation. In one series of irradiations, exposures were made through two grids which provided 1-mm-wide bars of irradiation area with one grid masking all except a third of the area and the other all except a sixth of the area.[23] In addition, a mask (sieve) with circular holes which permitted an exposed area of a third of the uniform area was used. From these data, it was noted that the response with the smaller areas was lower even though the total dose to the area (expressed in gram-rads) was in the vicinity of the uniform dose required to produce the maximum incidence of adnexal tumors. In other words, the delivery of a specific amount of energy to a given overall area of skin resulted in fewer tumors when the energy was delivered at higher doses but to smaller subareas. Geesaman correctly points out that this suppression occurred at 1700 rads but not at 2300 rads.[6] However, the 2300-rads value for the uniform irradiation is well past the dose of maximum tumor induction, and there has been a significant drop in the incidence for this condition. Therefore, it is difficult to attribute this effect to other than the oversaturation of the response. Albert *et al.* conclude from this work: "The experiments reported here indicate that, in a limited dose range, the non-uniform radiation pattern has the effect of reducing both chronic hair follicle damage and tumor formation."[23]

In the studies of the association between hair follicle damage and tumor formation, Albert *et al.* noted that the damage to the hair follicles across the irradiated area was not uniform, with the major damage occurring at the center of the area and considerably lower damage at the edges.[24] From other data, it appears that the dose across the area was reasonably uniform and that the effect was due to something other than non-uniform dose. From this and the preceding work, Albert *et al.*[24] conclude: "Two observations indicate the importance of the size of the irradiated area on the magnitude of hair damage: (1) the follicles along the margin of the irradiated area are relatively uninjured compared to the follicles in the center of the irradiated area..... (2) there is a suppression of follicle damage when the irradiation is delivered in a sieve pattern..... These observations strongly suggest that the pathogenic mechanisms for the development of both irreparable hair follicle damage and skin tumors depend upon both the dose and the *amount of skin irradiated*" (emphasis added).

Thus, the data and conclusions in the papers used by Geesaman to justify his work (and quoted by Tamplin and Cochran[2] as "biological evidence" supporting their contentions) strongly suggest that extrapolations to smaller tissue volumes may not be legitimate.

C. Species Dependence

We have alluded earlier to the difference in response curves for skin tumor formation occasioned by either the strain difference or the age of the rats used. In a paper subsequent to the Geesaman proposal, Albert *et al.*[25] repeated some of their studies using mice as the experimental animal, since it had been noted that the response of mouse skin is different, with relatively few tumors and most tumors being epidermoid carcinomas rather than adnexal tumors.

The results of this experiment confirmed the previous findings that adnexal tumors, noted as the most probable outcome in rats, were rare in mice and that the total number of tumors produced in mice was only 15 to 20% of those in rats for comparable conditions. The lack of adnexal tumors was attributed to the fact that the hair follicles in the mouse are more radiosensitive than those in the rat. As a result, little follicle atrophy is noted in the

mouse -- either the follicles remain intact or they are destroyed.

The results of this experiment indicate clearly the difficulties of applying results from one organ to another. Even though skin was the target in both cases, the differences in structure between rat skin and mouse skin caused a completely different outcome upon irradiation. The outcome upon comparison to a different organ such as the lung, where follicle structures or functions do not even exist, would seem to make the final conclusion by Geesaman one of sheer speculation.

D. Volume of Follicle Irradiated

In the original studies of rat skin response, Albert *et al.* used electron beams which had an approximate linear decrease in dose with depth.[8,22-24] The relation between dose at the tip of the hair follicle, lying at a depth of about 3 mm, was established by noting that the tumor incidence curves for electrons of various penetrations coincided when the dose was expressed as the dose at a depth of 0.3 mm.[8] However, it can be noted that the entire follicle was irradiated to this dose or greater.

To test the dependence of the effect of doses to various portions of the follicle, Heimbach *et al.* used the Bragg peak of alpha radiation produced by a cyclotron.[26] The energy of a 37-MeV alpha beam was adjusted by the use of aluminum absorbers in the experiments so that the Bragg peak fell at depths of 0.12, 0.35, and 0.55 mm. Since the Bragg peak can produce dose rates up to 5 times that along the early portion of the track, this enabled investigation of doses delivered to various parts of the follicle. The results indicated that the response curves coincided when the dose was expressed as minimum dose to any point along the hair follicle. The tumor types were identical with those found with electrons, and there was once again a correlation between tumors and atrophied hair follicles, with the ratio between tumors and atrophied hair follicles of about 1/9000.

From this experiment, the authors concluded that the entire hair follicle must be irradiated to produce tumors. The minimum penetration alpha radiation used did not irradiate the lowest part of the follicle and did not induce tumors. The authors then suggested: "The findings reported here can be explained on the basis that the hair follicle is reparable from cells originating at any point along its length, and that the capacity for such repair is inversely related to the degree of damage sustained by the part of the follicle minimally damaged. The existence of a 'critical depth' in skin of about 0.3 mm which was demonstrated with electron radiation ..... can be explained on the basis that the follicle tips, which received the minimum dose to the follicles, were the most protected part of the skin epithelium and, therefore, contained the critical reservoir of cells for replacing the more superficial and more heavily irradiated cells."[26]

Since the hair follicle is a few tenths millimeters long (several hundred μm) and the range of an alpha particle is about 50 μm, these results strongly suggest that a single alpha-emitting particle, even if it could be placed in rat skin, would not produce tumors. Thus, in the statement of the Geesaman hypothesis, "If the respiratory zone of the lung contains a structure analogous to the rat hair follicle, and if a radioactive particle deposited in the respiratory zone has the capacity to disrupt one or more of these structures....., then cancer risks of the order of 10^{-3} to 10^{-4} per particle can be expected."[6] The second conditional clause does not follow unless the first is modified to further redefine the hypothetical structure to a size where it will be fully irradiated by the particle (i.e., less than ~100 μm). A further necessary condition is that such structures be located throughout the lung with such a frequency that the particle will irradiate one with a probability approaching unity. This appears to be stretching an already tenuous theory beyond the realm of credibility.

V. THE TAMPLIN-COCHRAN APPLICATION

In the Tamplin-Cochran interpretation of the Geesaman work,[2] they introduce the concept of a "critical architectural unit" in the following passage: "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'."

There are significant differences, however, in the statement by Geesaman and that quoted above. Geesaman states his theory as conditional: i.e., "If the respiratory zone ..... contains a structure analogous to the rat hair follicle....."[6] Thus, in the Tamplin-Cochran version there is a progression from "if" to "when," with no evidence or attempt to indicate what this critical architectural unit may be. Further, they imply that *any* hair follicle will be a "critical architectural unit," while Geesaman carefully refers to structures ".....analogous to the rat hair follicle."[6] We have seen earlier that mouse skin hair follicles do not fit the Geesaman description, since they are not analogous in their response.

The second part of Geesaman's conditional statement indicates that ".....if a radioactive particulate deposited in the respiratory zone has the capacity to disrupt one or more of these structures and create a precancerous lesion....."[6] has been changed to indicate that when the structure is ".....irradiated at a sufficiently high dosage, the chance of it becoming cancerous is approximately 10^{-3} to 10^{-4}."[2] Thus, the hypothetical statement of the possibility of disruption and cancer formation has become, in translation, a statement of fact.

It is of interest that Tamplin and Cochran use the same probability of cancer formation for particles deposited in the lung that Geesaman states for the condition that the particle actually irradiates the hypothetical structure. We can deduce from this something of the character of this supposed structure. From Table III of the Tamplin-Cochran report, the mass of tissue irradiated to 1000 rems per year around a 0.07-pCi particle is 65 μg with the lung at half-inflation. Geesaman, for this condition and his cubical lattice lung model, estimates the range of an alpha particle to be between 335 and 1000 μm, depending upon the path through the lattice.[5] The experiments with alpha particles and rat hair follicles indicate that the full "analogous structure" must be irradiated,[26] which can only occur if the 65 μg of tissue surrounds the particle. Thus, we can conclude that the structure has a mass of 65 μg or less, since the probability of the particle lodging at the center would seem to be low. From the Tamplin-Cochran assumption that the probability of cancer for a particle lodged in the deep respiratory zone is the same as Geesaman's probability assuming the structure to be irradiated and damaged, it is apparent that the number and spacings of the structures must be assumed to be such that *each* particle will irradiate one. (Otherwise, the probability of the particle lodging close enough to irradiate the structure must be included in their estimates.) In a 1000-g lung, there must be greater than 10^7 such structures, each of which weighs less than 65 μg. It appears from this type of estimate that the "critical architectural unit" is any group of cells rather than an identified structure, as is implied by the comparison with the hair follicle.

The second change in interpretation introduced by Tamplin and Cochran is the minimum activity of a particle to produce cancer. This could logically follow from Geesaman's second conditional statement concerning the ability of the radiation to disrupt one or more of the structures.[6] However, the consequences of introducing such a threshold on the radiation response when the entire lung is irradiated are of interest. If one irradiates the full lung, obviously *all* of the hypothetical structures will be irradiated. If one assumes the disruption of these structures to be the sole cause of radiation-induced cancer and there were more than 1000 to 10 000 such sites in the lung, then the incidence would remain at zero until the threshold dose (1000 rems) was reached. The incidence would then increase rapidly above this to 100% or greater. If there are fewer than this number of sites (with a probability of 10^{-3} to 10^{-4} of producing cancer per site when irradiated), then obviously the probability of a particle irradiating the site must be included. There may be causes of radiation-induced cancer other than the mechanism of tissue disruption. These could result in a gradual increase in incidence below the threshold, but the response from the architectural unit mechanism postulated would still increase to 100% when the threshold is exceeded. This pattern does not conform to any known data on cancer incidence dose-effect relations for full lung irradiation.

It is of interest that the Tamplin-Cochran interpretations of the theory receive only minimal, if any, justifications. For example, there is no

attempt to identify the structure in the lung responsible for the effect, nor is it explained how one can extrapolate from the effects on a hair follicle to the effects in a lung which contains no unit even similar in function or structure to the hair follicle and sebaceous gland of the rat skin. Data on these tumors and their incidence, which have appeared since the original Geesaman postulation and which throw considerable light on the hypothesis, have been ignored. It can only be concluded that a more thorough and comprehensive study could have changed the conclusions of the document.

VI. THE HUMAN DATA

People have been exposed to plutonium during various uses of the material over the past 30 years. Tamplin and Cochran have chosen a few of these experiences, some to discount on the basis of their threshold theory and others to support their contention. Although we profess no special knowledge in the field of medicine, we will analyze their contentions on the basis of biological and health physics experience.

A. The Lushbaugh Report

In 1962, Lushbaugh and Langham reported on a lesion associated with plutonium in a wound.[7] The patient, while machining plutonium metal, received a wound which was later excised. Some 4 years after the accident, he noticed a nodule which, upon measurement, still contained some 0.08 μg of plutonium (~5000 pCi). Lushbaugh reported on the histological examination of the lesion, and the quotation appearing in the Tamplin-Cochran report arose from this paper: "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 pre-cancerous 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" (emphasis added). Following this quotation, Tamplin and Cochran indicated that ".....less than 0.1 μg of Pu-239 produced pre-cancerous changes in human tissue." They refer several sentences later to "this pre-cancerous lesion....." and state that this proves that a single ^{239}Pu particle ".....irradiates a significant (critical) volume of tissue and is capable of producing cancer." In other words, they manage, in the space of a few sentences, to move from ".....similarity to known pre-cancerous epidermal cytologic changes....." and expressed uncertainty on the part of the pathologist on the eventual outcome to a conclusion that cancer will result. We believe that the uncertainty expressed by the expert should be given proper weight in the conclusion.

In point of fact, examination of the autoradiograph in the Lushbaugh paper indicates very clearly that the lesion contained a number of small particles, since several points of origin of alpha particle "stars" can be discerned. Further, the author indicates the lesion containing the plutonium had a volume of 27×10^{-5} cm^3 or, for unit density tissue, a mass of some 27 μg. Reference to Fig. 1 would indicate that a single particle would deliver an alpha dose to only about 0.3 μg in unit density tissue.

In a subsequent paper, Lushbaugh *et al.* describe the result of the study of 8 such lesions resulting from plutonium in wounds in which the plutonium had resided for periods of time ranging from 0.5 to 8 years.[27] They indicate, "The lesions were found to vary morphologically in an orderly manner related roughly to the length of time the plutonium had been present. All were confined to the dermis. The size of the nodule depended on the dispersion of the particles present rather than the duration of the lesion. The largest nodule was about 2 mm in greatest dimension." They conclude in the discussion, "Although this study is based on too few small lesions to evoke much confidence in these retrospective interpretations, the conclusions may be warranted that metallic plutonium implanted in the skin in minute amounts elicits a foreign-body reaction of granulomatous type, which after subsiding in cellular activity becomes fibromatous." No reference is made in this paper to cancerous or similarity to pre-cancerous lesions.

These lesions are the most severe changes which have been reported in humans as a result of plutonium and, as such, require the question of wound

contamination to be taken seriously in radiation protection programs. However, to extrapolate these to cancers, in view of the uncertainty on outcome expressed by the pathologist, and especially to extrapolate to lung cancer seems to be an unjustifiable step.

B. The Gleason Case

The information available to the authors on the Gleason case is primarily that presented by Dr. Arthur R. Tamplin in the appendix of the Tamplin-Cochran document.[2] This involves the case of an individual who handled a crate containing a leaking carboy of ^{239}Pu solution and later developed a synovial sarcoma of the left hand.

In the initial analysis of this case, Tamplin indicates that the occurrence of this type of cancer is less than the total skin cancer death rate, since the prognosis for this type of cancer is poor. He concludes, "Thus it is highly unlikely that anyone who handled this crate would spontaneously develop this sarcoma on the contaminated hand....." This, of course, is not the question of interest, since the *a priori* condition that cancer did develop is given and the question is now whether there is evidence that indicates whether the plutonium was involved. Tamplin introduces evidence from animals that injection of 1 μg of ^{239}Pu into the skin of rats produced fibrosarcomas in 5% of the animals.[28] The relevance of this information appears remote, since these tumors were of a different type and arose from different tissues than the synovial sarcoma. (This is similar to the extrapolation from follicular tumors in the rat skin to lung tumors in the humans.) We know of no evidence, nor do Tamplin and Cochran produce any, that this type of tumor has been produced by radiation. However, in view of the ubiquitous nature of radiation as a carcinogenic agent, it would appear as a definite possibility providing that the proper critical tissue is irradiated (presumably the synovial membrane or the synoid capsule). It would appear that this would require something other than an injection into the dermis. Thus, the question to be examined is whether there is a reasonable probability that plutonium could have penetrated to the critical tissue under the conditions of the purported exposure.

Early in the discussion, Tamplin states: "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" (emphasis added). It is our experience that plutonium does not "find" its way through skin, even though there is water exchange across the skin. The skin has been shown to be an excellent barrier to prevent the passage of many materials,[29] including plutonium.[30] Thus, some mechanism such as a break in the skin (wound) must be postulated and of such a depth and location that the critical tissue is involved.

The incident occurred on January 8, 1963. According to the Tamplin account, a survey was conducted on Mr. Gleason's home, clothing, and automobile on January 19, 1963. The results apparently were negative, or they would have been mentioned. It is indicated earlier when referring to Mr. Gleason's handling of the crate: "This could not have occurred without contaminating the palmar surface of his left hand, which was bare." It is difficult to see why the contamination should preferentially go to the *left* hand. Other portions of the body and the shoes presumably would also be susceptible. However, if a sufficient quantity to deposit 0.1 μCi (0.01 ml of a 160-μg/ml solution) were on the left hand, experience has indicated that such contamination transfers rapidly to other objects, including clothing and items handled such as tools or even the automobile steering wheel. The fact that these surveys, even 11 days later, did not detect significant contamination would indicate that not much was initially present.

Tamplin further indicates that urine samples collected subsequent to January 20 gave negative results and, "The only thing that this demonstrates is that no detectable level of Pu-239 was found." Later he indicates that negative findings in the feces and urine were obtained in April 1970 and, again, dismisses the results on the grounds that little is absorbed into the body. The latter conclusion is, of course, dependent upon the type of material used. As an illustration of a worst case, Johnson *et al.* injected plutonium oxide particles with a count mean diameter of 7 μm subcutaneously into dogs.[31] They found that the translocation to

the body occurred rapidly, with on the order of 0.25% of the plutonium recovered from other tissues. Assuming this very low translocation of PuO_2 to apply to the nitrate and using Langham's equations[32] for the excretion, we find that, for the 0.1 μCi postulated by Tamplin, urine samples should have indicated on the order of 0.2 disintegration per minute in the period around January 20. This level is easily detectable by adequate analysis. Of greater applicability to the soluble nitrate case is a wound described by Schofield *et al.*[33] Here the material was plutonium oxalate, and they estimated that, without treatment, about 0.1% of the material in the wound would have been excreted in the 10- to 20-day period and 0.08% in the 20- to 30-day period. For a postulated wound burden of 0.1 μCi of this soluble material, one would expect, therefore, on the order of 20 disintegrations per minute per day excretion in the urine or some 200 to 1000 times the detectable level for most analyses. The later analyses are also significant in that they indicate the lack of a source of relatively insoluble material continually leaching into the blood.

The physical examination by Dr. Roy Albert seems to be significant in several respects. While the details are not given, there is no mention of a wound or other break in the skin through which plutonium could enter. Further, the solution was undoubtedly very acidic to retain the plutonium in solution. Such shipments are usually made in nitric acid. There is no indication given that the medical examination showed any signs of acid reaction with the skin. (Nitric acid can produce a yellow discoloration even when no overt burn occurs.) In a later conclusion, Tamplin indicates that the deposition ".....may have occurred through a small cut or via a sliver." One can only speculate on the size of cut required to introduce the plutonium in a position to irradiate the critical tissue, but it is important to note that the medical examination, which presumably included questioning of Mr. Gleason, did not reveal any indication of such a wound or sliver. (Tamplin presumably is referring to a contaminated sliver of material other than that of the carboy, since there is no indication that it was broken.)

From the above evidence, we can only conclude that the association between cancer and plutonium is speculation. The subject did handle the carboy, but subsequent examinations showed no contamination, and urine and medical history provided no indication of plutonium deposition.

C. The Los Alamos Cases

In referring to the exposures of 25 individuals exposed to plutonium some 30 years ago during the Manhattan Project,[34] Tamplin and Cochran indicated that the exposures were to insoluble plutonium and, hence, of interest. However, they discount this experience on the grounds that 14 of the 25 subjects worked in plutonium recovery operations and were exposed to droplets of plutonyl nitrate: "A droplet 1 u in diameter ($0.5\ u^3$) would therefore contain only 6×10^{-4} pCi compared with a 0.07 pCi particle of PuO_2." However, no justification is given for the assumed drop size, which appears to be very small based on attempts to produce particles by evaporating droplets from a nebulizer. For comparison, fog has a particle size of 5 to 50 μm and mists of 50 to 100 μm. If we assume the particles to be the size of fog particles, then the plutonium content would range from 0.16 to 160 pCi* -- well within the range of the definition of the "hot particle."

A summary of particle size measurements for various operations using plutonium is given in Table II.[35,36]

The aerosol from the Rocky Flats fire was generated by high-temperature condensation of PuO_2 in a manner perhaps not unlike fume formation in the wartime reduction processes. In addition, it is similar to those aerosols measured at the Los Alamos Scientific Laboratory in connection with the operations of fluorination and reduction. The lathe operation is not typical of the wartime operations, and the resuspension aerosol from cleanup is quite different from the others, although this distribution undoubtedly occurred during the wartime exposure. As a best estimate of the aerosol involved in the Los Alamos exposures, we have considered the 0.32-μm mass median diameter (MMD) with a σ_g of 1.83 μm, along with the estimates of deposition in these individuals.

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*For a 1-μm-diameter droplet containing 40 g/liter of ^{239}Pu with a specific activity of 0.0614 Ci/g but still assuming unit density for the solution, we obtain 1.3×10^{-3} pCi.

TABLE II

PARTICLE SIZE MEASUREMENTS FOR PLUTONIUM OPERATIONS

Source	Mass Median Diameter (μm)	Geometrical Standard Deviation, σ_g (μm)	Mass Fraction as "Hot Particles"[a]
Rocky Flats Fire	0.32	1.83	0.15
Fluorination of Nitrate	0.45	1.55	0.23
Reduction to Metal	0.32	1.62	0.10
Lathe Operation	0.26	1.44	0.01
Cleanup	1.90	1.80	0.97

[a]Diameter greater than 0.6 μm.

TABLE III

ESTIMATED "HOT PARTICLE" BURDENS OF LOS ALAMOS WORKERS

Diameter (μm)	Incremental Mass Fraction	Activity (pCi/particle)	Activity (nCi/man)	Particles (per man)
0.6 - 0.7	0.05	0.09	20.0	2.22×10^5
0.7 - 0.8	0.033	0.14	13.2	9.4×10^4
0.8 - 0.9	0.022	0.20	8.8	4.3×10^4
0.9 - 1.0	0.015	0.28	5.9	2.2×10^4
1.0 - 1.2	0.015	0.44	5.9	1.4×10^4
1.2 - 1.4	0.007	0.72	2.8	3.9×10^3
1.4 - 1.8	0.0057	1.34	2.3	1.7×10^3
			Total	4.0×10^5

The number of "hot particles" from an aerosol of this distribution was calculated by numerical integration in given particle size ranges above 0.6 μm. It was further considered that the total of 2.5 μCi of plutonium in these 25 men was 10 μCi at the time of exposure to allow for subsequent elimination. On this basis, the total number of particles in various size ranges is given in Table III.

The process of pulmonary deposition would not significantly distort the deposition in this range since, for more than 90% of the mass range represented, the pulmonary deposition fraction varies only in the ranges of 0.2 to 0.32. Thus, if the lung cancer per particle estimate of 10^{-3} to 10^{-4} given by Geesaman[6] were valid, we would expect some 1000 to 10 000 lung cancers in this group. Exposure has been for 30 years, so that a significant portion of the lifetime has passed with no cancers developing.

In a recent study, McInroy *et al.*[37] measured the distribution of plutonium particle size in a lymph node of a deceased worker by the autoradiographic technique. Although this individual was exposed at a later time than those discussed above, it is of interest that these estimates also indicated that 15% of the plutonium was in particles larger than 0.07 pCi.

D. The Rocky Flats Workers

Tamplin and Cochran discuss the 25 individuals exposed to plutonium during a fire in 1965.[35] They compare the lung burdens in these individuals with the lung burdens in the beagles which developed lung cancer by noting, ".....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 μCi, developed lung cancer. The highest burden in Table V is comparable to the lowest beagle exposure; the lowest exposure ....., the 19 cases with lung burdens in the 0.24 uCi range, are only an

order of magnitude less than the lowest beagle exposure." The fact that they are, in this case, using microcuries rather than numbers of particles leads to the conclusion that they are referring to radiation dose to the lung, yet they neglect to point out the difference in size between the beagle lung and the human lung -- a factor which would make the human dose about an order of magnitude lower than that of the dog with a comparable burden.

It is of passing interest that the lack of cancer in these Rocky Flats workers is dismissed on the grounds that only 9 years have passed, which is not adequate to produce cancer. We concur in this statement but note that Tamplin argues strongly for the production of a synovial sarcoma, in spite of the lack of evidence of exposure, in a matter of a few years after the incident. (Times are not given in his report, but the accident occurred in 1963 and the report of Dr. Wald, referred to by Tamplin and Cochran, was submitted in 1973, indicating that the cancer was well developed by this time.)

VII. EVIDENCE ON PARTICLE DOSE EFFECTS

As was indicated in an earlier section, those groups charged with providing safe limits for radiation exposure have consistently utilized the average dose to an organ as a basis for the limiting quantity of radioactive material. That is, the dose is calculated as though the energy were uniformly distributed through the organ. In the earliest of these recommendations, the opinion was undoubtedly based upon meager direct evidence plus the knowledge of radiation biology of those involved, and cautions as to the uncertainty of the procedure were appropriate (and still are, since full and complete data will require some years to accumulate). However, as evidence has accumulated, such cautions refer to a much narrower range of uncertainty. It is the purpose of this section of the report to summarize briefly some of the more pertinent information which can be used in assessing the question of particle dose but is not included in the Tamplin-Cochran document.

Two reviews on the question of particle dose have appeared in the past year.[38,39] The first[38] focused on the general question of whether the non-uniform dose distribution in an organ is more or less hazardous than the uniform distribution (i.e., in Tamplin-Cochran's appraisal, is the distribution factor appropriate to the particulate situation greater or less than one?). The conclusion, from the evidence available at that time was ".....that the preponderance of the evidence indicates that the use of an average lung dose is appropriate in limiting exposures and may well be conservative." The second review was a more complete examination of all of the information available on plutonium and other isotopes in the lung, with emphasis on the particle question. The conclusion of this review was similar to that of the first. We will not, here, pursue again all of the evidence but will provide a brief description of some of the pertinent results. While these experiments are selected because of the way in which they illustrate the results, we would also note that neither of the reviews found evidence which indicated the particle dose to be more harmful than the uniform dose.

Little *et al.*[40,41] administered ^{210}Po (an alpha emitter) intratracheally to hamsters both with and without iron oxide. The administration with iron produced agglomerations (effectively particles) of the ^{210}Po on the iron oxide particles, while the administration without iron produced a more uniform distribution as was shown by autoradiographs. Sanders[42] performed experiments with inhalations of both $^{238}PuO_2$ and $^{239}PuO_2$ prepared in the same manner in rats. The $^{238}PuO_2$ behaved in such a manner that it appeared to be more soluble and provided a more homogeneous dose to the lung. Both of these experiments led to the conclusion that the homogeneous distribution is more effective in producing cancer than the particulate distribution (i.e., the DF for the particulate is less than 1). Dolphin[43] quotes Lafuma as reporting ".....greater toxic effects including cancer in rats following deposition of curium-242 in lungs compared with equal amounts of plutonium-239 activity. This he attributes to the diffuse nature of the curium deposit and the particulate nature of the plutonium, as shown by autoradiographs."

In studies with beta emitters in the lung, Cember[44] concluded, ".....the carcinogenicity of a given amount of absorbed radiation energy increases up to a point, as the absorption of the energy is spread out, both time- and space-wise. From a practical point of view, this means that, for a given

total amount of absorbed energy, low-level, continuous exposure of the total lung may be more carcinogenic than the same amount of energy delivered acutely to a restricted volume." Thus, there is evidence that the same effect may be true for beta radiations.

Current experiments at the Los Alamos Scientific Laboratory provide a direct test of the Geesaman theory in that the particles are carried to the lung by the bloodstream and are lodged in immobile positions in the capillaries. Here they are in position to irradiate the surrounding tissue in patterns little, if at all, different from those administered by inhalation or intratracheally. However, they do not agglomerate or move about so that the results can be ascribed to a fixed particle and the dosimetry examined. In the first experiment,[45] particles of $^{238}PuO_2$ of 180-μm diameter were used in rats. Although a lesion similar to the one described by Lushbaugh[7] developed, it did not affect the well-being of the animal, and no cancers developed in 32 animals sacrificed from 120 to 400 days after implantation or in a group of 6 animals allowed to live out their lifetime. It is estimated that the radiation energy from this particle, if averaged over the lung of the latter 6 animals, would have delivered a dose of 2 500 000 rads (or 25 000 000 rems). Such a dose to the full lung would have caused very early death and is many orders of magnitude above that at which increased incidence of cancer is noted.

In an experiment currently in progress,[46,47] uniform-sized microspheres (10-μm-diameter) of ZrO_2 are used with intermixed PuO_2 to provide particles of differing activities, and these are introduced into the lungs of hamsters by the above technique. In the first study in this experiment, 8 groups of 60 animals each were injected with 2000 such particles, with the plutonium content of each particle ranging from 0.07 to 59.4 pCi. Essentially all of animals have now died, with only two lung cancers observed. (Three other cancers in the exposed animals occurred in organs other than the lung.) The dose rates to the lungs of those animals, when calculated as the average dose to the lung, ranged from 13 rads per year (130 rems per year) to 12 000 rads per year (120 000 rems per year). This is a range over which one would expect high tumor incidence and, in fact, premature death from pulmonary inefficiency if the material had been distributed homogeneously. Since the survival curves of the individual groups did not differ from those of the controls and the total tumor incidence was low, one can only conclude that the DF for plutonium in particulate form must be less than one. In the continuation of this study, some 1900 hamsters have received 1.6×10^8 microspheres.[48] As of October 1974, the minimum time of exposure has been 50 weeks,* which is comparable to or longer than the tumor induction times observed by Little *et al.* in their experiments with more uniformly distributed ^{210}Po. In fact, only three lung tumors (including the two observed in the first study) have, as yet, developed from the microsphere exposures. While this study is as yet incomplete, the very low tumor incidence again indicates a low effectiveness of the particles in inducing lung cancers as compared to more homogeneously distributed alpha emitters, as well as the failure of the Geesaman hypothesis to correctly forecast the results of this experiment.

VIII. DISCUSSION

There appear to be few further conclusions which can be drawn. The preceding review has indicated that the Tamplin-Cochran conclusions are based upon a hypothesis which requires considerable extrapolation of the data upon which it is based. Later evidence, of the same nature as was used in the derivation (i.e., rat skin data), does not support the assumptions of the original model. The Tamplin-Cochran interpretation of the model not only fails to take into account the later evidence but appears to present the hypothesis as fact. The supporting evidence on human data which they present are based upon unsupported assumptions and distortions of the words of the authors they quote. Most importantly, they fail to use or acknowledge direct evidence on the effect of radioactive particles. Such evidence indicates that the basic damage model which they use overestimates badly the carcinogenic effects of radioactive particles. We conclude,

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*Reference 48 indicates that ".....by the spring of 1974....." these exposures had been attained. The intent was to indicate progress to the time of preparation of the paper. The administrations were actually completed in September 1973.

therefore, that the application of the average organ dose to the establishment of limits is still appropriate, although experimentation to narrow existing uncertainties on the effects of non-uniform dose distribution should continue.

REFERENCES

1. Natural Resources Defense Council, "Petition to Amend Radiation Protection Standards as They Apply to Hot Particles," submitted to the U. S. Atomic Energy Commission and the Environmental Protection Agency (February 14, 1974).

2. A. R. Tamplin and T. B. Cochran, "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," Natural Resources Defense Council, 1710 N Street, N. W., Washington, D. C. (February 14, 1974).

3. P. N. Dean and W. H. Langham, "Tumorigenicity of Small Highly Radioactive Particles," Health Phys. 16, 79-84 (1969).

4. J. R. Coleman and L. S. Perez, Jr., "Considerations of a Tumor Probability Function and Micro-Dosimetry for the Deep Lung," Part II of Final Report of Radiological Safety Studies for the SNAP Program for Safety Branch, SEPO, USAEC, Environmental Safeguards Division, NUS Corporation report NUS-596, Rockville, Md. (1969).

5. D. P. Geesaman, "An Analysis of the Carcinogenic Risk from an Insoluble Alpha-Emitting Aerosol Deposited in Deep Respiratory Tissue," Lawrence Radiation Laboratory report UCRL-50387 (February 9, 1968).

6. D. P. Geesaman, "An Analysis of the Carcinogenic Risk from an Insoluble Alpha-Emitting Aerosol Deposited in Deep Respiratory Tissue: Addendum," Lawrence Radiation Laboratory report UCRL-50387 (October 9, 1968).

7. C. C. Lushbaugh and J. Langham, "A Dermal Lesion from Implanted Plutonium," Arch. Dermatol. 86, 461-464 (1962).

8. R. E. Albert, 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, 515-524 (1967).

9. S. Laskin, M. Kuschner, N. Nelson, B. Altshuler, J. H. Harley, and M. Daniels, "Carcinoma of the Lung in Rats Exposed to the β-Radiation of Intrabronchial Ruthenium^{106} Pellets. I. Dose-Response Relationships," J. Natl. Cancer Inst. 31, 219-231 (1963).

10. E. C. Anderson and P. N. Dean, "Effects of Internal Radiation on Living Organisms (Hot Particle Program). Dosimetry," in Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division, January through December 1973, Los Alamos Scientific Laboratory report LA-5633-PR (1974).

11. D. E. Lea, "Actions of Radiations on Living Cells," The MacMillan Company, New York (1947).

12. National Council on Radiation Protection and Measurements, "Basic Radiation Protection Criteria," NCRP Publication 39, Washington, D. C. (January 15, 1971).

13. J. Vaughan, "Distribution, Excretion and Effects of Plutonium as a Bone-Seeker," in Handbook of Experimental Pharmacology. Uranium, Plutonium, Transplutonic Elements (H. C. Hodge, J. N. Stannard, and J. B. Hursh, eds.), Springer-Verlag, New York-Heidelberg-Berlin (1973).

14. W. H. Langham and J. W. Healy, "Maximum Permissible Body Burdens and Concentrations of Plutonium. Biological Basis and History of Development," in Handbook of Experimental Pharmacology. Uranium, Plutonium, Transplutonic Elements (H. C. Hodge, J. N. Stannard, and J. B. Hursh, eds.), Springer-Verlag, New York-Heidelberg-Berlin (1973).

15. R. D. Evans, "The Effect of Skeletally Deposited Alpha-Ray Emitters in Man," Brit. J. Radiol. 39, 468; 881-895 (1966).

16. International Commission on Radiological Protection, "Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965)," ICRP Publication 9, Pergamon Press, Oxford (1966).

17. G. E. McMurtrie (Secretary), "Permissible Doses Conference Held at Chalk River, Ontario (September 1949)," Report RM-10 (May 1950).

18. National Committee on Radiation Protection, "Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water," National Bureau of Standards Handbook 52, U. S. Department of Commerce, Washington, D. C. (March 20, 1953).

19. International Commission on Radiological Protection, "Radiosensitivity and Spatial Distribution of Dose," ICRP Publication 14, Pergamon Press, Oxford (1969).

20. Subcommittee on Inhalation Hazards, Committee on Pathologic Effects of Atomic Radiation, "Effects of Inhaled Radioactive Particles," National Academy of Sciences-National Research Council Publication 848, Washington, D. C. (1961).

21. Advisory Committee on the Biological Effects of Ionizing Radiation, Division of Medical Sciences, National Academy of Sciences-National Research Council, "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," Washington, D. C. (November 1972).

22. R. E. Albert, W. Newman, and B. Altshuler, "The Dose-Response Relationships of Beta-Ray-Induced Skin Tumors in the Rat," Radiation Res. 15, 410-430 (1961).

23. R. E. Albert, 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, 525-540 (1962).

24. R. E. Albert, 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, 590-599 (1967).

25. R. E. Albert, F. J. Burns, and P. Dermott, "Radiation-Induced Hair-Follicle Damage and Tumor Formation in Mouse and Rat Skin," J. Natl. Cancer Inst. 49(4), 1131-1137 (1972).

26. R. D. Heimbach, F. J. Burns, and R. E. Albert, "An Evaluation by Alpha-Particle Bragg Peak Radiation of the Critical Depth in the Rat Skin for Tumor Induction," Radiation Res. 39, 332-344 (1969).

27. C. C. Lushbaugh, R. J. Cloutier, G. Humason, J. Langham, and S. Guzak, "Histopathologic Study of Intradermal Plutonium Metal Deposits: Their Conjectured Fate," Ann. N. Y. Acad. Sci. 145, 791-797 (1967).

28. H. Lisco, M. P. Finkel, and A. M. Brues, "Carcinogenic Properties of Radioactive Fission Products and of Plutonium," Radiology 49(3), 361-363 (1974).

29. R. T. Tregear, "Physical Functions of Skin," Academic Press, Inc., New York (1966).

30. W. D. Oakley and R. C. Thompson, "Further Studies on Percutaneous Absorption of Plutonium Solutions in Rats," in Biology Research-Annual Report 1955, Hanford Atomic Products Operation report HW-41500 (February 1956).

31. L. J. Johnson, R. L. Walters, J. L. Lebel, C. R. Lagerquist, and S. E. Hammond, "The Distribution of Pu and Am: Subcutaneous Administration of PuO_2 and the Effect of Chelation Therapy," in Radiobiology of Plutonium (B. J. Stover and W. S. S. Jee, eds.), J. W. Press, Salt Lake City, Utah (1972).

32. W. H. Langham, S. H. Bassett, P. S. Harris, and R. E. Carter, "Distribution and Excretion of Plutonium Administered Intravenously to Man," Los Alamos Scientific Laboratory report LA-1151 (September 20, 1950).

33. G. B. Schofield, H. Howells, F. Ward, J. C. Lynn, and G. W. Dolphin, "Assessment and Management of a Plutonium Contaminated Wound Case," Health Phys. 26, 541-554 (1974).

34. L. H. Hempelmann, C. R. Richmond, and G. L. Voelz, "A Twenty-Seven Year Study of Selected Los Alamos Plutonium Workers," Los Alamos Scientific Laboratory report LA-5148-MS (January 1973).

35. J. R. Mann and A. R. Kirchner, "Evaluation of Lung Burden following Acute Inhalations of Highly Insoluble PuO_2," Health Phys. 13, 877-882 (1967).

36. W. D. Moss, E. C. Hyatt, and H. F. Schulte, "Particle Size Studies on Plutonium Aerosols," Health Phys. 5, 212-218 (1961).

37. J. F. McInroy, M. W. Stewart, and W. D. Moss, "Studies of Plutonium in Human Tracheobronchial Lymph Nodes," in Proceedings of the Conference on Radiation and the Lymphatic System, Battelle-Northwest Laboratories, Richland, Washington (September 30-October 4, 1974), in press.

38. J. W. Healy, "A Proposed Interim Standard for Plutonium in Soils," Los Alamos Scientific Laboratory report LA-5483-MS (January 1974).

39. W. J. Bair, C. R. Richmond, and B. B. Wachholz, "An Assessment of Spatial Distribution of Radiation Dose Relative to Inhalation of Radionuclides," U. S. Atomic Energy Commission report WASH-1320 (September 1974).

40. J. B. Little, B. N. Grossman, and W. F. O'Toole, "Respiratory Carcinogenesis in Hamsters Induced by Polonium-210 Alpha Radiation and Benzo(α)-Pyrene," in Morphology of Experimental Respiratory Carcinogenesis (P. Nettesheim, M. G. Hanna, Jr., and J. W. Deatherage, Jr., eds.), AEC Symposium Series 21, U. S. Atomic Energy Commission report CONF-700501 (1970), Office of Information Services, Springfield, Va.

41. J. B. Little, B. N. Grossman, and W. F. O'Toole, "Factors Influencing the Induction of Lung Cancer in Hamsters by Intratracheal Administration of ^{210}Po," in Radionuclide Carcinogenesis (C. L. Sanders, R. H. Busch, J. E. Ballou, and D. D. Mahlum, eds.), AEC Symposium Series 29, U. S. Atomic Energy Commission report CONF-720505 (1973), Office of Information Services, Springfield, Va.

42. C. L. Sanders, "Carcinogenicity of Inhaled Plutonium-238 from Crushed Microspheres," in Pacific Northwest Laboratories Annual Report 1972, Battelle-Northwest Laboratories report BNWL-1750, Part 1 (1973), p. 28.

43. G. W. Dolphin, "Hot Particles," National Radiological Protection Board, Radiological Protection Bulletin 8, Harwell, Didcot, Berkshire, England (July 1974).

44. H. Cember, "Radiogenic Lung Cancer," Prog. Exp. Tumor Res. 4, 251-305, Hafner Publishing Co., Inc., New York (1964).

45. C. R. Richmond, J. Langham, and R. S. Stone, "Biological Response to Small Discrete Highly Radioactive Sources. II. Morphogenesis of Microlesions in Rat Lungs from Intravenously Injected $^{238}PuO_2$ Microspheres," Health Phys. 18, 401-408 (1970).

46. C. R. Richmond and G. L. Voelz, eds., "Annual Report of the Biological and Medical Research Group (H-4) of the LASL Health Division, January through December 1972," Los Alamos Scientific Laboratory report LA-5227-PR (March 1973).

47. C. R. Richmond and E. M. Sullivan, eds., "Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division, January through December 1973," Los Alamos Scientific Laboratory report LA-5633-PR (May 1974).

48. E. C. Anderson, L. M. Holland, J. R. Prine, and C. R. Richmond, "Lung Irradiation with Static Plutonium Microspheres," in Experimental Lung Cancer, Carcinogenesis and Bioassays, Springer-Verlag, New York-Heidelberg-Berlin, CONF-740648-1 (1974), in press.

LA-5483-MS

INFORMAL REPORT

A Proposed Interim Standard for Plutonium in Soils

los alamos
scientific laboratory
of the University of California
LOS ALAMOS, NEW MEXICO 87544

UNITED STATES
ATOMIC ENERGY COMMISSION
CONTRACT W-7405-ENG. 36

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November 22, 1974

Office of Radiation Programs
AW 560 E.P.A.
Washington, D.C. 20460

Gentlemen:

In the recent EPA Bulletin it stated that current levels of trans-uranium elements in the environment are very low,in the case of plutomium there is a very small amount in the environment. That statement is certainly open to discussion. The Natural Resource Defense Council states in its "Report on the Risks of Plutonium Recycle" that one millionth of a gram has been shown capable of producing cancer in animals.

The question here arises as to whether the small amount of plutonium already in the environment is more than, equal to, or less than one millionth of a gram. This same NRDC reports that an amount of plutonium the size of a football is enough for a nuclear explosion capable of mass destruction. This statement in itself would indicate that the production of plutonium should be kept to an absolute minimum.

The AEC impact statement on the assesment of plutonium concedes that the problem of plutonium toxins and nuclear theft are far from solved and suggests that they may never be solved in the near future and perhaps may never be solved at all. This impact statement certainly indicates that we should halt the further construction of nuclear reactors until a solution to this problem can be found. This is the procedure that EPA should use for meeting emergencies.

NRDC further stated in its report that microgram quantities of plutonium in skin wounds cause cancer and in the body it is a bone seeker where, once deposited it can cause bone cancer. But, plutonium is most dangerous when inhaled. This means that under a number of probable conditions plutomium forms aerosols of micro sized particulates which can remain suspended for a significant time. When inhaled it deposits itself in the lung tissue and can eventually cause lung cancer.

The AEC radiation protection standards governing the amount of plutonium to which members of the public can be exposed are roughly 100,000 times too lax. Judging from these statements I would advise EPA to call as immediate halt to all construction of any equipment which produces plutonium other than what the Defense Department deems to be absolutly necessary for national defense. This should be the method of preventing such releases and keeping that release to an absolute minimum. The cost of not preventing such releases could mean our lives.

I make an appeal to the office of radiation programs to petition the AEC and the EPA to reduce the present maximum permissable exposure levels by 100,000. Under the existing conditions mentioned above I can see no justifyable reason for either of these agencies to refuse this request.

Very truly yours,

Elmer F. Glueck

Elmer F. Glueck
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Chicago, Illinois 60613

cc:The task force against nuclear pollution
The Environmental Defense Fund
Citizens Alert
Senator Gravel
Bulletin of the Atomic Scientists

National Radiological Protection Board

Chairman: Sir Brian Windeyer Director: Dr A S McLean Secretary: L D G Richings

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29th November 1974

Dr. W. A. Mills,
Director,
Criteria & Standards Division (AW-560),
Office of Radiation Programs,
US Environmental Protection Agency,
WASHINGTON, D.C. 20460,
U.S.A.

Dear Bill

Thank you for the copy of the notice about public hearings in Washington next month. I enclose two copies of R-29. The purpose of this report was to draw attention to the many different aspects of radiological protection of people exposed to plutonium and as a result it is far from definitive on any one of the problems it reviews.

All the best,

Yours sincerely,

Encl.

G. W. Dolphin,
Assistant Director
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Radiological Problems in the Protection of Persons Exposed to Plutonium

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ABSTRACT

For the protection of persons exposed to plutonium isotopes and higher actinides there are five important requirements. First it is essential to evaluate all the appropriate data and develop basic dose standards for control of exposure of body organs, particularly bone, liver and lung; these must be comparable to standards for exposure to external gamma and X-radiation. Secondly, from these basic standards, values for maximum permissible concentrations in air and permissible annual intakes must be derived using metabolic models for plutonium in humans. As part of the biological monitoring of workers it is necessary to make assessments of the amount of plutonium in the body either by measurement of the excretion rate of plutonium in the urine or by external counting of gamma or X-radiations over the chest or contaminated wound site. For the treatment of cases of high over-exposure therapeutic techniques should be available for accelerating the excretion or removal of the radioactivity from the body. Finally, plans must be made to cover the possibility of a large release of plutonium into the environment and these should include acceptable values for ground contamination levels.

Relevant data on these five subjects are reviewed in this report.

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CONTENTS

		Page No.
1.	INTRODUCTION	1
2.	PRODUCTION AND SOME CHEMICAL AND PHYSICAL PROPERTIES	1
3.	METABOLISM	3
	3.1 Routes of entry into the body	3
	3.2 Metabolism at the site of entry	3
	3.3 Transport of plutonium from the site of entry	3
	3.4 Retention in the lung of inhaled plutonium	4
	3.5 Distribution in body tissue after intake by inhalation or through wounds	5
	3.6 Amount in gonads	6
	3.7 Summary of metabolism for radiological protection purposes	7
4.	PLUTONIUM DISTRIBUTION IN HUMANS	7
5.	BIOLOGICAL EFFECTS	8
	5.1 Bone	8
	5.2 Liver	9
	5.3 Lung	9
	5.4 Lymph nodes	10
	5.5 Blood	11
6.	BIOLOGICAL EFFECTS IN MAN	11
	6.1 Health of workers exposed to plutonium	11
	6.2 Pathological effects	12
7.	THERAPEUTIC PROCEDURES FOR THE REMOVAL OF PLUTONIUM FROM THE BODY	12
8.	THE RADIOLOGICAL PROTECTION OF PEOPLE EXPOSED TO PLUTONIUM	13
	8.1 Basic standards	13
	8.2 Derivation and adequacy of the basic standards	13
	8.3 Derived standards for air concentration and annual intake	17
	8.4 Determination of body content from bioassay measurements	18
	8.5 Dosimetric model for thoracic lymph nodes	20

		Page No.
9.	HOT PARTICLES	20
10.	PLUTONIUM-238 OXIDE	22
11.	PLUTONIUM IN THE ENVIRONMENT	22
	11.1 Sources and chemical form	22
	11.2 Routes to man	23
	11.3 Aquatic environment	24
	11.4 Experience at Palomares	25
	11.5 Plutonium in man	25
12.	CHEMICAL TOXICITY	26
13.	SUMMARY	27
14.	SUMMARY OF NRPB RESEARCH PROGRAMMES	28
15.	ACKNOWLEDGEMENTS	29
16.	REFERENCES	30

TABLES

1. Some important transuranic nuclides produced in nuclear reactors

2. The specific alpha activity of particles of PuO_2 and nitrate from fuel irradiated to a level of 2000 MWd/t and unit density equivalent aerodynamic diameter of 1 μm

3. Percentage distribution of plutonium in tissue of beagles after inhalation and intravenous injection, and in rabbits after intramuscular injection

4. Distribution of plutonium in humans as measured in tissues obtained at autopsy

5. Chromosome aberrations found in blood lymphocytes of individuals known to have been accidentally contaminated with plutonium

6. Basic dose standards expressed both as rem in a year and as rad in a year for organs of importance in the protection of persons exposed to plutonium, from ICRP Pub.9 (1966)

7. Estimated values of risk coefficient for various types of cancer in exposed population groups for external γ and α radiation

8. Percentage of inhaled plutonium-239 activity transferred to blood from lung and into bone and liver, half-lives of retention as used to calculate $(MPC)_a$ and MPAI for soluble, insoluble, class W and Class Y compounds

9. The variation with particle size of the number of particles and the number of cells irradiated for a constant activity of 0.016 μCi of plutonium-239 oxide

FIGURES

1. Plutonium induced osteogenic sarcomas in rat

2. Plutonium induced lung cancer in experimental animals

1. INTRODUCTION

Comprehensive reviews of the biological research done during the last two decades on the metabolism and effects of plutonium have been recently published by Bair (1974), Bair and Thompson (1974), Vaughan (1973), Bair et al. (1973) and Bair et al. (1974). The object of this report is to review briefly, in the context of a nuclear power programme, the problems of radiological protection of workers exposed to plutonium in the nuclear power industry and environmental populations who may be exposed to plutonium as a result of an accidental release from a nuclear establishment.

2. PRODUCTION AND SOME CHEMICAL AND PHYSICAL PROPERTIES

The production of plutonium in thermal reactors and its use as fuel in thermal or breeder reactors are unavoidable if the large quantities of uranium-238 in the earth's crust are to be used as a source of energy. The important nuclear transformations in which plutonium isotopes and some other actinides are produced in thermal nuclear reactors are shown in Table 1. The relative amounts of these radionuclides present depends on the irradiation of the fuel. This is proportional to the time in the reactor and the neutron flux density and is expressed as megawatt-days per tonne. Almost pure plutonium-239 is produced only if the fuel is given a low irradiation, say less than 1000 megawatt-days per tonne. At irradiation levels higher than this, alpha-emitting plutonium-240 and beta-emitting plutonium-241 are produced in significant quantities relative to plutonium-239. At the very high fuel irradiations planned for power reactors, other alpha-emitting actinides become important, namely americium-241 from the decay of plutonium-241, curium-242 and curium-244, as shown in the sixth column of Table 1, where the figures have been normalised to plutonium-239. It should also be noted that the nuclides in this table undergo spontaneous fission but the total amount of energy emitted in this type of transition is small compared with that in the alpha particle decay process.

There are two relatively short-lived plutonium isotopes, plutonium-238 (86.4 years) which decays by alpha emission to uranium-234 (2.5×10^5 years) and plutonium-241 (15 years) which decays by beta emission to americium-241 (458 years) which in turn decays by alpha emission to neptunium-237 (2.2×10^6 years). The short-lived curium-242 (163 days) isotope which accounts for most of the alpha activity in the fuel rods at the end of long irradiation periods decays to plutonium-238. In summary, as the irradiation of the fuel rods increases so isotopes of plutonium other than 239 become more important, as do the higher actinides, americium and curium.

The chemical separation of plutonium from irradiated uranium fuel is carried out in the UK at Windscale. The process involves dissolution of the fuel rods in nitric acid and a separation of plutonium from uranium and

fission products. Plutonium is obtained from this chemical process in the form of a nitrate solution which is used to produce insoluble plutonium oxalate. Most of this material is converted to plutonium dioxide. The chemical forms of plutonium most likely to be released into a factory working area are the oxide, the oxalate and the nitrate, the two former as a dust and the latter as droplets of solution. If plutonium is accidentally released into the environment it is most likely to be as the oxide.

Plutonium dioxide is chemically very inert. The material can be dissolved only by drastic chemical treatment with hot concentrated mineral acids containing complexing agents or by alkali fusion. Even the most highly polluted atmosphere is unlikely to have much chemical effect on a plutonium dioxide aerosol. Plutonium nitrate is water soluble and it undergoes rapid hydrolysis to produce plutonium in an insoluble form. Atmospheric droplets of plutonium nitrate or plutonium nitrate solution undergo hydrolysis to give insoluble plutonium hydroxide. The plutonium dioxide or the products of hydrolysis of plutonium nitrate deposited on soil and vegetation will be chemically inert.

An important physical property in radiological protection is the radioactivity associated with respirable particles. The number of particles with a unit density equivalent aerodynamic diameter of 1 micrometer, a representative size for respirable particles, having a total activity of 40 nCi is given in Table 2 for two compounds of plutonium. This value of 40 nCi is the present maximum permissible body burden (MPBB) recommended in ICRP Publication 2 (1959), and from the table it can be seen that this amount of activity is contained in about 10^6 particles of respirable size.

One other important physical property is that plutonium-239 emits an L X-ray of uranium in 4% of disintegrations with an energy of about 17 keV. It is possible to detect these X-rays through a few centimetres of tissue and this provides the basis for the detection and measurement of plutonium-239 in the lungs or in a wound. All the alpha-emitting isotopes of plutonium also emit L X-rays in varying amounts during decay. Plutonium-241 decays to americium-241 which emits a gamma ray with an energy of 60 keV in 40% of the disintegrations which can be readily detected outside the body from incorporated deposits.

3. METABOLISM

3.1 Routes of entry into the body

Plutonium may enter the body either after deposition in the lung or by penetration through the skin. There is no evidence of entry through the intact skin in humans; however there are records of humans being internally contaminated after a hand wound (Schofield et al., 1974) or acid burn (Lister et al., 1963). The transfer of orally-ingested plutonium across the

gut wall is very small and the ICRP Task Group on The Metabolism of Compounds of Plutonium and Other Actinides (ICRP Publication 19, 1972) summarised the data from animal experiments and concluded that the absorption of oxides and similar insoluble compounds was 10^{-4}% and for the more soluble compounds, such as nitrates, 3×10^{-3}%. However, it should be noted that in some animal experiments special forms of plutonium may be more readily absorbed, as in the case of an early experiment by Weeks et al. (1956) who measured an absorption of 1.9% in rats given a plutonium compound in the hexavalent form and at pH = 1. This represents an artificial situation because such a compound is unlikely to be met in factories or in the environment where plutonium dioxide or hydrolysed plutonium nitrate are the most commonly occurring compounds in the air.

3.2 Metabolism at the site of entry

For purposes of understanding the metabolism at the site of entry a simple model may be used in which the plutonium is considered to form three fractions. One fraction is rapidly formed and consists of a complex of monomeric plutonium stabilised by biological anions such as bicarbonate and citrate and by amino acids and possibly some proteins. This fraction diffuses away from the site into body fluids and is deposited mainly in bone and liver. The second fraction consists of colloidal or particulate material. Some colloids may be formed at the site of entry as the plutonium compounds come into contact with the biological milieu and some plutonium compounds may enter the body as colloids or particles. Much of this second fraction of material will become engulfed by cells within a few hours, especially if the site of entry is the lung (Morrow and Casarett, 1961; Sanders, 1969). The fate of this second fraction will depend on the physical and chemical properties of the compound and the biological conditions encountered but undoubtedly some will slowly erode away from the particles or colloids during the following months or years, become complexed by biological anions and diffuse into body fluids similar to the first fraction. That remaining in the cells will be distributed in the body according to the kinetics of the cell system involved but most will probably remain in the lymphatic system (Bair, 1974; Schofield et al., 1974) or become trapped in reticulo-endothelial cells, exemplified by the Kupffer cells. The cells containing plutonium will be heavily irradiated and eventually die and the particle will be engulfed by another cell. A third fraction may remain almost permanently at the site of entry, particularly in the case of wounds, and become isolated in a fibrotic region (Lushbaugh and Langham, 1962).

3.3 Transport of plutonium from the site of entry

Experimental studies in animals on the binding of plutonium in plasma after intravenous injection of plutonium citrate (Stevens et al., 1968) or plutonium nitrate (Boocock and Popplewell, 1965) show that about 90% of the plutonium is bound to transferrin, a large protein molecule which binds iron,

and the remainder is bound to smaller molecules, probably citrate. The same binding pattern is found when plutonium nitrate is mixed with human serum *in vitro*. Some evidence from work in the NRPB laboratories (Loveless, 1974) indicates that this pattern of distribution in blood may be different when the plutonium compound is originally deposited in the lung but more experimental work is needed to substantiate this observation. Recent experiments at NRPB (Popplewell et al., 1973) support the hypothesis that the plutonium excreted in the urine is bound to citrate molecules.

Plutonium citrate intravenously injected into humans is rapidly cleared from the blood. In a summary of available data Durbin (1972) records that 53%, 27%, 17% and 3% are cleared from the blood with half-lives of 20 minutes, 7 hours, 1.2 days and 5 days respectively. This rapid clearance from the blood may be influenced by the fact that it is direct injection of the plutonium citrate complex. In a human accident case the clearance of that fraction of plutonium which reaches the blood from the site of intake may be slower as the transporting molecule is predominantly transferrin. However, this rapid clearance of the blood, mainly to bone and liver, does necessitate immediate action after accidental intake if deposition in these organs is to be prevented. This will be discussed later.

Plutonium bound to molecules in the plasma and body fluids are exchanged on to binding sites, such as bone mineral, bone protein and cell membranes. The amount of plutonium exchanged at these sites will depend both on the ability of the site to bind plutonium and on the physiological processes of circulation and fluid movement which take the transporting molecule to the binding site.

3.4 Retention in the lung of inhaled plutonium

Inhaled plutonium deposited in the ciliated portion of the respiratory system, that is nasal passages, trachea, bronchi, bronchioles and terminal bronchioles, is cleared within a few hours to the external nares or to the throat for passage through the gut into the faeces. Some of the plutonium dust in the activity median aerodynamic diameter (AMAD) size range below 2 or 3 microns may penetrate deep into the lungs and become deposited in the alveoli, the non-ciliated region of the lung where gas exchange takes place. Particles deposited in this region are cleared very slowly by three main routes; transport in macrophages on to the ciliated epithelium and up to the throat, transport probably in cells into the lymphatic drainage pathways of the lung, and slow solubilisation and transfer to the blood, as described by Mercer (1967). Sanders and Adee (1970) and Sanders et al. (1971) showed that some of the inhaled plutonium particles in mouse lung are phagocytosed by type I alveolar epithelial cells. These cells appear to be radio-resistant and to be firmly attached to the epithelium wall so that plutonium trapped by this mechanism could remain in the lung alveoli for a long time.

Watts (1974) has analysed all the available data on the lung retention of inhaled plutonium dioxide in large animals, namely dogs, sheep, burro and man. She found that the retention could be represented by three exponential terms as in the equation:-

$$R(t) = A \exp - \frac{(0.69t)}{(1)} + B \exp - \frac{(0.69t)}{(30)} + C \exp - \frac{(0.69t)}{(500)}$$

The half-lives of the three exponential terms, 1, 30 and 500 days, are reasonably well established from the data, although it should be noted that 1000 day half-lives have been reported in long-term dog experiments by Park et al. (1972). The amplitudes of the three terms, A, B and C cannot be determined from the available data and these probably depend on the particle size distribution of the inhaled material. A possible physiological explanation of the three terms is that the first represents the rapid clearance of particles deposited on the ciliated epithelium, the second represents slower clearance of particles engulfed in free macrophages which enter the alveoli from the blood and pass out of the lung up the ciliated epithelium, and the third represents the long term retention of particles in cells of the epithelium.

3.5 Distribution in body tissue after intake by inhalation or through wounds

Plutonium cleared from the lung via the lymphatic drainage system may be retained for several years in the thoracic lymph nodes. In beagles, the important nodes, where the plutonium concentrates, are situated round the trachea and bronchi at the bifurcation but in man, many lymph node chains are found in the peripheral pulmonary tissues. Park et al. (1972) reported on the retention in tissues of beagles in an experiment involving inhalation of plutonium dioxide by nearly 100 dogs and lasting over 11 years. They found that the accumulated amount, after 11 years, in the thoracic lymph nodes was on average about 40% of the initial alveolar deposit which ranged from 1 to 50 μCi. This is a large amount of activity; it delivered a radiation dose to the lung so high that it could have affected the lung clearance mechanisms. Bair and Thompson (1974) describe another experiment underway at the Battelle Laboratories in which smaller lung deposits have been used and in about 10 years it should be possible to find out whether the large deposits used in the first experiment affect lung clearance.

In the experimental work reported by Park et al. (1972), mentioned above, the average accumulation after 11 years in liver and bone was 15% and 5% respectively of the initial deposit of plutonium dioxide. For the more soluble compounds of plutonium, such as the nitrate and citrate, the retention in the thoracic lymph nodes of both beagles and rats is less avid and more material is transported to other organs. Some data indicating this effect in beagles are shown in Table 3. By comparison with plutonium

dioxide, the amount of plutonium in the thoracic lymph nodes after inhalation of soluble compounds is very small. Furthermore, the amount reaching the skeleton is about twice that reaching the liver. The fifth column in Table 3 shows the distribution between the skeleton and liver 4 years after intravenous injection of plutonium citrate and the ratio of the amount in the skeleton and liver is about the same as that found after inhalation of this compound.

In the NRPB laboratories, Stather and Howden (1974) found that the extra pulmonary distribution in rats after pulmonary intubation of small amounts of activity, equivalent to about five times the maximum permitted amount in man, is the same for the three compounds of plutonium used, nitrate, citrate and oxide. They found that the skeleton to liver ratio was about 6 to 1 after 7 days. This indicates that the initial diffusible fraction of the plutonium compound is more readily deposited in the bone than in the liver. However, at longer times after intake some translocation from bone to liver occurs or there may be a direct transfer of particulate material inside cells from the lung to the liver.

Other experimental work by Stather et al. (1974) showed that the amount of plutonium dioxide transferred from the lung to other body organs depended markedly on the particle size. They found that plutonium dioxide filtered through a 25 millimicron Millipore behaved almost in the same way as the more soluble nitrate and citrate compounds. It has been known for decades that particle size influences deposition in the lung and now the work of Stather et al. shows that it also influences retention. Many years previously, Bair and Willard (1962) had noted a more rapid removal of small particles from beagle lungs.

Experiments have been made with animals to measure the tissue distribution after intramuscular or intradermal injection and the results of these experiments have been summarised in ICRP Publication 19 (1972). A typical set of values obtained by Taylor (1969) after intramuscular injection in rabbits are shown in the last column of Table 3. Again the skeletal accumulation is more than in the liver but 67% of the injected amount remained at the site of injection. The amount remaining at the site will depend markedly on the physical and chemical conditions during injection.

3.6 Amount in gonads

Measurements of the amount transferred from blood to gonads have been made in dogs injected with plutonium, americium or curium in citrate solution (Mays et al., 1974). The percentages transferred and retained for a very long time in the gonads were 0.017 for males and 0.0012 for females. In humans, small amounts have been measured in gonads obtained at post-mortem arising from the inhalation of plutonium in fallout (Richmond and Sullivan, 1974). The genetic effect of these small amounts is not known; the somatic effect is

likely to be relatively negligible because the concentration is lower than in other organs such as bone or liver.

3.7 Summary of metabolism for radiological protection purposes

All the available data on the metabolism of compounds of plutonium and other actinides were reviewed by a Task Group of ICRP (ICRP Publication 19, 1972). They concluded that the distribution in humans could be reasonably represented for all compounds by a distribution from blood to bone, liver and soft tissues plus urine of 45%, 45% and 10% respectively. They also suggested that the percentage transfer from the lung to blood was 12% for more soluble and 5% for less soluble compounds. Previously the recommendation for soluble compounds was 25% and insoluble compounds were not transferred to blood. Transfer from the gut to blood is taken to be 3×10^{-3}% and 10^{-4}% for soluble and insoluble compounds.

Retention is exponential with half-lives for the lung of 50 and 500 days for soluble and insoluble compounds and 100 and 40 years for bone and liver respectively. These latter values are independent of the compound entering the body.

4. PLUTONIUM DISTRIBUTION IN HUMANS

Data on the concentration of plutonium in human tissues are gradually becoming available from autopsy specimens. This is a very important aspect in the study of plutonium metabolism and every effort should be made to obtain such data. It is noted that the US authorities have set up at Richland a register of persons exposed to transuranic elements (Norcross and Newton, 1972).

Measurements of plutonium in human tissue obtained at post-mortem have been reported for more than 60 persons who were exposed at work. A summary of the data from the 12 highest exposed persons is given in Table 4. These cases have been selected for inclusion in the table by reference to the measured lung content which is over 2000 dpm/kg (900 pCi in lung) in all cases. As the analysis for plutonium at these levels should present no problems these data are expected to be reliable. In determining the tissue distribution in humans the problems mainly arise from the inadequacy of the tissue samples available. Both in the UK and USA, it is the practice to obtain as large a sample of liver, whole lung with trachea and thoracic lymph nodes as possible, together with a portion of bone from ribs, sternum and vertebrae (Schofield 1974; Parker 1973). Hence, the most reliable organ content estimates can be made for the lung and liver but in the case of the skeleton where non-uniform deposition among the bones may have occurred the estimate is less reliable. Naturally, dissection of the lymph nodes in the pulmonary and tracheobronchial region must be done with care to avoid extraneous tissue and to locate as many lymph nodes as possible. Nevertheless,

the values quoted of dpm/kg for these lymph nodes may be inaccurate.

From the data in Table 4 it can be seen that the ratio of thoracic lymph node to lung tissue concentration varies widely and there is no obvious increase in the ratio with the years of exposure. However, if the exposure pattern is non-uniform, as it usually is with plutonium, the years of exposure might not necessarily be a guide to the mean duration of the deposit in the lungs. Furthermore, the animal experiments, as typified by data in Table 3, indicate that plutonium nitrate is not concentrated in these lymph nodes to the same extent as plutonium oxide. The compounds to which humans are exposed during a working life cannot be specified with certainty.

The mean of the ratio of concentrations in the lymph nodes and lung weighted by the amount of plutonium in the nodes is 5.8 and the unweighted value is 12.5. These values are much lower than those measured in beagles exposed to plutonium dioxide. Park et al. (1972) found in 5 dogs, all surviving more than 7 years, a lymph node concentration relative to the lung of between 150 and 3000.

The ratios of the amounts in bone and liver given in Table 4 vary widely and again this is due partly to the duration of the exposure and partly to the type of compound taken into the body. The mean value of the ratio is 1.9 and this is well within the range of values found in animal experiments.

5. BIOLOGICAL EFFECTS

In animal experiments it has been well established that late biological effects occur in the bone, liver, lung, thoracic lymph nodes and blood. After the intake of soluble compounds of plutonium sufficient may be transferred from the site of entry, that is the lung or a wound site, to the bone and liver so that late effects may occur in these organs. There is little transfer from the lung and thoracic lymph nodes and the late effects after inhalation of insoluble plutonium dioxide mainly occur in these organs. The significant effect in blood observed in beagles following inhalation of relatively large amounts of plutonium dioxide is a chronic lowering of white cell count, particularly of lymphocytes, for which the count may be lowered by a factor of 2 (Park et al., 1972). No neoplasia of the haematopoietic system such as leukaemia have been observed in these dogs.

5.1 Bone

Plutonium is transported in the blood and deposited on the endosteal surfaces of bone where it is well situated to irradiate osteogenic cells. The deposited plutonium may later become buried in the bone by apposition of bone mineral. It may enter osteoclasts, bone cells concerned with resorption, during bone remodelling or it may be phagocytosed by macrophages and move into the bone marrow (Jee, 1972). Osteogenic sarcomas have been produced

in experiments with mice, rats and dogs after administration of plutonium by various methods; intravenous injection, intratracheal injection, inhalation and subcutaneous injection. A summary of the osteogenic sarcomas found in rats, as reported by several investigators, is plotted in Fig. 1. Data from intravenous injection experiments have been excluded because this is not a relevant method of intake for humans and it could lead to a micro-distribution on the bone surfaces different from that occurring when plutonium reaches the blood slowly after crossing membranes, as in inhalation experiments. On the basis of a linear analysis, the risk coefficient in the rats is 3.9% per 100 rads average dose to bone.

At the University of Utah, beagles were injected intravenously with plutonium citrate and 4 out of 12 developed osteosarcomas after about 9 years and they had an average dose to bone at death of 86 rads. At higher average bone doses, 190 rads and 310 rads, 8 out of 13 and 10 out of 12 developed osteosarcomas. Again on the basis of a linear analysis these data indicate a risk coefficient of about 40% per 100 rads which is about 10 times higher than the value for rats. This could be due to species differences, in which case it clearly demonstrates the uncertainties of extrapolation from one species to another. Alternatively, there may be a difference in osteogenic sarcoma induction depending on whether the plutonium is deposited rapidly by intravenous injection or is deposited slowly by transfer via the blood from the lung.

5.2 Liver

Liver tumours are rarely seen after administration of plutonium to rats and this is probably due to the fairly rapid clearance of plutonium from the rat liver to bile and into faeces. In beagles at the University of Utah, Taylor et al. (1972) reported 10 bile duct tumours in 96 experimental dogs and only 4 in 98 control dogs. In 2 dogs these liver tumours were the cause of death and the average radiation dose to their livers was about 60 rads. The tumours appeared at the end of the expected life span and they caused little life shortening. There are not enough data from animal experiments to calculate a risk coefficient for liver tumours.

5.3 Lung

Acute effects have been observed in dogs after inhalation of large amounts of plutonium, equivalent to more than 500 microcuries in man. These consist of severe inflammatory reactions, oedema and haemorrhage and death occurs within a few days. At lower intakes fibrosis is the critical change in the lung which reduces its capacity for gaseous exchange and within a few months death occurs due to pulmonary insufficiency and associated heart failure.

The important late effect is lung cancer and data from three experiments involving inhalation of insoluble plutonium by rats are plotted in Fig.2. On a linear analysis the risk coefficient is 7.5% per 100 rads, which is higher than the similar value for bone. In a long-term experiment started in the early 1960s, Bair and his colleagues at the Battelle Northwest Laboratory exposed nearly 100 beagles to aerosols of plutonium dioxide. In those animals which survived more than 1600 days, 20 out of 21 developed lung cancers and it is therefore not possible to evaluate a risk coefficient from these data (Bair and Thompson, 1974). It should be noted that the cancers observed by Bair and his colleagues were often multifocal and all in the peripheral region of the lung. Naturally occurring cancers in man are more frequently found in the primary and segmental bronchi than in the peripheral region of the lung (UICC, 1973).

5.4 Lymph nodes

Lymph nodes associated with the lymphatic drainage system of the lung and local lymph nodes near the site of subcutaneously-injected plutonium may have plutonium concentration levels higher than most other tissues. In the beagles exposed to plutonium dioxide aerosols at Battelle Northwest Laboratories 3 dogs developed thoracic lymph node malignancies out of 21 animals surviving more than 1600 days (Park et al., 1972). These malignancies were classified as haemangiosarcoma, lymphangiosarcoma and endothelioma but all 3 dogs also had primary cancers in the lung. The results of this large-scale dog experiment indicate that although these lymph nodes received a higher radiation dose than the lung, they were not the organ in which most cancers were observed. This view may change when the results from the low dose experiment now under way at the Battelle Laboratories become available in the next 10 years.

Experiments involving the implantation of plutonium dioxide in the fore-paws of dogs have shown that plutonium is transferred to the cervical and axillary lymph nodes. No dogs have shown any detrimental effect which can be unequivocally ascribed to this implant in the first three years of the experiment (Lebel et al., 1972). One dog with about 6 μCi of plutonium dioxide implanted in the paw died of a lymphosarcoma after 4 months. The estimated dose to the cervical lymph nodes was about 7000 rads but this single cancer developed very soon after implant and may not be due to the radiation. It is, therefore, not sufficient evidence for conclusions to be drawn.

Up to the present the experiments with beagles and with rodents indicate that the irradiation of lymph nodes with doses ranging from a few rads to a few thousand rads does not induce primary cancer of lymphoid tissues in significant numbers.

5.5 Blood

Park et al. (1972) have reported a chronic reduction of the lymphocyte cell count in the blood of dogs starting within a few months of exposure to plutonium dioxide aerosols. Similar observations have been made by other workers (Buldakov et al., 1969; Ballou et al., 1972).

Leukaemia and other haematopoietic neoplasia have not been found in dog studies at Battelle Northwest Laboratories during the last 20 years (Bair, 1974). The only leukaemias observed are in rodents. Bensteid et al. (1965) found 3 myeloid leukaemias in 26 rats given multiple intravenous injections of plutonium nitrate with a total equivalent amount for man of over 200 μCi; the permissible amount in man is 0.04 μCi. They found one leukaemia in 22 rats given a single intravenous injection of about the same amount of plutonium.

Brooks and Mewhinney (1973) found chromosome aberrations in blood lymphocytes of Chinese hamsters after inhalation of plutonium-238 oxide particles. Presumably this damage was produced in the lymphocytes by alpha irradiation as they passed through the lung capillaries or through the thoracic lymph nodes.

6. BIOLOGICAL EFFECTS IN MAN

6.1 Health of workers exposed to plutonium

Hempelmann et al. (1973) reported on the health of 25 males who were exposed to a range of plutonium compounds 27 years previously under the crude working conditions existing during the production of the original atomic bombs at Los Alamos Scientific Laboratory, New Mexico, USA. The body content estimated from the urinary excretion rates of plutonium for these men ranged from 5 nCi to 420 nCi, that is up to 10 times permissible amounts. They were recently subjected to a comprehensive medical examination and none of the findings could be attributed to the presence of plutonium in the body. Only the diseases and physical changes characteristic of a male population entering the sixth decade were observed.

More recently, Schofield and Dolphin (1974) have reported health statistics for workers at BNFL, Windscale. In a comparison with another BNFL establishment the number of days lost through sickness per year per 100 male employees was about the same. Hence no relevant general health problems appeared to exist at Windscale where large quantities of radioactivity, including tonnes of plutonium, are processed each year. Cancer deaths in the male population at Windscale Works were given for the six years 1967-72. In this period 23 cancers were observed in the working population and over 37 were expected as calculated from national statistics, which were adjusted for age distribution of the workers. The data given by Schofield and Dolphin cannot yet conclusively show that the working levels for plutonium presently accepted are correctly chosen but they do allow a certain cautious optimism.

6.2 Pathological effects

No cancers or detrimental biological effects in man can be unequivocally attributed to plutonium. Lushbaugh and Langham (1962) reported some histological changes they had found near a particle of plutonium embedded in the palm of a man's hand for about four years as showing "a similarity to known pre-cancerous epidermal cytologic changes". The meaning of pre-cancerous changes is far from precise but this statement certainly cannot be used to infer the presence of a cancer in the man's hand at the time of excision.

Dolphin (1971) reported an average finding of 5 dicentrics per thousand cells in blood lymphocytes from eight workers with an average time of exposure to plutonium at work of seven years. They had also received on average 14 rads from external radiation. The observed aberration yield was not significantly different from that of workers who were only exposed to external radiation of about the same amount.

The yields of chromosome aberrations in blood lymphocytes from workers known to be contaminated with plutonium are shown in Table 5. These aberrations are presumably produced as the lymphocytes move close to plutonium deposits; the range of the alpha particles is only 40 μm in tissue, about 4 cell diameters. At present the interpretation of these aberrations in terms of body content of plutonium or in terms of the possibility of a late effect is not yet possible.

7. THERAPEUTIC PROCEDURES FOR THE REMOVAL OF PLUTONIUM FROM THE BODY

It is now well established that intravenous injections or infusions of the chelating agent, diethylenetriaminepentaacetic acid (DTPA), will effectively clear all plutonium from the plasma and some from recent deposits in the bone or liver. However, little plutonium can be removed from these organs when the deposit is of long standing. Experimental work with animals has shown that DTPA must be given very quickly after intake, within minutes, if it is to prevent significant deposition in the bones and liver. This is in agreement with the human data summarised by Durbin (1972) on the rapid removal of injected plutonium citrate from the blood to the bone and liver with 50% leaving the blood within 20 minutes.

There are two main disadvantages of DTPA as a therapeutic agent. First, it is rapidly cleared from the body fluids by excretion through the kidney with a half-life of about 90 minutes in humans, so that multiple injections are needed to maintain effective blood levels for a few days; secondly, it does not cross cell membranes and as most of the colloidal and particulate plutonium is engulfed by cells within a few hours of intake, particularly in the lungs, it is not effective in removing this material.

Attention has been directed in the NRPB laboratories to both these problems and some progress is being made towards solving them. One other factor in the use of DTPA has been opposition to its use in humans on the grounds of toxicity but Smith and Morgan (1974) failed to demonstrate any toxic effects of the substance in animals under all likely conditions of use.

The other technique now being developed for the removal of plutonium after inhalation is lung lavage. This has already been used once on a man who inhaled plutonium following a glove box accident at Rocky Flats, Colorado (McClellan et al., 1972). The technique was only partially successful in this case for only 13% of the initial lung content was removed, but there is no doubt that it must be developed as it offers the best hope of reducing the radiation dose to the lung should a massive inhalation occur. In work with dogs at the Lovelace Foundation (McClellan et al., 1972) about 50% of inhaled plutonium oxide particles were removed by lavage, and Nolibe (1974) working in France has removed up to 90% from baboons.

8. THE RADIOLOGICAL PROTECTION OF PEOPLE EXPOSED TO PLUTONIUM

8.1 Basic standards

It is proposed to review only those dose standards applicable to plutonium. For workers the maximum permissible dose to the whole body, gonads and red bone marrow is 5 rem in a year, as shown in Table 6. This is subject to certain limitations given in ICRP Publication 9 (1966) but of no real consequence for control of plutonium exposures. The maximum permissible dose for individual organs of the body is 15 rem in one year except for the bone, thyroid and skin where the dose is 30 rem per year. A quality factor, Q = 10, to allow for the greater effectiveness of alpha emitters in producing biological damage is used in calculating the dose to individual organs and for bone a modifying factor, N = 5, is also used. In effect, the factor N is used to take into account the evidence from animal experiments that plutonium is between 5 and 10 times more radiotoxic in bone than radium. This point will be discussed in greater detail in the next section. For members of the public the dose limits are 10 times lower than the maximum permissible doses for workers. For purposes of comparison with animal and other data the dose limits are also expressed in rads in Table 6.

8.2 Derivation and adequacy of the basic standards

Radiological protection standards have evolved over the last 50 years since the first recommendation of Mutscheller and Sievert in 1925. They suggested an annual dose limit to the body from external radiation equal to 10% of the dose required to produce erythema. Erythema, skin reddening, was known to be produced by a single dose of about 500 rads from 200 kV X-rays (the term röntgen was used at that time for units of dose). Hence, the first

standard for external radiation was 50 rads per year; this was reduced in 1950 to 15 rads per year and again by ICRP in 1956 to 5 rads per year where the standard currently remains.

This standard is based essentially on experience of human exposure in medicine and in industry. Two other sources of information are available for consideration in setting standards. One source is epidemiological studies of irradiated population groups, such as atomic bomb survivors and radiotherapy patients. The other source is animal experiments. Although there is great uncertainty in extrapolation of data from animal experiments to man, these experiments do facilitate the study of the mechanisms of radiation carcinogenesis, genetic effects, metabolism and the relative toxicities of internally deposited radionuclides.

The first basic exposure standard set as a result of an epidemiological study was that for radium-226. By 1941 sufficient data had been accumulated on the skeletal effects of radium in persons accidentally contaminated during the period 1910-1930 to allow a choice of 0.1 μCi of radium-226 as the maximum safe skeletal content. Although many more data have been accumulated on the health of these radium patients since 1941 the value of 0.1 μCi still remains a radiological protection standard and corresponds to an average dose equivalent of 30 rem per year to the bone.

The toxicity of plutonium-239 relative to radium-226 has been studied at the University of Utah in long-term experiments with beagles. The results of these experiments (May and Dougherty, 1972) show that for equal toxicities in terms of the induction of osteogenic sarcomas, 5 to 10 times more energy must be deposited in the bone from radium-226 compared with plutonium-239. It is generally accepted that plutonium-239 is more effective in producing bone cancers because it is deposited and remains mainly on endosteal surfaces where it is well placed to irradiate osteogenic cells from which the sarcomas originate, whereas some radium becomes buried in bone mineral where there are only a few cells. In effect the modifying factor for calculation of dose equivalent in bone, $N = 5$ for plutonium and $N = 1$ for radium, takes account of the higher radiotoxicity of plutonium relative to radium and this leads to the equivalence of the maximum permissible body contents of 0.1 μCi of radium-226 with 0.04 μCi of plutonium-239, given in ICRP Publication 2. Hence the standard for plutonium-239 is linked with the human radium cases.

Lloyd and Marshall (1972) drew attention to differences between bones in beagles and humans. They made measurements on lumbar vertebrae from man and beagles and found that the surface to volume ratio was a factor of two higher in beagles. They suggested that bone turnover was higher in beagles and from this evidence they claimed that the potential risk for plutonium-239 relative to radium-226 would be expected to be a factor of two higher. In making this

claim several assumptions have to be made regarding, for instance, the representativeness of vertebrae for all bones and the numbers and distribution of cells on bone surfaces in man and in dogs. Hence the observations of Lloyd and Marshall are of great interest but their significance to the relative toxicity of radium and plutonium in man is difficult to assess.

The first quantitative study of the risk of cancer production in an irradiated group was for a group of children and adults who had received radiation to the thyroid, published by Beach and Dolphin (1962). On the assumption of a linear relation between the incidence of thyroid cancer and radiation dose they calculated a risk coefficient for the induction of thyroid cancer. Since this original work many quantitative risk evaluations have been published both by individual scientists and by committees such as UNSCEAR (1972) and BEIR (1972).

A summary of some risk coefficients pertinent to the radiological protection of persons exposed to plutonium is given in Table 7. Goss (1974) has analysed the mortality statistics for the atomic bomb survivors in Japan published by Jablon and Kato (1972) and he found risk coefficients of 100 cancers per million man-rads for all cancers and 30 for leukaemias. In making these risk estimates, allowance was made for 10% more leukaemias not already manifest by 1970. Similarly an allowance was made for all the other cancers but due to their long latent period the number occurring before 1970 was doubled to obtain the ultimate number expected to occur. Goss found that the RBE value for cancer induction by neutrons at Hiroshima was about 5 and it should be noted that the data given in the table are for gamma radiation only. For patients with ankylosing spondylitis treated with 250 kV X-rays data have been published on excess cancers by Court Brown and Doll (1965) and these have been analysed in terms of risk coefficients by Dolphin and Marley (1969) who gave a value of 10 leukaemias per million mad-rads and 5 times this value for all cancers.

In the next line of the table a risk coefficient has been calculated from data on the mean dose to bone for 1417 cases of radium contamination including 71 with bone sarcoma (Rowland, 1973). Most of these people were contaminated during the period 1910-30, often when they were quite young; the studied population therefore involves a large number of man-years at risk. The value for the risk coefficient, 80 cancers per million man-rads (mean dose to bone), may be taken as the upper limit; this is not a truly random survey because there is a greater likelihood of including cancer cases that were less affected by the radium contamination.

Spiess and Mays (1970) reported on the incidence of bone sarcoma in 708 adults injected, mainly during the years 1945-51, with radium-224 (half-life, 3.64 days) for the treatment of tuberculosis, ankylosing spondylitis and

other diseases. They calculated a risk coefficient for this group of 90 cancers per million man-rads (mean dose to bone). An important point made by them was that radium-224 is initially deposited on bone surfaces where it delivers most of the dose due to a short half-life. Hence the spatial distribution of the radiation in the bone is like that from plutonium but the dose from plutonium is spread out in time.

An increased incidence of lung cancer among miners exposed to radon in uranium mines, fluorospar mines, haematite mines and hard-rock mines has been observed for many years. A summary of these exposures of lung to radon together with exposures of lung to thoron in patients injected with thorotrast was given by Lundin et al. (1971). From their summary an average risk coefficient has been obtained by weighting the individual risks by the square root of the number of lung cancers attributed to radiation in each group. The value obtained in this way is 30 lung cancers per million man-rads (mean dose to lung) based on the assumption that the annual risk rates given by Lundin et al. existed for a period of 20 years. If the experimental observation by Lafuma (1974) applies to man then this risk coefficient for lung cancer where the dose would be diffusely distributed throughout the lung may represent a greater risk than could be expected from the same radiation dose from alpha-emitting particulate material. In summary this value of 30 lung cancers per million man-rads may well over-estimate the risk estimate following inhalation of radioactive particles.

If the risk coefficients for human cancers in Table 7 are taken to have equal reliability so that meaningful comparisons can be made, then the important question concerns the risk coefficients of alpha emitters relative to external radiation. With external radiation the exposure is usually prolonged in time and the risk coefficient given in the table must be reduced by a factor in the range 3 to 10 to allow for the lower effectiveness of chronic irradiation. This factor is derived from experimentally-induced cancers and genetic mutations in animals such as thyroid cancer in rats (Doniach, 1964), leukaemia in male mice (Upton et al., 1970) and specific locus gene mutations in the offspring of irradiated male and female mice (Russell, 1965). With this factor, the risk coefficient for leukaemia and all cancers is reduced to the ranges 3 to 10 and 10 to 30 per million man-rads respectively and with the associated permissible annual dose of 5 rads the maximum annual risks at equilibrium are 15 to 50 and 50 to 150 per million per year.

For incorporated alpha emitters the biological effect is less dependent on the dose rate as indicated by the risk coefficients for radium-224 and radium-226 in Table 7. Hence for bone sarcomas induced by radium-226 the upper risk estimate remains unmodified at 80 per million man-rads associated with a permissible annual dose of 3 rads. The permissible annual risk to bone

from radium-226 is 240 and is higher than that for external X- or γ-radiation. The greater effectiveness of plutonium in producing bone sarcomas is taken into account by the lower permissible annual dose, 0.6 rads. The risk coefficient for radium-224 is about the same as that for radium-226 but the dose to bone is quite different in distribution and dose rate. No inference can therefore be drawn from the radium-224 data until studies on its effectiveness in producing bone sarcomas relative to plutonium-239 have been completed in beagles at the University of Utah.

For the lung, the risk coefficient 30 per million man-rads is associated with a permissible annual dose of 1.5 rads and the permissible annual risk at equilibrium for alpha emitters, 45 per million per year, is within the range given above for external γ-radiation. The effectiveness of radon and thoron per unit of radiation dose in producing lung cancer relative to plutonium is not known but can be assumed to be about the same.

In summary, the risk coefficients of the radium alpha emitters in bones of humans appear to be higher by a small factor than those for whole body irradiation by external γ-radiation. It is for general consideration whether this should be taken into account in setting current maximum permissible levels.

From Table 7 is can be seen that the risk coefficients for the rats exposed to plutonium for both bone sarcoma and lung cancer (Figs. 1 and 2) are considerably higher than those derived from observation of human populations. There appears to be no reason for these differences other than species differences. The risk coefficient which may be derived from the beagles injected with plutonium-239 citrate are about 10 times higher than the rat data. At present there is no explanation for the differences between animal and human data but clearly great care must be exercised when extrapolating from one species to another.

8.3 Derived standards for air concentration and annual intake

The important parameters required in the derivation of exposure standards from the basic radiation doses are given in Table 8. The presently used values from ICRP Publication 2 (1959) are given in the first and second lines. Other values which could be derived using the later data given in ICRP Publication 19 (1972) are given in the third and fourth lines. It should be noted that data from Publication 19 have not yet been incorporated in ICRP recommendations. It can be seen in the table that some changes would be introduced if the later data were used for lung clearance to blood and 45% each to liver and bone from blood; these changes are not very significant. The two values from Publication 19 for soluble and insoluble compounds are closer than those in Publication 2. Dolphin (1972) has pointed out that from a practical stand-point the most convenient change in derived standards would be to use a single value for $(MPC)_a$ applicable to all plutonium compounds. This single value could be

somewhere between those for class W and class Y compounds given in the third and fourth lines of the table.

The annual permitted intakes of activity by the oral route are relatively high due to the low gut absorption as already noted in the section on Metabolism.

8.4 Determination of body content from bioassay measurements

Two techniques are available for estimation of the amount of plutonium in the body; the measurement of the daily rate of urinary excretion of plutonium and the measurement of the counting rate over the chest of L X-rays from plutonium-239 and plutonium-240 or 60 keV gamma rays from americium-241, the decay product of plutonium-241.

The body content estimation from urinary excretion data is based on human cases injected with known amounts of plutonium in 1944 and 1945 by Langham (1957) and his associates. The data from these experiments were recently reviewed in depth by Durbin (1971). There are many limitations to these data; first, they were obtained after intravenous injection of plutonium citrate or nitrate compounds into 16 patients and this is not a normal route of entry into the body except in experimental conditions; second, the excreta were collected for only a few days in many cases and for more than 138 days in only 4 cases; third, the data were obtained from terminal patients whose metabolism was in differing degrees abnormal. Nevertheless these are the only data available for relating excretion rate to body content.

The use of these excretion data in estimating body content has been described by Beach and Dolphin (1964), Beach (1973) and Adams and Watts (1974). The formula derived from Langham's original equation by Beach and Dolphin is as follows:-

$$Y_u = (b-1)\ 0.16 \int^{t} \tau^{-b}\ (t-t)^{-0.68}\ d\tau$$

where Y_u is the urinary excretion rate expressed as a percentage of the plutonium intake, τ^{-b} is a function of time representing the transfer of plutonium from the lung to the body, t is in days and b is a constant. To use this formula for the function for transfer into the body a value of b must be assigned, if possible, from knowledge of the intake conditions.

The formulae based on Langham's equation are known to over-estimate the body content in most cases where the exposure occurred some years previously. Schofield and Dolphin (1974) found that the amount of plutonium at death estimated from autopsy tissue samples obtained from persons exposed to plutonium for a number of years was between 1.2 and 8.3 times lower than that calculated from measurements of the excretion rate in all 6 cases for which urine data were available. Lagerquist et al. (1973) also reported the same over-estimation from the urinary excretion data. In addition, Dolphin (1972) has shown that

use of the Langham equation to estimate body content within a few weeks of intake can lead to an over-estimation by a factor which could be as high as 10.

Recently, data on urinary excretion rates have been obtained from two of the original cases injected with plutonium designated as HP-3 and HP-6 (Durbin, 1972). HP-3, a white female, was injected with 0.3 μCi of plutonium citrate in November 1945. She is now 77 years old and her plutonium excretion rate is 1.4×10^{-3}% per day. The other case, HP-6, a white male, was also injected with 0.3 μCi of plutonium citrate and is now 73 years old with a plutonium excretion rate of 2.5×10^{-3}% per day (Rundo et al., 1974). The equation proposed by Langham (1957) predicts a value of 2.9×10^{-4}% per day. Hence the excretion rate is much higher than expected in these two cases and if these data were included in the Langham equation the effect would be to lower the estimates made from urinary measurements on those people who have a long standing body content.

In summary, body content estimates based on Langham's data are likely to be an over-estimate of the true content but many more autopsy data correlated with urinary excretion data are required before Langham's equation can be confidently amended or replaced.

Techniques for the measurement of plutonium in the chest by external counting have been developed over the last 15 years, first using large proportional counters and more recently by a system of combined thin and thick crystals. For either technique the limit of detection is between 5 and 10 nCi of plutonium-239 plus the other alpha-emitting isotopes depending on the amount of absorbing tissue over the rib cage of the individual. This lower limit of detection corresponds to a total radiation dose of about 15 rem to the lung during the following few years. Hence the chest monitoring systems available at the moment are just adequate; more development work is required to increase the sensitivity. As the alpha-emitting isotopes of plutonium, which vary in relative amounts depending on fuel irradiation, emit varying numbers of L X-rays per disintegration, this adds another difficulty in the interpretation of chest monitoring results.

The presence of americium-241, the decay product from plutonium-241, increases the efficiency of detection due to the emission of 60 keV gamma-rays which are less absorbed in the chest wall. If a sample of the inhaled material is available then the ratio of americium-241 to alpha-emitting plutonium isotopes plus plutonium-241 may be determined outside the body and used to estimate the total plutonium plus americium content of the lung. However, caution is necessary for it is known that americium-241 is more transportable than plutonium in the lungs and therefore it cannot be considered as a true tracer for plutonium, particularly over long periods.

Holliday et al. (1970) have described a comprehensive system for the radiological protection of workers from airborne plutonium particles. This

involves routine chest measuring for insoluble plutonium in the lung at six-monthly intervals and annual or six-monthly urine samples taken over five consecutive days for estimation of systemic plutonium.

8.5 Dosimetric model for thoracic lymph nodes

One of the outstanding problems in radiological protection related to all insoluble materials concerns the high accumulation in lymph nodes associated with lung drainage, as indicated by animal experiments. The experiments with beagles by Park et al. (1972) show that on average 40% of the alveolar deposit passes into these lymph nodes and as there appears to be little clearance, this leads to tissue concentrations between 150 and 3000 times higher than those in the lung. In man, the small amount of information available, reviewed in Table 4, does not match such high relative concentrations and an average value is about 10 for lymph nodes relative to lung. This lower value may be due either to the human exposures being more predominantly to soluble compounds of plutonium or to routes of clearance in man following chronic inhalation which do not involve lymph drainage.

The lymph nodes are part of the lymphoid tissue in the body which are estimated to have a total mass of over 2000 g. The kinetics of lymphoid tissue are not well understood but they consist broadly of two fractions; about 700 g of stationary reticulum cells and about 1500 g of mobile lymphocytes (ICRP Reference Man, 1974). Due to the mobility of the lymphocytes the amount of lymphoid tissue irradiated is considerably more than the 16 g of lymph nodes associated with human lung, as reported by Pochin (1966). Hence, the effective concentration in lymphoid tissue is less than that calculated for the thoracic lymph nodes as individual tissues.

The most appropriate dosimetric model to use for the thoracic lymph nodes has not yet been resolved by ICRP but it certainly should not be based on the very high concentrations found in the dogs exposed to large amounts of plutonium dioxide at the Battelle Laboratories. The question of tissue mass to be used in the model and the residence time of the material in the nodes must be carefully chosen and above all the absence of large scale late effects in these lymph nodes, as observed in animal experiments, must be given due regard as no human data are available.

9. HOT PARTICLES

The radiological protection problems associated with insoluble particles of alpha-emitting radionuclides have been known and considered for a number of years (Dolphin, 1964) but recently public attention has been drawn to these problems by a petition submitted to the USAEC by Tamplin and Cochran (1974) which caused comment in the national press. The problems concern the biological effect of high but localised doses. Alpha radiation from

plutonium-239 only penetrates about 40 microns in unit density tissue and consequently only a small volume of cells around the plutonium particles is irradiated, about 2.7×10^{-7} cm^3. The average radiation dose to such a volume around a one micron particle of plutonium-239 oxide is 300 rads per day or about 90000 rads per year. However, radiological protection dose standards cannot be applied to small volumes as they have been derived from observation of the biological effects in the whole body or in body organs.

The arguments used by Tamplin and Cochran were essentially based on the data of Albert et al. (1967) who induced skin cancers in rats either by high doses of beta particles or by electron irradiation. These skin cancers originated in hair follicles and Tamplin and Cochran argued that high doses of the order of 1000 rads or more to the small number of cells in the follicles could induce these cancers. They then transferred this concept from rat hair follicles to the irradiation of small numbers of cells in human lung after the inhalation of plutonium particles. They concluded that lung cancer in humans might be expected following the inhalation and deposition of 2000 particles but they were unable to present evidence of cancer of the lung following such small intakes either in animals or in humans.

The amount of plutonium-239 in the lung which would deliver a mean dose at the permissible rate of 15 rems per year is 0.016 μCi. Some estimates of the number of cells irradiated by this amount of activity are given in Table 9 for three particle sizes. The table shows clearly that if 0.016 μCi is divided into large particles then fewer cells would be irradiated, considerably less than if this amount of activity was divided into very small particles. In making this calculation the cell volume was taken to be 10^{-9} cm^3 and no allowance was made for energy absorption by the air inside the lung. Hence, if the radiotoxicity is related to the number of cells irradiated then particles are less hazardous than diffuse deposits in the lung or elsewhere in the body. Lafuma (1974) has studied life-shortening and lung cancer in rats following inhalation of various actinides. He reported finding greater life-shortening and more lung cancer (Lafuma, 1974) in animals with curium-242 compared with those given an equal average lung dose from plutonium-239. Lung autoradiographs show that the curium-242 is diffusely distributed in the lung whereas the plutonium-239 in his experiments was in particulate form. This experimental finding supports the view that the risk of biological damage from a given mean dose to lung increases as the number of cells irradiated increases and is at variance with the "hot spot" hypothesis advocated by Tamplin and Cochran.

Other evidence which shows that particles of plutonium dioxide are less effective at producing biological damage than equal amounts of diffusely distributed plutonium comes from the work of Brooks et al. (1973). They studied the chromosome aberrations induced by plutonium in hamster liver cells,

in vivo. Plutonium citrate produced a linear increase of aberrations with an average radiation dose to the liver which was greater than the aberration yield produced by plutonium dioxide particles at the same average dose levels.

There are more data from animal experiments available to support the hypothesis that for a given average organ dose the particles are less effective than uniform distributions. These have been reviewed by Bair et al. (1974).

In summary, there is no biological evidence available at present which suggests that "hot spots" carry a higher risk of cancer induction. Hence there is no necessity to change from the present system of using average dose to organs or tissues. However, it would be prudent to continue research into the biological effects of non-uniform dose distributions within organs.

10. PLUTONIUM-238 OXIDE

Plutonium-238 with a half-life of 86 years is an important heat source used to drive long-lasting thermoelectric generators which have applications as implanted batteries for human heart pacemakers, for the development of artificial hearts and for space exploration. Worldwide public attention was attracted to this material when 17,000 Ci were dispersed into the atmosphere during an abortive rocket launch in 1964 by the USA.

The radiation dose rate in an 80 micron diameter sphere of unit density tissue around a one micron diameter plutonium-238 oxide particle is about 93000 rads per day. The effect of this high emission of energy is not only to raise the temperature of the particle but also to cause radiolytic action in and around it which tends to break up the particle. Experimental work with animals shows that inhaled material diffuses freely into body tissues, for example, Willard and Park (1970) found only 4% of the terminal body content in the lungs of dogs 23 months after inhalation of plutonium-238 oxide; 63% was in the skeleton and 23% in the liver which is a translocation even greater than that expected for plutonium-239 citrate.

For radiological protection purposes all plutonium-238 compounds should therefore be considered as soluble.

11. PLUTONIUM IN THE ENVIRONMENT

11.1 Sources and chemical form

Some sources of plutonium either worldwide or local in the environment are:-

(a) from nuclear weapons tests which have dispersed over 320 kCi of plutonium-239 and plutonium-240 throughout the world;

(b) dispersal of 17 kCi of plutonium-238 from a nuclear-powered battery in a space rocket which burnt up in the atmosphere in 1964;

(c) local dispersal of plutonium-239 at Palomares and Thule as a result of nuclear weapons breaking up in crashes of US bombers;

(d) local dispersal at the nuclear weapons test site in Nevada; this includes experimental release in operation "Rollercoaster" (Wilson and Terry, 1968);

(e) liquid waste disposal into the Irish Sea from Windscale.

The predominant chemical form of the plutonium in the atmosphere and on the ground from these sources is oxide and in the marine environment, from liquid waste discharge, hydroxide. Sufficient data are available from these dispersals to enable some predictions to be made of the effects of an accidental release of plutonium oxide from a reactor, chemical plant or in a transport accident. Some of these effects will be discussed in the succeeding sections.

11.2 Routes to man

There are two principal routes for plutonium in the environment back to man; by inhalation, either from a cloud of airborne particles during the initial dispersal or from resuspension of plutonium already deposited on the ground or vegetation; by oral ingestion of foodstuffs with surface contamination or possibly incorporated in vegetation.

The amount of plutonium inhaled directly from the cloud immediately after the release will depend on factors such as air concentration, its variation with time and breathing rate of the exposed persons. If the release is of very short duration there is little that can be done to avoid or reduce exposure. In a release of longer duration some exposure to the population could be avoided in the immediate vicinity by evacuating the area.

In operation "Rollercoaster" (Wilson and Terry, 1968) sheep, dogs and burros, 300 animals in all, were exposed to airborne plutonium oxide generated by a non-nuclear explosion. Measurements of initial lung deposition and accumulation due to continued exposure to resuspended activity agreed well with the amounts predicted from measurements of airborne plutonium concentrations and particle size distributions. This was reassuring, for in previous experimental work of this type, code named Test Group 57, the animals had accumulated more in the lung than predicted from measurements of concentration in the air (Cowan, 1960). One important result from this work was that animals left in the test area for 160 days after the release did not accumulate plutonium in the lung due to resuspension of ground deposits. It had been expected that exposure to resuspended plutonium from the surface would result in a significant increase in lung content. From this work it seems that the amount of plutonium resuspended cannot be correlated with the levels of ground deposition (Stannard, 1973).

The amount of resuspension may be measured in terms of a resuspension factor which is defined as the ratio of the airborne concentration, $\mu g/m^3$ to the average ground contamination $\mu g/m^2$. Measured values of this factor on the Nevada test site ranged from 10^{-2} to 10^{-11} m^{-1} (Langham, 1971) and this wide variability shows the difficulty of predicting airborne concentrations from resuspension factors and measurements of ground surface concentrations.

The route back to man via plants and vegetation is not favourable for plutonium transfer. Fractional uptake by plants from contaminated soil or other growth media is of the order of 10^{-3} to 10^{-4} on a dry weight basis (Langham, 1971). This discrimination coupled with gut uptake of only 10^{-4}% in humans makes this food pathway highly selective against plutonium.

One potential pathway through the food chain to man is being investigated by Wildung et al. (1974). Although previous studies indicate that plutonium is insoluble and not accumulated by plants, there is a remote possibility that naturally occurring chelating agents arising from organic decomposition could complex plutonium in the soil into a soluble form. More research is needed on this topic and it must be extended to include feeding the contaminated plants to animals in order to ascertain if the high gut discrimination applies to this plutonium complex.

11.3 Aquatic environment

Plutonium normally found in the aquatic environment is associated with particles and sediments probably adsorbed in an insoluble form. Marine invertebrates which form a food supply both to man and to fish may spend much time close to or in these sediments and could be exposed to high concentrations. At Bikini Atoll where many nuclear weapons were tested invertebrates were sampled and the mean level was 2350 pCi/kg wet weight and at Thule after the accident in which a large amount of plutonium was dispersed in 1968, the levels ranged from 95 to 8000 pCi/kg. In a more normal situation around Cape Cod in 1970 the mean values were found to be between 0.1 and 3.5 pCi/kg. Concentration factors in these invertebrates can only be satisfactorily estimated for an equilibrium situation which does not exist after an accident but values have been estimated to lie between 300 and 3000.

In fish, concentration factors relative to sea water range between 20 and 500 for bone, 50 and 170 for liver and 1 to 13 in muscle (Noshkin, 1972). It is noted that the normally edible parts of the fish have the lowest concentration but there are special situations where some bone may be eaten such as in tinned salmon or sardines.

Thus in the marine environment concentration of plutonium can occur. Further information is needed however about the chemical form of the plutonium in those species of fish eaten by man and about the degree of absorption of these forms from the human gut.

11.4 Experience at Palomares

Following the dispersal from nuclear weapons by a non-nuclear explosion at Palomares, crops were removed from the area where plutonium contamination was above 5 $\mu g/m^2$ and the ground was ploughed to a depth of 25 cm to minimise the possibility of resuspension. The area involved was 550 acres. At ground contamination levels over 500 $\mu g/m^2$, the vegetation and top soil were stripped off and put into drums for return to the USA. About 5.4 acres were involved (Langham, 1968).

In follow-up studies of 100 potentially contaminated residents no detectable amounts of plutonium were found in the chest but the lower limit of detection was initially 40 nCi, and only later reduced to 16 nCi. Urine samples collected over 24-hour periods were all found to be negative but this is not surprising as the plutonium was insoluble. Thus residents may have been contaminated but not at detectable levels.

Measured air concentrations in the village even under conditions of high wind velocities were low. In 1966 a value of 0.38×10^{-15} $\mu Ci/cm^3$ was obtained and in 1967 the value was lower at 0.09×10^{-15} $\mu Ci/cm^3$. For continuous exposure to insoluble plutonium the air levels given for adult workers in ICRP Publication 2 (1959) are 1×10^{-11} $\mu Ci/cm^3$ which is 10^4 to 10^5 times higher than the measured value at Palomares. If the air concentration measurements are representative of continuous exposure then the hazard is very low. By implication the consequences of dispersal of a few kilograms of plutonium dioxide in a limited area of undeveloped or agricultural land are not too serious but in a developed area the cost of decontamination could be very high.

11.5 Plutonium in man

Worldwide contamination of the atmosphere from nuclear weapons tests has produced measurable amounts of plutonium in the air at ground level. For example, in 1965 and 1966 in mid-northern latitudes the measured air concentration was 10^{-4} pCi/m^3 (Parker, 1973). Using the metabolic models in ICRP Publication 19, it is possible to calculate tissue deposition from continuous breathing of this concentration. The daily alveolar deposition would be 3×10^{-4} pCi and if the retention half-life is 500 days then the equilibrium amount in the lung is about 0.2 pCi. Transfer from lung through the blood to the bone would amount to 0.5×10^{-4} pCi per day and in 10 years the accumulated amount in the skeleton would be about 0.2 pCi.

Harley (1971) published data on tissue concentrations in humans. He found an average concentration in the lung of 0.45 pCi/kg, the range in bone 0.04 to 0.12 pCi/kg and a daily dietary intake of 7×10^{-3} pCi. The values for lung and bone are in reasonable agreement with those calculated from the air concentration levels in the previous paragraph. This agreement gives some confidence in the ICRP models.

The daily transfer from the diet to the blood is the daily intake, 7×10^{-3} pCi, multiplied by the gut discrimination factor, 10^{-6}, which equals 7×10^{-9} pCi. This is an extremely low daily intake to the blood and even after 50 years this would only lead to a bone level of 8×10^{-6} pCi/kg according to the ICRP metabolic model. However, dietary contamination from plutonium in fallout under most circumstances has probably had a negligible effect on the concentrations found in the bone of humans.

12. CHEMICAL TOXICITY

Statements have recently been made in the popular scientific press that plutonium is not only toxic due to its radioactivity but also due to its chemical nature. There are no experimental data available on the chemical toxicity of plutonium-239, 240 and 242, the long-lived alpha-emitting isotopes, but some inference can be drawn from experimental data on related elements.

The most stable oxidation state of plutonium is +4 in biological fluids and it has an ionic radius of 0.90 Å. Related elements are cerium and neptunium which both have stable oxidation states at +4 and both have ionic radii of 0.92 Å which is close to that of plutonium. It is noted that neptunium has no stable isotopes but neptunium-237 has a half-life of 2.2×10^6 years and this is sufficiently long to allow the acute symptoms of chemical toxicity to be separated from the late effects of irradiation.

The acute and sub-acute toxic effects of cerium in the liver of rats are just detectable after the intravenous injection levels of 4 mg/kg of body weight (Snyder et al., 1959) and for neptunium at 3 mg/kg of body weight (Markham, 1967). These levels correspond to an intravenous intake in a 70 kg man of over 200 mg and may be compared with the maximum permissible body burden (ICRP Publication 2, 1959) of 0.64 μg (0.04 μCi) of the alpha-emitting isotopes plutonium-239, 240 and 242. Hence, the acceptable plutonium mass in the body from radiological protection considerations is 330,000 times less than that which might be expected to produce detectable chemical effects in the liver.

The mass of inhaled plutonium, determined from dog experiments, which might be expected to cause death within days due to severe oedema and haemorrhage in the lung is over 8 mg (500 μCi). This mass of plutonium required to produce acute effects due to radioactivity is still less than the estimated 200 mg which might be expected to just cause toxic effects when injected into the blood. It should be noted that it would not be possible to transfer from the lungs into the liver sufficient plutonium to cause chemical toxicity.

It must be concluded that the mass of plutonium-239, plutonium-240 or plutonium-242 required to produce either acute or late biological effects due to alpha activity is considerably lower than that expected to cause toxic effects in the liver due to its chemical nature.

13. SUMMARY

For the proper protection of persons exposed to plutonium isotopes and higher actinides, there are five important requirements:-

(1) Evaluation and acceptance of basic dose standards for the exposure of body organs, particularly bone, liver and lung

(2) Derivation of standards for maximum permissible air concentrations and annual intakes

(3) Facilities for the assessment of the amount in the body by measurements of urinary excretion rate, or by external counting of X- or γ-rays emitted by incorporated plutonium

(4) Availability of methods for the treatment of over-exposed cases

(5) Derivation of acceptable ground contamination levels in areas where plutonium has been accidentally released.

All these topics have been discussed in this report. It is noted that knowledge is better founded in some of these topics than in others.

More consideration of the basic dose standards is required, especially in terms of the relative effectiveness of external radiation and incorporated alpha emitters. It is unlikely that information on cancer incidence due to exposure of groups of people to plutonium will become available in the next few decades therefore the greatest effort must be made to study the biological effect of plutonium relative to radium in many animal species. In this way the fullest use can be made of the human experience with radium-226 and radium-224.

The second topic involves knowledge of a metabolic model for the translocation to and retention in body organs of plutonium entering the body after deposition in the lung or at a wound site. The model proposed by the ICRP Task Group and given in ICRP Publication 19 (1972) is adequate and has the advantage of not over-emphasising a single organ for it implicates bone, liver and lung, whereas in ICRP Publication 2, the liver was not considered a very vulnerable tissue. The major problem in the metabolic model concerns the lymph nodes and lymphoid tissue associated with lung clearance where there are uncertainties both about the retention and about the mass of these tissues in the human.

Measurement of the amount present in the body, the third topic, gives rise to difficulties, but improvements are continually being made in the techniques. Reliability of the interpretation of urinary excretion rate in terms of plutonium body content will improve as more data become available on body content at death, made from analyses of tissues obtained at post mortem,

and its relation to the urinary excretion rates measured before death. So far all the indications are that the current methods overestimate body content. The technique of measuring body content by external counting of X-rays from plutonium or gamma-rays from americium-241 are improving continually but still more improvements are required to obtain a satisfactory sensitivity for detecting the amounts of these materials in the lung.

The fourth topic is very important in cases of accidental over-exposure. The new technique of lung lavage offers a good possibility of removing insoluble compounds from the lung. Inside the body, the problems of removal of plutonium from intracellular sites has not been successfully solved and more research is required in this field.

The final topic listed above will be important if large quantities of plutonium are accidentally released to the environment. There is some experience of ground contamination at the sites of weapons tests and from the accidents at Palomares and Thule, and this will be very useful in setting acceptable ground contamination levels. Naturally, these levels would vary from place to place and country to country depending upon factors such as climate, ground usage, population, density, etc.

Finally, the best indicator of the adequacy of radiological protection standards is the health of those people who are exposed to these materials. It is therefore important that health statistics as well as information on work histories and smoking habits be collected and analysed for these individuals.

14. SUMMARY OF NRPB RESEARCH PROGRAMMES

The Board recognised the biological problems posed by the large-scale use of plutonium and the associated higher actinides in the nuclear power programme and consequently set up an extensive biological research programme. The main areas of research are:-

1. Study of the comparative metabolism of plutonium, americium and curium in animals following entry by four main routes; inhalation, ingestion, wounds and intravenous injection. The amounts of activity used are comparable with a few body burdens in humans. The effect of factors such as particle size, chemical form, solubility and binding in biological fluids on the translocation process are being studied. In addition, attempts are being made to elucidate the fundamental mechanisms of urinary and faecal excretion.

2. Study of the biological consequences of deposited plutonium in lung, bone, liver, lymph nodes, skin and gonads.

3. Development of techniques and regimes for the removal of plutonium from the body. These include:
   (i) removal of plutonium by injection or inhalation
   (ii) development of techniques for washing insoluble particles from the lung
   (iii) development of methods for increasing the release of intracellular plutonium.

4. Assessment of dose from internally-deposited actinides:
   (i) improvement of methods for estimating body content by external counting over the chest and by measurement of urinary excretion rate
   (ii) analysis of selected autopsy samples in order to estimate body content at death.

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16. REFERENCES

Adams, N. and Watts, L.M.
NRPB Report (In Preparation)

Albert, R.E., Burns, F.J. and Heimback, R.D. The effect of penetration depth of electron radiation on skin tumour formation in the rat. Radiat. Res., 30, 515 (1967).

Bair, W.J. Toxicology of Plutonium. Advan. Radiat. Biol., 4, 255, (1974).

Bair, W.J. and Thompson, R.C. Plutonium: Biomedical Research. Science, 183, 715 (1974).

Bair, W.J., Richmond, C.R. and Wachholz, B.W. A radiobiological assessment of the spatial distribution of radiation dose from inhaled plutonium. Wash. 1320, DBER, USAEC, Washington. (1974).

Bair, W.J., Ballou, J.E., Park, J.F. and Sanders, C.L. Plutonium in soft tissues with emphasis in the respiratory tract. IN Uranium, Plutonium and Transplutonic Elements. Edited by H.C. Hodge, J.N. Stannard and J.B. Hursh, Berlin, Springer Verlag (1973).

Bair, W.J. and Willard, D.H. Plutonium inhalation studies. IV Mortality in dogs after inhalation of $Pu^{239}O_2$. Radiat. Res., 16, 811 (1962).

Ballou, J.E., Park, J.F. and Morrow, W.G. On the metabolic equivalence of ingested, injected and inhaled ^{239}Pu citrate. Health Phys., 22, 857, (1972).

Beach, S.A. SEBEACH, a digital computer program for the estimation of body content of plutonium from urine data. Health Phys., 24, 9 (1973).

Beach, S.A. and Dolphin, G.W. A study of the relationship between X-ray dose delivered to the thyroids of children and the subsequent development of malignant tumours. Phys. Med. Biol., 6, 583 (1962).

Beach, S.A. and Dolphin, G.W. International Atomic Energy Agency, Assessment of radioactivity in man. Proc. Symposium on the Assessment of Radioactive Body Burdens in Man, Heidelberg, May 11-16, 1964, Vienna, IAEA/ILO/WHO, p.603 (1964).

BEIR Report. National Academy of Sciences/National Research Council. The effects on populations of exposure to low levels of ionising radiation. Report of the Advisory Committee on the Biological Effects of Ionising Radiations, Washington, National Academy of Sciences (1972).

Berstead, J.P.M., ~~Chiu, B.~~, Taylor, D.M. and Sowby, F.D. The carcinogenic effects of americium-241 and plutonium-239 in the rat. Brit. J. Radiol., 38, 920 (1965).

Boocock, G. and Popplewell, D.S. Distribution of plutonium in serum proteins following intravenous injection into rats. Nature 208, 282, (1965).

Brooks, A.L. and Mewhinney, J.A. The frequency of chromosome aberrations in the blood lymphocytes of the Chinese hamster following inhalation of plutonium-238 dioxide particles. Annual Report of the Inhalation Toxicology Research Inst. for 1972-73, p.230, LF-46, Lovelace Foundation, Albuquerque (1973).

Brooks, A.L., Retherford, J.C. and McClellan, R.O. The effect of plutonium-239 dioxide particle, number and size on the frequency and distribution of chromosome aberrations in the liver of the Chinese hamster. Annual Report of the Inhalation Toxicology Research Inst. for 1972-73, p.233, LF-46 Lovelace Foundation, Albuquerque (1973).

Buldakov, L.A. and Lyubchansky, E.R. Report ANL-trans-864, Argonne National Laboratory, Argonne, Illinois. (translated from a Russian Report) (1970).

Buldakov, L.A., Lyubchansky, E.R., Moskalev, Yu.I. and Nifatov, A.P. IN Problemy Toksikologii Plutoniya Atomizdat, Moscow, 1969. Translated (Problems of plutonium toxicology) by A.A. Horvath, Lovelace Foundation, Albuquerque, New Mexico. USAEC LF-7R-41, p.149 (1970).

Buldakov, L.A., Nifatov, A.P., Erokhin, R.A. and Filippova, L.G. IN Remote after effects of radiation damage. Ed. Yu.I. Moskalev. pp.350-355. Atomizdat, Moscow. (Translated in AEC-tr-7387, 381-387)(1971)

Campbell, E.E., Milligan, M.F., Moss, W.D., Schulte, H.F. and McInroy, J.F. Plutonium in autopsy tissue. Los Alamos Scientific Laboratory Document, LA-4875. (1973).

Court Brown, W.M. and Doll, R. Mortality from cancer and other causes after radiotherapy for ankylosing spondylitis. Brit. Med. J., ii, 1327 (1965).

Cowan, M. Plutonium contamination from one-point detonation. Report WT-1510, Sandia Corp. Albuquerque, New Mexico, (1960).

Dolphin, G.W. International Atomic Energy Agency. Assessment of radioactivity in man. Proc. Symposium on The Assessment of Radioactive Body Burdens in Man, Heidelberg, May 11-16, 1964, Vienna, IAEA/ILO/WHO, p.589 (1964).

Dolphin, G.W. and Marley, W.G. Risk evaluation in relation to the protection of the public in the event of accidents at nuclear installations. Harwell, UKAEA Health and Safety Branch, AHSB(RP)R93 (1969).

Dolphin, G.W. The biological problems in the radiological protection of workers exposed to ^{239}Pu. Health Phys., 20, 549, (1971).

Dolphin, G.W. Problems associated with setting safe levels for working with plutonium. Health Phys., 22, 937 (1972).

Doniach, I. Effects including carcinogenesis of iodine-131 and X-rays on the thyroid of experimental animals. Biology of Radioiodine p.277 (Ed . L.K. Bustad) Pergamon Press, Oxford. (1964).

Durbin, P.W. Plutonium in man: A twenty-five year review. California University, Berkeley. Donner Laboratory, Report No. UCRL 20850, June (1971).

Durbin, P.W. IN Radiobiology of Plutonium, p.469, Edited by B.J. Stover and W.S.S. Lee. J.W. Press, University of Utah (1972).

Erokhin, R.A., Koshurnikova, N.A., Lemberg, V.K., Nifatov, A.P. and Puzyrev, A.A. Gig. Tr. Prof. Zabol., 13 (5), 61, (1969).

Goss, S.G. The risk of death from radiation-induced cancer as estimated from the published data on the Japanese atomic bomb survivors. Harwell, National Radiological Protection Board, NRPB-R20 (1974).

Harley, J.H. Worldwide plutonium fallout from weapons tests. IN Environmental Plutonium Symposium. Los Alamos Scientific Laboratory, Report LA-4756 (1971).

Hempelmann, L.H., Richmond, C.R. and Voelz, G.L. Twenty-seven year study of selected Los Alamos plutonium workers. Los Alamos Scientific Laboratory, Report LA-5148-MS (1973).

Holliday, B., Dolphin, G.W. and Dunster, H.J. Radiological protection of workers exposed to airborne plutonium particulate. Health Phys., 18, 529 (1970).

International Commission on Radiological Protection. Permissible Doses for Internal Radiation. ICRP Publication 2, Pergamon Press (1959).

International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection. ICRP Publication 9, Pergamon Press (1966).

International Commission on Radiological Protection. Metabolism of Compounds of Plutonium and Other Actinides. ICRP Publication 19, Pergamon Press (1972).

International Commission on Radiological Protection. Reference Man. (To be published).

Jablon, S. and Kato, H. Mortality among A-bomb survivors 1950-70. Hiroshima Atomic Bomb Casualty Commission, JNIH-ABCC Life Span Study, Report No. 6 (1972).

Jee, W.S.S. Distribution and toxicity of ^{239}Pu in bone. Health Phys., 22, 583 (1972).

Lafuma, J. La contamination radioactive interne. Paper presented at the French Society for Radiation Protection Paris (1974) and Personal Communication.

Lagerquist, C.R., Hammond, S.E., Bokowski, D.L. and Hylton, D.B. Distribution of plutonium and americium in occupationally exposed humans as found from autopsy samples. Health Phys., 25, 581 (1973).

Langham, W.H. The application of excretion analyses to the determination of body burden of radioactive isotopes. IN The Measurement of Body Radioactivity, papers read at a conference held at Leeds April 16-17, 1956 p.95. Brit. J. Radiol., supplement no. 7 (1957).

Langham, W.H. Plutonium distribution as a problem in environmental science IN Proc. Symposium on Environmental Plutonium, Los Alamos Scientific Laboratory, August 4-5, 1971. Los Alamos Scientific Laboratory Report LA-4756, December (1971).

Langham, W.H. The problem of large area plutonium contamination. Seminar paper 002 Bureau of Radiological Health, Public Health Service US Department of Health, Education and Welfare (1968).

Lebel, J.L. and Watters, R.L. Study of the translocation of plutonium and americium from puncture wounds. Fourth International Progress Report, June 16, 1971 - June 15, 1972. Colorado State University, Fort Collins, Dept. Radiology and Radiation Biology, COD-1787-16 (1972).

Lister, B.A.J., Morgan, A. and Sherwood, R.J. Excretion of plutonium following accidental skin comtamination. Health Phys., 9, 803 (1963).

Lloyd, E. and Marshall, J.H. IN Radiobiology of Plutonium. p.377. Edited by B.J. Stover and W.S.S. Lee. J.W. Press, University of Utah (1972).

Loveless, B.W. Personal Communication (1974).

Lundin, F.E., Wagoner, J.K. and Archer, V.E. Radon daughter exposure and respiratory cancer quantitative and temporal aspects. Virginia USA, National Institute for Occupational Safety and Health and National Institute of Environmental Health Sciences, Joint Monograph No. 1 (1971).

Lushbaugh, C.C. and Langham, J. A dermal lesion from implanted plutonium. Archs. Dermat. 86, 461, (1962).

McClellan, R.O. and Rupprecht, F.C. Fission product inhalation program. Annual Report (1972), October 1, 1971 - September 30, 1972. Report No. LF-45, Albuquerque, Lovelace Foundation (1972).

Mahlum, D.D. Neptunium-237 toxicity in rats II Intracellular distribution of neptunium and cerium in rat liver. Toxicology and Applied Pharm. 11, 264-271 (1967).

Mays, C.W. and Dougherty, T.F. Progress in the beagle studies at the University of Utah, Health Phys. 22, 793 (1972).

Mays, C.W., Lloyd, R.D. and Atherton, D.R. Personal Communication (1974).

Mercer, T.T. On the role of particle size in the dissolution of lung burdens. Health Phys., 13, 1211 (1967).

Morrow, P.E. and Casarett, L.J. An experimental study of the deposition and retention of plutonium-239 dioxide aerosol. IN Inhaled Particles and Vapours, p.167. Edited by C.N. Davies, New York, Pergamon Press (1961).

Mutscheller, W.H. Am. J. Roentgenol. Radium Therapy 13, p.65 (1925).

Nolibe, D. CEA, Montrouge. Personal Communication (1974).

Norcross, J. A. and Newton, C.E. Jr. US transuranium registry: a progress report. Health Phys., 22, 887 (1972).

Norwood, W.D., Norcross, J.A., Newton, C.E., Hylton, D.B. and Lagerquist, C. IN Radionuclide Carcinogenesis. Proceedings of the 12th Annual Hanford Biology Symposium at Richland, Washington, May 10-12, 1972. Oak Ridge, Tennessee, USAEC Technical Information Centre, AEC Symposium Series 29, p.465 (1973).

Noshkin, V.E. Ecological aspects of plutonium dissemination in aquatic environments. Health Phys., 22, 537 (1972).

Park, J.F., Bair, W.J. and Howard, E.B. IN Pacific Northwest Laboratory, Annual Report for 1967 to the USAEC Division of Biology and Medicine, Vol. 1. Biological Sciences, p.322. BNWL-714 Vol. 1. Battelle Northwest Laboratories, Richland, Washington (1968).

Park, J.F., Bair, W.J. and Busch, R.H. Progress in beagle dog studies with transuranium elements at Battelle Northwest. Health Phys., 22, 803 (1972).

Parker, H.M. Plutonium, Industrial Hygiene, Health Physics and related aspects in Uranium, Plutonium and Transplutonic Elements, Chapt. 14, p.613, Ed. by H.C. Hodge, J.N. Stannard and J.B. Hursh, Berlin, Springer Verlag, (1973).

Pochin, E.E. The mass of the tracheobronchial lymph glands. Health Phys., 12, 563 (1966).

Popplewell, D.S., Stradling, G.N. and Ham, G. The determination of plutonium in urine by ultrafiltration. Harwell, National Radiological Protection Board, NRPB-R17 (1973).

Richmond, C.R. and Sullivan, E.M. Annual Report of Biomedical and Environmental Research Programme LA-5633-PR p.30, Los Alamos Scientific Laboratory, New Mexico (1974).

Rowland, R.E. Radiological and Environmental Research Division Annual Report, Argonne National Laboratory Report ANL-8060, Part II, Appendix A Exposure data for radium patients. p.289, Argonne, Illinois (1973).

Rundo, J., Starzyk, P.M. and Sedlet, J. The excretion rate of plutonium 10,000 days after acquisition. Fifth International Congress of Radiation Research, Seattle, July (1974).

Russell, W.L. Studies in mammalian radiation genetics. Nucleonics, 23, p.53, (1965).

Sanders, C.L. The distribution of inhaled plutonium-239 dioxide particles within pulmonary macrophages. Archives of Environmental Health, 18, 905 (1969).

Sanders, C.L. and Adee, R.R. Ultrastructural localisation of inhaled $^{239}PuO_2$ particles in alveolar epithelium and macrophages. Health Phys., 18, 293, (1970).

Sanders, C.L., Adee, R.R. and Jackson, T.A. Fine structure of alveolar areas in the lung following inhalation of $^{239}PuO_2$ particles. Arch. Environ. Health, 22, 525 (1971).

Sanders, C.L. and Jackson, T.A. Induction of mesotheliomas and sarcomas from "hot spots" of $^{239}PuO_2$ activity. Health Phys., 22, 755 (1972).

Sanders, C.L. Personal Communication (1972).

Schofield, G.B. Personal Communication (1974).

Schofield, G.B. and Dolphin, G.W. UK experience on the medical aspects of radiological protection of workers handling plutonium. Annals. Occup. Hyg., (In Press).

Schofield, G.B., Howells, H., Ward, F., Lynn, J.C. and Dolphin, G.W. Assessment and management of a plutonium contaminated wound case. Health Phys. 26, 541 (1974).

Smith, H. and Morgan, R.M. The effect of acute and sub-acute treatment with diethylenetriaminepentaacetic acid on the hepatic function of mice. Toxicology Vol. 2 p.43 and p.153 (1974).

Snyder, F., Cress, E.A. and Kyker, G.C. Liver lipid response to intravenous injection of rare earths in rats. Journal of Lipid Research 1, 125-131 (1959).

Spiess, H. and Mays, C.W. Bone cancers induced by ^{224}Ra (Th X) in children and adults. Health Phys., 19, 713 (1970).

Stannard, J.N. Plutonium in the environment. IN Uranium, Plutonium and Transplutonic Elements, p.669. Edited by H.C. Hodge, J.N. Stannard and J.B. Hursh, Berlin, Springer Verlag (1973).

Stather, J.W. and Howden, S. The effect of chemical form on the clearance of plutonium-239 from the respiratory system of the rat. Health Phys. (In Press).

Stather, J.W., Carter, R.F. and Howden, S. A method for investigating the metabolism of the transportable fraction of plutonium aerosols. Phys. Med. Biol. (In Press).

Stevens, W., Bruenger, F.W. and Stover, B.J. In vivo studies on the interactions of PuN with blood constituents. Rad. Res., 33, 490 (1968).

Stover, B.J., Atherton, D.R., Bruenger, F.W. and Buster, D.S. Further studies of the metabolism of Pu^{239} in adult beagles. Health Phys., 8, 589 (1962).

Tamplin, A.R. and Cochran, T.B. 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. Washington, National Resources Defense Council (1974).

Taylor, D.M. The metabolism of plutonium in adult rabbits. Brit. J. Radiol., 42, 44 (1969).

Taylor, G.N., Jee, W.S.S., Williams, J.L. and Shabestari, L. IN Radiobiology of Plutonium, p.105. Edited by B.J. Stover and W.S.S. Lee. J.W. Press, University of Utah (1972).

UICC Committee on Professional Education (eds). p.167 IN Clinical Oncology, A Manual for Students and Doctors. Springer-Verlag, Berlin (1973).

UNSCEAR Ionising Radiations: Levels and Effects. New York, United Nations, (1972).

Upton, A.C., Randolph, M.L. and Conklin, J.W. Late effects of fast neutrons and gamma rays in mice as influenced by the dose rate of irradiation; induction of neoplasia. Rad. Res. 41, 467 (1970).

Vaughan, J. Distribution, excretion and effects of plutonium as a bone seeker IN Uranium, Plutonium and Transplutonic Elements. Edited by H.C. Hodge, J.N. Stannard and J.B. Hursh, Berlin, Springer Verlag (1973).

Watts, L.M. Determination of the rates of clearance of insoluble compounds of plutonium from the lung. Harwell, National Radiological Protection Board, NRPB-R22 (1974).

Weeks, M.H., Katz, J., Oakley, W.D., Ballou, J.E., George, L.A., Bustad, L.K. Thompson, R.C. and Kornberg, H.A. Further studies on the gastrointestinal absorption of plutonium. Rad. Res. 4, 339 (1956).

Wildung, R.E., Garland, T.R., Dracker, M. and Schneiderman, G.S. IN Pacific Northwest Laboratory Annual Report for 1973 to the USAEC Division of Biology and Medicine, Richland, Washington, Battelle Northwest, BNWL-1850 (Part 2) (1974).

Willard, D.H. and Park, J.F. IN Pacific Northwest Laboratory Annual Report for 1969 to the USAEC Division of Biology and Medicine. Vol. 1, Life Sciences. Part 1. Biomedical sciences edited by R.C. Thompson, Richland, Washington, Battelle Northwest, BNWL-1306 (Part 1) (1970).

Wilson, R.H. and Terry, J.L. Plutonium uptake by animals exposed to non-nuclear detonation of plutonium-bearing weapon simulant. Report No. POR-2512. Washington, Defense Atomic Support Agency (1968).

TABLE 1. SOME IMPORTANT TRANSURANIC NUCLIDES PRODUCED IN NUCLEAR REACTORS

Isotope	Half-life	Mode of Formation	Principal Mode of Decay	Relative Activities at fuel irradiations		Spontaneous fissions/min per d/min
				1000 Mwd/t	24000 Mwd/t	
^{238}Pu	86.4 yr	$^{238}U(n,2n)^{237}U \xrightarrow[6.7\ d]{\beta} {}^{237}Np(n,\gamma)^{238}Pu$; $^{239}Pu(n,2n)^{238}Pu$	α, γ-rays	-	3.8	1.8×10^{-11}
^{239}Pu	24,400 yr	$^{238}U(n,\gamma)^{239}U \xrightarrow[23\ m]{\beta} {}^{239}Np \xrightarrow[2.3\ d]{\beta} {}^{239}Pu$	α, X-rays	1	1	4.4×10^{-12}
^{240}Pu	6,600 yr	$^{239}Pu(n,\gamma)^{240}Pu$; $^{239}Np(n,\gamma)^{240}Np \xrightarrow[\leq 67\ m]{\beta} {}^{240}Pu$	α	0.35	1.9	4.9×10^{-8}
^{241}Pu	15 yr	$^{240}Pu(n,\gamma)^{241}Pu$	β	23	560	1.3×10^{-29}
^{242}Pu	380,000 yr	$^{241}Pu(n,\gamma)^{242}Pu$	α	-	0.01	5.3×10^{-6}
^{241}Am	458 yr	$^{241}Pu \xrightarrow{\beta} {}^{241}Am$	α, γ-rays	-	1.3	2.3×10^{-12}
^{242}Cm	163 d	$^{241}Am(n,\gamma)^{242}Am \xrightarrow[16\ hr]{\beta} {}^{242}Cm$	α	-	190	6.2×10^{-8}
^{244}Cm	17.6 yr	$^{243}Pu \xrightarrow[5\ hr]{\beta} {}^{243}Am(n,\gamma)^{244}Am \xrightarrow[10\ hr]{\beta} {}^{244}Cm$	α	-	7.8	1.3×10^{-6}
^{246}Cm	4700 yr	$^{244}Cm(n,\gamma)^{245}Cm(n,\gamma)^{246}Cm$	α	-	-	3.2×10^{-4}

TABLE 2. THE SPECIFIC ALPHA ACTIVITY OF PARTICLES OF PuO_2 AND NITRATE FROM FUEL IRRADIATED TO A LEVEL OF 2000 MWd/t AND UNIT DENSITY EQUIVALENT AERODYNAMIC DIAMETER OF 1 μm

Compound	Density g/cm^3	Mass of Pu/particles (g)	dpm(^{239}Pu + ^{240}Pu)	No. of particles equivalent to 40 nCi
PuO_2	11.5	1.4×10^{-13}	0.03	2.9×10^{6}
$Pu(NO_3)_4 5H_2O$	2.9	1.3×10^{-13}	0.03	3.1×10^{6}

TABLE 3. PERCENTAGE DISTRIBUTION OF PLUTONIUM IN TISSUE OF BEAGLES AFTER INHALATION AND INTRAVENOUS INJECTION AND IN RABBITS AFTER INTRAMUSCULAR INJECTION

	Inhalation (beagle)			Intravenous Injection (beagle)	Intramuscular Injection (rabbit)
	PuO_2	Pu nitrate	Pu citrate	Pu citrate	Pu nitrate
Duration	11 yr	~250 days	100 days	4 yr	1 yr
Percentage distribution:					
Lungs	9	33	29	-	-
Thoracic Lymph Nodes	40	1.5	0.4	-	-
Skeleton	5	26	38	44	31
Liver	15	14	16	13	1
Reference	Park et al. 1972	Park et al. 1968	Ballou et al. 1972	Stover et al. 1962	Taylor et al. 1969

TABLE 4. DISTRIBUTION OF PLUTONIUM IN HUMANS AS MEASURED IN TISSUES OBTAINED AT AUTOPSY

Years of Exposure	Lung dpm/kg	Liver dpm	Concentration Ratio TLN*/Lung	Bone/Liver	Reference
5	97600	527	0.009	2.3	B
-	34100	505	0.3	0.8	B
21	14700	5460	13.3	0.4	D
13	8540	4880	17.7	0.88	A
-	8460	-	66.7	-	A
11	6330	16700	13.4	1.0	A
6	5930	102	0.4	2.3	B
12	4050	15900	0.8	3.2	C
9	3960	1470	1.7	1.3	D
7	2690	425	6.0	0.6	B
11	2520	-	25.5	-	A
4	2510	132	4.4	6.7	D

*TLN = Thoracic Lymph Nodes

References:
A = Campbell et al. (1973)
B = Lagerquist et al. (1973)
C = Norwood et al. (1973)
D = Schofield and Dolphin (1974)

TABLE 5. CHROMOSOME ABERRATIONS FOUND IN BLOOD LYMPHOCYTES OF INDIVIDUALS KNOWN TO HAVE BEEN ACCIDENTALLY CONTAMINATED WITH Pu

Cells Scored	Dicentrics	Acentrics	Exposure
1000	2	12	Six years after inhalation of about 0.3 μCi
500	14	22	Wound puncture contaminated with 14 μCi. Schofield et al., 1974
1000	12	34	15 years exposure to Pu with an estimate of about 0.04 μCi. External radiation 10 rads
500	2	8	Second sample 7 years later with no further exposure

TABLE 6. BASIC DOSE STANDARDS EXPRESSED BOTH AS REM IN A YEAR AND AS RAD IN A YEAR FOR ORGANS OF IMPORTANCE IN THE PROTECTION OF PERSONS EXPOSED TO PLUTONIUM, FROM ICRP PUB. 9 (1966)

Organ	MPD for adults at work		Dose Limits for Members of the Public	
	Rem in a year	Rad in a year*	Rem in a year	Rad in a year*
Gonads, red bone marrow (whole body irradiation)	5	-	0.5	-
Liver) Lung	15	1.5	1.5	0.15
Bone	30	0.6	3	0.06

* The radiological protection units are derived from the energy deposition units by the use of a quality factor Q = 10 for liver and a modifying factor N = 5 and Q = 10 for bone.

TABLE 7. ESTIMATED VALUES OF RISK COEFFICIENT FOR VARIOUS TYPES OF CANCER IN EXPOSED POPULATION GROUPS FOR EXTERNAL γ AND α RADIATION

Biological Effect	Population Group	Type of Radiation	Risk Coefficient Cancers/10^6 man-rad	Reference
Leukaemia	A-bomb survivors	γ	30	Goss (1974)
All cancers	A-bomb survivors	γ	100	Goss (1974)
Leukaemia	Ankylosing spondylitics	X-rays	10	Dolphin and Marley (1969)
All cancers in X-ray field	Ankylosing spondylitics	X-rays	50	Dolphin and Marley (1969)
Bone sarcomas	Radium-226 cases	α	80[1]	This paper
Bone sarcomas	Radium-224 cases	α	90	Spiess and Mays (1970)
Lung cancer	Miners exposed to radon Thorotrast patients	α	30[2]	Lundin et al. (1971)
Bone sarcoma	Rats exposed to Pu	α	400	This paper
Lung cancer	Rats exposed to Pu	α	650	This paper

1. Calculated from data given by Rowland (1973) assuming a linear relationship for cases with average accumulated dose to bone of less than 10,000 rad.

2. Calculated from data on risk coefficients taken from Lundin et al. (1971), for US uranium miners, Newfoundland, fluorospar miners, British haematite miners, US hard-rock miners, Portuguese thorotrast patients and Danish thorotrast patients weighted by the square root of the number of lung cancers attributed to radiation in the survey.

TABLE 8. PERCENTAGE OF INHALED PLUTONIUM-239 ACTIVITY TRANSFERRED TO BLOOD FROM LUNG AND INTO BONE AND LIVER, HALF-LIVES OF RETENTION AS USED TO CALCULATE $(MPC)_a$ AND MPAI FOR SOLUBLE, INSOLUBLE, CLASS W AND CLASS Y COMPOUNDS

Compound Inhaled	Percentage Transfer to Blood	Percentage of Inhaled Activity in Organ and Half-Life						Maximum Permissible Concentration (MPCa)	Maximum Permissible Annual Intake[3]	
		Bone (7000 g)		Liver (1700 g)		Lung (1000 g)				
		Amount %	Half-life (days)	Amount %	Half-life (days)	Amount %	Half-life (days)	$\times 10^{-12}$ µCi/cm^3	Inhalation µCi	Oral µCi
Soluble	25	20	7.3×10^4	3.8	3×10^4	-	-	2	5×10^{-3}	27.5
Insoluble[1]	-	-	-	-	-	12.5	365	40	0.1	220
Class W[2]	12[4]	5.4	3.6×10^4	5.4	1.5×10^4	15	50	8	0.02	-
Class Y[2]	5[4]	2.25	3.6×10^4	2.25	1.5×10^4	15	500	18	0.04	-

1. As defined in ICRP Publication 2 (1959)
2. Solubility classes and lung model defined in ICRP publication 19 (1972)
3. Only half this amount is permitted in a single intake (vide ICRP Publication 9)
4. Deposition fractions in the lung compartments for 1 µm AMAD particles given in ICRP Publication 19 (1972)

TABLE 9. THE VARIATION WITH PARTICLE SIZE OF THE NUMBER OF PARTICLES AND THE NUMBER OF CELLS IRRADIATED FOR A CONSTANT ACTIVITY OF 0.016 μCi OF PLUTONIUM-239 OXIDE.

Particle Diameter (micron)	Particles per 0.016 μCi	Irradiated Cells Per Particle[1]	Total Cells Irradiated
0.1	4.9×10^{7}	270	1.3×10^{10}
1	4.9×10^{4}	280	1.4×10^{7}
10	4.9×10	380	1.9×10^{4}

[1]Cell volume assumed to be 10^{-9} cm^{3}

Figure I. Plutonium induced osteogenic sarcomas in rat

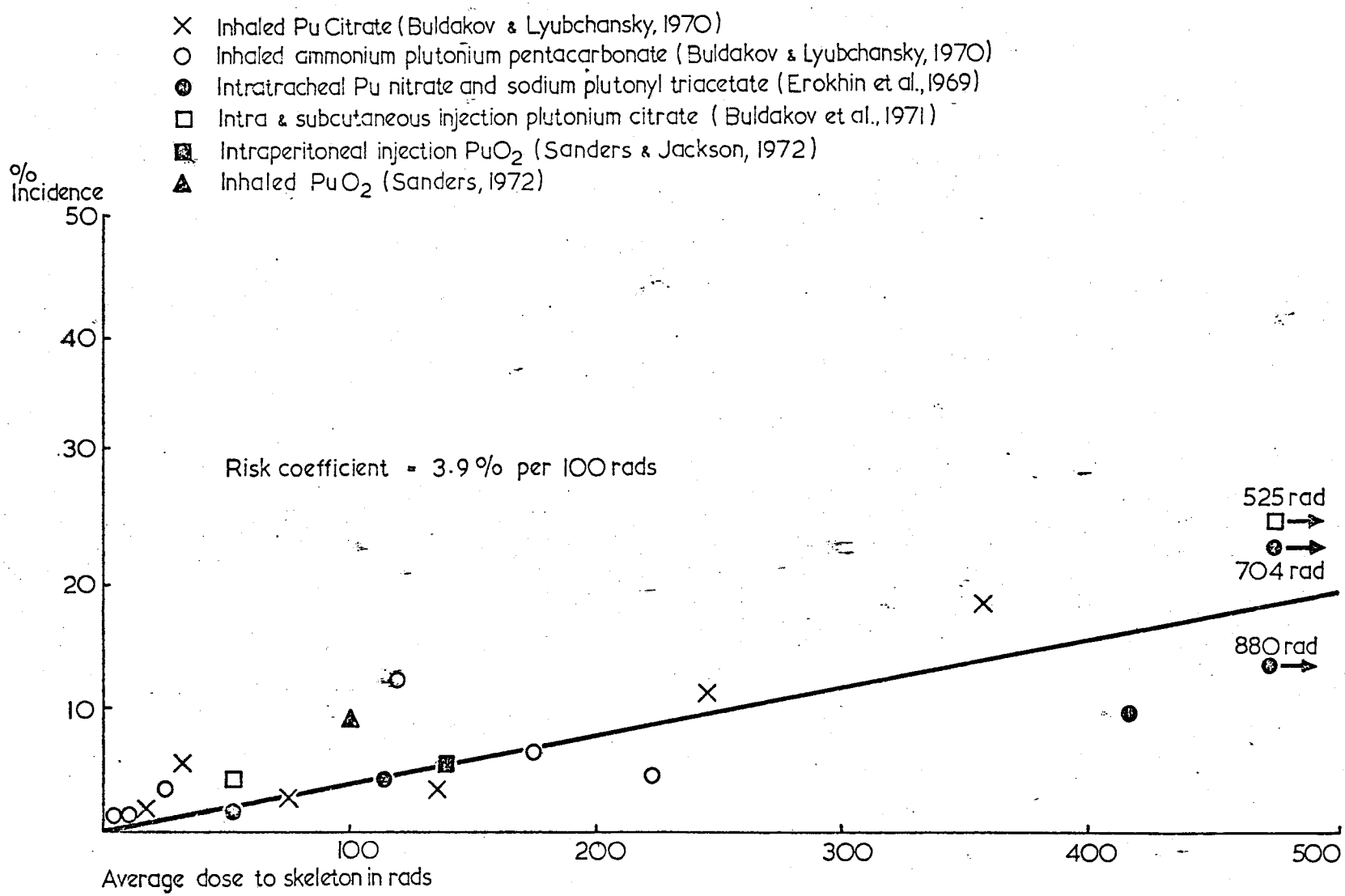
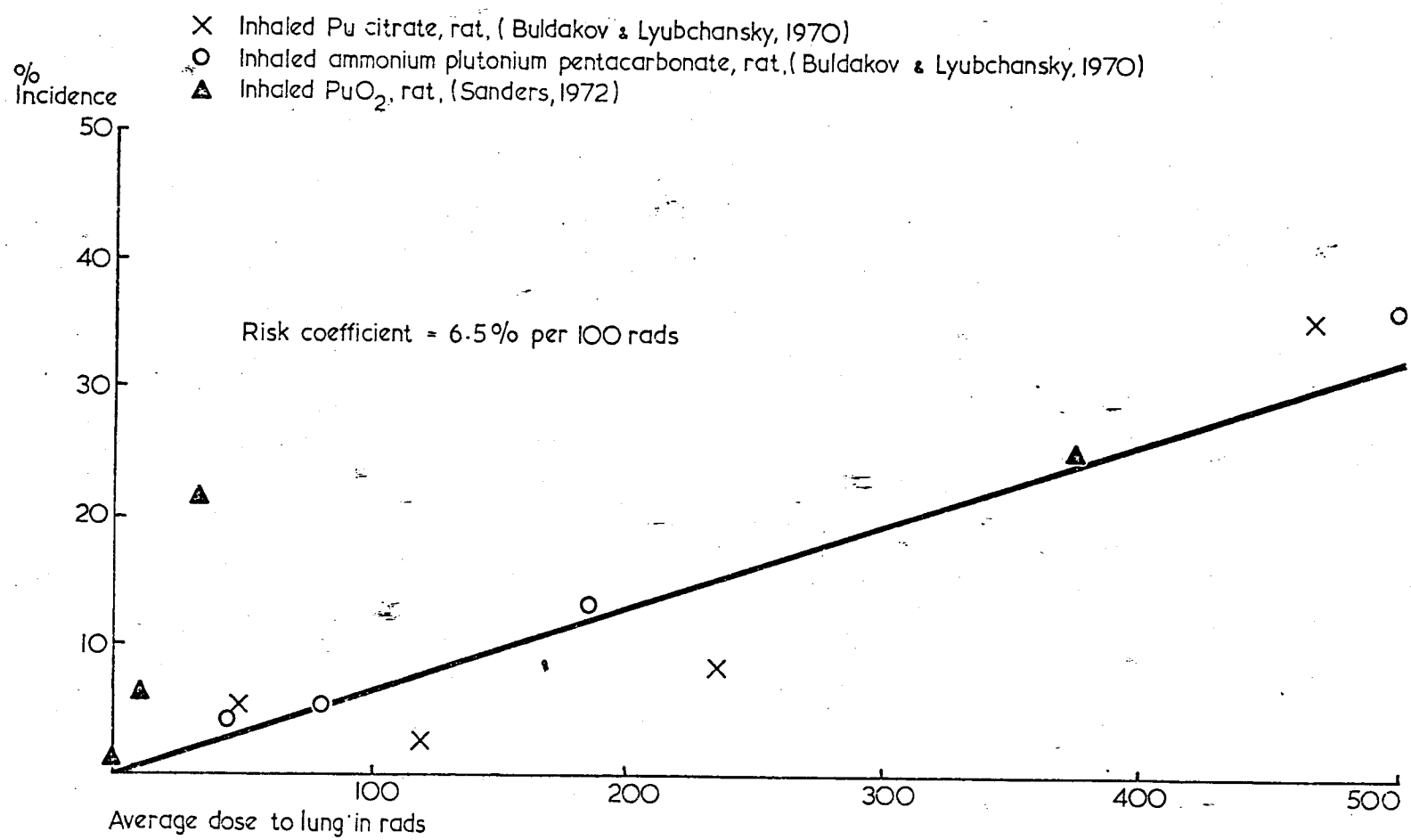


Figure 2. Plutonium induced lung cancer in experimental animals



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Hot particles

Dr G W Dolphin
NATIONAL RADIOLOGICAL PROTECTION BOARD, HARWELL

This is a brief critical review of "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" by A R Tamplin and T B Cochran, 14 February 1974.

It should be noted that no human cancers have been positively associated with exposure to insoluble particles or soluble compounds of plutonium. Hence the conclusions in the Report are based on implication or extrapolation from animal experiments.

The authors have no new biological evidence. The Report is based on data published during the last 15 years by other scientists.

Tamplin and Cochran refer to the only well-documented human case of biological changes in cells surrounding a particle of plutonium embedded in the palm of a man's hand. This was reported by Lushbaugh and Langham in 1962. A 5 nCi particle of plutonium was excised from the palm 4 years after it had become embedded as a result of an accident. Lushbaugh, a pathologist, carefully described the cell changes as having "a similarity to known pre-cancerous epidermal cytologic changes". Tamplin and Cochran acknowledge this wording in the first reference to it in their Report. However, in the second reference the wording is altered slightly to "pre-cancerous changes in human tissue" and on the third reference another alteration is made and it becomes "particle induced cancer". In summary, the authors use the old trick of progressively changing words to arrive at a dramatic conclusion.

From this alleged "cancer" in the palm of the hand, they go to the conclusion that the risk of cancer from a hot particle is 1 in 1,000 for they assume that there are 1,000 men with plutonium embedded in tissues as a result of wound accidents who have not developed cancers at the wound site. Clearly this is an absurd conclusion from the available human data.

Albert *et al.* (1967) reported the incidence of skin cancer in rats exposed to doses of between 500 and 7,000 rads of beta particle radiation. The cancers observed were in hair follicles and they showed a sharp increase at doses above 1,000 rads. From these data Tamplin and Cochran conclude that doses of over 1,000 rads to small volumes of cells, as in hair follicles, can produce cancers and it therefore follows that hot particles can produce cancers if the dose to a small number of surrounding cells exceeds about 1,000 rads.

From a paper by Geesaman (1968), the authors extracted a value for the risk of cancer in a hair follicle of a rat in the range 1 in 1,000 to 1 in 10,000 at doses over 1,000 rads to follicles. From these data and the human data quoted above, Tamplin and Cochran conclude that the risk of cancer developing in cells surrounding a hot particle is 1 in 2,000. It should be noted

here that the follicles were irradiated together with the rest of the epidermis and some of the dermis and the effects of these irradiated tissues on the development of the follicular cancer cannot be assessed in these experiments.

The hair follicle cancers found by Albert *et al.* in rats were not found in similar experiments carried out by by Hulse (1969) using mice. He found only epidermal and dermal cancers. Hence the hair follicle cancers described by Albert *et al.* may be peculiar to the rat species. If extrapolation from rat skin to mice skin is not possible in this work, then little confidence can exist in the extrapolation from rat skin to human lung tissue.

Some human experience on the incidence of skin cancer comes from 11,000 children treated by 100kV X-rays for ringworm of the scalp with doses of about 400 rads. One skin cancer has developed in these children in a period up to 21 years after irradiation (Modan *et al.*, 1974). Furthermore, no deaths from skin cancer have been reported by Court Brown and Doll (1965) among patients treated with doses of about 1,500 rads for ankylosing spondylitis. The field size on the skin of these patients was about 450 cm^2.

This human experience of skin irradiation indicates that large doses to a few hundred square centimetres of skin do not produce significant numbers of skin cancers and therefore it is unlikely that a comparable dose of alpha-radiation to a small number of cells in the skin would produce cancer as suggested by the hot particle concept of Tamplin and Cochran.

Bair (1974) reports the finding in dogs studied for periods up to 4068 days after deposition in lungs of 10 million to 100 million plutonium oxide particles (0.2 μCi to 3.3 μCi). One dog out of 21 living for more than 1,600 days has not developed a lung cancer. Tamplin and Cochran note this finding but dismiss it and make no estimate of the risk of cancer per particle in the lung. If they had made an estimate, it would be that the cancer risk in this dog that survived was in the range 1 in 500,000 to 1 in 5 million per particle.

Lafuma (1974) has reported greater toxic effects including cancer in rats following deposition of curium-242 in lungs compared with equal amounts of plutonium-239 activity. This he attributes to the diffuse nature of the curium deposit and the particulate nature of the plutonium, as shown by autoradiographs. This is in direct contradiction to the Tamplin and Cochran hot particle hypothesis.

It is noted that the basis of ICRP recommendations is the average radiation dose to an organ and not the number of radioactive particles in the organ. This dosimetric basis of radiological protection has been established for many years by observation of humans and experimental work with animals. A better evaluation than that offered by Tamplin and Cochran would be needed for this system to be set aside in favour of the hot particle concept. Their estimate that there is a risk of cancer being generated in cells surrounding a hot particle of 1 in 2,000 cannot be substantiated by our present knowledge.

References

Albert, R E, Burns, F J and Heimbach, R D *Radiation Res.*, **30**, 590, 1967.
Bair, W J *Advances in Radiation Biol.*, **4**, 255, 1974.
Court Brown, W M and Doll, R *Brit. med. J.*, Dec. **4**, 1327, 1965.
Geesaman, D P UCRL-50387, *Op. cit.*, p.11, 1968.
Hulse, E V *Brit. J. Cancer*, **21**, 531, 1967.
Lafuma, J Paper presented at the French Society for Radiation Protection, Paris, March 1974 on "La Contamination Radioactive Interne".
Lushbaugh, C C and Langham, *J Archs. Dermat.*, 86, 461, 1962.
Modan, B, Baidatz, D, Mart, H, Steintz, R and Levin, S G *Lancet*, Feb. **23**, 7852, 1974.

Hillsboro, N.H. 03244

November 29, 1974

Office of Radiation Programs
AW-560
EPA
Washington, D.C. 20460

Dear Sirs,

In reply to a recent EPA Citizen's Bulletin, inviting comments "from all interested parties" on setting plutonium standards", I enclose a recent clip (Octoberll, 1974) from the New York Times on Plutonium Found in Plants' Roots. The Battelle Laboratories research would tend to prove that no amount of plutonium should be introduced into our biosphere. In which case, as well as for many other reasons, a moratorium on future development of nuclear fission products should be declared.

I call to your attention a book published in 1973: Nuclear Energy - Its Physics and Its Social Challenge by David R. Inglis of the University of Massahcusetts (Addision-Wesley Publishing Co., Reading, Mass.). He devoted pages to the question of plutonium.

Having attended AEC Liscencing Board hearings on the Vermont Yankee plant in Vermont and the Seabrook Plant proposed in our state, I would urge your more active participation and involvement - not to say control - of nuclear plant development.

With all good wishes, I am,

Sincerely,

Annette B. Cottrell

Annette B. Cottrell, Honorary Trustee
New England Coalition on Nuclear Pollution

P.S.
I assume you are in touch with The Union of Concerned Scientists at MIT as well as Ralph Nader

enc:
N.Y. Times clip (Note: not printed because copyright release not obtained.)

BIOMEDICAL DIVISION
L-523

November 20, 1974

Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

This letter is in response to EPA's request for interested parties to submit testimony or materials relevant to a review of the need for establishing new rules for contamination limits for plutonium and the transuranium elements.

Although the data are equivocal, it is frequently assumed that the food-chain transport of Pu to man is so low that the most significant pathway to man is via inhalation. We have recently presented a paper entitled "Evaluation of the Resuspension Pathway Toward Protective Guidelines for Soil Contamination with Radioactivity" at an International Atomic Energy Agency Seminar held in Portoroz, Yugoslavia, May 20-24, 1974. This paper summarizes experimental studies concerning the resuspension of ground-deposited radioactivity, presents models useful for predictive purposes, applies these models to rules development for plutonium in soil, and discusses cost-benefit aspects of clean-up procedures

We believe the topics considered in this paper are highly pertinent to your review of the need for establishing new rules for plutonium and transuranic elements. This paper is currently in press, but we are enclosing 20 copies of the Preprint for your consideration.

Sincerely yours,

L. R. Anspaugh, Ph. D.
Group Leader
Applied Environmental Science Group
Bio-Medical Division

LRA:sc
Enc: (20)

EVALUATION OF THE RESUSPENSION PATHWAY TOWARD PROTECTIVE GUIDELINES FOR SOIL CONTAMINATION WITH RADIOACTIVITY*

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Lawrence Livermore Laboratory, University of California
Livermore, California 94550 U.S.A.

ABSTRACT

The resuspension and subsequent inhalation of surface-deposited radioactivity released to the environment can be a significant mode of exposure for a few radionuclides such as ^{239}Pu. Two simple, interim models which may be used to predict the average concentration of resuspended aerosols are developed on an empirical basis. One method uses the time-dependent resuspension factor approach, and differs from previous work in placing more emphasis on resuspension at late times. The second method is appropriate only for aged sources, and uses a straight-forward mass-loading approach. The relative significance of the resuspension pathway is also modeled in comparison to the initial exposure resulting from a nonnuclear explosion which disperses radioactivity. Two hypothetical ^{239}Pu contamination situations are modeled. In the first case the 50 yr dose commitment resulting from an initial deposition of 1 $\mu Ci/m^2$ is calculated as a function of time post deposition. Half of the total dose commitment is accumulated in the first 100 d. In the second situation, the reoccupation of an area contaminated many years previously is considered. Protective guidelines for ^{239}Pu soil contamination are derived from these studies — 1 $\mu Ci/m^2$ for a freshly deposited source and 7 nCi/g in the top 10 mm of soil for a source which has aged several years. An estimate of the biological cost of not cleaning up contaminated areas is compared with the engineering and agricultural costs of soil removal.

*This work was performed under the auspices of the U.S. Atomic Energy Commission.

A convenient way to model the airborne concentration of resuspended contaminant over long periods of time is to make the resuspension factor a function of time to account for the observed decrease in air concentration which has been noted to occur in the abscence of a significant net loss of the deposited contaminant. Conceptionally, it would be more appropriate to define a time-dependent fraction of the total deposition which is available for resuspension. However, there is no realistic way in which such a fraction can be experimentally determined, so this approach will be avoided for the present purpose. With the time dependency inherent in the resuspension factor, it follows that the average airborne concentration, $\overline{\chi}$, of resuspended contaminant will be given by

$$\overline{\chi}\ (t) = K\ (t)\ S_A$$

where S_A is the total amount of contaminant deposited per unit area. S_A is therefore considered a constant although the actual distribution of the contaminant with soil depth will change with time.

Kathren [15] and Langham [16] have each formulated predictive resuspension models which, when expressed in the above format, give the following time dependency

$$K\ (t) = K_o\ \exp\ (-\lambda t)$$

with values of λ corresponding to half-times of 45 d and 35 d. Such a formulation appears to simulate reasonably well the available observations up to several weeks post deposition [4, 8]. After a few years, however, such a formulation underestimates by many orders of magnitude the airborne concentration of resuspended contaminants which have been measured over aged sources [5, 7, 9]. For example, the Kathren and Langham models would predict values for K (t) of 10^{-29} and 10^{-38} m^{-1} respectively 10 yr after a contaminating event whereas an average value determined from 236 individual air concentration measurements at a location contaminated with plutonium 17 yr previously was found to be 10^{-9} m^{-1} [9].

We have derived a different formulation of the time dependency of the resuspension factor which more accurately reflects the resuspension process as it is observed in the proximity of aged sources. This model was empirically derived to conform to the following constraints: 1) The apparent half-time of decrease during the first 10 weeks should approximate a value of 5 weeks and should approximately double over the next 30 weeks; 2) The initial resuspension factor should be 10^{-4} m^{-1}; and 3) The resuspension factor 17 yr after the contaminating event should approximate 10^{-9} m^{-1}.

A simple model which closely approximates these constraints is

$$K\ (t) = 10^{-4}\ \exp\ (-0.15\ d^{-\frac{1}{2}}\sqrt{t})\ m^{-1} + 10^{-9}\ m^{-1}$$

The second term was added based upon the assumption that there may be no further measurable decrease in the resuspension process after 17 yr which is the longest period post deposition for which measurements have been reported. This was deemed appropriate because such a "model" was derived empirically to simulate experimental measurements, and contains no fundamental understanding of the resuspension process. A graphical representation of this model, both with and without the second term is given in Fig. 1; the model equations used by Kathren and Langham are also shown for comparison.

This model is an attempt to provide protective guidance for the evaluation of the resuspension pathway over long time periods beginning with the initial contaminating event. It assumes that resuspension is a local phenomenon and that the concentration in air drops off rapidly downwind of the deposited source; this is consistent with experimental observations [3, 9]. The model is suitable only for the prediction of long-term averages of airborne concentrations; extreme short-term fluctuations are to be expected and suitable long-term averages of experimental data were used to define the model contrainsts. The initial value for the resuspension factor, however, was deliberately chosen to be sufficiently high to include the effects of artificial disturbance.

2.2 Mass-loading approach

Nearly all of the experimental measurements of the concentration of resuspended contaminants have been conducted in the vicinity of freshly deposited sources. This is appropriate for most situations of practical concern such as accidental events. However, there are some situations such as the contemplated reoccupation of test areas contaminated many years previously where an alternate method of predicting the concentration of resuspended contaminant may be used to supplement the results derived from the resuspension factor model.

It has been observed by many authors [17-20] that radionuclides deposited on the earth's surface in either solution or particulate form penetrate within a few months to depths of more than 10 mm. Eventually their distribution with depth is well approximated by an exponential function characterized by a relaxation depth of 10 to 100 mm. Such a distribution with depth implies an intimate mixing of the contaminate with the host soil. Therefore, a method of predicting the average airborne concentration of the contaminant several years after the contaminating event is to simply multiply the measured activity of the contaminant per unit weight of soil taken from the top 10 mm by the concentration of particulate matter in the atmosphere.

For predictive purposes, an average atmospheric concentration of 100 $\mu g/m^3$ appears to be reasonable [9]. The choice of this value is partly based upon measurements of particulate concentration reported for 30 nonurban

locations in the United States [21]. Annual arithmetic averages varied from 9 to 79 $\mu g/m^3$ with a mean for all 30 stations of 38 $\mu g/m^3$.

Several experimental results are available to check the accuracy of this simple prediction method [9, 22-26]. These values are tabulated in Table I; the agreement between the predicted and measured values is generally excellent.

2.3 Relative importance of resuspension in the inhalation pathway

Some accident situations may produce an initial contaminant aerosol cloud with a resulting dose commitment to an exposed population. In such cases, it is of practical interest to compare such an unavoidable dose commitment to that predicted via the resuspension pathway.

If the initial integrated air activity is A_o (activity-time/volume), the ground deposition may be calculated by multiplying by a deposition velocity, V, (length/time). The integrated air activity, A, due to resuspension is then given by

$$A = VA_o \int K(t)\, dt$$

which may then be compared conveniently with the initial integrated air activity, A_o. One problem in such a comparison, however, is the choice of an appropriate value for the deposition velocity. This varies as a function of particle size and wind speed, and in close proximity to an explosion the actual deposition would be strongly influenced by the ballistic effects of the explosion itself. At such close-in distances, however, the initial resuspension factor is also lower [2, 4, 10]. Because the initial resuspension factor chosen for use in the model is relatively high and is presumed to be appropriate for smaller particle size distributions of the contaminant aerosol, it is assumed that an appropriate value for V is 40 m/h. This is an average value which can be derived from several years of fallout data [27].

2.4 Dose commitment due to the resuspension of ^{239}Pu

In order to derive a protective guideline for soil contamination, some reference must be made to a primary standard. For ^{239}Pu, which will be used as an example, it will be assumed that the desired primary reference standard is the accumulated 50 yr dose commitment. The dose commitment calculations were made with the additional assumptions that the plutonium is non-transportable, the lung is the critical organ [28], 25% of the inhaled plutonium is deposited in the pulmonary region, and that the metabolic parameters listed by Morgan [29] for Class Y compounds were appropriate.

Representative calculations are presented in Tables II and III for two hypothetical situations. The first assumes a nonnuclear explosion which produces a ^{239}Pu deposition of 1 $\mu Ci/m^2$. The 50 yr dose commitment due to

the initial cloud passage and resuspension of deposited material as a function of time post deposition were calculated using the models given in sub-sections 2.1 and 2.3. The results in Table II indicate that any protective action must be undertaken fairly rapidly if a significant reduction in the 50 yr dose commitment is to be achieved.

The second situation assumes the reoccupation of landscape contaminated many years previously and where the ^{239}Pu concentration in the top 10 mm of soil is 1 μCi/g. Calculations of the 50 yr dose commitment as a function of time post reentry were made using the model given in sub-section 2.2, and are presented in Table III.

If we assume that an acceptable 50 yr dose commitment in such situations is 50 x 1.5 rem, or 75 rem, then protective guidelines of 1 $\mu Ci/m^2$ and 7 nCi/g are derived for the two hypothetical situations. It is emphasized, however, that this analysis has considered only the inhalation of ambient air. Other pathways such as personal contamination and ingestion may be more restrictive in some situations.

3. Cost-benefit aspects of protective guideline application

Models developed in this study can be applied to a general analysis of costs and benefits associated with the clean-up of environmental plutonium. In this instance, costs are defined as all expenditures and monetary losses derived from clean-up action. Benefits are the reductions in potential health costs which may be derived from any proposed clean-up action.

Comparative costs have been derived for clean-up of a hypothetical km^2 of agricultural land containing one Ci of ^{239}Pu in the soil surface (Table IV). The objective of this exercise is to provide a semi-quantitative analysis of the economic trade-offs between clean-up and no clean-up.

Costs of removal of soil depend upon availability of equipment, costs of transportation, and the complexity of terrain. In 1974, U.S. public works projects involving soil surface pick-up cost U.S. $0.15/m^3$ of soil; associated transportation costs for long hauls were U.S. $ 0.06/m^3 - km [30]. Clean-up costs for removing the top 30 mm of soil from a km^2 of land would be approximately U.S. $4500. Transportation of contaminated soil to a storage site 500 km away would be approximately U.S. $ 1 x $10^6/km^2$ of surface removed.

Thus, clean-up costs are likely to be small compared to transportation costs for clean-up in impacted areas which are remote from a radioactive waste management area. Costs associated with long-term waste management will be ignored here.

Land values are highly variable and depend upon the specific land use. Currently, U.S. agricultural land is valued at approximately U.S. $50,000/$km^2$ [31]. Restriction against crop production would result in

this economic loss as well as losses of annual crop production, valued at U.S. $ 2 x 10^4/km^2 [31]. Clean-up could result in costs from temporary decreased agricultural production due to interruptions in the crop cycle or decreased soil fertility. For locations with marginal food supply, restrictions in agricultural production could lead to more serious biological costs than costs from radiation exposure.

Potential health costs arising from radiation exposure in populations have been evaluated in monetary terms by others [32]. Such figures are useful for comparison with the monetary costs of remedial action against radiation exposure. Health cost estimates are developed in this study using a value of U.S. $250/man-rad. At a dose commitment level of 75 rem, the calculated cost per person exposed is U.S. $ 2 x 10^4. On typical agricultural land, one could reasonably expect a population density of one to ten persons per km^2. Potential biological costs could be approximately U.S. $ 2 x 10^5. The benefit-cost ratio for clean-up is therefore approximately 0.2. This ratio is very sensitive to the costs of transportation and the number of human receptors which are involved. Alternatives, such as restriction against crop production incur costs which are comparable to the benefits of reduced radiation exposure; for example, the economic crop loss of creating an exclusion area for ten years is approximately U.S. $ 2 x 10^5.

In summary, the deposition of a Ci per km^2 of ^{239}Pu on agricultural land can be evaluated as leading to potential health costs which are comparable in economic value to the costs of remedial actions which remove the potential exposure. The judgement regarding proper action involves consideration of qualitative features, such as economic policies and societial priorities for utilizing finite monetary resources to promote human welfare. Some consideration should also be given to an implied long-term commitment should a decision be made not to clean-up a contaminated area. If such an area is used for agricultural production, some monitoring program will be necessary to assess the level of ^{239}Pu in food crops. While the food-chain transport of ^{239}Pu is generally believed to be negligible based upon short-term studies, it cannot be stated with certainty that this will remain true over long periods of time [19].

The short time period available for making an effective decision is also apparent from the results in Table II. By 100 d after the contaminating event, one-half of the total 50 yr dose commitment has already been accumulated. An alternate couse of action would be to evacuate the area and/or plow the affected ground surfaces. This should be effective in greatly reducing the biological costs, but also substantially increases the ultimate cost of clean-up should it eventually be deemed necessary.

Finally, some mention should be made of urban environments. Here, the potential for rapid spread of the material and the large population density appear to offer no choice but to contain and remove the contamination as rapidly as possible.

REFERENCES

[1] LANGHAM, W.H., Plutonium distribution as a problem in environmental science, Environmental Plutonium Symposium (Proc. Symp. Los Alamos, New Mexico, 1971) USAEC Rep. LA-4756 (1971) 3.

[2] LARSON, K.H., NEEL, J.W., HAWTHRONE, H.A., MORK, H.M., ROWLAND, R.H., BAURMASH, L., LINDBERG, R.G., OLAFSON, J.H., KOWALEWSKY, B.W., Distribution, Characteristics, and Biotic Availability of Fallout, Operation Plumbbob, USAEC Rep. WT-1488 (1966).

[3] SHREVE, J.D., JR., Summary Report, Test Group 57, USAEC Rep. ITR-1515 (DEL) (1958).

[4] WILSON, R.H., THOMAS, R.G., STANNARD, J.N., Biomedical and Aerosol Studies Associated with a Field Release of Plutonium, USAEC Rep. WT-1511 (1960).

[5] OLAFSON, J.H., LARSON, K.H., Plutonium, Its Biology and Environmental Persistence, USAEC Rep. UCLA-501 (1961).

[6] STEWART, K., The resuspension of particulate material from surfaces, Surface Contamination (Proc. Symp. Gatlinburg, Tenn., 1964) Pergamon, London (1964) 63.

[7] VOLCHOK, H.L., Resuspension of plutonium-239 in the vicinity of Rocky Flats, Environmental Plutonium Symposium (Proc. Symp. Los Alamos, New Mexico, 1971) USAEC Rep. LA-4756 (1971) 99.

[8] ANSPAUGH, L.R., PHELPS, P.L., KENNEDY, N.C., BOOTH, H.G., Wind-driven redistribution of surface-deposited radioactivity, Environmental Behaviour of Radionuclides Released in the Nuclear Industry (Proc. Symp. Aix-en-Provence, France, 1973) IAEA, Vienna (1973) 167.

[9] ANSPAUGH, L.R., PHELPS, P.L., KENNEDY, N.C., BOOTH, H.G., GOLUBA, R.W., REICHMAN, J.M., KOVAL, J.S., Resuspension element status report, The Dynamics of Plutonium in Desert Environments, USAEC Rep. NVO-142 (in press).

[10] ANSPAUGH, L.R., PHELPS, P.L., HOLLADAY, G., HAMBY, K.O., Distribution and redistribution of airborne particulates from the Schooner cratering event, Health Physics Aspects of Nuclear Facility Siting (Proc. Symp. Idaho Falls, Idaho, 1970) 2, Eastern Idaho Chapter, Health Physics Society, Idaho Falls (1971) 428.

[11] MORK, H.M., Redistribution of Plutonium in the Environs of the Nevada Test Site, USAEC Rep. UCLA-12-590 (1970).

[12] SEHMEL, G.A., Particle resuspension from an asphalt road caused by car and truck traffic, Atmos. Environ. 7 (1973) 291.

[13] VOLCHOK, H.L., KNUTH, R.H., The respirable fraction of plutonium at Rocky Flats, Health Phys. 23 (1972) 395.

[14] MISHIMA, J., A Review of Research on Plutonium Releases during Overheating and Fires, USAEC Rep. HW-83668 (1964).

[15] KATHREN, R.L., Towards interim acceptable surface contamination levels for environmental PuO_2, Radiological Protection of the Public in a Nuclear Mass Disaster (Proc. Symp. Interlaken, Swit., 1968) EDMZ, Bern (1968) 460.

[16] LANGHAM, W.H., Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads, USAEC Rep. UCRL-50639 (1969).

[17] BECK, H.L., Environmental gamma radiation from deposited fission products, 1960-1964, Health Phys. 12 (1966) 313.

[18] ROGOWSKI, A.S., TAMURA, T., Erosional behavior of cesium-137, Health Phys. 18 (1970) 467.

[19] ROMNEY, E.M., MORK, H.M., LARSON, K.H., Persistence of plutonium in soil, plants and small mammals, Health Phys. 19 (1970) 487.

[20] KREY, P.W., HARDY, E.P., Plutonium in Soil Around the Rocky Flats Plant, USAEC Rep. HASL-235 (1970).

[21] NATIONAL AIR POLLUTION CONTROL ADMINISTRATION, Air Quality Data from the National Air Sampling Networks and Contributing State and Local Networks, 1966 Edition, USHEW Rep. APTD 68-9 (1968).

[22] GUDIKSEN, P.H., LINDEKEN, C.L., GATROUSIS, C., ANSPAUGH, L.R., Environmental Levels of Radioactivity in the Vicinity of the Lawrence Livermore Laboratory, January through December 1971, USAEC Rep. UCRL-51242 (1972).

[23] GUDIKSEN, P.H., LINDEKEN, C.L., MEADOWS, J.W., HAMBY, K.O., Environmental Levels of Radioactivity in the Vicinity of the Lawrence Livermore Laboratory, 1972 Annual Report, USAEC Rep. UCRL-51333 (1973).

[24] SILVER, W.J., LINDEKEN, C.L., MEADOWS, J.W., HUTCHIN, W.H., MCINTYRE, D.R., Environmental Levels of Radioactivity in the Vicinity of the Lawrence Livermore Laboratory, 1973 Annual Report, USAEC Rep. UCRL-51547 (1974).

[25] SEDLET, J., GOLCHERT, N.W., DUFFY, T.L., Environmental Monitoring at Argonne National Laboratory, Annual Report for 1972, USAEC Rep. ANL-8007 (1973).

[26] HAMILTON, E.I., The concentration of uranium in air from contrasted natural environments, Health Phys. 19 (1970) 511.

[27] KLEINMAN, M.T., VOLCHOK. H.L., Radionuclide concentrations in surface air: Direct relationship to global fallout, Science 166 (1969) 376.

[28] ICRP COMMITTEE 4, The Assessment of Internal Contamination Resulting from Recurrent or Prolonged Uptakes, ICRP Publication 10A, Pergamon, Oxford (1971).

[29] MORGAN, K.Z., Proper use of information on organ and body burdens of radioactive material, Assessment of Radioactive Contamination in Man (Proc. Symp. Stockholm, 1971) IAEA, Vienna (1972) 3.

[30] MCMAHON, L.A., Dodge Estimating Guide for Public Works Construction, 1974 Annual Ed., No. 6, McGraw-Hill, New York (1974).

[31] US DEPT. OF AGRICULTURE, Agricultural Statistics, 1973, USGPO, Washington (1973).

[32] COHEN, J.J., HIGGINS, G.H., The socioeconomic impact of low-level tritium releases to the environment, Tritium (Proc. Symp. Las Vegas, 1971) Messenger Graphics, Las Vegas (1973) 14.

Table I. A comparison of observed and predicted air concentrations based upon a simple mass loading model.

Location, etc.	Radionuclide	Air Concentration Predicted[a]	Air Concentration Measured[b]
GMX site, USAEC Nevada Test Site [9]			
NE, 1971-1972	^{239}Pu	7200 aCi/m^3	6600 aCi/m^3
GZ, 1972, 2 weeks	^{239}Pu	120 fCi/m^3	23 fCi/m^3
Lawrence Livermore Laboratory			
1971 [22]	^{238}U	150 pg/m^3	52 pg/m^3
1972 [23]	^{238}U	150 pg/m^3	100 pg/m^3
1973 [24]	^{238}U	150 pg/m^3	86 pg/m^3
1973 [24]	^{40}K	1000 aCi/m^3	980 aci/m^3
Argonne National Laboratory [25]			
1972	^{232}Th	320 pg/m^3	240 pg/m^3
1972	^{nat}U	215 pg/m^3	170 pg/m^3
Sutton, England [26]			
1967-1968	^{nat}U	110 pg/m^3	62 pg/m^3

[a]Predicted value is equal to the soil concentration (activity/g) x 10^{-4} g/m^3.

[b]Most values are annual averages.

Table II. Calculated dose commitment to the lung from a hypothetical accident dispersing ^{239}Pu in the environment which produces a ground deposition of 1 $\mu Ci/m^2$.

Time	Accumulated 50 y dose commitment, rem	
	Resuspension	Total
Intial cloud passage		6.1
1 d	0.53	6.6
5 d	2.4	8.5
10 d	4.3	10.
50 d	15.	21.
100 d	23.	29.
1 y	41.	47.
10 y	52.	58.
50 y	52.	58.

Table III. Calculated dose commitment to the lung for population reentry into an area of aged ^{239}Pu contamination with 1 μCi/g in the top 10 mm of soil

Time	Accumulated 50 yr dose commitment, rem
1 y	210
10 y	2,100
30 y	6,400
50 y	10,000

Table IV. Estimates of the costs and benefits resulting from the clean-up of agricultural land contaminated with ^{239}Pu. All values are in US $.

Factor	System value	Cost	Benefit
Biological effect[a]			2.0×10^5
Soil pick-up		4.5×10^3	
Waste transportation[b]		1.0×10^6	
Agricultural land[c]	5.0×10^4		
Annual crop yield[c]	1.6×10^4		

[a]Population density of $10/km^2$ and a 50 yr dose commitment of 75 rem.

[b]Transportation of the top 30 mm a distance of 500 km.

[c]US average.

FIGURE CAPTION

Figure 1. A graphical representation of several time-dependent resuspension factor models. The two curves on the far left represent the models of Langham [16] and Kathren [15]. The remaining two curves represent models developed in this paper, both with and without a constant term of 10^{-9} m^{-1}. The hatched area represents measurements recently reported at an aged source [9].

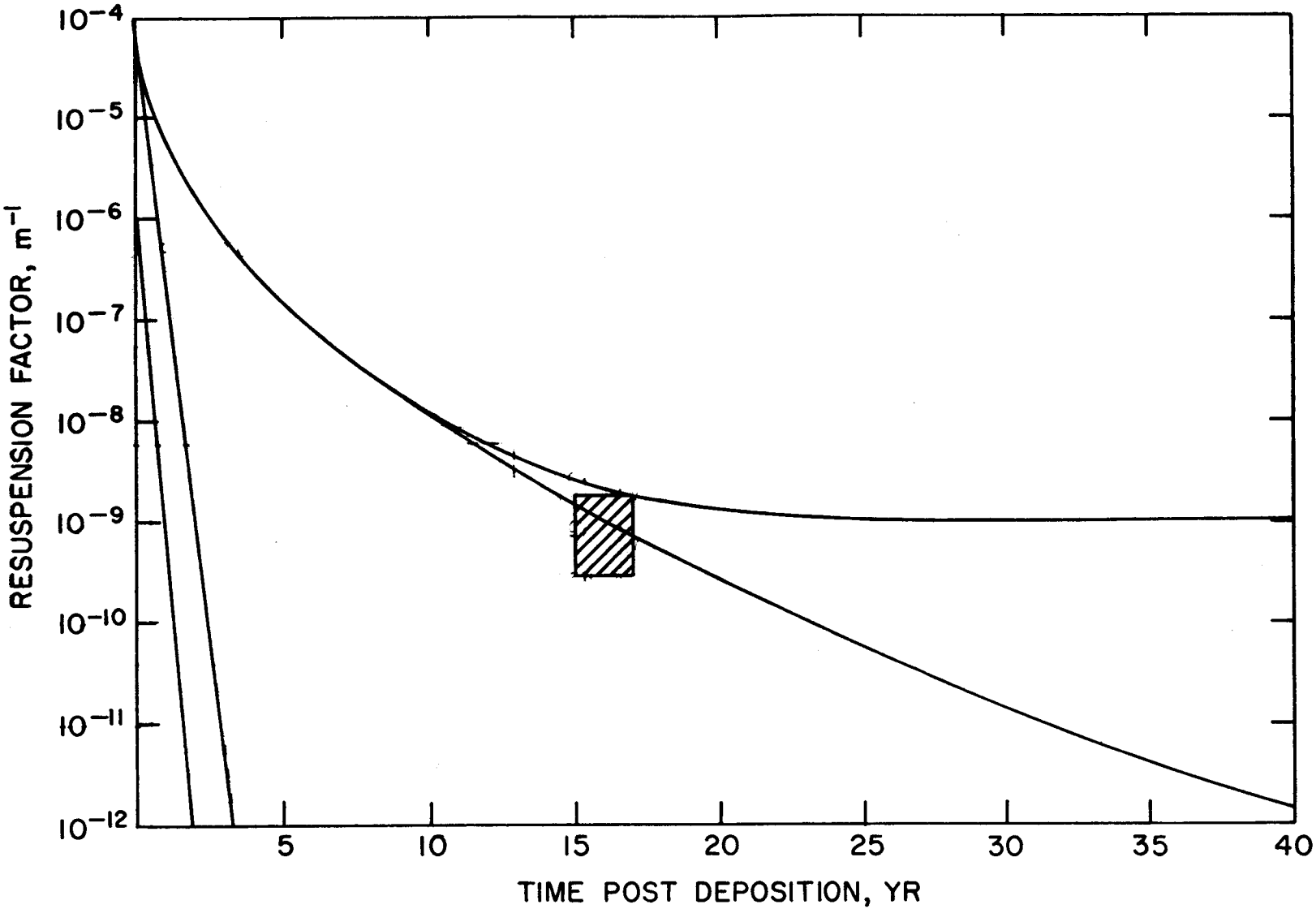

Fig. 1. A graphical representation of several time-dependent resuspension factor models. The two curves on the far left represent the models of Langham [16] and Kathren [15]. The upper two curves represent models developed in this paper, both with and without a constant term of 10^{-9} m^{-1}. The hatched area indicates values recently measured at an aged source [9].

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467

20 November 1974

W. Mills
EPA, Criteria and Standards Div.
401- M Str. SW.
Washington, D.C. 20460

Dear Dr. Mills:

This is to offer my testimony as chairman of ANSI's N 46-4 at the forthcoming hearings on Plutonium, starting December 10, 1974. The subject matter of sub-committee N46-4 concerns fuel fabrication plants. Since early in 1972 this committee has worked on the development of an A+ priority standard N 287 "Criteria for Siting, Design, and Operation of Plants for the the Manufacture of Mixed Oxide (U-Pu) Fuels."

The Purpose of the standard is defined by Sectionl.0. "These criteria establish the necessary siting, design, fabrication, testing, and performance requirements for structures, systems and components important to safety, to the physical security and accountability of special nuclear materials, and to the protection of the environment; thus to provide reasonable assurance that a facility, meeting these criteria, can be operated without undue risk to the health and safety of employees and the public, to the national security, and the natural environment."

The standard has gone through five drafts. Recently , the standard was balloted by the full N46 committee (Fuel Cycle Facilities) and the subcommittee has just finished the resolution of all the comments received.

The finished standard contains many quantitative design bases, some of which have not yet been adopted by any regulatory agency. A series of design basis accidents and events constitute a novel aspect of this standard.

My testimony (20 minutes) will discuss the reasons behind the development of this specific standard, and give some explanation for the numerical limits as recommended by the consensus of the committee.

Sincerely,
Frederick Forscher
Frederick Forscher
Chairman, N 46-4

468

Department of Environmental Health Sciences
Harvard School of Public Health
665 Huntington Ave., Boston, MA 02115
21 November 1974

Director, Criteria and Standards Division (AW-560)
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

Attached is a written statement which I wish to submit for your hearings on "Plutonium and the Transuranium Elements". I do not intend to make an oral presentation.

Sincerely,

William V. Lipton

William V. Lipton

William V.Lip n, Doctoral Candidate 21 November 1974
Department of Environmental Health Sciences, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115

Comments for EPA hearings on "Plutonium and the Transuranium Elements"

Category 3: "Environmental Levels and Pathways"

In the event of widespread Pu-239 contamination of an agricultural area, population exposure through the food chain must be considered. Presently, plant uptake is not considered a significant pathway because of observed soil to plant concentration factors of around 10^{-4}.[1] However, there are indications of an increasing plant uptake over time, as shown in a recent plutonium plant uptake experiment by Romney, Mork, and Larson.[2] Because of the 24,400 year half-life of Pu-239, even a slight increase from year to year could indicate a significant long-term problem.

Among the hypotheses advanced to account for this increase in Pu-239 uptake over time are: chelation of the plutonium in the soil, an increase in plutonium colloid size over time,[3] and a more intimate contact of the plant roots with Pu-239 over time, as the Pu-239 moves down into the soil.[4] In order to test these hypotheses, we are conducting Pu-239 plant uptake experiments where the following variables are controlled:

a. Chelation: Some of the plants are grown with DTPA, a chelating agent, added, and some are grown without added DTPA.

b. Colloid size: A hydrated PuO_2 colloid is created through the titration of plutonium nitrate. This colloid is then separated into 3 size groups, using Nuclepore filters, with different groups being applied to different plants.

c. Depth in soil: The plutonium is applied as a layer of contaminated sand buried at various depths below the surface.

The plants are grown indoors, under artificial lighting. The growing medium is washed, sterilized sand, since this would have a minimum of possibly confounding factors. The containers used are cylindrical jars, 3½" in diameter and 6" tall.

470 Colloidal plutc um, as described above, is used, since this is the form most likely to result under accident conditions.[5]

The principal result, so far, is that chelation has a significant effect on uptake. Comparisons of samples which were similar, except for DTPA application, indicate that,on the average, DTPA applied at a level of 100 ppm (based on dry soil weight) increased uptake by a factor of about 50. Some of the chelated samples showed soil to plant concentration factors greater than 1.

The results for colloid size are inconclusive,so far. Additional experiments are being undertaken, in order to increase the precision of the results and, hopefully, show any effect of this variable.

The results show a decreasing uptake with increasing depth of burial. This is the reverse of what was expected and could be an artifact of the experimental conditions, since the plants in this investigation are grown in a confined space, and the shallower plutonium is incontact with the roots for a longer period of time.

Although the relevance of these results to an environmental situation is open to question, they certainly indicate that at least one factor can significantly increase plant uptake, and that all possibly significant factors should be thoroughly studied. Environmental plutonium can no longer be dismissed, out of hand, as a food chain hazard.

Notes

1. Langham, WH, "Biological considerations of nonnuclear incidents involving nuclear warheads," UCRL-50639 (1969).

2. Romney, EM, Mork, HM, and Larson, KH, "Persistence of plutonium in soil, plants, and small mammals," Health Physics 19:487-491 (1970).

3. Price, KR, "A review of transuranic elements in soils, plants, and animals," Journal of Environmental Quality 2:62-66(1973).

4. Romney et. al., Health Physics 19:487-491(1970).

5. Langham, WH, UCRL-50639 (1969).

Nov. 21 1974

MRS. D. GORDON SHARP
307 GRANVILLE ROAD
CHAPEL HILL, N. C. 27514

EPA
Washington DC 20460

Gentlemen:

Having read thousands of pages on nuclear and breeder reactors and having hoped against hop that clean, safe energy was at hand, I believe coal is cheaper; and - with scrubbers, gassification & liquification, will be safer than nuclear. Surely the breeder is a "Faustian bargain" - "No," I say. Men are yet too undependable to use such dangerous sources. Proponents are whistling in the dark.

If solar & fusion are better in the long run, they're better now. Let us not waste more manpower and resources on nuclear.

We'd rather conserve and live for solar and fusion.

Sincerely,

Jane Sharp

MORATORIUM
ON NUCLEAR
NOW

I CANNOT MAKE THE HEARINGS BUT WISH TO BE COUNTED ON RECORD.

GEORGIA INSTITUTE OF TECHNOLOGY
ATLANTA, GEORGIA 30332

SCHOOL OF
NUCLEAR ENGINEERING

November 22, 1974

Criteria and Standards Division,
Director (AW-560)
Office of Radiation Programs
United States Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

Please find enclosed a copy of the testimony I wish to present before the hearings on Environmental Impact of Releases of Pu and Other Transuranium Elements. I do not wish to give oral testimony since I have accepted membership on the panel. I have sent twenty (20) copies of this paper to you under separate cover.

Thank you for your assistance in this matter.

Sincerely,

Karl Z. Morgan
Neeley Professor

KZM:f
Enclosure

cc: Mr. Claire C. Palmiter

SUGGESTED REDUCTION OF PERMISSIBLE EXPOSURE TO PLUTONIUM AND OTHER TRANSURANIUM ELEMENTS

by

Karl Z. Morgan
School of Nuclear Engineering
Georgia Institute of Technology
Atlanta, Georgia 30332

INTRODUCTION

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

the public domain of large quantities of radioactive material from nuclear power plants, from spent fuel operations, or from shipping accidents.

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

We were constantly resisting pressures of engineers and production supervisors to relax what they called our ridiculous conservatism. Sometimes we were forced to set exposure limits that were lower than our management wanted and perforce they were often little better than guesses because in some areas we had almost no experience or supporting experimental data. For example, one of the earliest papers[1] showing how to calculate dose from internally deposited radionuclides and giving values of permissible body burden and permissible concentration of some 20 radionuclides was delayed for almost a year when I presented it for publication in 1945 because some of the permissible occupational exposure values I calculated were much lower than those in use in weapons production operations. I had at that time almost no metabolic data for some of these radionuclides. For the most part

I had to rely on a series of publication by J. G. Hamilton et al.[2] on the metabolism of fission products, plutonium, and the other actinide elements in mice and rats and in a few cases data on only 3 or 4 rats were available. The maximum permissible internal dose rates for occupational exposure that I used in making these early calculations were 36 R/y for β and γ radiation and 3.6 rep/y (~ 3 rad/y) for α radiation. On this basis and using available metabolic data the value I obtained for ^{239}Pu for maximum permissible lung burden of the occupational worker was 0.035 μCi and for bone burden was 0.42 μCi. The standard man data I used were based on typical human values collected and summarized for me by M. J. Cook.[3]

The first semiofficial values for body burden of the radionuclides were developed at the Chalk River Canada Conference[4] in 1949. These values were later reviewed at the Harwell, England Conference in 1930. From about 1940 to 1973 I was chairman of the Internal Dose Committees of both the International Commission on Radiological Protection (ICRP) and of the National Council on Radiation Protection (NCRP) and so must assume some of the blame for shortcomings of our Handbooks on Internal Dose. During this period there were four principal publications of our Internal Dose Handbooks giving values of organ burden (qf_2) and body burden (q) and maximum permissible concentrations in air $(MPC)_a$ and water $(MPC)_w$ for a large number of radionuclides including values for ^{239}Pu and some of the other actinide elements. Table I summarizes these values of q and qf_2 for ^{239}Pu. Similar values to those in Table I have been given in these same publications for the other actinide radionuclides and for the most part there have been few changes since 1953. In most cases the ICRP and NCRP recommended dose limits are identical. In 1964, ICRP[9] made a few revisions for the actinide ele-

ments but the values for ^{239}Pu remained unchanged.

TABLE I

MAXIMUM PERMISSIBLE BODY BURDENS FOR ^{239}Pu

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

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

Changes Being Considered for Revised ICRP Internal Dose Handbook

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

The present ICRP and NCRP values[7,8,9] of q, qf_2, $(MPC)_a$, and $(MPC)_w$ were calculated on the basis of uniform distribution of the radionuclides in the critical body organ (e.g. uniform deposition in the skeleton) and irradiation only from the deposits of the radionuclide within this organ. These assumptions were made because of a lack of biological information. The assumption of uniform distribution of a radionuclide may have given rather reliable results in some cases for gamma and high energy β-emitting radionuclides that are fairly uniformly deposited in an organ but the risk (of bone cancer) from ^{239}Pu could have been seriously underestimated because most of the α-emitting ^{239}Pu is deposited on bone surfaces of the trabecular matrices adjacent to the thin layer of endosteal tissue which happens to be the most critical tissue in this case. Obviously, the inclusion in the calculation of dose only from the radionuclide deposited within the critical tissue itself could lead to underestimates of the risk except for α and low energy β-emitting radionuclides that are highly localized in the critical organ so that cross irradiation from other organs is insignificant. The decision of

the ICRP has been to consider the critical tissues of the skeleton the endosteal tissue (as it relates to bone cancer) with an average thickness of 10 μm and the active (red) bone marrow (as it relates to leukemia), and to limit the maximum permissible annual dose (MPAD) to these tissues to no more than 15 rem/y (a limit of 1.5 rem/y for members of the general public). Unfortunately our knowledge of the microdeposition of ^{239}Pu in the bone probably is too limited at the present time to apply these refinements and so it is likely the present practice will be continued; namely, calculate the dose from ^{239}Pu to the entire skeleton, as is done with some justification for ^{226}Ra, and apply an N-factor (= 5) to the absorbed dose (rad) as well as the usual Q factor (= 10) for α-radiation in obtaining the dose equivalent (rem) dose.

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

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

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

in which A,B and (MPAD) are defined above, f_2 is the fraction of the radio-

nuclide in the critical tissue of that in the total body, $\mathcal{E}$(MeV) is the total energy deposited in the critical tissue of mass m(g) per disintegration of the radionuclide in the entire body.

The Linear Hypothesis May Not Be Sufficiently Conservative

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

1. The linear hypothesis is based on extrapolations to zero dose of effects of radiation on animals or humans at intermediate to high doses. The points used on the curves at high doses may be on the descending part of the curve, i.e. from portions of the curve where there was overkill or where a large fraction of the highly exposed died of other types of radiation damage and did not survive to die of the radiation effect under study.

2. Extrapolations are made on human data which in general relate human damage such as bone cancer from ^{239}Pu for observation periods of no more than about 20 years. Many of the conclusions are based on studies of animals of life spans less than 10 years. Since man lives for more than 70 years, the slopes of these curves can only increase as more human data are accumulated over his entire life span.

3. The linear hypothesis assumes that man is a uniform and more or less homogeneous population. It applies to the average man and may not be sufficiently conservative for the fetus and for old people. It never takes into consideration special groups such as those studied by Bross[11] where he found

that children of age 1-4 had 3.7 times the risk of developing leukemia if they have allergic disease such as asthma and 24.6 times the risk of the children of this age group if they had both allergic disease and had received intrauterine x-ray exposure.

4. There may be cell sterilization at intermediate and high doses. By this we mean there may be many cells in the body which are likely targets to become precursors of a clone of cells which are malignant but they are killed by the higher doses. In other words, these cells may already have two of the "series cancer switches" closed and a low dose of radiation would likely close the final switch in the step toward cancer production. A high dose such as that from which extrapolations usually are made, however, might kill most such cells as it does in radiation therapy which is used to destroy a cancer.

5. For many types of radiation damage the best fit curve is a plot of equation $E = CD^n$ in which E = effect, C = constant, D = radiation dose, and n = constant. For the linear hypothesis $n = 1$. In some cases $n > 1$ indicating lesser damage per rad at low doses but in many cases the best fit to experimental data is obtained when $n < 1$. Baum[12] recently showed a best fit for cancer induction when $n = 1/2$. In such case the linear hypothesis would be non-conservative.

6. As pointed out above ^{239}Pu is an α-emitting, bone seeking, radionuclide like ^{226}Ra, but unlike ^{226}Ra, it is deposited on the bone surfaces adjacent to the radiosensitive endosteal and periosteal tissues. The use of the N-factor equal to 5 for all α-emitting radionuclides in bone except ^{226}Ra somewhat compensated for this increased risk from surface deposition but has always left some questions to be answered when we determined all q and qf_2

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

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

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

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

No one knows the answer to this question at the present time. Certainly we would like to have more information. Tamplin and Cochran[15] suggest that because of the very large dose (thousands of rem/y) in the vicinity of a micron size particle of ^{239}Pu lodged in lung tissue, the present q for lung (~ 0.015 μCi) and the corresponding values of $(MPC)_a$ for occupational exposure as well as those for members of the public should be lowered by a factor of 10^5. Perhaps they are right, but I believe they have not made a strong case for this factor simply because adequate biological data are not available and much of that which we have seems to give contradictory information. Early experiments of Lisco, Finkel, and Brues[16] have indicated there is a high probability (about 50%) of a malignancy at the site of injections of as little as one μg (~ 0.06 μCi) of ^{239}Pu in the skin of animals and data of

Cember[17] perhaps suggest a higher risk due to localized doses in the lungs. On the other hand, later experiments of Brues[18] have shown when plaques of radioactive materials are placed on the skin of an animal, the risk of skin carcinoma is greater for a uniform distribution of a μCi than for a μCi localized in hot spots. The outstanding research of Bair and Thompson[19] shed much light on the hot particle problem but unfortunately they do not provide us with unequivocal proof that there is or isn't a hot particle problem. They[19] leave the question as one still to be resolved when they state "The mean dose to a tissue may be less important, however, than the dose to localized regions within the tissue." There is no question that epithelial cells of the skin are very radiosensitive and local doses such as are produced by μg quantities of ^{239}Pu in wounds are very carcinogenic. The tissues at risk in the lungs also are epithelial and the most important question remaining is whether or not this large localized dose to the epithelial cells of the lung can likewise result in a high incidence of lung tumors when small dust particles of the highly insoluble $^{239}PuO_2$ are inhaled and find their way to the terminal bronchioles, alveolar epithelial cells, or are translocated to thoracic and abdominal lymph nodes. It certainly is encouraging that there is no clear evidence at the present time that human occupational exposure to plutonium and other transuranium elements has resulted in any form of cancer. We should realize, however, that no extensive epidemiological and autopsy study of the exposed human populations has been completed and with man the average incubation period for tumors of the lung, bone, liver, or lymph nodes may be 40 to 50 years.

In theory at least the occupational exposure values of q and qf_2 for α-emitting radionuclides that are bone seekers have not been set by the use of

equations 1 and 2 in the past but by direct comparison with the value of $q = 0.1\ \mu Ci$ of ^{226}Ra in the human body. It develops, however, that the same values of q and qf_2 as are given by NCRP[7] and ICRP[8] can be obtained by setting (MPAD) in equation 1 equal to 30 rem/y for bone seeking radionuclides. This standard of 0.1 μCi of ^{226}Ra was set by the U. S. Advisory Committee on Safe Handling of Radioactive Luminous Compounds[20] in 1941. The ICRP[8] stated, "At the present time, it would be difficult to say which is more harmful to man a) the dose rate to the total body of 0.1 rem/wk or b) the dose rate to the bone resulting from a body burden of 0.1 μCi of ^{226}Ra . . . . Although tumors have not been observed in persons with body burdens of radium as low as 0.1 μCi, the factor of safety may not be as large as 10 because tumors have been observed in persons having a body burden less than 1 μCi of radium at the time the tumor was first detected. . . . Several workers have described changes in skeletal density and/or histopathological changes in the bone of patients who had 0.1 μCi or less of radium, and more pathological changes may be expected as these individuals become older." In spite of uncertainties regarding the 0.1 μCi standard for ^{226}Ra, it is based on over 50 years of human (not other animal) experience. With proper adjustments to determine the equivalent dose (rem) to the critical body tissue from α-emitting actinide radionuclides, I believe comparison with ^{226}Ra and ^{224}Ra provides the best method now available for setting suitable radiation protection standards for these radioactive materials.

I believe the most reliable values of q based on bone as the critical tissue can be obtained for ^{239}Pu and some other transuranium radionuclides by making use of the comparative data on bone carcinoma and sarcome incidence

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

a) The value of q = 0.04 μCi makes use of an N-factor of 5 for the α-radiation of ^{239}Pu and other α-emitting radionuclides in the skeleton. As pointed out above, this N is intended to be the relative risk from bone seeking, α-emitting radionuclides (e.g. ^{239}Pu) in comparison with ^{226}Ra on the basis of absorbed dose (i.e. on a per rad basis). Data of Daugherty and Mays[21] have shown that this value of N for dogs is somewhere between 5 and 15. If we accept the value of 15, the appropriate value of $q_1 = 0.04 \times 5/15 = 0.01$ μCi.

b) The surface to volume ratio for the trabecular bone of the dog (the tissue in which it is believed most of the bone cancers originate) is about twice that for man. Thus the same amount of ^{239}Pu in man would have twice the concentration of ^{239}Pu near the trabecular surfaces as that in the dog. Making this correction we have $q_2 = 0.01 \times 1/2 = 0.005$ μCi.

c) The rate of turnover (burial) by apposition of new bone of the deposits of α-emitting radionuclides on the trabecular surfaces is probably about ten times that in the dog of that in man. Making this correction we have $q_3 = 0.005 \times 1/10 = 0.0005$ μCi.

d) Studies of Metivier et al.[22] on the survival time of baboons relative to the dog for various concentrations of $^{239}PuO_2$ in the lungs suggest that the baboon is about 4 times as radiosensitive as the dog. Assuming this same ratio would apply for bone burden of ^{239}Pu (perhaps a poor assumption) and that the radiosensitivities of the baboon and man are the same we have then applying this correction factor $q_4 = 0.0005 \times 1/4 \simeq 10^{-4}$ μCi ^{239}Pu.

The above would correspond to an overall reduction in q of $0.04/10^{-4} = 400$ when endosteal tissue of the bone is the critical tissue. Insufficient data are available to attempt any such correction to the value of q for the lungs other than apply correction (d) above. Thus we would have $q = 0.015/4 \simeq 0.004$ μCi when total lung is the critical tissue. This of course does not address the hot particle problem but rather shelves it until we have more data.

A somewhat similar problem, namely the possible use of pulmonary lymph nodes as the critical body organ for $^{239}PuO_2$, has been under discussion for many years by Committee 2 of ICRP. There is no question but that when dogs inhale $^{239}PuO_2$ in finely divided particles a major fraction ends up in the thoracic lymph nodes. Park et al.[23] for example give the percents of alveolar-deposited $^{239}PuO_2$ 11 years after exposure of about 40% for thoracic lymph nodes, 13% for liver, and 5% for bone. After many years of consideration of this question the ICRP finally decided not to use the lymph nodes

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

REFERENCES

1. K. Z. Morgan, "Tolerance Concentrations of Radioactive Substances," J. of Physical and Colloid Chem. 51, 984-1003 (1947).

2. J. G. Hamilton et al. A series of 80 papers published by Hamilton and his group at the University of California from 1937 to 1947. Summarized in the Bibliography for Biological Data by M. J. Cook, Health Physics 3, 235-380 (1960).

3. M. J. Cook. These data on the standard man collected by Ms. Cook of Oak Ridge National Laboratory in 1946 were never published but these data together with data collected by H. Lisco of the Argonne National Laboratory were the basis of the standard man data in the 1949 Chalk River Reports.

4. Chalk River, Canada Conference of members of radiation protection committees from the United States, United Kingdom, and Canada, September 29 and 30, 1949. Minutes of this meeting were never published but separate sets of minutes by K. Z. Morgan, W. H. Langham, G. J. Neary, and G. E. McMurtrie were rather widely circulated in 1950.

5. Maximum Permissible Amounts of Radioisotopes in the Human Body and Maximum Permissible Concentrations in Air and Water, NCRP - Handbook 52 - NBS - March 20, 1953.

6. "Recommendations of the International Commission on Radiological Protection," British J. of Radiology, Sup. No. 6, 1-92 (1955)

7. Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and Water for Occupational Exposure, NCRP - Handbook 69 - NBS - June 5, 1959.

8. Permissible Dose for Internal Radiation, ICRP - Pub. 2, Pergamon Press (1959).

9. Recommendations of the International Commission on Radiological Protection, ICRP Pub. 6, Pergamon Press (1964).

10. K. Z. Morgan, "Proper Use of Information on Organ and Body Burdens of Radioactive Materials," IAEA/WHO Symposium on the Assessment of Radioactive Organ and Body Burdens, Stockholm, Sweden, Nov. 22-24, 1971, IAEA/SM/150-50.

11. I. D. J. Bross, "Leukemia from Low-Level Radiation," New Eng. J. of Med. 287, 107-110 (July 20, 1972).

12. J. Baum, "Population Heterogeneity Hypothesis on Radiation Induced Cancer," given orally at Houston, Tex. meeting of the Health Physics Society, July 10, 1974.

13. C. W. Mays and T. F. Daugherty, "Progress in the Beagle Studies at the University of Utah," Health Physics 22, 793-801 (June 1972).

14. C. W. Mays, H. Spiess, and A. Gerspach, "Skeletal Effects Following ^{224}Ra Injections into Humans," Reported at the Symposium on Biological Effects of Injected ^{224}Ra and Thorotrast, Alta, Utah, July 21-23, 1974. To be published in Health Physics early in 1975.

15. A. R. Tamplin and T. B. Cochran, "Radiation Standards for Hot Particles," publication of Natural Resources Defense Council, 1710 N. Street, N.W., Washington, D. C. 20036, February 14, 1974.

16. H. Lisco, M. P. Finkel, and A. M. Brues, "Carcinogenic Properties of Radioactive Fission Products and of Plutonium," Radiology 49, 361 (1947).

17. H. Cember, "Radiogenic Lung Cancer," Progress in Experimental Tumor Research, Vol. 4, p. 251 (1964); H. Cember, J. A. Watson, and T. B. Brucci, "Pulmonary Effects from External Radiation," presented at AIHA meeting in Philadelphia, Pa., April 26, 1956.

18. A. M. Brues, unpublished experiments, Argonne National Laboratory (1953).

19. W. J. Bair and R. C. Thompson, "Plutonium: Biomedical Research," Science 183, 715-722 (Feb. 22, 1974).

20. National Bureau of Standards Handbook 27, "Safe Handling of Radioactive Luminous Compounds" (1941).

21. T. F. Daugherty and C. W. Mays, "Bone Cancer Induced by Internally-deposited Emitters in Beagles," in Radiation Induced Cancer, International Atomic Energy Agency, Vienna, 361-367 (1969).

22. H. Metivier, D. Nolibe, R. Masse, and J. Lafuma, "Excretion and Acute Toxicity of $^{239}PuO_2$ in Baboons," Health Physics 27, 512-514 (Nov. 1974).

23. J. F. Park, W. J. Bair, and R. H. Busch, "Progress in Beagle Dog Studies with Transuranium Elements at Battelle-Northwest," Health Physics 22, 803-810 (June 1972).

490 GENERAL ELECTRIC

GENERAL ELECTRIC COMPANY, 175 CURTNER AVENUE, SAN JOSE, CALIFORNIA 95114
Phone (408) 297-3000, TWX NO. 910-338-0116

NUCLEAR ENERGY DIVISION

BWR PROJECTS DEPARTMENT

November 22, 1974

Director, Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D. C. 20460

SUBJECT: PUBLIC HEARING - PLUTONIUM AND THE TRANSURANIUM ELEMENTS

Dear Sir:

In accordance with the Federal Register notice of October 24, 1974 and Dr. W. D. Rowe's letter of October 25, 1974, the General Electric Company requests the opportunity to present testimony at the subject hearing scheduled for December 10, 1974. We plan to submit a written statement at that time, and we also request the 20-minute period suggested in the hearing notice for the presentation of an oral summary of our statement.

The General Electric statement will address the subjects of:

1. The views of the General Electric Company for the utilization of plutonium for power production from domestic fuel sources both for recycling in light water reactors and in fast breeder reactors;

2. The need for environmental standards which are based on reasonable and proper evaluation of risk, with due regard for costs and benefits, so that the beneficial use of plutonium for power production may be realized;

3. The experience of the General Electric Company on the environmental aspects of plutonium, both in the operation of the SEFOR experimental facility and in the handling of plutonium over the past fifteen years at our Vallecitos Nuclear Center.

Our statement will be sponsored by Mr. George J. Stathakis, Vice President and General Manager, Nuclear Energy Products Division, General Electric Company, San Jose, California.

Sincerely,

Ivan F. Stuart

Ivan F. Stuart, Manager
Safety and Licensing
Mail Code 683 Ext. 2791

/lml

BE SURE TO INCLUDE MAIL CODE ON RETURN CORRESPONDENCE

Atomic Industrial Forum, Inc.
475 Park Avenue South
New York, New York 10016
Telephone: (212) 725-8300
Cable: Atomforum Newyork

Carl Walske
President

November 22, 1974

The Director
Criterion Standards Division (AW-560)
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

In accordance with the notice published in the Federal Register, October 24, 1974, time is hereby requested to make an oral presentation at the public hearing on plutonium and the transuranium elements scheduled for December 10. Our presentation will be in the form of a panel discussion which will include several experts in the plutonium field. The names of the panelists will be forwarded to you as soon as final arrangements are concluded and in any event, prior to commencement of the proceedings. The panelists will discuss in depth the items designated in paragraphs 1 through 5 of the October 24 Federal Register notice, with particular emphasis on adequacy of standards, the health effects of plutonium and experience with plutonium to date. It is anticipated that 60 minutes will be required for this presentation.

Communications with respect to this matter should be directed to me at the above address.

Sincerely,

Carl Walske

CW:gjh

Westinghouse Electric Corporation Power Systems

PWR Systems Division
Box 355
Pittsburgh Pennsylvania 15230

November 22, 1974
NS-RS-447

Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

In response to the invitation of the Environmental Protection Agency ("EPA") appearing in 39 F.R. 37810 on October 24, 1974, the Westinghouse Electric Corporation ("Westinghouse"), P. O. Box 355, Pittsburgh, Pennsylvania 15230, wishes to notify the EPA of its desire to present an oral statement (and to submit a written statement for the record) at the public hearing being conducted by EPA to ascertain whether any new standards are needed to assure protection of the environment and of the public health from potential contamination by radionuclides of the transuranium elements. Mr. Frederick W. Kramer, Engineering Manager, Nuclear Fuel Division, and Dr. J. H. Wright, Director, Environmental Systems Department, will make the presentation on behalf of Westinghouse.

We estimate that about twenty (20) minutes will be needed to complete our presentation. A summary of the areas to be covered in our presentation follows:

1. General

Westinghouse is pleased to have this opportunity to participate in EPA's effort to obtain further information on projected releases to the environment of transuranic elements, the costs associated with these projected releases, suggestions on possible alternative actions, and the resulting potential environmental and public health impacts for the purpose of evaluating the need to establish, at this time, new environmental standards. Westinghouse suggests that generally applicable standards for environmental levels of plutonium and the other transuranium elements must be based on health effects data and, insofar as possible, on cost-benefit analyses which compare alternative control options in activities comprising the plutonium fuel cycle in the context of present fallout

levels, as well as with alternative energy options. With respect to data on health effects, we urge that only authoritative, documented, and well reviewed sources be used by the EPA in developing its standards. We recommend that whatever risk estimates or radiation effects data are utilized by EPA to develop new standards, that such factors be balanced by careful consideration of the total environmental risks associated with nuclear and non-nuclear options relative to the magnitude of radiation from natural background, ambient levels of plutonium from fallout, and medical applications in determining the total dose commitment to the population.

Westinghouse believes that the current regulations and procedures for controlling releases of plutonium and the other transuranium elements are adequate and that there is merit in obtaining further operational experience and R&D before developing revised or additional standards. We fully endorse EPA's policy of issuing environmental impact statements in connection with formulating generally applicable radiation standards under the Atomic Energy Act of 1954, as amended.

2 & 3. Dosimetry, Health and Environmental Effects; Environmental Levels and Pathways

With respect to the construction and operation of facilities handling plutonium, Westinghouse identifies important pathways for any possible releases of transuranics through the ecosystem. Westinghouse also demonstrates how limits are placed on permitted plant effluents, and how monitoring of plant effluents in the environment is performed to assure that these limits are not exceeded.

In this connection, it is suggested that considerable information exists to permit projections of health effects from measured concentrations of plutonium, and of the pathways for transport of plutonium through the ecosystem.

Admittedly, such projections are necessarily approximate, and continuance of the already extensive research programs is encouraged. However, it is suggested that use of this information in conjunction with careful monitoring of the effluents from existing and near-term projected plutonium handling facilities will verify that the impact of such operations is kept acceptably small.

4 & 5. Applications Using Plutonium; Control and Cleanup Technology

The statement by Westinghouse presents current and projected uses of plutonium as fuel in the light-water and breeder reactors for commercial power generation. We present the Westinghouse estimate of the projected quantities of plutonium available from the overall light-water reactor industry during a period through approximately 1990. The Westinghouse projections of the plutonium available from the light-water reactors are consistent with the data on plutonium availability set forth in GESMO (WASH-1327, August 1974). On the assumption that plutonium recycle will constitute the most prominent use of plutonium until the Liquid Metal Fast Breeder Reactor makes its substantial contribution to the quantities available and used, we believe that the magnitude of the release of transuranium elements can be quantified by consideration of the actual operating experience of the Westinghouse Plutonium Fuels Development Laboratory (PFDL) and on the basis of the projected effects of the proposed Westinghouse Recycle Fuels Plant (RFP). Our presentation includes data on the magnitude of possible releases to the environment from the Westinghouse PFDL and the projected possible releases from the proposed Westinghouse RFP.

The Westinghouse statement also identifies the plant protection devices and installations utilized in fuel fabrication facilities for minimizing and restricting possible releases of transuranium elements to the environment. Methods for decommissioning fuel fabrication facilities are described. Release data, demonstrating the effectiveness of currently available technology, for the Westinghouse PFDL are also presented.

Very truly yours,

Romano Salvatori

Romano Salvatori, Manager
Nuclear Safety Department

/smh

2677 Ellendale Place
Los Angeles, Calif. 90007

November 24, 1974

Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

Pursuant to the hearings beginning December 10, 1974 on plutonium and the transuranium elements, I most strongly urge the Environmental Protection Agency to set forth definitive and detailed maximum permissible levels of ground contamination for these elements (1) within occupied structures, (2) outdoors in inhabited areas, (3) on cropland, (4) on grazing areas for livestock. In those cases where significant solubility is involved, MPC's for public water supplies are also requisite.

The importance of setting definite standards before the first major releases of these substances cannot be overstressed. Following a large accidental or malevolent dispersion in an urban area, there would be tremendous political pressure not to evacuate, or to engage in less than adequate decontamination efforts, lest citizens be alarmed or discommoded by such measures. This cannot be permitted to occur. The first urban incident, in the absence of clear EPA guidelines, will no doubt set a norm of negligence with perhaps cumulatively devastating effects.

As an indicator of the importance of the subject, I enclose some recent correspondence, in which the Atomic Energy Commission did not dispute my estimate that one pound of power-reactor-grade plutonium, if dispersed as oxide particles, could bring 57 square miles of downtown Washington to levels requiring some evacuation and cleanup. EPA is deserving of the highest commendation for addressing this most vital topic.

Very truly yours,

L. Douglas DeNike

L. Douglas DeNike, Ph.D.

Enclosures
Letter to President 8-24-74
Reply from AEC 10-3-74
Letter to President 10-7-74

2677 Ellendale Place
Los Angeles, Calif. 90007

August 24, 1974

President Gerald R. Ford
The White House
Washington, D. C. 20500

Dear President Ford:

So-called "reactor grade" plutonium, as recovered from nuclear power plant spent fuel, is 6.6 times as radiocontaminative as fairly pure plutonium-239. The numerical proof of this is appended to this letter.[1]

Two eminent nuclear scholars, Mason Willrich and Theodore B. Taylor, state that dispersion of 100 grams (less than ¼ pound) of plutonium-239 could produce "significant contamination requiring some evacuation and cleanup" over 5,000,000 square meters (1.93 square miles).[2] This works out to 8.74 square miles per evenly distributed pound of Pu-239. But since reactor-grade plutonium is 6.6 times as hazardous, one pound of the latter could bring 57 square miles to levels requiring some evacuation and cleanup.

This area is equivalent to a circle with its center at the White House and its perimeter, 4.3 miles distant, enclosing virtually all the essential government facilities of Washington, D. C. and adjoining Virginia.

Does it require an atomic explosion to destroy the nation's capital as a center of government? It appears that the answer is in the negative. It appears that the only requirement is possession of one pound of the material (recycled plutonium) which the Atomic Energy Commission hopes to make the <u>primary energy source of the nation</u>.

Questions will occur to the reader with regard to this conclusion. Would not plutonium be difficult to obtain by theft? No; as recently as last April the AEC's own consultants called for immediate and drastic upgrading in the safeguarding of special (fissionable) nuclear materials.[3]

Would not the handling and dispersion of plutonium be difficult and dangerous for unauthorized persons? No; the alpha-particle radioactivity characteristic of plutonium is non-penetrating. Thus it would not threaten criminals or terrorists who handled it in improvised gloveboxes. Its dispersion could be simply effected in three ways: (1) By burning in an ordinary fire. Plutonium metal is pyrophoric; i.e., it tends to ignite spontaneously, forming fine oxide particles which present an unparalleled lung-cancer hazard if inhaled. (2) By attaching the packaged metal or oxide to an ordinary chemical explosive charge. (3) By attaching a leaky container of an insoluble plutonium compound to the underside of a public transit vehicle having a known route.

But granting the foregoing, could not dispersions of radionuclides be readily removed by hosing, or rainfall? Hosing would be expensive, and would presuppose evacuation until suited and masked decontamination teams could complete it. Whether plutonium particles could be sufficiently removed by hosing, rainfall, or any other means within acceptable dollar and time limits is very uncertain. A large number of surfaces, ranging from lawns to rooftops, would tend to trap and hold radioactive particles for later resuspension (and thus possible inhalation) by wind, earth-moving operations, etcetera. Maleficent persons could compensate in advance for the estimated efficacy of decontamination procedures by releasing a proportionately greater amount of radioactivity per unit of area. In that regard, it should not prove significantly more difficult for evil-doers to divert or smuggle from abroad ten or more pounds of plutonium than a single pound of the material. Roughly eighteen pounds of reactor-grade plutonium would suffice for the construction of a "crude" atomic bomb, of course.[4]

My contention here is that the radiocontamination of Washington, D. C. with long-lived, extremely carcinogenic mixed isotopes of plutonium in particulate form might be nearly as disruptive to the nation as its destruction by a nuclear explosion. A "crude" low-yield implosion bomb might be expected to scatter particles of unfissioned plutonium downwind, producing much the same effect as a dispersal device.

Mr. President, let me be quite candid. If the nuclear power reactor enterprise, involving the uranium-plutonium fuel cycle, is permitted to expand as projected, the fulfillment of the above scenario is only a matter of time. It is beyond the puissance of the government of the United States to establish adequate safeguards for the handling of these materials when fifteen, soon to be thirty, sovereign nations are involved. However, the government does have the power to seek a bilateral declaration with the Soviet Union, stating that the two superpowers will take any steps necessary to prevent additional nations from acquiring nuclear-power capacity.

I am writing a popular-level book on radiological terrorism, crime, and warfare. Nothing would give me greater pleasure than to be dissuaded from publishing it by prompt government action toward the dismantling of the fission-power aberration here and abroad. However, nothing short of that will dissuade me from what I feel is necessary to prevent otherwise certain national suicide. Needless to say, if I do not write the book, another will do so. I do not enjoy the acute moral dilemma involved in presenting such information as this to a general readership. Yet, circumstances have brought us to the point at which patriotism must take unusual forms.

Very truly yours,

L. Douglas DeNike

L. Douglas DeNike, Ph.D.

Enclosures

References
Radioactive Malevolence

Footnotes

1. Each metric ton of spent power-reactor fuel contains about 13.04 kilograms of plutonium isotopes, with activity as follows:

Isotope	Activity (curies)	Air concentration to lung dose conversion factor
Pu-238	4,000	1
Pu-239	500	1
Pu-240	650	1
Pu-241	150,000 (times .001=150)	.001
Total activity	5,300 curies of Pu-239 equivalent lung-dose activity.	

Each 16.2 grams of Pu-239 contain one curie of alpha activity; thus 5,300 curies of Pu-239 would weigh 85.86 kilograms, or 6.6 times the weight of the equivalent in power reactor grade plutonium, 13.04 kilograms.

Reference: Environmental Protection Agency report EPA-520/4-73-002, Environmental Radiation Dose Commitment: An Application to the Nuclear Power Industry, February 1974, Tables B.4 and D.2.

2. Willrich, M., and Taylor, T. B. Nuclear Theft: Risks and Safeguards, Ballinger, 1974, p. 25, Table 2-2.

3. "The Threat of Nuclear Theft and Sabotage," Congressional Record, April 30, 1974, pp. S 6621-6630.

4. Willrich & Taylor, op. cit., p. 13.

UNITED STATES
ATOMIC ENERGY COMMISSION
WASHINGTON, D.C. 20545

OCT 8 1974

Dr. L. Douglas DeNike
2677 Ellendale Place
Los Angeles, California 90007

Dear Dr. DeNike:

Thank you for your letter to President Ford dated August 24, 1974, which has been referred to me for response. Your letter, as well as articles and other correspondence you have had with the AEC, evidences a good knowledge of and concern for the possibilities of nuclear related terrorism.

Some of the various problems to which your letter refers certainly require vigorous and effective action in order to assure that the benefits of nuclear energy may be obtained with minimum risk. Your view that the spread of nuclear-power capacity in the U.S. and abroad presents so great a hazard that this industry should be dismantled, is not universally shared. There is broad concensus here and abroad that nuclear energy is an essential element of the power industry of the future. Recognizing, however, that the future development of this nuclear capacity may increase the possibilities of nuclear terrorism, the AEC, (as well as other government agencies), is expanding its safeguards efforts. Your letter mentioned the expansion of nuclear power operating capacity in sovereign nations. The U.S. will participate in such expansion only when we are confident that proper safeguards are to be adopted. However, there are options open to foreign nations to develop their nuclear power capacity without using U.S. (or Soviet) nuclear technology. We are not unmindful of the problems arising from this situation, and we are trying to increase the effectiveness of safeguards in pertinent areas so far as we are able.

The book you propose to write may serve a useful purpose. You are evidently aware of some of the problems of dealing effectively with these subjects without contributing to an increase in the hazards. If it would be helpful to you, we would be happy to communicate further with you about questions you may have as to the direction and effectiveness of our safeguards program.

Sincerely,

A. B. Siebert
(FOR)

Edward B. Giller
Assistant General Manager
for National Security

500

2677 Ellendale Place
Los Angeles, Calif. 90007

October 7, 1974

President Gerald R. Ford
The White House
Washington, D. C. 20500

Dear Mr. President:

On August 24, I wrote to you providing my calculation that a single pound of plutonium recovered from nuclear power plant spent fuel would suffice to contaminate, to levels requiring evacuation and cleanup, virtually all the essential government facilities of Washington, D. C. and adjoining Virginia. I am now in receipt of the Atomic Energy Commission's reply to this letter, dated October 3, over the name of Edward B. Giller. Copies of both pieces of correspondence are enclosed herewith.

General Giller, the AEC's security chief, did not even attempt to refute my claim. He could not have done so successfully, for the contamination of Denver from the plutonium fire at Rocky Flats (September 11, 1957) testifies adequately to the environmental mobility of wind-borne plutonium particles. Indeed, the AEC has conducted extensive studies on the transport of plutonium in the environment; see, for example, pages 4.G-23 through 4.G-26 of the draft AEC document, WASH-1535, in which plutonium releases at smokestack level in Illinois are predicted to deposit throughout the eastern United States.

One could not guess the subject matter of my letter from General Giller's reply, and his failure to address it can be construed as a tacit admission of its accuracy. He goes on to credit me with "good knowledge of and concern for the possibilities of nuclear related terrorism." The basic truth of my assertion appears in itself to be adequate cause for the White House to hold in abeyance the AEC's proposal for the large-scale use of recycled plutonium reactor fuel, which is presently in the stage of draft environmental-statement review.

General Giller states that there is "broad concensus (sic) here and abroad that nuclear energy is an essential element of the power industry of the future." Herein I will indicate why any existent consensus is spurious, inasmuch as it is based upon unexamined hopes regarding the possible efficacy of safeguards in the atomic power field. This topic is further explored in my enclosed review from the October issue of the Bulletin of the Atomic Scientists.

There exists only one possible precedent for the level of security presumed to be maintainable for fuels in a fully developed fission-power economy. This is the relatively good record held thus far by the nuclear weapons industry in preventing the theft and deliberate dispersion of radioactive materials. For several reasons, the past record provides insufficient assurance

for the future. (A) Initially, the safeguarding of American nuclear weapons abroad has recently been criticised as "deplorable" by Senator John O. Pastore, Vice-Chairman of the Joint Committee on Atomic Energy. This suggests that the security of such weapons inheres to a significant degree in the fortuitous ignorance of would-be thieves and terrorists regarding their vulnerability. (B) In contrast to the six sovereign nations now known to be involved in the manufacture of nuclear explosives, by 1980 there will be thirty nations expected to have nuclear power stations (M. Willrich and T. B. Taylor, Nuclear Theft: Risks and Safeguards, page 197). (C) As demonstrated in my letter of August 24, the plutonium utilized in this burgeoning industry will be 6.6 times as radio-contaminative, weight for weight, as pure plutonium-239--capable of bringing 57 square miles per pound to evacuation levels.

(D) Roughly 200 tons of plutonium-239 have thus far been produced for U.S. nuclear weapons. The quantity of plutonium anticipated to be generated in U.S. civilian power reactors will exceed this within ten years. (E) Information concerning safeguards and security measures in the plutonium-fuels industry will over time leak irreversibly to the public, and to public enemies. Coordinately, the information-gathering abilities and weapons-construction talents of public enemies can be expected to improve.

In clear recognition of the inadequacy of present safeguards-security standards for the future, the AEC has proposed creation of a federal guard force, and the requirement of AEC security clearances for plutonium workers, in its generic environmental statement on plutonium recycle (August, 1974). Such tightened precautions will not directly affect the practices of other nuclear nations, whose diverted materials may promptly reappear in criminal or subversive possession within the United States. The clear international ramifications of the fissile-materials problem are for some a signal for despair. For the government of this country, they should be a signal for top-priority efforts to remove the peril.

Exhaustive study reveals only one way by which this peril can be removed. Even if a paper-perfect safeguards system is implemented on a planetary basis, actual practice will fall short of the regulations--and shortfalls are unacceptable. We note that in the American nuclear power industry during fiscal 1974, the AEC logged a total of 3,333 safety violations. We may look forward to violations of safeguards regulations. Perfection is necessary, and perfection is unattainable.

Another approach will demonstrate that the manageability of the nuclear safeguards problem is an article of faith, not a scientifically grounded judgment. It suffices to note the absence of any formal or informal, public or secret "stop criterion" for nuclear-power development. Every nuclear plant has an elaborate set of pre-specified quantitative limits, whose breach results in automatic shutdown, lest continued operation result in an unmanageable situation. Yet no such advance set of shutdown criteria exists for the nuclear industry as a whole. There may be good reasons why such criteria, if they exist, should not be made public. Yet it is my confident assertion that they do not exist, even as top government secrets.

There is, furthermore, no assurance that should shutdown of the fission power industry be undertaken (at such future [illegible] as criminal acts involving it have become clearly unacceptable), that this can be accomplished with reasonable safety from further depredations. In brief, there appears to be no contingency planning whatever for the orderly phaseout of nuclear power, should untoward events show this to be necessary. Perhaps the government fears that such contingency planning, if undertaken, would leak out to the public, further demoralizing the atomic energy industry. If so, the government might do well to apply the same concern about information leakage to the utilities' proprietary facts relevant to nuclear sabotage and theft, which are bound to seep out via loose-lipped and disgruntled employees.

Considerably more could be written to the effect that the fission-energy enterprise is an outgrowth of faith, and not a technology tied to defensible suppositions about the nature of human beings and their social arrangements. But for now, I submit the necessity of the Executive Branch's developing the contingency planning delineated above. This should include: (1) Unambiguous quantitative criteria for reaching the conclusion that nuclear safeguards and security measures are unmanageable in principle, based upon analysis of possible future events. (2) Plans for the orderly and reasonably secure phaseout of fission-based energy systems, on a transnational scale, should the first condition materialize. Even with its broadened mandate as ERDA, I doubt that the Atomic Energy Commission can be entrusted with the development of these necessary contingency plans. Its unwillingness to address such issues objectively is legendary. Rather, the White House must seek a source of expertise which is independent of the AEC or its successor.

Sincerely yours,

L. Douglas DeNike

L. Douglas DeNike, Ph.D.

Enclosures

CORPORATION

LEON LEVENTHAL
GENERAL MANAGER

LFE ENVIRONMENTAL
ANALYSIS LABORATORIES DIVISION

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18 November 1974

Office of Radiation Programs
East Tower
Environmental Protection Agency
401 M Street, S.W.
Washington, D. C. 20460

Gentlmen:

I recently noted there will be public hearings starting December 10th, 1974 to examine the need for establishing radiation protection standards for plutonium and other transuranium elements in the environment. I would appreciate a copy of the detailed agenda and schedule. I would also like to get a copy of the hearing transcript when it is available.

Our organization has been involved in the analysis of the transuranium elements for a number of years. For the record, I gave a paper on "Analytical Considerations Connected with the Transuranium Elements", at the American Nuclear Society Meeting in Washington, D, C. on October 31st. This was part of an invited session on Environmental Levels of the Transuranium Elements, which I chaired. I also gave a paper the previous week at the Analytical Conference at Gatlinburg entitled, "A Survey of Radiochemical Techniques for the Assessment of Plutonium and Americium in Environmental Samples". So you see, we are very much interested in this subject.

Very truly yours,

Leon Leventhal

LL/kc

504

11 Brown Road
Great Neck, N. Y. 11024

November 19, 1974

Environmental Protection Agency
Office of Radiation Programs
AW - 560
Washington, D.C. 20460

Gentlemen:

This is with reference to the item entitled "EPA to Weigh Setting Plutonium Standards", Citizens Bulletin, October 1974.

An attempt to set standards for release of plutonium into the environment would be tantamount to legitimizing the spread of a very toxic and longlasting pollutant as a legacy for our children and their children.

The public should not be exposed to even minute quantities of plutonium. It is inconceivable that so poisonous, pernicious and irreversible a poison as plutonium should be accepted, even in microgram quantities, as "limits" by EPA.

Mankind can no more coexist with plutonium than it can with botulin toxin. Setting standards for either poison would make no sense and would be equally irrelevant.

Please do not help legitimize the ultimate pollutant.

Sincerely yours,

Nat. H. Sauberman, P.E.

UNITED STATES
ATOMIC ENERGY COMMISSION
WASHINGTON, D.C. 20545

November 29, 1974

Director
Criteria and Standards Division
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

Pursuant to the Federal Register Notice of Thursday, October 24, 1974 (39 FR 37810), this is to give notice of the Atomic Energy Commission's (AEC) intent to participate in the public hearing to be held by the Environmental Protection Agency beginning December 10, 1974, on plutonium and transuranium elements.

In order to cover adequately the overall activities of the AEC and to reflect the division of responsibilities and programs within the AEC, separate testimony will be presented by the General Manager's operating side and the Regulatory side. Testimony from the General Manager's side will be presented first since it does reflect the scope of the entire subject of plutonium and as such better sets the stage for the Regulatory input. For your information, please find enclosed two summary outlines which identify the major topics to be addressed in the AEC testimony.

As indicated in the outline, there are a number of individuals under the leadership of Dr. James L. Liverman, Assistant General Manager for Biomedical and Environmental Research and Safety Programs, scheduled to address the General Manager's program. In addition, supplementary information will also be submitted for the hearing record. The General Manager testimony will include an introductory statement which provides an overview and scoping of the subject. This will be followed by a series of presentations by AEC Headquarters and laboratory representatives which address: (1) potential source terms and control measures; (2) environmental levels and pathways; and (3) biomedical effects.

Lester Rogers, Director of Regulatory Standards, will make a brief statement, approximately 30 minutes long, on the regulatory program. This statement will summarize the detailed written information being submitted by Regulatory for the record. Mr. Rogers will describe the manner in which the AEC develops its regulations and exercises its regulatory responsibilities for commercial activities involving plutonium and transuranium elements. The testimony will summarize AEC regulations already issued or under development which concern the releases of plutonium and transuranium elements to the environment. In addition, it will summarize licensing experience and future projections concerning the magnitude of the quantities of plutonium and transuranium elements in commercial fuel cycle facilities and products.

As the General Manager testimony is still in preparation, specific times for each presentation have not been assigned as yet. However, it is anticipated that the total time necessary for both General Manager and Regulatory presentations will approximate four hours. Your office will be advised as soon as possible of more definitive time requirements.

Sincerely,

Paul C. Bender

Paul C. Bender
Secretary of the Commission

Enclosures:
1. Outline of General Manager Testimony
2. Outline of Regulatory Testimony

OUTLINE OF GM TESTIMONY FOR EPA HEARING

December 10, 1974

A. Headquarters Introduction - James L. Liverman

1. General scoping of the subject
2. Existing information, location, how to find it
3. How this information has been used
4. What additional information is needed
5. Current and projected research to address those needs

B. Potential Source Terms and Control Measures - Robert E. Yoder

1. Transuranium fuels and recycle
2. Weapons and production
3. SNS operations
4. Waste handling
5. Medical uses

C. Environmental Levels - Burton Bennett/McDonald E. Wrenn

1. Location, composition, and origin

   a. worldwide
   b. local
   c. media

2. Disposition with time
3. Environmental pathways

D. Biomedical Effects - William J. Bair, Chet Richmond, William Burr, Roy Thompson

1. Health effects - experimental
2. Health effects - human experiences
3. Implications with respect to protection criteria

OUTLINE OF REGULATORY TESTIMONY FOR EPA HEARING

December 10, 1974

A. Regulations Controlling Population Exposures to Radiation

B. Relative Regulatory Roles of AEC and EPA

C. Examples of the Exercise of Regulatory Responsibility by AEC

D. Characterization of Commercial Facilities Processing Large Quantities of Plutonium and Transuranium Nuclides

E. Releases of Plutonium and Transuranium Nuclides to the Environment from Commercial Facilities

F. Use of Technical Specifications and Monitoring to Assure Safe Operation of Facilities Processing Large Quantities of Plutonium and Transuranium Nuclides

G. Standards for Controlling Levels of Plutonium and Transuranium Nuclides in the General Ambient Environment

NATIONAL AUDUBON SOCIETY
CENTRAL MIDWEST REGIONAL OFFICE
ROUTE 1, BOX 19 • MAUCKPORT, IN. 47142 • (812) 732-4349

November 21, 1974

Director, Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

As per your request for comments, either written or oral, re: the potential adverse environmental impact of releases of plutonium and other transuranium elements, would you please enter the enclosed written comments into the record on behalf of the:

Central Midwest Regional Office of the National Audubon Society
Ohio Audubon Council
Indiana Advisory Committee to the National Audubon Society
Kentucky Audubon Council
Audubon Council of Illinois

Special credit should be given to our specialist, Dr. Owen Davies of the Black River Audubon Society, Elyria, Ohio.

Sincerely,

Myron J. Swenson
Central Midwest Representative

MJS/tma

The U. S. Environmental Protection Agency would not be conducting a public hearing on plutonium and the transuranium elements if major uses of these elements were not planned. Certainly among the applications being considered is the fast-breeder nuclear reactor and the use of plutonium produced therein as a fuel for the light water reactors. The major question really should be : "Can our society in any way accept the wide spread use of plutonium?" For plutonium is not just another run-of-the-mill potential environmental pollutant.

The Atomic Energy Commission states that:

(1) The alpha radiation given off by plutonium is especially damaging to the blood-forming organs of the bones and can produce bone diseases many years later.

(2) Plutonium may enter the body through cuts or abrasions of the skin, by being swallowed, or most importantly by inhalation. (Small amounts of inhaled plutonium can cause lung cancer.)

(3) Once in the body, plutonium is eliminated so slowly that as much as 80% of any amount taken in will still be there 50 years later.

(4) The maximum permissible body burden, or the total amount of plutonium that can be accumulated in an adult without producing undue risk to health, has been set at 0.6 microgram.

(The reference for items 1 through 4 is "Plutonium", a booklet of the Understanding the Atom Series of the Division of Technical Information of the Atomic Energy Commission.)

(5) Plutonium-239 has a half-life of approximately 24,000 years and thus will take hundreds of thousands of years to decay to an innocuous level of radiation.

(6) There will be an unavoidable loss rate of 1 to 2% in handling plutonium. (Science, April 9, 1971, pp. 143-146) This is most serious, since the plutonium inventory has been projected to be 720,000 kilograms by the year 2000. (Science, April 9, 1971, pp. 143-146)

The tons of plutonium that would be produced by the fast-breeder reactors would be transported across the nation. Such shipments will inevitably be involved in highway accidents and will be subject to hijackings and diversion for the production of nuclear bombs.

It would seem inevitable that large groups of citizens would be subjected to security clearance and security procedures. The present situation, where Texas state police maintain files on nuclear power plant opponents, would be minor by comparison.

The Environmental Protection Agency is attempting to perform a cost-benefit analysis on plutonium. Yet how can such an analysis take into account the certain loss of individual freedom and rights to individual privacy? How can the value of human life be evaluated in a cost-benefit analysis?

The recent disclosure of the leakage of plutonium at the Mound Laboratory at Miamisburg, Ohio does not inspire confidence that humans can maintain the necessary vigilance to operate a plutonium industry successfully. In 1974, unexpectedly high plutonium levels were found in nearby water areas and on portions of the land at the laboratory. The

source of the plutonium has subsequently been indicated to be a pipe which reportedly had been leaking since 1969. One wonders how many incidents of a like nature have occurred and have either remained undetected or have been "covered-up". There apparently is no possible way to evaluate the consequences of the Miamisburg leak, since there is no reliable means to establish that the plutonium that leaked has been completely contained. The monitoring program at the Mound Laboratory obviously was not proper or the leak would not have gone undetected for such a length of time. With a widespread plutonium industry, such lax monitoring techniques would eventually lead to a major contamination problem which just would not go away within the lifetimes of humans. How could this be evaluated in a cost-benefit analysis?

The question that we should be asking would seem to be "Should we proceed at all with the development of applications using plutonium when problems raised by the toxicity of plutonium and problems of nuclear theft are apparently far from being solved and may remain so for a great number of years?"

BASIC REQUIREMENTS FOR STANDARD SETTING ON THE CONTROL OF TRANSURANIUM ELEMENTS IN THE ENVIRONMENT

by

R. A. Karam
School of Nuclear Engineering
Georgia Institute of Technology
Atlanta, Georgia 30332

Like all standards, environmental standards for the protection of the general environment from transuranium elements require: (1) that society define the cost (risk) it is willing to pay for the benefits it derives from the use of plutonium, and (2) that the technical know-how is at hand to assure that that cost is not exceeded.

Cost (Risk) - Benefit

The cost (risk) society is willing to pay for a given benefit is by no means easy to establish. It involves, among other things, putting a monetary value on human lives and on ecological changes which may not be reversible. In many cases the detriments to people and to the environment resulting from all the activities involved in producing the benefit are not known. This is especially true of effects on future generations. The identification and quantification of costs (risks) in the broad societal sense and the synthesis of these costs into decision making machinery should be a continuing goal of responsible society.

The term cost-benefit analysis correctly implies that there is a price that society pays for every benefit it derives from its industrial activities. The price may not be readily equated to a lump sum when one considers the price associated with the death of Lake Erie, the hazards of vinyl chloride,

such benefits as the automobile and the risk associated with events such as lightning, falls, drowning, firearms, and natural diseases. The price varies between 10^{-2} and 10^{-6}. It should be noted that although the probability of death per person due to natural causes is 10^{-2}, this value may be largely due to people in the old age category where death is a natural end. Death at birth when susceptibility to disease is high may also be a contributing factor.

Table I

U. S. Accident Death Statistics - 1967, 1968

Accident	Total Deaths		Probability of Death per Person per Year	
	1967	1968	1967	1968
Motor Vehicle	53,100	55,200	2.7×10^{-4}	2.8×10^{-4}
Falls	19,800	19,900	1.0×10^{-4}	1.0×10^{-4}
Fires, burns	7,700	7,500	3.9×10^{-5}	3.8×10^{-5}
Drowning	6,800	7,400	3.4×10^{-5}	3.7×10^{-5}
Firearms	2,800	2,600	1.4×10^{-5}	1.3×10^{-5}
Poisoning	2,400	2,400	1.2×10^{-5}	1.2×10^{-5}
Cataclysm	155	129	8×10^{-7}	6×10^{-7}
Lightning	110	162	6×10^{-7}	8×10^{-7}
Natural diseases				1×10^{-2}

The probability of death due to motor vehicle accidents is not the total price that society pays for the benefit of the automobile. Motor vehicle air pollution contributes directly to an increase in the probability of death and this increase may be currently attributed unjustly to natural causes.

Regardless of the precise cost (risk) society pays for the benefit of the automobile, its magnitude is at least a probability of 10^{-4} death per person per year. Should this value of cost (risk) be adopted as a standard

for the control of transuranium elements? Would society accept a death probability of at least 10^{-4} per person per year for the benefit of using plutonium as a major source of energy for the generation of electricity?

According to reference (1), Otway and Erdmann (ref. (2)) point out that when the probability of death risk approaches 10^{-3} per person per year society takes immediate action to reduce the hazard. This level of fatality risk is not acceptable to anyone. At a risk of 10^{-4} per person per year, people are less inclined to concerted action but are willing to spend money to control the hazard. At a level of 10^{-5} per person per year society is still concerned and may accept a certain amount of inconvenience to avoid such risks. For example, rules are adopted by community swimming pools against anyone swimming alone and by himself. At a level of 10^{-6} per person per year, the risk is not of great concern to the average person.

Suggested Standard for Cost (Risk)

If the assessment of Otway and Erdmann of what society accepts in terms of risk is correct, then it is reasonable to propose a draft of a standard on the control of transuranium elements defining the cost (risk) society is willing to pay for the benefit it derives from the use of plutonium, such as:

1. For occupational exposure, the cost (risk) society pays for the use of transuranium elements shall be a probability of death not to exceed 10^{-4} per person per year.
2. For the general public, the cost (risk) society pays for the use of transuranium elements shall be a probability of death not to exceed 10^{-6} per person per year.

The occupational cost (risk) recommended here is comparable to occupational risks from other industrial activities (see reference (1), pp. 6-18).

The general public cost (risk) is somewhat arbitrary and requires full public scrutiny.

Technical Know-how Needed

Once the cost (risk) is defined, the technical know-how needed to assure that the cost (risk) is not exceeded, must be supplied through research and development. The goals of the research should be providing answers to the following problems:

1. Identification in man of the critical organ which is most sensitive to transuranium radiations (lungs, lymph nodes, bones, liver, etc.)
2. Determination as closely as possible of the relationship between dose and the probability of death per person per year. Here the "hot particle" theory must be scrutinized by experiments. Until and unless experiments discount the "hot particle" theory, it would be foolhardy to ignore it!
3. Identification of the environmental pathways of the transuranium elements released during normal and/or during off-normal operations of nuclear power plants and the relationship of the release to man.
4. Realistic projection of the accumulation of the released transuranium elements and an assessment of their impact on the environment as a function of time.

Agencies charged with the development of nuclear power could also be charged with supplying the technical know-how needed to assure that the cost (risk) of such technology does not exceed adopted standards.

References

1. "The Safety of Nuclear Power Reactors (Light Water-Cooled) and Related Facilities," U. S. Atomic Energy Report WASH-1250 (1973).

2. H. J. Otway and R. C. Erdmann, "Reactor Siting from the Risk Viewpoint," Nuclear Engineering and Design, 13, 365-376 (1970).

Karl H. Puechl
Nuclear Consultant
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Atlanta, Georgia 30328
Telephone 404/252-8821

December 10, 1974

Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D.C. 20460

Subject: Plutonium and the Transuranium Elements

Gentlemen:

In your review of the subject environmental standards, I wish to alert you to the fact that for reactor use, the plutonium is significantly diluted with uranium in almost all processing operations. For example, mixed oxide fuel for light water reactors is expected to contain less than 5% plutonium. As a consequence of this dilution and the chemical stability of the oxide, generalized models of long-term exposure pathways relative to pure plutonium and/or soluble compounds can be misleading. I urge that particular attention be paid to the specific material involved and its associated chemistry and radiological behavior.

I suspect that technical information relative to this subject (including material compositions, particle sizes, etc.) will be further developed during the anticipated AEC hearings on the Generic Environmental Statement on Mixed Oxide Fuel, consequently, this comment is not elaborated upon herein.

Very truly yours,

Karl H. Puechl

Karl H. Puechl

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

DEC 19 1974

Dr. Richard Perkins
Battelle Northwest Laboratories
Richland, Washington 99352

Dear Dr. Perkins:

At the EPA hearings on plutonium on December 10 and 11 in Washington, a graph (enclosed) attributed to you was introduced by Dr. Wrenn. The lack of units on the ordinate axis was questioned by one of the panel members and, in my capacity as chairman of the technical hearing panel, I am writing to you to attempt to resolve this difficulty. If you can supply any additional information on this graph, or wish to supplement the information presented, your contribution would be most welcome. You might wish to reply through Dr. Liverman, Director of the Division of Biomedical and Environmental Research, who coordinated the AEC presentations.

Sincerely yours,

W. A. Mills

William A. Mills, Ph.D.
Director
Criteria & Standards Division (AW-560)

Enclosure

cc: Dr. J. L. Liverman, AEC
Dr. McD. E. Wrenn, DBER

Battelle
Pacific Northwest Laboratories
Battelle Boulevard
Richland, Washington 99352
Telephone (509)
Telex 32-6346

December 18, 1974

Dr. William Mills
Criteria and Standards Division
AW_560
Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Dr. Mills:

Enclosed is a copy of a section of our Annual Report for 1972, BNWL-1751 PT2 UC-48, which includes the information used in a viewgraph by Dr. M. E. Wrenn in the hearing last week. It was called to my attention by both Herb Parker and Ed Wrenn that the absolute plutonium-238 and plutonium-239 concentrations which are included in this report, but were not on the viewgraph, would be important for inclusion in the proceedings of the hearing.

I am also sending a copy of this material to Dr. Edward P. Radford who I am told showed particular interest in this data.

Sincerely,

Richard W. Perkins

Richard W. Perkins, Manager
Radiological Chemistry Section
RADIOLOGICAL SCIENCES DEPARTMENT

RWP/liz

Enclosure

cc: Dr. Edward P. Radford
Dr. M. E. Wrenn
Dr. H. M. Parker

Plutonium Concentrations in Surface Air at Richland, Washington

C. W. Thomas

The plutonium isotopes released by atmospheric nuclear testing are a combination of unreacted plutonium in the device and isotopes of plutonium formed by neutron capture in plutonium and any ^{238}U in the device. The most abundant isotope of plutonium is ^{239}Pu, with lesser amounts of ^{238}Pu, ^{240}Pu, ^{241}Pu and ^{242}Pu. The ^{238}Pu is produced by either the reaction of fast neutrons with ^{239}Pu or by the decay of ^{238}Np or ^{242}Cm. Plutonium-238 to ^{239}Pu ratios vary from detonation to detonation depending upon the burst conditions and the material used.

In April 1964, a transit navigational satellite was launched from Vandenberg containing a System for Nuclear Auxiliary Power (SNAP-9A) generator. The satellite failed to achieve a stable orbit and subsequently burned upon re-entry, releasing approximately 17 kCi of ^{238}Pu ($\sim$1 kg) at 46 km over the Indian Ocean. This ^{238}Pu almost tripled the global atmospheric ^{238}Pu inventory. Plutonium from this device was first detected in a sample collected by balloon at 33 km over Australia in August 1964. Since that time, it has been detected in stratospheric and tropospheric air from both hemispheres.

The atmospheric concentrations of ^{238}Pu and ^{239}Pu were measured in surface air samples collected at Richland, Washington from 1963 to 1972 as a part of a program to define the rates of long term stratospheric processes in the northern hemisphere as well as the rates of interhemispheric mixing. The seasonal variations in the concentrations of ^{238}Pu and ^{239}Pu in surface air at Richland were similar to those of other nuclear-weapons-produced radionuclides of stratospheric origin; maximum concentrations occurred in the late spring and minimums occurred in the winter. The rate of decrease in the ^{239}Pu concentration from 1963 through 1967 corresponded to a stratospheric half-residence time of 10 to 11 months, which is the same as the half-residence times calculated from measurements of other radionuclides of stratospheric origin. The ^{239}Pu concentrations remained fairly constant from 1967 to 1972, primarily because of yearly injections of plutonium by thermonuclear tests conducted by the Chinese at Lop Nor (35° N); the contribution from the French tests in the South Pacific (23° S) may also have been significant. Fission product yields indicate that the Chinese used ^{235}U as the fissile material in their high-yield tests of 1967 through 1970. However, large amounts of ^{237}U, ^{239}Np, and ^{239}Pu were produced, indicating that ^{238}U was used as a component part of the devices.

From 1962 through 1965 both the ^{238}Pu and the ^{239}Pu in surface air at Richland came primarily from the 1961-62 U.S.-Russian test series. The $^{238}Pu/^{239}Pu$ ratio averaged 0.015 in 1964. The ratio increased slightly in 1965, and by the spring of 1966 it had increased to 0.042,

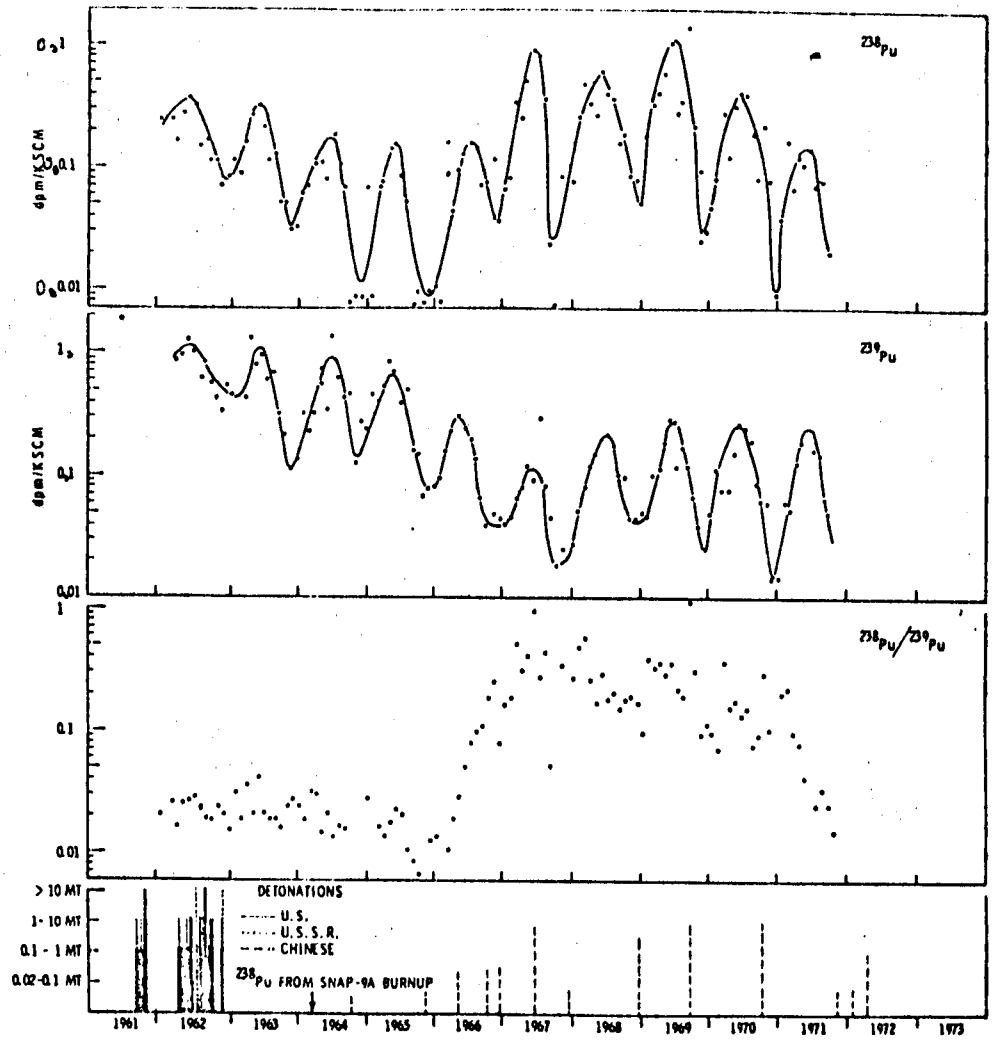
indicating that ^{238}Pu from the SNAP-9A burnup was present. The amount of SNAP-9A ^{238}Pu present was calculated from the ^{238}Pu concentrations and the $^{238}Pu/^{239}Pu$ ratios, assuming that the ratio in debris from nuclear weapons tests was 0.015. These calculations indicate that the ^{238}Pu in Richland air from 1967 to 1971 came primarily from SNAP-9A. From 1967 to 1969 the concentrations of SNAP-9A plutonium at Richland remained fairly constant, indicating that the ^{238}Pu was being transferred across the equator into the northern hemisphere at a rate comparable to the rate at which ^{238}Pu was being deposited on the earth's surface.

The ^{238}Pu concentrations showed seasonal variations typical of radionuclides of stratospheric origin, so the transfer was probably taking place primarily in the stratosphere. Concentrations of SNAP-9A plutonium at Richland have decreased rapidly since that time. These results indicate that the stratospheric debris injected into the high stratosphere of one hemisphere will produce high concentrations of the debris in ground level air in the other hemisphere in the spring 1 to 2 years later. These concentrations will then remain constant for about three years before beginning to decrease.

The Average Yearly Ratios of ^{239}Pu, ^{138}Pu, and ^{90}Sr in Surface Air at Richland, Washington

dpm/KSCM

	^{90}Sr	^{239}Pu	^{238}Pu	$^{239}Pu/^{90}Sr$	$^{238}Pu/^{239}Pu$
1962	90.6	1.15 ± 0.169	.0264	0.0127	.023
1963	119	1.20 ± 0.02	.0229	0.010 ± 0.0043	.019
1964	80.5	1.02 ± 0.015	.0158	0.0127 ± 0.012	.015 ± 0.0015
1965	40.0	0.704 ± 0.012	.0108	0.0176 ± 0.0072	.015
1966	22.9	0.325 ± 0.007	.0203	0.0142 ± 0.0055	.062
1967	7.08	0.125 ± 0.002	.0467	0.018 ± 0.0049	.374
1968	10.3	0.230 ± 0.003	.0564	0.022 ± 0.0068	.245
1969	11.4	0.310 ± 0.004	.111	0.027 ± 0.0076	.358
1970		0.284 ± 0.004	.0410		.144
1971		0.275 ± 0.003	.0162		.059



Neg 726084-2

^{238}Pu and ^{239}Pu Concentration in Surface Air at Richland, Washington

Atmospheric Radionuclide Concentrations at Richland, Washington and at Point Barrow, Alaska

C. W. Thomas and J. A. Young

The radionuclide concentrations in surface air are being measured continuously at Richland, Washington (45° N) and at Point Barrow, Alaska (71° N) by filtering large volumes of air through membrane filters and then analyzing the filters by gamma-ray spectrometry. A few of the radionuclides must be separated chemically

UNITED STATES
ATOMIC ENERGY COMMISSION
WASHINGTON, D.C. 20545

January 15, 1975

Dr. William A. Mills, Director
Criteria and Standards Division
(AW-560)
Environmental Protection Agency
401 M. St., S.W.
Washington, D.C. 20460

AEC SUBMISSIONS FOR THE HEARING RECORD

Dear Dr. Mills:

The following documents are transmitted hereby for inclusion in the record of the EPA public hearing concerning plutonium standards. These materials will supplement the AEC-Operations presentations in Washington, D.C. on December 10-11, 1974.

(1) A figure concerning Plutonium Concentration in Surface Air at Richland, Washington and a table concerning Preliminary Estimates of Local Sources of Plutonium in the Environment. These materials supplement the testimony of Dr. McDonald E. Wrenn on December 10, 1974. The hearing board at that time expressed interest in the numerical values of the figure, which was presented as Figure 3 of Dr. Wrenn's testimony. These values have been added and are expressed in disintegrations per minute per thousand standard cubic meters of air (dpm/KSCM). This work is referenced in BNWL-1751, PT2- UC48, page 2, 1974. The enclosed figure is a copy of the referenced page. The table is a correction of Table 4 of Dr. Wrenn's testimony. The major change is the deletion of the first three entries under Rocky Flats, which were typographical errors.

(2) A response to panel member, Dr. Melvin First's question at the hearing in Washington, D.C. concerning the effectiveness of cleanup activities. This document is in the form of a memorandum from Robert E. Yoder, Assistant Director for Facilities Safety, Division of Operational Safety to Gregory A. Thomas, Office of the General Counsel, both of the AEC.

(3) A response to a question of panel member Dr. Radford to Mr. Edward Hardy, who testified on behalf of the AEC, concerning the relationship of plutonium in soils in the vicinity of AEC's Rocky Flats plant to existing radiation standards. Additionally, this material presents a response

to Dr. Radford's comment, addressed to AEC spokesmen Drs. W. J. Bair and C. Richmond, concerning AEC's performance in monitoring the environment.

Additional materials to supplement the record of the second hearing in Denver, Colorado will be forthcoming under separate cover.

Sincerely,

Gregory A. Thomas, Attorney
Office of the General Counsel

Enclosures:
As stated

UNITED STATES
ATOMIC ENERGY COMMISSION
WASHINGTON, D.C. 20545

DEC 23 1974

Gregory A. Thomas
Office of the General Counsel

SUPPLEMENTARY INFORMATION FOR THE RECORD OF THE EPA HEARINGS CONCERNING PLUTONIUM ENVIRONMENTAL STANDARDS, DECEMBER 10-11, 1974

In response to Dr. Melvin First's question in which he asked for an indication of the improvement of the effectiveness in cleanup activities as a result of experiences at Palomares and Thule, the following information is presented.

Because the United States has not had another incident such as those which occurred at Palomares, Spain, and Thule, Greenland, the answer can only reflect anticipated results. The following improvements have been incorporated into our accident response posture.

1. Instrumentation to evaluate the extent and degree of contamination was developed following the Palomares incident and prototypes were used at Thule. The difficulties experienced in the arctic have led to further refinements so that now portable instrumentation is available to monitor specifically for plutonium at the 0.1 $\mu Ci/m^2$ level. This instrumentation is capable of operating under any climatic condition and in any terrain. These devices are available in sufficient numbers and locations to allow good initial surveys basic to establishing control lines to prevent the spread of contamination. This equipment also is sufficiently sensitive to permit a field analysis of bioassay specimens which can indicate a significant internal exposure to plutonium contamination (1/2 to 1 lung burden) so that cleanup personnel or the affected population can receive prompt medical attention.

2. Techniques to fix contamination, prior to removal, to prevent its translocation by wind and water are under development. These techniques have been used in a few instances in protected environments (indoors) and are being evaluated for other environments.

3. Aerial mapping, including a capability to remotely determine radioactivity surface contamination levels at the 0.3 $\mu Ci/m^2$ level, and downwind air sampling to allow an assessment of windborne debris movement are now available. This capability complements the improvements noted in 1., above, so that the entire accident area can be more accurately defined.

The methods for removal or fixation (i.e., rendering contamination unavailable to man) of radioactivity still require excavation, watering, and plowing. The improvements in executing these functions are derived from our improved ability to delineate the degree of dispersed radioactive materials and our preventive techniques to temporarily fix, "tie down," contamination. Where previous remedial operations have removed between 50 and 90 percent of the radioactivity, future operations may allow removal of 70-95 percent of the dispersed material.

Robert E. Yoder
Assistant Director for
Facilities Safety
Division of Operational Safety

Question by Dr. Radford to Mr. Edward Hardy, AEC-HASL, concerning the relationship of plutonium in soils in the vicinity of the AEC's Rocky Flats plant to existing radiation standards. Mr. Hardy disqualified himself from responding to the question and it was not answered.

Answer: Extensive soil sampling conducted by the RF plant and the AEC's Health and Safety Laboratory has permitted the definition of the distribution and levels of plutonium in soils in the vicinity of the plant. The concentrations of Pu observed in soil, which vary with location and depth, cannot be reliability translated by a calculation into an expected dose to man. The difficulty of translating observed soil concentrations into expected dose is probably the major reason why no national or international concensus standards exist relative to Pu levels in soil. Accordingly, the usual method of evaluating the significance of such contamination is to conduct site specific air sampling programs over periods of time sufficiently long for comparing concentrations of Pu in air with established consensus standards for airborne activity. Concentrations of Pu observed in air can then be compared with established concensus standards for airborne activity. The radioactivity concentration guides (RCG's) legally established for annual average levels of plutonium in air by AEC are based on recommendations of the National Council on Radiation Protection and Measurement and the International Commission on Radiological Protection. The RCG of 3×10^{-13} μCi/ml for plutonium oxide, the chemical form of plutonium encountered at the plant, is the standard that would normally be used in assessing the significance of plutonium air concentrations around the plant. However, the more restrictive RCG of

2×10^{-14} μCi/ml for soluble forms of plutonium was used by the Rocky Flats plant as the reference standard since it results in the most conservative assessment of potential public exposure.

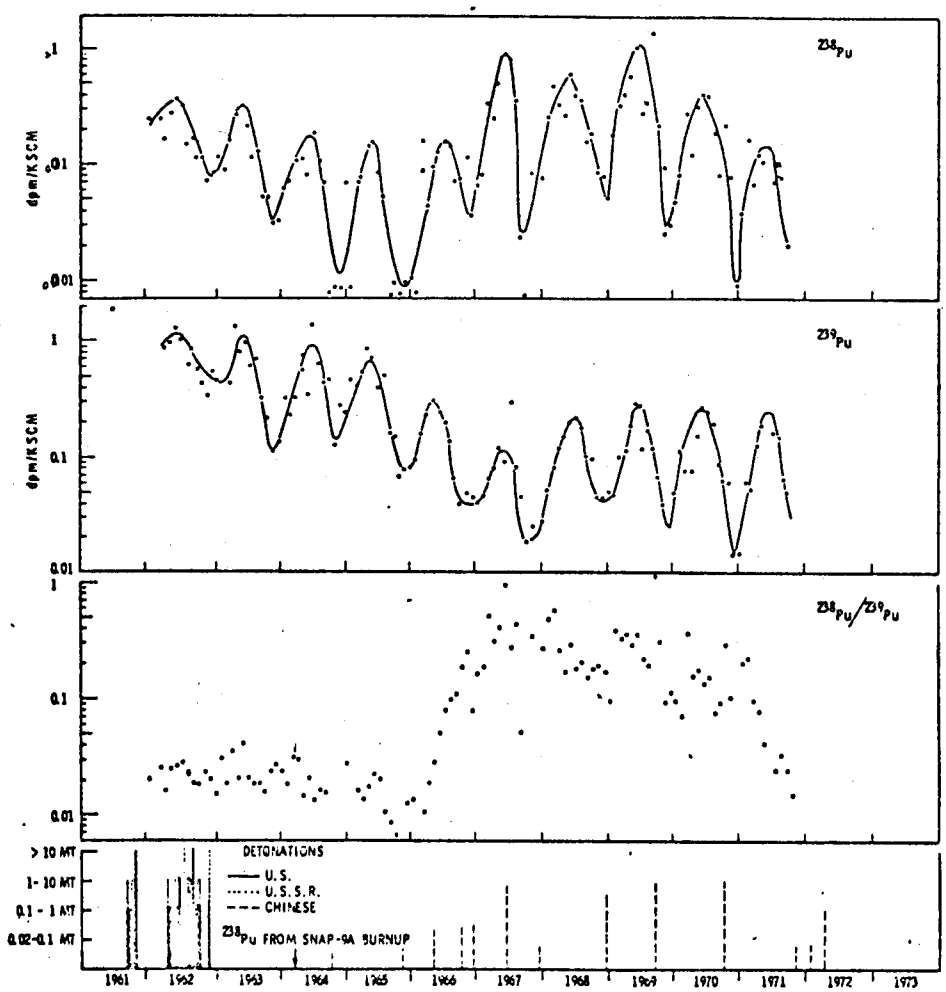
In the vicinity of the Rocky Flats plant, the public health significance of the soil contamination (estimated to be about 14 curies of Pu distributed in surface soils, mostly onsite) has been evaluated by operating air sampling stations located around the plant and analyzing the samples for radioactivity. The 1973 annual average concentration of plutonium in air among the 12 stations located between 2 and 4 miles distant averaged less than 0.3 percent of the reference RCG. The maximum annual average concentration observed at a single station in 1973 was about 0.7 percent of the reference RCG.

It should be noted that air concentrations of plutonium as indicated from sampling in the near vicinity of the plant would reflect contributions of plutonium from worldwide fallout, plant stack emissions and any resuspension of plutonium present in surface soils. Direct contribution of plutonium in air in the RF plant area from worldwide fallout is estimated to be $1\text{-}2 \times 10^{-17}$ μCi/ml of air, about 0.1 percent of the reference RCG.

Comment: Dr. Radford commented to Drs. W. J. Bair and C. Richmond that whereas the AEC's performance has been commendable in regard to studies and research conducted towards protecting employees at AEC facilities, the AEC's performance in monitoring the environment has not been good. AEC was not given an opportunity during the hearing to respond to Dr. Radford's comment.

Response: Environmental radioactivity monitoring programs have been conducted at each AEC site since at least one year prior to the initiation of nuclear operations at the site. Beginning in 1959, each AEC site has been required to issue a public report, at least annually, summarizing the results of the site environmental monitoring program. The monitoring programs and reports have always placed emphasis on radioactivity levels in air, water and foods to which members of the public might have been exposed. The data have indicated that members of the public have not been exposed to radioactivity in excess of a small fraction of the applicable NCRP radiation standards. In recent years, exposures to the public near AEC facilities have generally been well below one percent of the applicable radiation standards. The 1973 site environmental monitoring reports not only include summarizations of monitoring results, but also the total annual curie releases of specific radionuclides and estimates of individual and population doses due to site operations.

534



Neg 726084-2

^{238}Pu and ^{239}Pu Concentration in Surface Air at Richland, Washington

Atmospheric Radionuclide Concentrations at Richland, Washington and at Point Barrow, Alaska

C. W. Thomas and J. A. Young

The radionuclide concentrations in surface air are being measured continuously at Richland, Washington (45° N) and at Point Barrow, Alaska (71° N) by filtering large volumes of air through membrane filters and then analyzing the filters by gamma-ray spectrometry. A few of the radionuclides must be separated chemically

Table 4

Preliminary Estimates of Local Sources of Plutonium in the Environment (Quantities > 0.1 Ci)

Facility	Quantity	Location
NV NTS + Pacific	~ 1,000 Ci	Pu-239 to Onsite Soil
Thule	~ 25 Ci	Pu-239 in Marine Sediments and Surface Soils
Rocky Flats	0.04 Ci	Pu-239 to Atmosphere
	0.1 Ci	U + Pu-239 to Offsite Streams
	4.0 Ci	Pu-239 to Offsite Soil
	10.0 Ci	Pu-239 to Onsite Soil
Mound Laboratory	0.4 Ci	Pu-238 to Atmosphere
	0.5 Ci	Pu-238 to River - Routine Releases
	5.0 Ci	Pu-238 to Canals and Ditches
	0.5 Ci	Pu-238 to Soil
Savannah River	2.9 Ci	Pu-239 to Atmosphere
	0.6 Ci	Pu-239 to Atmosphere
	1.5 Ci	Pu-239 to Onsite Soil
	0.1 Ci	Pu-239 to Onsite Soil
LASL	1.3 Ci	Pu-239 to Atmosphere
	0.3 Ci	Pu-239 to Soil

536

UNITED STATES
ATOMIC ENERGY COMMISSION
WASHINGTON, D.C. 20545

JAN 10 1975

William A. Mills, Ph.D., Director
Criteria and Standards Division (AW-560)
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Dr. Mills:

In addition to the formal statement which the AEC presented at the EPA Hearing on Transuranium Elements on December 10, 1974, we would like to submit the following for the record.

The National Conference of Radiation Control Program Directors is an organization of the Program Directors of the 50 States. This organization includes the representatives of the 25 Agreement States (those States which have entered into agreements with the AEC pursuant to Section 274 of the Atomic Energy Act of 1954, As Amended). These agreements provide for the transfer of certain regulatory authority over byproduct material, source material and small quantities of special nuclear material to States. Both of these groups have requested that we indicate their interest in the establishment of uniform soil contamination limits for transuranium elements.

Sincerely,

G. Wayne Kerr

G. Wayne Kerr, Chief
Agreements and Exports Branch
Directorate of Licensing

COLORADO DEPARTMENT OF HEALTH

4210 EAST 11TH AVENUE • DENVER, COLORADO 80220 • PHONE 388-6111

Edward G. Dreyfus, M.D., M.P.H., Executive Director

December 13, 1974

William A. Mills, Ph. D., Director
Criteria and Standards Division (AW-56)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

In reference to the announcement of the public hearing on the information required for standards development for Plutonium and the other transuranic elements, your letter of November 26 and a phone call from Carl Miller, (EPA), on December 12, the Department wishes to make a presentation at the hearing continuance in Denver.

We understand that the Denver session is scheduled for January 10, 1975 beginning at 9 a.m. at the Post Office Building Auditorium. Our presentation will last approximately 30 minutes.

Your agency's attention to our request is appreciated.

Sincerely,

Robert D. Siek
Assistant Director of Public Health
Environmental Programs

RDS/ljw
cc: A. J. Hazle, Occupational and Radiological Health, CDH
Paul Smith, Environmental Protection Agency, Region VIII

NATIONAL CENTER FOR ATMOSPHERIC RESEARCH
P. O. Box 1470 • Boulder, Colorado 80302
Telephone: (303) 494-5151 • TWX: 910-940-3245 • Telex: 45 694

16 December 1974

Dr. William Mills, Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D. C. 20460

Dear Dr. Mills:

I was unable to participate at the recent EPA public hearings on plutonium standards in Washington, D. C., on December 10 and 11, 1974. However I am advised by Dr. Gordon Burley of your staff that another public hearing will take place in Denver, Colorado, on January 10, 1975. I wish to present written and oral testimony at these Denver hearings on the subject of plutonium standards applicable to the long term inhalation exposure of the general public.

My oral and written testimony will include a brief critique of the basis of current plutonium standards, followed by a discussion of the published evidence on the nature and effects of alpha interactions with cells and the trends in the relative distribution of such effects at low dose rates. My presentation also will include a brief discussion of the possible mechanisms of tumor induction for "hot" plutonium oxide particles. It will be shown that even the incidence of tumors associated with "hot" particles may be explained by the irradiation of large numbers of cells which have been subjected to only a limited number of alpha interactions. On this basis a significant tumor risk can be attributed to very small burdens of alpha emitting particles of low activity per particle. Similarly, unacceptable risks can be attributed to as little as tens of picocuries of low activity particles. These conclusions imply serious health risks for tobacco radioactivity, for fallout plutonium and for other insoluble alpha emitter contaminants and, if confirmed, will call for a drastic downward revision of plutonium standards and other alpha emitter standards.

In addition I plan to discuss plutonium standards applicable to surface soils and urban dusts, including discussion of a new approach to the prediction of organ burdens that will result from long-term inhalation exposure to such sources. Based on these and the above considerations I shall propose interim standards applicable for public exposure to insoluble alpha emitting particles in surface soils and urban dusts.

The National Center for Atmospheric Research is Operated by the University Corporation for Atmospheric Research under sponsorship of the National Science Foundation.

For my oral presentation I would appreciate it if you could allocate at least 30 minutes, and preferably 45 minutes. Thank you.

Sincerely yours,

Edward A. Martell

Edward A. Martell

UNIVERSITY OF MINNESOTA
TWIN CITIES

School of Public Affairs
Social Sciences Building
Minneapolis, Minnesota 55455

18 December 1974

Dr. William Mills
Director, Criteria and Standards
Division
Office of Radiation Protection
Environmental Protection Agency
Washington, D.C. 20460

RE: Public Hearing/Plutonium Contamination Limits

Dear Dr. Mills:

I ask that I be given the opportunity to testify at your Public Hearing on Plutonium Contamination Limits (scheduled for Denver, Colorado on January 10, 1974). My testimony would be approximately 30 to 40 minutes in duration. If it would not inconvenience you, it might be advisable for my testimony to be scheduled no earlier than 10 A.M., since I may have to take an early morning flight, Minneapolis to Denver, on the 14th.

Thank you.

Yours truly,

Donald P. Geesaman
Associate Professor

DPG/lam

UNIVERSITY OF COLORADO
MEDICAL CENTER
4200 EAST NINTH AVENUE
DENVER, COLORADO 80220

SCHOOL OF MEDICINE
DEPARTMENT OF PREVENTIVE MEDICINE
AND COMPREHENSIVE HEALTH CARE

DIVISION OF HEALTH ADMINISTRATION
JOHN EDWARD KRALEWSKI, Ph.D.
DIRECTOR

December 20, 1974

Director, Criteria and Standards
Division (AW-560)
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

I request about 15 minutes for a statement at the plutonium standards hearings in Denver on January 10th, 1975. The information requested follows:

Name: John C. Cobb, M.D., M.P.H.
Professor of Preventive Medicine
University of Colorado Medical School
Denver, Colorado 80220

Affiliations: Member, Governor's Scientific Advisory Council
Member, Governor-elect's Task Force on Rocky Flats
Member, Denver Medical Society, Public Health Commission
Chairman, Rocky Flats Action Committee of the American Friends Service Comm.

Topic: "Value Decision in the Face of Scientific Uncertainty."

Summary: I would briefly summarize critical evaluations by various members of the Medical School faculty of the report by Arthur R. Tamplin and Thomas B. Cochran, entitled, "Radiation Standards for Hot Particles" dated February 14, 1974, which was prepared for the Natural Resources Defense Council in support of their petition to the EPA and AEC. (I presume it is not necessary for me to summarize the content of that report, but would be glad to do so if you desire, and want to give me more time to do it.)

I would then point out the implications of this report and other related research findings to the control of hot insoluble plutonium particles emanating from The Rocky Flats Plutonium Project.

Finally, I would review the possible dangers to populations living now and in the future in the area surrounding the Rocky Flats Project, and make a recommendation of how the scientific data, with its recognized degree of uncertainty, should be utilized in making a decision regarding standards for plutonium in the environment.

I would appreciate hearing from you regarding how much, if any, of the above you would like me to present orally; and in particular whether you want me to summarize the content of the report by Tamplin and Cochran, which I presume you already have in your files. I will, in any case, prepare a written statement for the record.

Yours sincerely,

John C. Cobb

John C. Cobb, M.D., M.P.H.
Professor

JCC/ma
cc: Paul Smith

AMERICAN FRIENDS SERVICE COMMITTEE

Staff:
KAY JOHNSON, *Administrative Secretary*
JUDY DANIELSON, *Peace Education*
PAM SOLO, *Peace Education*
BEN REYES, *Justice Program*

Colorado Area Executive Committee:
ROBERT HUBBARD, *Chairman*
MIDORI ABE
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JACK JOURGENSEN
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ROSINA OLSON
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MARCIA WARNER
PAUL WEHR
GLENN WILLIAMS

COLORADO AREA OFFICE
2801 E. COLFAX AVE., #304
DENVER, COLORADO 80206
(303) 388-5896

December 22, 1974

Director, Criteria and Standards Division
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

I am writing to request time to give testimony at the Plutonium Standards Hearings to be held in Denver on January 10, 1975.

The testimony of the American Friends Service Committee will require five to eight minutes only. Our testimony will be concerned with the social and moral impact of plutonium.

We look forward to your response in this matter. Thank you.

Sincerely,

Pam Solo

Pam Solo
Co-Director
Peace Education

544

AMERICAN FRIENDS SERVICE COMMITTEE

Staff:
KAY JOHNSON, *Administrative Secretary*
JUDY DANIELSON, *Peace Education*
PAM SOLO, *Peace Education*
BEN REYES, *Justice Program*

Colorado Area Executive Committee:
ROBERT HUBBARD, *Chairman*
MIDORI ABE
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MURIEL ASHMORE
LEE SANNE BUCHANAN
GINNY COWLES
JIM GRAHAM
LEE HAMBY
JACK JOURGENSEN
CURTIS KING
JERRY KRENZ
HELEN LUCERO
MARIAGNES MEDRUD
KAREN MOORE
LARRY NAVES
ROSINA OLSON
ROY SMITH
TONY UMILE
MARCIA WARNER
PAUL WEHR
GLENN WILLIAMS

COLORADO AREA OFFICE
2801 E. COLFAX AVE., #304
DENVER, COLORADO 80206
(303) 388-5896

January 22, 1975

Director, Criteria and Standards Division
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

We were dismayed and shocked by the defensive, hostile and patronizing attitude of several members of the hearing panel at the Environmental Protection Agency hearings on plutonium standards in Denver on January 10. We commend the opening remarks by Dr. W. D. Rowe, Deputy Assistant Administrator for Radiation Programs. He encouraged those giving testimony to address themselves to the value judgments involved in assessing risks, costs, and benefits which, he said, " . . . must be made by society as a whole and not by the technical community alone . . ." But citizens who expressed concern about the futility of making more atomic bombs and the dangers of plutonium pollution coming from the Atomic Energy Commission projects involved in manufacturing bombs, such as Rocky Flats plutonium processing plant near Denver, were treated as if their opinions were of second class importance compared with the technical testimony of others.

While the panel members were selected by the EPA, presumably to further its public charge of protecting our environment, some of them responded as if their charge were rather to protect the Atomic Weapons Establishment.

We have no objections to such a hearing panel asking questions and arguing about points raised by the testimony, provided that the purpose is clarification of the testimony and that the witness has a chance to respond. This is necessary to bring out the truth. But when EPA hearing panelists make derogatory comments, not even phrased as questions, and address themselves directly to the public, evidently attempting to discredit the testimony of the witness, they reveal their true colors.

Even worse, when they argue ad hominem attempting to discredit a witness by such questions as "Doctor, do you consider yourself a scientist?" this suggests that they lack substantial defense against the testimony itself and therefore have to resort to the tactics of trying to discredit the witness through sarcasm. While they may be technically competent, each in his own scientific field, there were those on the hearing panel who displayed a lack of breadth and depth

of human understanding, indicating an unfortunate limited point of view. They evidently were most reluctant to hear discussion of risk-cost-benefit value judgments which necessarily involve humanistic or moral considerations. An example is the question of whether, for the benefit of a few people who are living today, we should run even a slight risk of endangering the health and life of every living thing for thousands of generations. They branded the consideration of such questions as "emotionalism" and proceeded immediately to readjust their blinders in order to focus only on the technical questions. This, in spite of the specific request by Dr. W. D. Rowe, in his opening remarks on behalf of EPA, requesting comment on this type of value judgment. Since the hearings were to be "evidentiary" in nature, persons giving testimony before the panel hardly expected to be cross examined by panelists--and for them to be so treated was unfortunate and unfair.

Perhaps the panelists guilty of such conduct forget that in the history of mankind, attempts to resolve moral problems of humanity simply by technological means have generally resulted in the creation of worse moral problems. The testimony given before the panel rightly addressed itself not only to protection of the immediate environment of the Denver metropolitan area, but to the global environment as well. The risks of local radiation pollution intersect with those represented by the nuclear weapons produced at Rocky Flats.

All nations will have to realize soon that this multi-national atomic arms race provides more danger to humans and their environment than security. The Pentagon has a name for it, "Mutual Assured Destruction," which goes by the appropriate acronym "MAD."

Sincerely,

Paul Wehr

Paul Wehr
Co-chairman, Peace Education
Committee

PW:kj

546

CITY OF BOULDER

OFFICE OF THE CITY MANAGER

December 24, 1974

Dr. William A. Mills
Director for Criteria and Standards Division
AW 560
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

The City of Boulder would appreciate an opportunity to make a ten-minute presentation at the public hearing regarding the environmental impact of plutonium and transuranium elements, being held in Denver on January 10, 1975. Our presentation will present some of the concerns relevant to the City of Boulder, particularly those resulting from a brief staff study of the potential impact of the Atomic Energy Commission's Rocky Flats Plant.

Our City Council has not yet had the opportunity to establish its position regarding the potential impact of plutonium but plans to address the questions involved prior to the public hearing. Until that time, it will not be possible to provide a complete outline of our concerns or an official statement of our position. However, if any additional information is needed prior to the public hearing I would be pleased to supply it.

Thank you for your consideration and for the opportunity to participate in the public hearing.

Sincerely,

Louis F. Dow

cc: John Green
Regional Administrator
U.S.E.P.A.

Ruth Correll
Boulder City Councilmember

LFD:jw

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JOHN C. SELNER, M.D.
ALLERGY AND PEDIATRIC ALLERGY

5800 E. EVANS — TEL. 756-3614
DENVER, COLORADO 80222

2020 WADSWORTH — 234-1067
LAKEWOOD, COLORADO 80215

December 30, 1974

Director, Criterion and Standards Division
(AW--560) Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

This note is to notify you of our desire to offer a statement at the hearings to be held at the Post Office building on Stout Street in Denver January 10 regarding plutonium standards. I would identify myself as John C. Selner, M.D., 5800 East Evans representing the Colorado Medical Society. Our testimony should take not more than ten minutes and be offered here in Denver. The topic of the presentation is regarding plutonium standards and their specific application to the Rocky Flats plant.

The Colorado Medical Society appreciates your allowing us this time. I am uncertain as to how I will know that this time is being made available to us, as I am not sure of your confirmation procedures. Perhaps we could attain this by inquiring with the local EPA office. Thank you very much for your attention to this matter.

Sincerely,

JOHN C. SELNER, M.D.

JCS:ks

CURE
CITIZENS UNITED FOR RESPONSIBLE ENERGY
500 E. 6th
DES MOINES, IA. 50309
PH. (515) 282-8191

December 31, 1974

Dr. William Mills
Director of Criteria and Standards Division (AW 560)
Office of Radiation Program
Environmental Protection Agency (EPA)
Washington, D.C. 20460

Dear sirs:

Citizens United for Responsible Energy most strenuously objects to the proposed policy of plutonium recycle. Our objections are well based because of the present insecure method of handling plutonium and uranium. The recent New York Times story indicating that plutonium is missing from 15 commercial reactors only points up the validity of our contentions. We must be a mad society to even contemplate adding to the plutonium risk when we obviously have not assured control of it now.

The use of plutonium recycle in the present reactors will only escalate the risks of nuclear power, risks that we feel are unbearable even now. The plutonium risk is not necessary and puts a grave cloud over the future of man's activity and genetic base. No way exists to allow this technology to proliferate without jeopardizing man's freedom, and literally putting us under military control.

Instead we feel our energy should be moving toward decentralized, less complex sources that do not require the extensive, expensive backup and safeguard systems that essentially make us captives of our own technology.

Enclosed is the essence of our thinking as expressed by three experts, J. Gustave Speth, Arthur Tamplin and Thomas Cochran, in their article, "Plutonium Recycle: The Fateful Step", printed in the Bulletin of the Atomic Scientists November, 1974. We ask that the entire article be entered as part of the record.

Copies to: Iowa Congressmen
Iowa Dept. Environ. Quality
25 Iowa citizen groups

Sincerely,

Jane Magers

Jane Magers, chairperson, CURE

THE bulletin OF THE ATOMIC SCIENTISTS

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Plutonium Recycle: The Fateful Step

Impending move to reprocess fuel would escalate the risks of nuclear power

I fear that when the history of this century is written, that the greatest debacle of our nation will be seen not to be our tragic involvement in Southeast Asia but our creation of vast armadas of plutonium, whose safe containment will represent a major precondition for human survival, not for a few decades or hundreds of years, but for thousands of years more than human civilization has so far existed.

James D. Watson
Nobel Laureate, Medicine

J. GUSTAVE SPETH, ARTHUR R. TAMPLIN and THOMAS B. COCHRAN

The Atomic Energy Commission, if unchecked, is about to sow the seeds of a national crisis. The Commission now proposes to authorize the nuclear power industry to proceed to use plutonium as fuel in commercial nuclear reactors around the country. The result of a decision approving this commercial use of plutonium will be the creation of a large civilian plutonium industry and a dramatic escalation in the risks posed by nuclear power.

This decision to launch what the AEC calls the plutonium economy is the conclusion of the AEC's recently released draft environmental impact statement for *plutonium recycle*: the recycling of plutonium as fuel in the present generation of light water reactors [1, 2]. The final version of the impact statement, which is expected to confirm the decision to authorize plutonium recycle, is due in a few months.

Plutonium is virtually unknown in nature; the entire present-day inventory is man-made, produced in nuclear reactors. Plutonium-239, the principal isotope of this element, has a half-life of 24,000 years, hence its radioactivity is undiminished within human time scales. It is perhaps the most toxic substance known. One millionth of a gram has been shown capable of producing cancer in animals [3]. Plutonium is also the material from which nuclear weapons are made. An amount the size of a softball is enough for a nuclear explosive capable of mass destruction. Scientists now widely recognize that the design and manufacture of a crude nuclear explosive is no longer a difficult task technically, the only real obstacle being the availability of the plutonium itself [4].

We believe that the commercialization of plutonium will place an intolerable strain on our society and its institutions. Our unrelenting nuclear technology has presented us with a possible new fuel which we are asked to accept because of its potential commercial value. But our technology has again outstripped our institutions, which are not prepared or suited to deal with plutonium. Those who have asked what changes in our institutions will be necessary to accommodate plutonium have come away from that enquiry profoundly concerned. And the AEC's environmental impact statement does not allay these concerns. It reinforces them.

The AEC concedes that the problems of plutonium toxicity and nuclear theft are far from solved and indicates that they may not be for some years. Yet it concludes, inexplicably, that we should proceed. Whether stemming from blind faith in the technology it has fostered or from callous promotion of the bureaucratic and industrial interests of the nuclear power complex, the AEC's proposal cannot be justified in light of what we know and, just as important, what we do not know.

The fuel now used in present-day reactors, the light water reactors, is uranium which has been enriched; the uranium-235 content is increased from 0.7 percent present in natural uranium to about 3 or 4 percent. Uranium-235 is a fissionable isotope of uranium, the remainder being non-fissile uranium-238. Unlike plutonium, uranium fuel is not extremely toxic, and it is not sufficiently rich in uranium-235 to be fashioned into nuclear weapons. The uranium

J. Gustave Speth (attorney), Arthur R. Tamplin (biophysicist) and Thomas B. Cochran (nuclear physicist) are on the staff of the Natural Resources Defense Council in Washington, D. C. Dr. Tamplin is on leave of absence from the Lawrence Livermore Laboratory of the University of California.

The current AEC radiation protection standards governing the amount of plutonium to which members of the public can be exposed are roughly 100,000 times too lax.

can be enriched to weapons grade material only with extremely sophisticated technology which is not available to the public, notably gaseous diffusion plants.

While present-day reactors are operating, however, they are also producing as a by-product moderate amounts of plutonium, principally plutonium-239. A typical large reactor produces about 200 to 250 kilograms of plutonium each year. Since this plutonium is easily fissioned, it can be used as reactor fuel. Plutonium recycle is the nuclear industry-AEC proposal to recover the plutonium produced in light water reactors, process it and recycle it as fuel back into these reactors.

Several critical steps are involved in recycling this plutonium. First, the used or spent fuel from the reactor must be shipped to a fuel reprocessing plant where the plutonium is recovered from the spent fuel, converted to oxide form and shipped to the next fuel cycle stages—the fuel fabricating and assembly plants. At a fuel fabricating plant the plutonium oxide will be mixed with uranium oxide into mixed oxide fuel. This mixed oxide fuel will be fabricated into fuel pellets, the pellets will be placed in fuel rods, and these rods will be collected into fuel assemblies. These assemblies will then be sent to the reactors for use, thus completing the fuel cycle.

At this point plutonium recycle has not yet begun, and there is no major industrial commitment of resources to it [5]. No major commercial plutonium fuel fabricating plants are operating or under construction.* No commercial reprocessing plants are operating now.** Reprocessing plants, in addition to recovering plutonium and other fission products from the spent fuel, are supposed to solidify high-level wastes and ship them to a permanent AEC repository for perpetual management. As yet, however, the AEC has no such repository. Nor does the AEC know whether the technology and social institutions for isolating these high-level wastes for geologic periods can be made available.

If the plans of the AEC and the nuclear industry are permitted, however, a major plutonium industry will develop quickly. Some 140 tons of plutonium could be recovered from commercial reactors by 1985 and some 1,700 tons by the year 2000 [7]. A plutonium industry by the turn of the century could involve hundreds of light water reactors fueled with plutonium, perhaps a score of fuel reprocessing and fabricating plants, and thousands of interstate and international shipments containing hundreds of tons of plutonium.

Plutonium Toxicity

The most pernicious product of the nuclear industry is plutonium. Microgram quantities in skin wounds cause cancer, and in the body plutonium is a bone seeker where, once deposited, it can cause bone cancer. But plutonium is most dangerous when inhaled. Donald Geesaman explains this hazard:

> Under a number of probable conditions plutonium forms aerosols of micron-sized particulates. When lost into uncontrolled air these particulates can remain suspended for a significant time, and if inhaled they are preferentially deposited in the deep lung tissue, where their long residence time and high alpha activity can result in a locally intense tissue exposure. The lung cancer risk associated with these radiologically unique aerosols is unknown to orders of magnitude. Present plutonium standards are certainly irrelevant and probably not conservative. Even so, the fact that under present standards, the permissible air concentrations are about one part per million billion is a commentary on plutonium's potential as a pollutant [3].

To determine whether the AEC's radiation protection standards for plutonium are inadequate, as Geesaman suggests, two of the authors of this article undertook a review of the biological evidence for the Natural Resources Defense Council (NRDC). Their report, *Radiation Standards for Hot Particles* [9], concludes that plutonium particulates or hot particles are uniquely virulent carcinogens and that the current AEC radiation protection standards governing the amount of plutonium to which members of the public can be exposed are roughly 100,000 times too lax.

The lung cancer risk associated with hot particles of plutonium, as estimated by Tamplin and Cochran, is comparable to the lethal dose of botulin toxin, a biological warfare agent. Certainly one would hope that this nation would give careful consideration and pursue all alternatives before implementing an energy policy based on such toxic materials.

As a result of this study, NRDC formally petitioned the AEC and the Environmental Protection Agency to reduce the present maximum permissible exposure levels by 100,000. Neither the AEC nor the EPA have responded finally to NRDC's petition, but the petition is now being considered by National Council on Radiation Protection and Measurements, National Academy of Sciences, Biophysical Society and several AEC national laboratories. Moreover, EPA will

*There are, however, several small commercial facilities that process plutonium for research and development purposes.

**The first commercial reprocessing plant built in the United States, Nuclear Fuel Services in West Valley, New York, was shut down in 1972 for repairs and enlargement. The Midwest Fuel Recovery Plant under construction near Morris, Illinois, has been declared an almost total loss due to faulty design and construction [6]. The Barnwell Nuclear Fuel Plant in South Carolina is 70 percent complete. Thus, since mid-1972, all spent fuel from light water reactors has been simply stored and not reprocessed.

shortly commence a series of hearings and other initiatives on plutonium-related issues, including the hot particle controversy.

Although the adequacy of the AEC's plutonium standards is thus a matter of considerable doubt and great controversy, the AEC's draft environmental impact statement for plutonium recycle simply assumes that the present standards are adequate. The entire risk analysis of the statement, as well as the ultimate decision to proceed with plutonium recycle, are based upon a premature and unexplained rejection of the hot particle hypothesis. Yet, the AEC is forced to concede that this hypothesis "is being given careful consideration in a separate proceeding" [2, chap. 4, pp. 5-7].

We submit that the AEC has no basis whatever to conclude that plutonium recycle will not cause undue risk to the public health and safety until it has either satisfactorily resolved the hot particle issue or calculated the impacts of plutonium recycle using the assumption that hot particles are uniquely carcinogenic. The AEC's draft environmental impact statement for plutonium recycle does neither. However, the more basic issue is whether we want our energy system based on a material of unprecedented toxicity.

Some plutonium contamination of the environment has already occurred, due principally to the atomic weapons program. The leakage of plutonium from contaminated oil at the AEC's plutonium weapons plant at Rocky Flats, 10 miles west of Denver, Colorado, led to an uncontrolled source of plutonium which was much larger than the integrated effluent loss during the 17 years of plant operation. Tens to hundreds of grams of plutonium went off-site, 10 miles upwind from Denver [3, p. 59].

The Nuclear Materials and Equipment Corporation (NUMEC) of Apollo, Pennsylvania, was recently fined $13,720 for a 16 count violation of AEC regulations ranging from failure to follow radiation monitoring procedures to failure to comply with certain safeguards requirements [9]. Production workers at Nuclear Fuel Services, Inc. in Erwin, Tenn., a fuel processing and fabricating facility, met with AEC inspectors on August 13, 1974 to complain about the absence of even the rudiments of accepted health physics practices at that plant. Occurrences such as these can reasonably be expected to multiply greatly if plutonium is made a major article of commerce.

Nuclear Theft

On May 18 of this year the world was made dramatically aware of the relationship between nuclear power and nuclear weapons when India exploded a nuclear device made from plutonium taken from a peaceful reactor built with Canadian assistance. The magnitude of the threat posed by the availability of plutonium from power reactors is set out by Willrich and Taylor in their book *Nuclear Theft: Risks and Safeguards:*

> As fuel for power reactors, nuclear weapon material will range in commercial value from $3,000 to $15,000 per kilogram—roughly comparable to the value of black market heroin. The same material might be hundreds of times more valuable to some group wanting a powerful means of destruction. Furthermore, the costs to society per kilogram of nuclear material used for destructive purposes would be immense. The dispersal of very small amounts of finely divided plutonium could necessitate evacuation and decontamination operations covering several square kilometers for long periods of time and costing tens or hundreds of millions of dollars. The damage could run to many millions of dollars per gram of plutonium used. A nuclear explosion with a yield of one kiloton could destroy a major industrial installation or several large office buildings costing hundreds of millions to billions of dollars. The hundreds or thousands of people whose health might be severely damaged by dispersal of plutonium, or the tens of thousands of people who might be killed by a low-yield nuclear explosion in a densely populated area represent incalculable but immense costs to society [4, pp. 107-108].

In our troubled world, terrorist activity and other forms of anti-social violence are an almost daily occurrence. A recent AEC study identified more than 400 incidents of *international* terrorism carried out by small groups during the past six years [10]. In an age of bombs and bomb threats, of aircraft hijacking, of the ransom of diplomats and the murder of Olympic athletes, the risks of nuclear theft, blackmail and terrorism are not minimized even by some of the most ardent supporters of nuclear energy. Thus former Atomic Energy Commissioner Clarence Larson has described the evolution of a plutonium black market:

> Once special nuclear material is successfully stolen in small and possibly economically acceptable quantities, a supply-stimulated market for such illicit material is bound to develop. And such a market can surely be expected to grow once the source of supply has been identified. As the market grows, the number and size of thefts can be expected to grow with it, and I fear such growth would be extremely rapid once it begins. . . .Such theft would quickly lead to serious economic burdens to the industry, and a threat to the national security [11].

The critical point here is that these tremendous risks will become real with the advent of plutonium recycle. Unless plutonium is reprocessed and recycled, the possibility that it will be stolen is small. If the plutonium has not been detoxified by separating it from the high-level wastes in the spent fuel at a reprocessing plant, it is very effectively protected from theft, at least for hundreds of years. Willrich and Taylor explain these relationships:

> In the light water reactor (LWR) fuel cycle without plutonium recycle, plutonium which is produced in a power reactor, if reprocessed, might be stolen at the

Is the American public willing to accept the risks of plutonium in exchange for the promised benefits?

output end of a reprocessing plant, during transit from the reprocessing plant to any separate storage facility used, and from a long-term plutonium storage facility. *Until irradiated fuel is reprocessed, the theft possibilities in the LWR fuel cycle are minimal.* (Emphasis added.)

In the LWR fuel cycle with plutonium recycle, in addition to possibilities without recycle, plutonium might be stolen during transit from any separate long-term storage facility, and from a fuel fabrication plant. Complete LWR fuel assemblies, each containing a significant quantity of plutonium might also be stolen during transit from a fuel fabrication plant to a power reactor, and at a power plant prior to loading into the reactor, although the weight of each assembly makes this difficult [4, p. 168].

In sum, plutonium recycle will bring with it all the risks associated with nuclear theft that numerous authors have described [12]. Reasonable prudence dictates, therefore, that we have adequate answers to the problem of nuclear theft well in hand before we begin plutonium recycle.

Safeguards and the AEC

In the language of the nuclear industry, the various programs and techniques to prevent nuclear theft and recover stolen nuclear material are called 'safeguards.' There is now widespread agreement—at least among those outside the nuclear industry—that present safeguards against nuclear theft are woefully inadequate [13]. The AEC's Rosenbaum Report concluded:

> In recent years the factors which make safeguards a real, imminent and vital issue have changed rapidly for the worse. Terrorists groups have increased their professional skills, intelligence networks, finances and level of armaments throughout the world. . . .Not only do illicit nuclear weapons present a greater potential public hazard than the radiological dangers associated with power plant accidents, but. . .the relevant regulations are much less stringent [13].

The problem is not simply that the AEC has not implemented the necessary safeguards programs; rather the agency has not even developed an adequate program on paper.

On the subject of safeguards, the AEC's draft impact statement on plutonium recycle is a marvel of clouded reasoning and breezy optimism. The statement concedes that the objective of keeping the risk of nuclear theft small "will not be fully met for the recycle of plutonium by current safeguards measures" [2, pp. 5-6]. Steps which might be taken to correct current inadequacies are then summarized in the statement as follows:

> 1. Minimization or elimination of the transportation of plutonium from reprocessing plants to mixed oxide fuel fabrication facilities which is the operation most vulnerable to an attempted act of theft or sabotage. To the extent that such shipments are minimized or eliminated, the safeguarding of plutonium would be enhanced. This objective can be accomplished by locating mixed oxide fuel fabrication plants in close proximity to or adjacent to reprocessing plants in Integrated Fuel Cycle Facilities. . . .
>
> 2. Further protection of transportation functions by use of massive shipping containers, special escort or convoying measures, vehicle hardening against attack, improved communications and response capabilities.
>
> 3. Additional hardening of facilities through new barrier requirements, new surveillance instrumentation, new delaying capabilities (e.g., incapacitating gases).
>
> 4. Upgrading of operating and guard functions through the use of personnel security clearance procedures, a federally operated nuclear security system, more advanced systems for monitoring and searching of personnel, and closer liaison with law enforcement authorities.
>
> 5. Improving the timeliness and sensitivity of the system of internal control and accountability of plutonium.
>
> 6. Use of 'spiked' plutonium which would be less susceptible to theft and would be more difficult to manufacture into a nuclear explosive because of the required elaborate handling procedures [2, pp. 5-7].

Despite the facts that: (1) these proposals are preliminary and their content not well defined, (2) they are still being studied, some apparently for the first time, (3) some would require Congressional action, (4) some would necessitate substantial changes in the structure of the U.S. utility industry, and (5) a sophisticated safeguards program would pose a major threat to civil liberties and personal privacy—despite all these facts the draft impact statement nevertheless recommends that we proceed now with plutonium recycle because "the Commission has a high degree of confidence that through implementation of some combination of the above concepts the safeguards general objective set forth earlier can be met for plutonium recycle" [2, pp. 5-7]. The Commission's faith, unfortunately, is hardly reassuring.

The AEC's lead safeguards suggestion—the Integrated Fuel Cycle Facility concept—merits special comment. It actually represents a major watering down of a far more significant concept, that of nuclear power parks where reactors as well as fuel reprocessing and fabricating plants are all located at one site [14]. In our judgment, a safeguards system which does not require nuclear parks is not addressing the problem of theft during transportation in a serious and responsible way. Moreover, the nuclear industry's current plans, already well advanced, do not call for the implementation of even the Integrated Fuel Cycle Facilities concept.

Adequate Safeguards?

While it may be possible to devise an adequate safeguard system in theory, there is little reason to believe that such a system would be acceptable in practice [15]. This is true for several reasons.

First, the problem is immense. The illegal diversion of weapons material is only one type of antisocial behavior a safeguards program must protect against. Terrorist acts against the reactors, shipments of radioactive wastes, fuel reprocessing facilities and waste repositories can result in catastrophic releases of radioactivity. Such threats against nuclear facilities have already occurred [16]. Moreover, a safeguards system would have to exist on a vast, worldwide basis. Some 1,000 nuclear reactors are projected for the United States in the year 2000, with hundreds of shipments of radioactive materials daily. Hundreds of tons of plutonium will be in the commercial sector of our economy by that date.

To accommodate plutonium we shall have to move toward a more intimidated society with greatly reduced freedoms.

Abroad, American firms are constructing nuclear reactors in countries that have little political stability and in countries, such as Japan, who have not signed the Non-Proliferation Treaty. Safeguarding nuclear bomb material would ultimately require a restructuring of the socio-political institutions on a worldwide scale. The United Nations unfortunately gives us little reason to believe that this is a practical reality.

Second, safeguards measures are strongly opposed by the nuclear industry. The degree to which the industry is sensitive to the diversion hazards and is likely to be an effective partner in the enforcement and implementation of safeguards programs was apparent in the vociferous industry opposition to the modest strengthening of the AEC safeguards rules which were first published in the February 1, 1973, *Federal Register* [17].

Third, experience with present safeguards is hardly reassuring. Nuclear Materials and Equipment Corporation, over several years of operation, was unable to account for six percent (100 kilograms) of the weapons grade material that it handled. As noted previously, it was also fined by the AEC, in part, because of safeguards violations. At a safeguards symposium the director of the AEC's Office of Safeguards and Materials Management observed that "we have a long way to go to get into that happy land where one can measure scrap effluents, products, inputs and discards to a one percent accuracy" [3, p. 59]. This statement takes on particular significance when it is realized that only one-half of one percent of the plutonium utilized by the commercial sector in the year 2000 is enough to make hundreds of atomic bombs. The editors of the *Bulletin* have noted that the frequent 'misroutings' of shipments of weapons grade materials highlights a key safeguards problem —hijacking [18].

A spot-check by General Accounting Office investigators at three AEC-licensed contractors showed that in some cases access to easily portable quantities of special nuclear material could be gained in less than a minute using the simplest of tools. At two of the three plants checked, GAO found weak physical barriers, ineffective guard patrols, ineffective alarm systems, lack of automatic-detection devices, and the absence of an action plan should material be stolen or diverted. AEC's inspectors, however, were giving the same facilities good marks on virtually every security category [GAO, 13].

Fourth, and perhaps most basically, there is little reason to believe that safeguards will work when little else does. For example, the AEC supports the creation of a federal police force to provide an immediate federal presence whenever the use of force may be needed to protect these incredibly dangerous materials from falling into the hands of would-be saboteurs and blackmailers. But is there anyone who believes that police are effective at a level commensurate with the potential nuclear hazard? The New York City police department was proven incapable of maintaining security over confiscated heroin. Are similar losses of plutonium acceptable?

The general point here is that our safeguards system must be essentially infallible. It must maintain what Alvin Weinberg, former director of the Oak Ridge National Laboratory, has called "unaccustomed vigilance" and "a continuing tradition of meticulous attention to detail" [19]. Yet our human institutions are far from infallible. Our experience indicates that rather than sustaining a high degree of esprit, vigilance and meticulous attention to detail, our governmental bureaucracies instead become careless, rigid, defensive and, less frequently, corrupt. A basic question, then, is whether we want to entrust so demanding and unrelenting a technology as plutonium recycle to institutions which are negligent of their own responsibilities and insensitive to the rights of others and to technical fixes which are untried and unproven.

Threat to Civil Liberties

One principal reason for our believing that an adequate safeguards system would not be acceptable in practice is the tremendous social cost of such a system in terms of human freedom and privacy. Safeguards necessarily involve a large expansion of police powers. Some one million persons have been trained in the handling, moving and operation of nuclear weapons. The projected growth of the nuclear industry will give rise to a parallel and, ultimately, a much larger group of persons, in this case civilians, who will be subjected to security clearance and other security procedures now commonplace in the military weapons program. Indeed, the AEC makes the following disturbing statement in its draft environmental impact assessment of plutonium recycle:

> Security problems are much simplified when it can be established with high probability that the persons who are responsible for the handling of plutonium or implementing of related safeguards programs are trustworthy. Various court rulings in recent years have been favorable to the protection of individual privacy and of individual right-to-work. These rulings have made it difficult to make a personnel background check of an individual in commercial activities to assure with high probability that he is trustworthy and, hence, potentially acceptable as a steward for the protection of plutonium. The AEC has requested legislation which would allow background checks of individuals with access to plutonium and related material accountability records [2, chap. 5, p. 42].

The keeping of police dossiers will not be limited to nuclear industry personnel. The New York Times reported August 11 that Texas state police maintain files on nuclear power plant opponents. How much more government investigation into the private lives of individuals can be tolerated by a free society? Security and surveillance procedures at best infringe upon the privacy of families and their friends. At worst, they are the instruments of repression and reprisal.

A second AEC safeguards proposal is the creation of a federal police force for the protection of plutonium plants and shipments. The draft impact statement for plutonium recycle justifies such a federal force in the following terms:

> A federal security system would be less apt to have the variations in staff and capability that would be encountered in use of private security guards. In addition, it should be noted that the consequences of a successful theft or diversion of plutonium would undoubtedly have nationwide impacts and could best be handled by Federal authorities; certainly, with Federal participation, there is the potential for a larger force, more effective weapons, and better communications [2, chap 5, p. 42].

How large would such a force be? What standards should govern and restrain its operations? The Washington Post reported in October 1973 that the AEC issued shoot-to-kill orders to personnel directing the production, shipment and storage of atomic weapons at the height of the Yom Kippur War.

Once a significant theft of plutonium or other weapons material has occurred, how will it be recovered? To prevent traffic in heroin, police have asked for no-knock search laws. This infringes upon one of our most cherished freedoms. To live with plutonium we may have to abandon this freedom along with others. In the presence of nuclear blackmail threats, the institution of martial law seems inevitable. It has been said that the widespread availability of weapons material and terrorists targets in the nuclear fuel cycle will radically alter the power balance between large and small social units (De Nike [16]). It should be added that the threatened society will undoubtedly attempt to redress that balance through sophisticated and drastic police action.

In sum, to accommodate plutonium we shall have to move toward a more intimidated society with greatly reduced freedoms. In this respect the following passage from the report of the distinguished international group of scientists attending the 23rd Pugwash Conference on Science and World Affairs is instructive:

> The problem of theft of nuclear material by internal groups of individuals intent on sabotage, terrorism or blackmail was agreed to be a very serious one, although there was some sentiment expressed that the possibility of such activity was much smaller in socialist states.

We believe that sentiment to be true. It is also apparent that that is the direction in which we must move to accommodate the nuclear industry. After having spent billions of dollars for our nuclear deterrent, our civilian nuclear industry might well accomplish that which our defense system is trying to prevent.

Alvin Weinberg is one of the few persons closely associated with the nuclear power complex who has looked carefully at the political and regulatory institutions that will be necessary to support a plutonium-based nuclear power economy, and his views on this subject merit close attention [19]. Weinberg's basic premise is that nuclear power will place unprecedented strains on our society. In an unpublished paper circulated prior to a conference in June 1973 at the Woodrow Wilson International Center for Scholars in Washington, D.C., Weinberg set out his views on the type of new institutions required to cope with the plutonium economy:

> One suggestion (proposed by Sidney Siegel) that is relevant to the situation in the United States would be to establish a national corporation patterned after COMSAT to take charge of the *generation* of nuclear electricity. Such an organization would have technical resources that must exceed those available to even a large utility: and a high order of technical expertise in operating reactors and their sub-systems is essential to ensuring the continued integrity of these devices. [Here Dr. Weinberg suggests nationalization of the industry.]
>
> Each country now has its own AEC that sets standards or, in some cases, actually monitors or operates reactors. Perhaps this will be sufficient forever. Yet no government has lasted continuously for 1,000 years: only the Catholic Church has survived more or less continuously for 2,000 years or so. Our commitment to nuclear energy is assumed to last in perpetuity—can we think of a national entity that possesses the resiliency to remain alive for even a single half-life of plutonium-239? A *permanent* cadre of experts that will retain its continuity over immensely long times hardly seems feasible if the cadre is a national body.
>
> It may be that an International Authority, operating as an agent of the United Nations, could become the focus for this cadre of expertise. The experts themselves would remain under national auspices, but they would be part of a worldwide community of experts who are held together, are monitored, and are given long-term stability by the International Authority. The Catholic Church is the best example of what I have in mind: a *central authority* that *proclaims* and to a degree *enforces* doctrine, maintains its own long-term social stability, and has connections to every country's own Catholic Church. (Emphasis added.)

These are far-reaching concepts presented by Weinberg. The basic question they pose is: Will the plutonium economy raise socio-political problems of such magnitude that their resolution will be unacceptable to society? In attempting to do the impossible—live with plutonium—we may create the intolerable.

Super-Human Requirements

The commercialization of plutonium will bring with it a major escalation of the risks and problems already associated with nuclear power. Plutonium will further strain the already weakened regulatory fabric of the nuclear industry.

Hannes Alfven, Nobel laureate in physics, has described the regulatory imperatives applicable to the nuclear industry:

> Fission energy is safe only if a number of critical devices work as they should, if a number of people in key

positions follow all their instructions, if there is no sabotage, no hijacking of the transports, if no reactor fuel processing plant or reprocessing plant or repository anywhere in the world is situated in a region of riots or guerrilla activity, and no revolution or war—even a "conventional one"—takes place in these regions. The enormous quantities of extremely dangerous material must not get into the hands of ignorant people or desperados. No acts of God can be permitted [20].

Weinberg similarly stresses the need ". . . of creating a continuing tradition of meticulous attention to detail" and suggests that "what is required is a cadre that, from now on, can be counted upon to understand nuclear technology, to control it, to prevent accidents, to prevent diversion" [19].

The public and its decisionmakers must seriously question whether it will be possible to attract, train and motivate the personnel required for these functions. These must be highly qualified persons who will maintain a tradition of "meticulous attention to detail" even when the glamorous aspects of a new technology become the commonplace operations of an established industry. We suggest that it is beyond human capabilities to develop a cadre of sufficient size and expertise that can be counted upon to understand nuclear technology, to control it, and to prevent accidents and diversion over many generations.

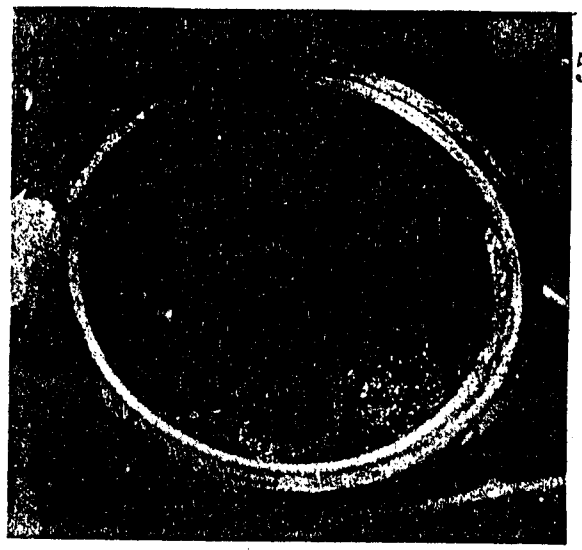
There is considerable evidence at the present time to suggest that the fledgling nuclear industry is already unmanageable. Consider, for example, that a previously secret AEC study released by Ralph Nader concluded that:

> The large number of reactor incidents [850 abnormal occurrences], coupled with the fact that many of them had real safety significance, were generic in nature, and were not identified during the normal design, fabrication, erection, and preoperational testing phases, raises a serious question regarding the current review and inspection practices both on the part of the nuclear industry and the AEC [21].

In addition, consider the tritium that recently appeared in the drinking water of Broomfield, Colorado. Consider the 115,000 gallons of high-level radioactive wastes that leaked from the tank at Hanford, Washington, over a period of 51 days while no one monitored the tank. Consider that the radioactive releases from the famed Shippingport reactor in Pennsylvania were higher than recorded. Consider that the executives of Consumers Power Corporation in Michigan failed to notify the AEC that their radioactive gas holdup system was not functioning. Consider that two reactors in Virginia were half completed before the AEC was informed that they were being constructed over an earthquake fault. Consider that the GAO found security at plutonium storage areas totally inadequate after the AEC inspectors had certified the facilities.

Considering all this, there is good reason to suggest, because of the meticulous attention to detail that will be required at every stage of plutonium recycle, that a decision to proceed with plutonium recycle will precipitate an already unmanageable situation into a national crisis.

Given that the risks of plutonium recycle are unacceptably high, particularly in light of the present



Plutonium in cake form. This batch was produced at the AEC's Savannah River Plant near Aiken, S.C.

uncertainties, a key question is what are our options? What are the alternatives to the AEC's proposal to proceed now with plutonium recycle? We believe that there are essentially three options, each of which is preferable to the AEC's announced plan.

Alternatives to Plutonium Recycle

• We could phase out nuclear power reactors. There is mounting apprehension among knowledgeable persons concerning the human and societal hazards of fission reactors, which would only be compounded by plutonium recycle. The 23rd Pugwash Conference on Science and World Affairs in September, 1963, concluded:

> 1. Owing to potentially grave and as yet unresolved problems related to waste management, diversion of fissionable material, and major radioactivity releases arising from accidents, natural disasters, sabotage, or acts of war, the wisdom of a commitment to nuclear fission as a principal energy source for mankind must be seriously questioned at the present time.
> 2. Accordingly, research and development on alternative energy sources—particularly solar, geothermal and fusion energy, and cleaner technologies for fossil fuels—should be greatly accelerated.
> 3. Broadly based studies aimed at the assessment of the relation between genuine and sustainable energy needs, as opposed to projected demands, are required.

This third recommendation implies the implementation of energy conservation measures. It is important to recognize that energy conservation can be our major energy source between now and the year 2000. Conservation means using our present energy more efficiently; it need not mean a change in life styles. Coupled with the use of solar and geothermal energy, energy conservation could eliminate the need for new nuclear power stations.

• We could continue with the present generation of light water reactors but strictly prohibit plutonium recycle for the foreseeable future. Such a decision would be premised upon a judgment that plutonium

556

is too dangerous because of its toxicity and explosive potential to be allowed to become an article of commerce. Of course, we would still have plutonium to cope with because it is produced in present-day reactors. But without plutonium recycle there should be little incentive to reprocess the plutonium out of the spent fuel, so the plutonium could remain in the spent fuel where it is effectively protected from theft and, hopefully, confined and contained.

The benefits of plutonium recycle are small. Plutonium recycle would reduce the annual uranium requirements by about 10 to 15 percent and reduce the light water reactor fuel cycle cost by about the same amount. But the nuclear fuel cycle cost represents less than 20 percent of the total cost of power from nuclear plants, and nuclear plants by 1985 will represent less than 40 percent of the electric, or about 15 percent of the total, domestic energy supplied. In other words, plutonium recycle involves an economic savings of less than one-half of one percent.

Plutonium differs from the high-level wastes in the spent fuel in one critical respect: whereas the radioactivity of high-level wastes will continue for thousands of years, that of plutonium will continue for hundreds of thousands. Thus, while the problem of effectively storing both these materials and preventing their entering the environment are unprecedented in human history, plutonium must be contained for eons longer. For this reason, an argument can be made that, ultimately, the safest thing that can be done with plutonium is to burn or fission it in reactors, thus making it into high-level wastes rather than plutonium. But that is an activity that is best left for decades or even centuries hence—for a society more capable and less violent than today's.

- We could defer for several years the decision regarding plutonium recycle until present uncertainties regarding safeguards and plutonium toxicity are satisfactorily resolved and a basis has been laid for a more intelligent judgment regarding the risks and benefits of the commercialization of plutonium. We believe that this option must command general support. Too many questions, both technical and social, are unanswered today. And until these questions are answered it would be a grave error, we believe, to rush into the AEC's plutonium economy.

Is the American public willing to accept the risks of plutonium in exchange for the promised benefits? The national debate which must occur on this basic question has hardly begun.

NOTES

1. Glenn T. Seaborg, "The Plutonium Economy of the Future," Release No. S-33-70 (Washington, D.C.: Atomic Energy Commission, October 5, 1970).

2. Atomic Energy Commission, "Draft Generic Environmental Statement on the Use of Mixed Oxide Fuel," WASH-1327 (Washington, D.C.: The Commission, July 1974).

3. Donald P. Geesaman, "Plutonium and the Energy Decision," in *The Energy Crisis*, ed. R.S. Lewis and B.I. Spinrad (Chicago, Ill.: Bulletin of the Atomic Scientists, 1972), pp. 58-59.

4. Mason Willrich and Theodore B. Taylor, *Nuclear Theft: Risks and Safeguards* (Cambridge, Mass.: Ballinger, 1974).

5. The AEC's attempt to recycle plutonium into the Big Rock Point (Mich.) reactor was stopped by a lawsuit. *West Michigan Environmental Action Council v. AEC* (W. D. Mich. Dkt. No. G-58-73).

6. *Weekly Energy Report*, "GE Fuel Recovery Plant 'Inoperable,'" II (July 15, 1974), 1.

7. Atomic Energy Commission, "Nuclear Power Growth: 1974-2000," WASH-1139 (Washington, D.C.: The Commission, 1974), p. 34 (Case D projection). The year 2000 figure includes plutonium produced in liquid metal fast breeder reactors.

8. Arthur Tamplin and Thomas Cochran, *Radiation Standards of Hot Particles* (Washington, D.C.: Natural Resources Defense Council, Feb. 14, 1974). Copies of this report are available from NRDC (1710 N St., N.W., Washington, D.C. 20036) for $3 per copy.

9. Atomic Energy Commission, press release, August 14, 1974.

10. W. C. Bartels and S. C. T. McDowell, quoted in *Nuclear News*, 17 (Aug. 1974), 46.

11. Clarence E. Larson, "Nuclear Materials Safeguards: A Joint Industry-Government Mission," in *Proceedings of AEC Symposium on Safeguards Research and Development*, Oct. 27-29, 1969, WASH 1147 (Washington, D.C.: The Commission, 1969); and Deborah Shapley, "Plutonium: Reactor Proliferation Threatens a Nuclear Black Market," *Science*, 172:3979 (April 9, 1971), 143.

12. See, for example, Bernard T. Feld, "The Menace of a Fission Power Economy," *Bulletin*, 30 (April 1974), 32-34; Lawrence Scheinman, "Safeguarding Nuclear Materials," *Bulletin*, 30 (April 1974), 34-36; David T. Rose, "Nuclear Electric Power," *Science*, 184:4134 (April 19, 1974), 351-359. See also Robert L. Heilbroner, *An Inquiry into the Human Prospect* (New York: W. W. Norton, 1974), pp. 40-43.

13. See, for example, Atomic Energy Commission, "The Threat of Nuclear Theft and Sabotage" (Rosenbaum Report), *Congressional Record*, April 30, 1974, p. S 6621; General Accounting Office, "Protecting Special Nuclear Material in Transit: Improvements Made and Existing Problems," B-164105 (Washington, D.C.: U. S. Government Printing Office, 1973).

14. Dean E. Abrahamson, "Energy: Nuclear Theft and Nuclear Parks," *Environment* (July/August, 1974), 5.

15. Taylor and Willrich believe that "a system of safeguards can be developed that will keep the risks of theft of nuclear weapon materials from the nuclear power industry at very low levels" [4, p. 171]. Yet they also emphasize that "regardless of its effectiveness, a nuclear safeguards system applicable to the nuclear power industry in this country cannot provide complete assurance that unannounced fission explosions will not occur in the United States in the future." They point out that "no future safeguards system that will be practical can offer 100 percent assurance against theft" [4, p. 123]. They never say what level of nuclear theft, or what size plutonium black market or how many unauthorized nuclear explosions are in fact acceptable to them.

16. L. Douglas DeNike, "Radioactive Malevolence," *Bulletin*, 30 (February 1974), 16. See also the story on the bomb threats that have occurred at the Zion nuclear power plant in northern Illinois reported in *Environment*, "Spectrum" (October 1974).

17. *Nuclear Industry*, "Industry Inundated by Proposed New Safeguards Rules" (February 1973), pp. 45-47.

18. R. S. Lewis and B. I. Spinrad, eds., *The Energy Crisis*, (Chicago, Ill.: Bulletin of the Atomic Scientists, 1972), p. 59.

19. Alvin Weinberg, "Social Institutions and Nuclear Energy," *Science*, 177:4043 (July 7, 1972), 32-34.

20. Hannes Alfven, "Energy and Environment," *Bulletin*, 29 (May 1972), 5.

21. AEC Task Force Report, dated October, 1973, page 16, released in testimony presented to the Joint Committee on Atomic Energy by Ralph Nader and the Union of Concerned Scientists, January 29, 1974.

ENVIRONMENTAL ACTION OF COLORADO

January 1, 1975

Dr. William Mills, Director
Criteria and Standards Division
Office of Radiation Programs
Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Dr. Mills:

I am writing to request an opportunity to provide testimony at the upcoming plutonium standards hearing to be held in Denver on the 10th of this month. It is my understanding from a conversation with Paul Smith of E.P.A., Denver regional office, that it is necessary for those who wish to testify to submit a written request prior to the 3rd of January outlining the gist of one's testimony.

It is also my understanding that the Denver hearing is a continuation of a one-day hearing held in Washington, D.C. on the 10th of December. Paul didn't have any reports that had resulted from that first section of this hearing, but he said that he believed the testimonies from both were going to be compiled and distributed sometime after January 10th and that to the best of his awareness there was not going to be any other opportunity for public participation or input (i.e. no other hearings or public forums). Is this true? Are there any other hearings or programs planned that involve the public at large? I would also greatly appreciate any documentation of the Washington hearing that may be accessible, including a list of who testified about what, who makes up the E.P.A. panel, etc.

I plan on giving a curt statement, not longer than 10 minutes, regarding the overall health hazards to the public, particularly from plutonium releases in the past and potentially in the future in this area, and how they relate to Denver regional planning. I will also touch upon the federal government's and industry's approach to public awareness and educational efforts that have been made in the past and how these efforts have fallen short of what is needed. I would like, if possible, to utilize a slide presentation and would also like to submit reprinted material to be included in the record.

If you have any questions or comments, please don't hesitate to contact me. We are very happy to see that this important subject is being dealt with and that the public is allowed to participate. I look forward to your response.

Sincerely,

Albert Nunez, Jr.
1100 14TH STREET
DENVER, COLORADO 80202
PHONE (303) 534-1602

AN:jmj

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Solar Energy Coalition of Texas ..... S.E.C.T. ... P.O. Box 28183, San Antonio, Texas 78228 •

(512) 732-4181 • (512) 733-0557

MATERIAL SUBMITTED RE: HEARING ON PLUTONIUM

January 1, 1975

Dr. William Mills
Director of Criteria and
Standards Division
Office of Radiation Program
Environmental Protection Agency
Washington, D.C. 20460

Dear Sir:

We submit the enclosed items for your record and also wish to express our opposition to the building and operation of nuclear fission reactors, since they produce radioactive wastes including plutonium 239 and since they are being designed to use plutonium as fuel.

Sincerely yours,
Stephanie Kolodziej
Stephanie Kolodziej, Research Secretary

Enclosures: Physicians letter, Plutonium items.

"Nuclear fission is one of the main obstacles to other new energy systems. If we were not in the middle of a sales campaign for nuclear fission, we would be in the middle of a sales campaign for the alternatives - and solar energy is the outstanding alternative. The moment a moratorium on the operation of nuclear power plants seems certain, we will see solar energy systems blossom into commercial readiness with breath-taking speed." Egan O'Connor, energy consultant, Committee for Nuclear Responsibility, Washington, D.C. 20003.

HERMAN R. LEVINE, M.D.
1017 DONALDSON AVENUE
SAN ANTONIO, TEXAS 78228

732-4181

January 1, 1975

Dr. William A. Mills, Director of
Criteria and Standards Division
Office of Radiation Program
Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

As a physician, I wish to take this opportunity to express my deep concern regarding the proposed move to use plutonium 239 as a power source.

With just a comparatively few fission reactors operating, there are already numerous accidental plutonium contaminations and spills. With increased use of this chemical as fuel, accidents will multiply and plutonium pollutants will eventually reach out and cause widespread tragic disease and deformities.

It is therefore essential that plutonium be banned from use as nuclear fuel and that the operation of nuclear reactors, since they produce plutonium, be halted.

Sincerely yours,

Herman R. Levine

Herman R. Levine, M.D.

A. E. C. Release

No. T-613
Contact: Carl Gustin
Tel. 301/973-7771

FOR IMMEDIATE RELEASE
(Monday, December 16, 1974)

AEC PROPOSES FINING COMMONWEALTH EDISON $25,500
FOR APPARENT VIOLATIONS OF AEC REGULATIONS

The Atomic Energy Commission's Director of Regulatory Operations has proposed fining Commonwealth Edison Company of Chicago $25,500 for 18 apparent violations of AEC Regulations which occurred at the three-unit Dresden Nuclear Power Plant in Morris, Illinois, between June and September.

The apparent violations involved management of the plant's radioactive waste (radwaste) system, an unplanned and uncontrolled release of radioactivity from Dresden Unit 1 and implementation of the facility's security plan. None of the violations involved an immediate threat to public health and safety.

The release of radioactivity occurred on August 25 when 1,130 gallons of water from the unit's laundry tank were pumped inadvertently into the Illinois River through a valve that should have been closed. The radioactivity was undetectable above natural background levels and posed no public health or safety hazard.

Director of Regulatory Operations, Dr. Donald F. Knuth, in a letter to the Company, said the incident was the latest example of failure to properly manage radioactive waste operations.

Other apparent violations involving the radwaste system include the Company's failure to exercise control over the use of valves through which radioactive liquids can be discharged, to conduct required analyses for radioactive isotopes and to calibrate monitoring instrumentation. The systems are designed to keep the routine release of radioactivity from nuclear plants as low as practicable.

Violations of the security plan involved control of limited access areas, training of guards and the adequacy of communications between security force personnel. The Company's security plan is intended to meet substantially strengthened AEC security requirements which went into effect on March 6, 1974.

In his letter to the Company, Dr. Knuth said the AEC plans to continue to conduct unannounced inspections to assure full compliance in the future with AEC requirements.

no immediate threat to public health + safety!
this technology is SILENT VIOLENCE

Not Man Apart June 1974
(Friends of Earth publication)

(If shows that have [illegible] Dept. Lic. [illegible])

NEWS

(Reprinted by permission of Not Man Apart.)

Radiation Leaks In Ohio, South Carolina, and Illinois

JEFF KNIGHT

The past month has seen three accidental releases of various kinds of radiation into the environment.

The most serious was the discovery that Plutonium-238 had leaked out of the AEC's Mound Weapons Laboratory in Miamisburg, Ohio (population 14,000) into a portion of the Erie Canal near the factory. The plutonium was discovered by city officials taking samples of the sediment in the canal.

"We have no idea how the plutonium leaked out of the factory into the mud," an AEC spokesman told the *Washington Post*. "This comes as a complete surprise." The AEC said it is investigating the leak but did not know how long it had been going on or how extensive it was. The AEC said there is no danger as long as the Pu-238 remains in the mud. (*The Cincinnati Post* reported that the plutonium may have been leaking for 16 years.)

Plutonium-238 is about 250 times more hazardous than Pu-239. It has a half-life of 90 years and is less hazardous if it is in water than in the atmosphere. Like Pu-239, it is most dangerous if taken into the lungs. Nevertheless, some scientists feel that the AEC's standards for plutonium in water are much too high.

The second "incident" occurred at O'Hare Airport in Chicago, where a container with one millicurie of Iodine-131, a medical isotope, fell to the runway and was run over by a fork-lift. It contaminated 1,000 square feet of runway, which was hosed down. While the danger this accident created may have been negligible (Iodine-131 is normally disposed of by discharge into the sewer system), the accident highlights the problems in safely transporting radioactive materials.

The third release was on May 8, at the AEC special nuclear weapons production facility on the Savannah River in South Carolina. Fifty grams of radioactive tritium gas were released from a 200-foot stack on the 200,000-acre site. The "cloud" of gas moved northward into a rainstorm as it dispersed. Extensive testing by state and federal officials revealed virtually no radiation on the ground the next day, according to the AEC. Apparently, the gas had dispersed and diffused into the upper atmosphere, out of harm's way. The amount of radiation released was about half a million curies. Tritium has a half life of 12.2 years.

While the AEC reported that the local release caused no harm, it did add radiation to the environment. The dangers of this were highlighted in a letter by Dr. Ernest Sternglass of the University of Pittsburgh School of Medicine to *The New York Times* on May 23.

"Recent experimental studies of the effects of very low doses of radiation on human cell membranes now indicate that the small releases of radioactive gases and liquids in the course of normal operations appear to produce biological damage at a rate thousands of times greater than had been expected on the basis of our experience with medical X-Rays."

Dr. Sternglass cited experiments by Dr. A. Petkau at the Canadian Atomic Energy Laboratories in Manitoba and by Dr. E.G. Scott at the University of California Medical Center in San Francisco. Dr. Petkau confirmed that the more protracted the exposure, the smaller dose of radiation is needed to damage the membranes of living cells. Dr. Scott's experiments "were able to detect an increase in the leakage rate of blood-cell membranes at only 0.008 rads per month, comparable to the dose received from natural background radiation previously believed to be biologically very nearly harmless."

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San Luis Express 11-8-74

Workers' bodies are radioactive

DENVER, Colo. (AP) — Radioactive plutonium, a dangerous substance linked to cancer, has been detected in the bodies of 171 present and former employes at the Rocky Flats nuclear weapons plant.

The figure, compiled by the Dow Chemical Co., represents an increase from 105 in March 1972.

The figures indicate that eight present employes and six former workers show more plutonium in their lungs than is considered permissible, and 12 others have more than the permissible body burden, based on urine analysis.

Dow, which operates the plant for the Atomic Energy Commission, said the overall figure of 171 is for employes showing a measurable amount of plutonium in their lungs.

The plant employs 2,940 persons altogether.

Plutonium, used at Rocky Flats in the manufacture of components for nuclear weapons, is known to cause cancer in persons who inhale large amounts, and a study is being conducted of the effect on plant workers.

Some 178 Rocky Flats employes have already signed contracts permitting autopsies to aid the study.

The plant's medical director says there have been no deaths traced to the exposures so far.

(Two articles were also submitted from the New York Times but are not printed here because copyright releases were not obtained. They are:

1. "2 Senators Warn on Plutonium as Fuel" by Anthony Ripley, 9-28-74, and
2. "Plutonium Found in Plants' Roots" by Walter Sullivan 10-11-74.)

"Transient" Nuclear Workers: A Special Case for Standards

(BY R. Gillette)

Buffalo, New York. For the Buffalo area's unemployed laborers, for the moonlighters, college students, and the young men recruited from small farming towns south of the city, the guarantee of half a day's pay for a few minutes' work was an offer they couldn't refuse. Attracted by the prospect of easy money, they flocked by the hundreds to the Nuclear Fuel Services company between 1966 and the middle of 1972 to perform some of the dirtiest jobs in what one official of the Atomic Energy Commission (AEC) calls "the dirty end of the nuclear business."

The business of Nuclear Fuel Services (NFS) is the chemical extraction of uranium and plutonium from the highly radioactive spent fuel rods of nuclear power reactors. Situated in pastoral, wooded hills 40 miles south of Buffalo, the chemical plant was the nation's first commercial fuel processing facility. Although the technology it used was far from experimental, the NFS plant proved less than a smashing technical success. Almost from the time it opened in 1966 until it ceased operating in June of 1972 (for a major repair and enlargement program to be finished in 1977) the plant suffered repeated breakdowns and leaks of radioactivity. To clean things up and make repairs, the company relied heavily on the Buffalo area's abundant labor pool.

During 5½ years of operation, according to correspondence between NFS and the AEC, the company each year hired an average of 1400 "supplemental" workers from surrounding communities, making up a temporary, continually changing work force that outnumbered the plant's permanent, trained operating staff by more than 10 to 1. With an apparent minimum of instruction in safety procedures and the potential hazards of their jobs, the supplemental men were put to work decontaminating equipment and working areas, burying low-level nuclear waste, and repairing radioactive equipment.

Some of these workers were as young as 18 and others are alleged to have been recruited from bars for an afternoon's work. Some would last a week or more on the job. Others reached legal exposure limits within minutes and were promptly paid off—half a day's pay (at around $3 an hour)—and replaced, in the derisive phrase of a former full-time employee, by "fresh bodies."

On the average, according to AEC inspection reports, the plant's temporary workers received a whole-body radiation dose of 1.73 to 2 rems, an amount not considered harmful, but the equivalent nevertheless of five chest x-rays. This is less than the maximum the AEC allows for full-time radiation workers but much more than the industrywide average of 0.2 rem per year and more than the 0.5 rem allowed for members of the general public.*

The temporary workers, like the plant's permanent staff, also were exposed to small airborne concentrations of plutonium and other radioactive fission products whose hazards are under debate (*Science*, 20 and 27 September).

At one time the plant and its radioactive effluents were the focus of environmental protests, but these objections largely subsided, first as waste treatment improved and later when the plant closed. The company's public relations efforts have generally been effective, and a predominantly blue-collar region now seems to regard NFS as a welcome source of jobs. Local opposition to a planned tripling of the plant's capacity thus have been limited to a handful of conservationists and a few families whose sons worked at the plant. It is expected to reopen in about 3 years, at which time, AEC officials say, the plant will be much cleaner. If it isn't, one official adds, "we're in trouble."

Dormant as it is right now, the NFS plant provides a particularly vivid example of a common and long-standing practice in the nuclear industry. The AEC has long condoned the use of

* Federal radiation protection guidelines in force since 1960 recommend that individuals in the general population receive no more than 0.5 rem per year of nonmedical radiation to the whole body. Nuclear workers are limited to 5 rems per year, but the guidelines allow a worker to accumulate unused exposure according to the formula $5(n - 18)$ where n is his age. The worker may draw on his "body bank" at a rate up to 3 rems per quarter or 12 rems per year.

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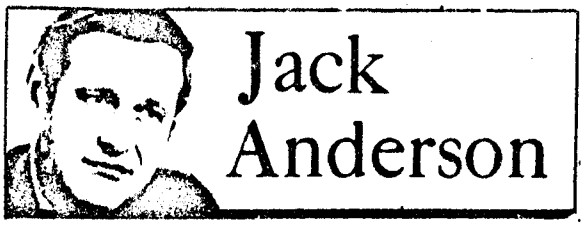
THE CITIZEN REGISTER

Ossining, N.Y., Fri., July 12, 1974



WASHINGTON —

CONTAMINATED YOUTHS

A nuclear plant run by billionaire J. Paul Getty has h red youths of 18 and older, worked them in radiation areas, and then allegedly cut them loose in two days after they got radiated.

In some cases, environmentalists charge, the young people were never given forms telling them how much radiation they got, as required by the Atomic Energy Commission.

The AEC is now holding hearings on a request by Getty's Nuclear Fuel Services to expand the plant, which has been out of major operation since 1972. At present, the sprawling facility at West Valley, N.Y., is doing mainly waste burial, decontamination and storage work.

Already, 1,300 residents of the West Valley area have petitioned against the expansion, according to the antinuclear National Interveners. The local Sierra Club's energy chief, physicist Dr. Marvin Resnikov, is collecting affidavits from some of the young people who worked in the Getty plant.

RESNIKOV HAS already produced a horror gallery of the plant inspection reports showing workers skin accidentally punctured by discarded plutonium needles, a worker whose head was so contaminated it left radiation on his pillow, and other incidents.

Spokesmen for the company insist that the plant is well run and safe, that the doses received by workers were well within the allowable lifetime range for radiation set by the AEC.

Since its reprocessing shutdown for expansion in 1972, the spokesman said, the plant has had virtually no exposure problems, which previously were minimal, they insisted. The hiring of young people is done through a contractor, they explained, and all get close radiation monitoring.

"Reprinted by permission of United Feature Syndicate"

"Enough to kill us all," says Huver

by Lloyd Nelson

"There is more than enough nuclear waste stored now to kill every individual in the world," said Charles W. Huver, associate professor of the University of Minnesota. He lectured on the ecological environmental effects of power plants on Wednesday, Jan. 23, at the Collins Classroom Center.

Huver has done special research on radiation physics and presented testimony at congressional and state hearings on environmental effects of power plants.

Nuclear wastes are from one million to one billion times more toxic than poisons such as cyanide on a per weight basis, he said. At present there are 90 million gallons of waste on hand.

Huver countered Atomic Energy Commission (AEC) claims that these wastes are being converted to salts by saying that only 1 per cent of these nuclear wastes have been successfully treated.

Wastes have even been released into open waters, he said.

According to Huver, the most important question is what should be done with radioactive waste materials. The costs alone are very high. At present it is costing between two to three million dollars for waste storage.

After World War II we felt a moral need to develop a 'good' use for atomic power, according to Huver. This need was felt as a result of guilt feelings after dropping nuclear bombs on Japan. It was a means of atonement, Huver said.

Because of these feelings we rushed into nuclear power production without proper regard to the safety aspects. Nuclear power was at first viewed as a savior, he said.

Of the first generation of nuclear power plants, seven or eight have been declared failures and have either been termed inoperative or have been closed for safety reasons, said Huver.

Huver went on to say that there are still many unanswered safety problems and what we are left with is a number of "dead white elephants."

If a major accident occured at a plant such as the one proposed at Rudolph, Wis., immediate damage would amount to $7 million property damage and a human life loss of about 3,400. These are old figures and have been updated to even higher numbers, he said.

Huver said that even at present, nuclear contaminants have been linked with genetic mutations and occurances of stillborn babies.

Although nuclear power appears to be the answer to our present energy crisis, it is not the savior that it seems to be, commented Huver. Energy consumption in the building and operating of these plants should be considered, Huver said. As of 1970, 10 times the energy was consumed in the building and operation of nuclear power plants than all that had been produced, he added.

Nuclear power production is dependent on fossil fuels. We are therefore burrowing deeper into our already declining fuel reserves, Huver stated.

According to Huver, it is not only health hazards that present a problem. National security problems also arise. He said that extremist groups may sabotage nuclear reactor plants and-or use them as scare tactics to their advantage.

Huver cited a case that arose in Argentina in which a guerilla group machine-gunned a nuclear reactor plant. Luckily the plant was inoperative and the storage tanks empty, he said.

THE POINTER January 31, 1974

(Reprinted by permission of The Pointer.)

The Cleveland Press, Thursday, May 23, 1974 (Reprinted by permission of the Cleveland Press)

AEC probing radioactive water in Erie Canal near Dayton

By RICHARD GIBEAU
Press Ohio Bureau

DAYTON — Bob Wainwright, the Atomic Energy Commission's manager for Monsanto's Mound Laboratory in Miamisburg, has a problem, and it's highly radioactive.

It's called plutonium-238, a deadly man-made radioactive element.

A concentration of the hot plutonium particles, which emit Alpha rays, has been found in the deceptively peaceful-looking Old Erie Canal and three small ponds below the hilltop atomic production plant operated by the Monsanto Research Corp. It is about 10 miles from Dayton.

The problem is compounded by the possibility that the plutonium may have been flowing into the canal and ponds for 16 years through a pipe that was discovered only recently.

Wainwright's job is to find out how much of the plutonium has accumulated in the silt and mud at the bottoms of the canal and ponds, and then to determine what must be done with the material.

The plutonium-238, which has been used in Mound Laboratory's production since 1958, is not supposed

to be in the canal and pond beds.

It was only in recent weeks that Monsanto's environmental monitoring personnel discovered that what they described as the plant's "effluent stream" was being diverted by the pipe from its intended patthway to the Great Miami River.

Samples were taken of the silt content, including some obtained by driving a pipe four feet deep into the sediment.

"We got something of a surprise, something we didn't anticipate," Wainwright said.

Wainwright refuses, however, as do others at the Mound Laboratory, to be specific about the plutonium concentrations found, emphasizing that a "very limited number" of random samples was taken.

"In the bottom, the levels appear to be higher than in the water and vegetation," he said.

Because the follow-up investigation will be an intensive effort in the canal and pond area, owned by the city of Miamisburg, Wainwright said Mound Laboratory announced the findings to allay public suspicion about the activities to come.

The announcement, made last Tuesday, was couched in cautious language, describing the "small amount of plutonium-238" found as presenting "no health problem."

Even that admission was an unusual break in the tone of serene perfection in safety and engineering that characterizes Mound Laboratory's annual environmental reports.

A radiological chemist in Cincinnati, a recognized authority in the nuclear field, said, "It must be pretty big if they are making a special announcement."

Monsanto's announcement triggered a flurry of news reports across the country, including some containing the statement that plutonium 238 "can cause instantaneous death if inhaled."

Plutonium-238 is among the deadliest elements, but the degree of its effect is proportionate to the exposure. Microscopic quantities will not cause instanteous death.

Just three weeks ago, Mound Laboratory's 1973 monitoring report was distributed. It cited Mound's "effective systems for the containment of radioactive materials," and "on-site and off-site monitoring programs which verify the integrity of the control systems."

The case of the effluent stream gone astray deflates those claims.

Mound's monitoring program was fairly intensive, Wainwright said, but added, "We did not, however, sample the bottoms of these waterways."

Now, Mound and AEC face the task of determining how much of the plutonium-238 has settled below those waters in the last 16 years.

"We got something of a surprise, something we didn't anticipate."

An AEC operation safety officer in Washington speculated earlier in the week that tons of radioactive silt and mud may have to be excavated, sealed in containers and buried in a disposal site.

Plutonium-238 has a half life of about 86 years, which means half of it decays in that time. It is highly radioactive, the reason it has been used as a heat-producer in the auxiliary power systems for the Apollo space vehicles and for satellites.

But Wainwright refuses to speculate about what will have to be done to eliminate whatever radioactive material may lie in Miamisburg's ponds. They are planned as part of a future city park.

He also reserves for later consideration the question of whether the effluent stream might also be depositing hot plutonium in silt in a drainage ditch leading to the river, and in the bottom of the Great Miami itself.

His immediate task is to develop "a basic outline of how to comprehensively sample the whole area so we can have a statistical base . . . to define the problem, if there is a problem, and then decide what we're going to do," he said.

Wainwright took that plan this week to AEC's operations office in Albuquerque, headquarters for its atomic weapons complex for approval.

He estimates the investi**gation of the canal and pond sediment will get underway next Friday, with sampling being don** / Mound personnel.

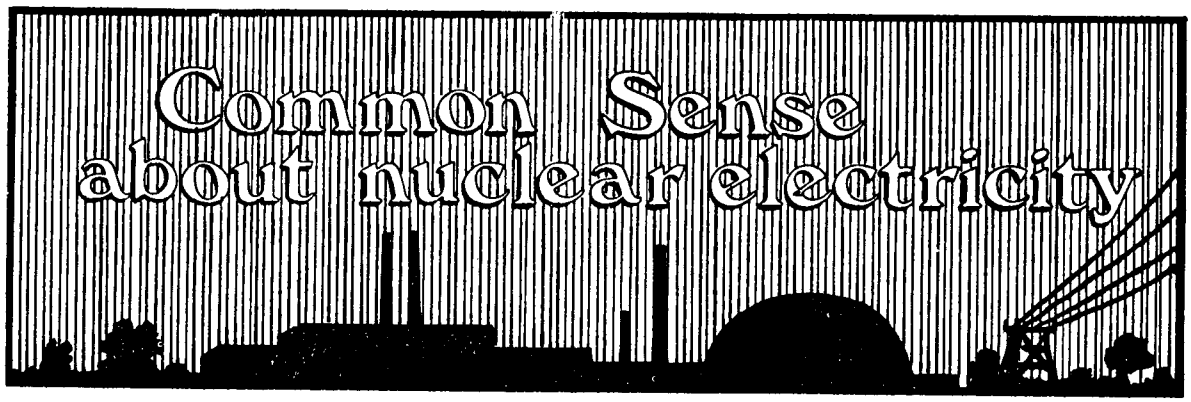
Other agencies will be involved in addition to AEC, including the U.S. Environmental Protection Agency.

Gary Bramble, environmental evaluation group chief in the Dayton office of the Ohio EPA, said the state agency also intends to be involved.

"We want to be on hand when they sample and we want to take our own samples and obtain an independent analysis," he s

Amid all the furor about the plutonium threat in the midst of his future park for peaceful Miamisburg, that city's manager, John Laney, refused to be dismayed.

"We don't feel the least bit concerned about our park development. We expect it to proceed," he said.



* **It's** time to listen to your own common sense when you hear the claim that nuclear electric power will be "clean" and "safe", or the odds on a catastrophic nuclear power accident are "one-in-a-billion".

* **In** order to keep those important promises – when nuclear plants will produce as much radioactivity as *a million Hiroshima atomic explosions* every year – the nuclear industry will have to contain its radioactive poisons with *better than* 99.99% success. Total poisoning of the planet is a certainty if just 1% of the long-term radioactivity escapes into the environment. There is no disagreement over that.

* **No** one denies the *requirement* for perfect performance in the nuclear power industry. The argument is over the likelihood of *meeting* the requirement and *keeping* the promises.

1. How many industries come *close* to 99.99% perfection in performance; Or even 99%?
2. What about mistakes and carelessness *already* appearing in the civilian nuclear program?
3. What about the performance record with nuclear *submarines*? (Please see inside page.)

Some Mistakes Already

"Review of the operating history associated with 30 operating nuclear reactors indicated that during the period 1/1/72 to 5/30/73 approximately 850 abnormal occurrences were reported to the Atomic Energy Commission (AEC). Many of the occurrences were significant and of a generic nature requiring follow-up investigations at other plants. Forty percent of the occurrences were traceable to some extent to design and/or fabrication-related deficiencies. The remaining incidents were caused by operator error, improper maintenance, inadequate erection control, administrative deficiencies, random failure and combination thereof . . .

"The large number of reactor incidents, coupled with the fact that many of them had real safety significance, were generic in nature, and were not identified during the normal design, fabrication, erection, and pre-operational testing phases, raises a serious question regarding the current review and inspection practices both on the part of the nuclear industry and the AEC." (Source: *AEC Task Force Report: Study of Reactor Licensing Process*, by AEC Asst. Director of Regulation, L. V. Gossick and 7 additional AEC experts, the *October* 1973 version; see also *Study of Quality Verification*, AEC, Jan. 1974, p15-17)

From years of living, YOU know that the very things which fine, intelligent people work hard to prevent often happen ANYWAY. Even statistically "impossible" events have occurred in YOUR lifetime.

WOULD YOU BELIEVE . . .

The *Guinness Book of World Records* lists park ranger Roy Sullivan as the "only living man to be struck by lightning four times". On August 7, 1973, he stepped out of his truck and was zapped for the *fifth* time; he suffered second-degree burns.

"The bolt struck me right on the head, set my hair on fire, traveled down my left arm and leg, knocking off my shoe but not untying the lace," he explained to the Associated Press in Waynesboro, Virginia.

Sullivan was first hit in 1942, when a bolt clipped off a toenail. In 1969, lightning burned off his eyebrows and knocked him unconscious. In 1970, lightning struck and burned his shoulders. In 1972, another bolt burned off his hair.

(*Washington Post*, August 27, 1973)

Short Amazing Stories

1 NOT VERY LIKELY: What are the odds that, in an 8-vehicle collision on Los Angeles' Golden State Freeway, *four* of the vehicles will be fuel trucks? It happened, on January 8, 1974 . . . and in the middle of a fuel-shortage.

2 THEN THERE WERE TWO: "With what one observer called a 'tremendous cracking sound', a 9-month-old, $12-million tanker operated by the Ingram Corp. split down the middle and sank in Port Jefferson Harbor on New York's Long Island. The 620-foot ship, which had cruised unscathed through two hurricanes, had already unloaded its cargo of 6 million gallons of gasoline and fuel oil, and authorities suggested that the tanker might have been under unusual stress amidships from improper ballasting." (*Newsweek*, 1/24/72) The odds? Human error somewhere?

3 DESIGNED TO PRODUCE A BETTER PRODUCT: After several months of investigation and 300 controlled tests, the Campbell Soup Co. discovered how lethal bacteria got into some chicken vegetable soup in the summer of 1971. On Nov. 20, 1971, Campbell's president, W. B. Murphy, announced that the botulism occurred by "several unusual conditions happening simultaneously — above average viscosity of the can contents, over-fill of the can, and incomplete hydration of the dry ingredients, coupled with a new process designed to produce a better product."

Said Dr. Willis Irvin, USDA inspector at the Campbell plant, "We have our men spread pretty thin. . . . It's a very large and complex plant, but I doubt if having another inspector would have prevented this. . . . It was a combination of very obscure things that no human being would have picked up." (*Washington Evening Star*, 11/20/71)

4 COULD NEVER HAPPEN TO US: Remember the hapless bank clerk in Idaho who inadvertently shredded 8,000 *un*processed checks worth an estimated $850,000? The bank president appeared on national news saying, "We never dreamed it could happen to us," while a team of clerks were seen in the background trying to fit the right pieces back together with scotch tape. (From the CBS evening news, 7/21/71)

5 THIS IS AN EMERGENCY ACTION: If there is one system whose credibility must be protected at all cost, it is the Army's system to warn the nation of a military emergency. Nevertheless, in February 1971, the Army's National Warning Center sent out this fully authenticated national emergency alert instead of the routine test-message: MESSAGE AUTHENTICATOR, HATEFULNESS - HATEFULNESS. THIS IS AN EMERGENCY ACTION NOTIFICATION DIRECTED BY THE PRESIDENT. NORMAL BROADCASTING WILL CEASE IMMEDIATELY. Scores of radio and TV stations broadcast the emergency.

It took the Associated Press and United Press International only 10 minutes to find out the alert was a dud, but it took the Army 37 minutes to cancel it. The first cancellation message had no authenticator — the necessary code-word. The second cancellation message had the *wrong* authenticator. On the third try, the Army got the right code-word: IMPISH - IMPISH, and officially called off the national "emergency", which was nothing more than one human who had selected the wrong pre-punched teletype tape.

6 TENDER LOVING CARE: Although thousands of Americans, from people soldering circuits to computer programmers, felt personally responsible for the safety and success of the Apollo astronauts, two out of seventeen Apollo missions failed not from outer-space surprises, but from simple human errors (1967, 1970).

7 WIDE OPEN SPACES: Although pilots try harder than anything not to run into other airplanes, two airliners traveling in the same direction collided in the mid-morning skies over the Grand Canyon in July 1956. The odds? Pilots also try to avoid well-known buildings. On an August morning in 1945, a bomber flying *under* a 900-ft. ceiling, flew smack into the Empire State Building.

8 A PERFECT RECORD . . . UNTIL: Leon Moisseiff, the famous engineer who designed and built the Manhattan, Triborough, and George Washington bridges, also built a giant suspension bridge across the Tacoma Narrows. The bridge collapsed 4 months after completion. "Moisseiff said only that the bridge failed because engineers do not yet know enough about aerodynamics." (*Time* magazine, 11/18/40)

THE MOLASSES TIDAL WAVE: In all of human history, there may have been only one killer tidal-wave made of molasses. On Jan. 15, 1919, an "incredible" 15-foot wave of molasses killed 21 people and demolished a block area of Boston immediately after the rivets began popping from a tank holding 2.2 million gallons of the sticky goo. Damage was $2 million. (*Boston Evening Globe*, 8/12/71) Even the AEC might have said, when the 21 victims were born, that the odds were EXCEEDINGLY REMOTE that they would die in a molasses tidal-wave. But that is how they died. Many more died in 1912 when, in defiance of all mathematical probabilities, the "unsinkable" Titanic sank on her maiden voyage.

Nuclear Submarines

The captains of our nuclear submarines are key-pins in our policy of deterring nuclear war, and surely they are more alert than the average man-in-the-street. You would expect them to know when a freighter is right on top of them. Nevertheless, on Oct. 6, 1972, one of our nuclear submarines – the USS TULLIBEE – collided with the West German merchant ship HAGEN 150 miles off the coast of North Carolina. "First reports indicate the collision involved a glancing blow on the upper part of the submarine bow with no internal damage. At that time, the TULLIBEE apparently was operating just below the surface. . . ." (Release issued in Norfolk, Virginia, by the Commander in Chief, Atlantic.)

Nuclear submarines, which are supposed to survive the problems of wartime attack, are not supposed to sink under peacetime conditions. However, in 1963, the USS THRESHER vanished and was found at the bottom of the sea. The causes: faulty pipe joints and an inadequate deballasting system, according to the Navy. In 1968, the USS SCORPION also went down forever. And imagine the Navy's dismay when a nuclear submarine sank at *dockside* in May 1969. Fortunately, the nuclear power plant had not yet been installed in the USS GUITTARO when it suddenly flooded and sank at the Mare Island Shipyard in California. The odds?

As for the submarine *missiles*, in August 1973, Rear Admiral Levering Smith acknowledged that 58% of the Poseidon missiles on nuclear submarines had failed their operational tests, and that "essentially all of the missiles" would have to be recalled.

Nuclear submarines have even sent false alarms about attack. On Jan. 16, 1974, the United Press carried this story: "Emergency transmitters on Polaris submarines mistakenly signaled they had been 'sunk by enemy action' on two occasions in 1971, and raised the threat of accidental nuclear war, Rep. Les Aspin (D-Wis) said yesterday. The signals set off general military alerts until the subs themselves surfaced and advised by regular radio signals that the buoy-borne emergency transmitters had malfunctioned, Aspin said. . . . The Navy confirmed that the two incidents took place. . . . Spokesmen declined direct comment, however, on whether alerts resulted."

THE ACCIDENT RECORD IN THE NUCLEAR SUBMARINE PROGRAM IS A REAL-LIFE WARNING ABOUT WHAT WE MUST EXPECT IN THE CIVILIAN NUCLEAR POWER PROGRAM. When 2 out of 125 operating nuclear submarines have completely failed to perform (just stay afloat) during peacetime, what are the odds that *no* civilian nuclear power plant will *ever* experience a disastrous failure to contain its radioactivity?

THE ANSWER: As of January 1, 1974, there were 42 nuclear reactor plants licensed to operate, 56 under construction, and 101 on order (planned). The AEC plans to license 280 for operation by 1985, and 1000 by the year 2000 (average: 20 in every state). *If* we permit a thousand plants to operate, and *if* the probability of a major accident were really as low as one-in-a-million per reactor per year, then the probability of a major accident during the 40-year lifespan of the plants would be about one-chance-in-25.

Committee for Nuclear Responsibility, Inc. P.O. Box 2329, Dublin, California 94566

Lenore Marshall
Founder (1899-1971)

Richard E. Bellman, Ramsey Clark, John T. Edsall, Paul R. Ehrlich, John W. Gofman, Charles E. Goodell, David R. Inglis, Richard Max McCarthy, Ian McHarg, Lewis Mumford, Linus Pauling, Harold Urey, George Wald, James D. Watson

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One-Chance-in-a-Billion

Several stories inside this flyer show that statistically "impossible" events happen frequently, and that extremely complex systems like moon-rockets (or nuclear power plants) can be destroyed by something as common as faulty wiring or valves, a bad welding job, or a person simply doing the wrong thing. Recently, the AEC paid professors at M.I.T. two million tax-dollars to estimate the probability of a nuclear power catastrophe. The report, which is known as "the Rasmussen study," provides the AEC with figures like one-chance-in-a-billion per plant per year, according to the AEC.

Such Figures Have No Meaning

FIRST REASON is the difficulty of predicting either the frequency or the consequences of human error (and malice). Error or malice could instantly reduce the catastrophe-odds from one-per-billion to *near certainty*. Estimates about the small chance of a nuclear disaster depend on the reckless assumption that *operators* of nuclear plants will make no serious errors during emergencies; also, that no demented or hostile people will try to destroy the plants.

SECOND REASON is the lack of experience with operating nuclear hardware. Since the very first 1,000-megawatt nuclear plant went into operation in *June, 1973*, experts have hardly one reactor-year of experience to examine. They can do little better than guess when they assign reliability estimates to nuclear hardware of this type. Furthermore, for 4 years in a row, the AEC has had to scold and to fine nuclear equipment firms, engineering firms, and utilities for *unacceptably sloppy quality-control*, but according to a report in the *Los Angeles Times*, Dec. 26, 1973, the industry is *still* unresponsive.

THIRD REASON is the unjustifiable assumption that nuclear safety-systems (some of them never tested) have been properly designed. This assumption denies all the recent nuclear "surprises" which show that nuclear engineers are *failing* to foresee all the design problems. If the *design* of a safety-system is defective, even perfectly working hardware will not make it effective.

FOURTH REASON is the flaw of assuming that all possible paths leading to a catastrophe have been recognized and considered. As recently as October 1973, the AEC's Director of Regulation, L. Manning Muntzing, admitted to a Congressional Committee (JCAE): "I'm really concerned about some of the surprises we see." How many *un*suspected paths to catastrophe are still waiting to be *discovered?*

HUMAN ERROR

Reactor Operating Experiences in the journal "Nuclear Safety" show over and over that a human decision is required on a number of occasions.

When there are enough reactors operating, the total number of occasions requiring a human judgment will increase appreciably.

Let's play with some numbers which I think grossly minimize the danger to be faced.

Suppose you have 500 nuclear power reactors in operation. Let's further suppose that a serious human decision is required once in ten years for each reactor; a study of the literature might show that the frequency is far greater. This means 50 serious decisions requiring human judgment per year.

Suppose further that the judgments are 98 percent right and 2 percent wrong, which is a very optimistic estimate.

This finally gives us one reactor per year suffering the consequences of wrong judgment on a serious matter. Clearly if the judgmental error results in a nuclear melt-down, that's a disastrous event. Even less than melt-down accidents could be very, very serious.

The requirement for a human judgment is a source of immense weakness in nuclear safety. Even if all other safety problems could be solved, the human factor all by itself means that nuclear power makes no sense.

John W. Gofman, Ph.D., M.D.

SOME NUCLEAR 'SURPRISES'

Discovery in 1972 that nuclear engineering firms have built the Prairie Island and Kewaunee plants with steam lines running underneath the control rooms, where a rupture of a line could destroy the controls and kill the nuclear plant operators; extensive modifications will be required in about six plants.

Unexpected densification of nuclear fuel, one of the most tested elements of the whole nuclear power system; this discovery forced the AEC to cut back permissible power levels by 5 to 25 percent at 10 nuclear power plants on August 24, 1973.

Failure of the vital emergency core cooling system to provide AEC experts with assurance of effective performance; the system, which has never had a large-scale test, failed six out of six miniscale tests in late 1970.

As of Spring 1974, the emergency cooling system has never had a successful large-scale test.

SURPRISE! SURPRISE!

Discovery in 1971 that the allegedly watertight salt mine chosen for radioactive waste storage in Kansas was full of holes; the AEC has been forced to improvise "surface storage" plans.

Confirmation by the National Academy of Sciences in November 1972 that low-level radiation exposure is at least 500 percent more harmful than the experts had previously admitted; this surprise had already forced the AEC to suggest drastically reduced "permissible emissions" from nuclear power plants.

Discovery by the North Anna Environmental Coalition in August 1973 that two nuclear power plants in Virginia have been built on an earthquake fault in undeniable violation of AEC policy.

Apparently nuclear experts did not foresee, either, that on November 11, 1972, three skyjackers would threaten to bomb the nuclear reactor at Oak Ridge, Tenn.; helpless, the AEC shut down its reactor and evacuated. The skyjackers did not carry out their threat.

DO *YOU* WANT NUCLEAR POWER? **Consult Your Common Sense.**

Jan. 3 1975
15 Westminister Rd.
Summit, N.J. 07901

Dr. William A. Mills
Director of Criteria and Standards Division (AW 560)
Office of Radiation Program
Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

I ask that the attached article The Hazards of Plutonium by J.G. Speth be read and made part of the Hearings on environmental plutonium standards which are being held by the EPA in Denver, on Jan. 10, 1975.

In place of an insane plutonium power source our government should crash program wind generator development, using the best engineering minds and all needed investment capital. 150 miles of wind generators could produce 60% of New Jersey's electric needs estimates Heronemus of U. of Mass., Amherst Mass. Let us stop paying tribute to OPEC. Once wind generator prototypes are built, the price of OPEC oil will fall. The development of the fly wheel for power storage and automotive power would also free us from dependence on dwindling natural resources.

Money for such development can become available if we stop the production of useless atomic weapons, and inefficient atomic power plants.

Sincerely
Frances Tyson
Frances Tyson

100% recycled paper earth color

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The Hazards of Plutonium

by J. Gustave Speth

From Natural History Magazine - by
Amer. Museum of Natural History
Central Park West at 79th Street
New York - N.Y. 10024

The recycling of this element—the stuff of nuclear bombs and one of the most toxic substances known—is highly controversial

The Atomic Energy Commission, if unchecked, is about to sow the seeds of a national crisis. The commission proposes to launch what it calls the "plutonium economy," which would authorize the nuclear power industry to use recycled plutonium as fuel in commercial nuclear reactors around the country. The result of such a decision would be the creation of a large civilian plutonium industry and a dramatic escalation in the risks posed by nuclear power.

Plutonium barely exists in nature: the entire present-day inventory is man-made, produced in nuclear reactors. Plutonium-239, the principal isotope of this element, has a half-life of 24,000 years; hence its radioactivity is undiminished within human time scales. That isotope is one of the most toxic substances known. One millionth of a gram has been shown capable of producing cancer in animals. Plutonium-239 is also the material from which nuclear weapons are made. An amount the size of a softball is enough for the production of a nuclear explosive capable of mass destruction. Scientists now widely recognize that the design and manufacture of a crude nuclear explosive is no longer a technically difficult task; the only real obstacle is the availability of plutonium itself.

We believe that the commercialization of plutonium will place an intolerable strain on our society and its institutions. Our nuclear technology has presented us with a possible new fuel that we are asked to accept because of its potential commercial value. But in our opinion, technology has outstripped our institutions, which are not prepared or suited to deal with plutonium. And those of us who have asked what changes in our institutions will be necessary to accommodate plutonium have come away from the inquiry profoundly concerned.

The AEC's recently released draft environmental impact statement assessing the effects of recycling plutonium reinforces these concerns. It concedes that the problems of plutonium toxicity and nuclear theft are far from solved and indicates that they may not be for some years. Nevertheless, the statement concludes that we should proceed. The AEC decision, whether it stems from blind faith in the beneficence of the technology the commission has fostered or from a callous promotion of the bureaucratic and industrial interests of the nuclear power complex, cannot be justified in light of what we know and, just as important, what we do not know about the implications of a plutonium industry.

The fuel used in today's nuclear reactors—light-water reactors, or LWRs—is uranium that has been enriched so that its uranium-235 content is increased from 0.7 percent, the amount present in natural uranium, to about 2 to 4 percent. Uranium-235 is a fissionable isotope of uranium. The remainder of the fuel is nonfissile uranium-238. Unlike plutonium, this uranium fuel is not extremely toxic and not sufficiently rich in uranium-235 to be fashioned into nuclear weapons. When the LWRs are in operation, however, they also produce as a by-product moderate amounts of plutonium, principally plutonium-239. A typical large reactor produces about 200 to 250 kilograms of plutonium isotopes each year. Since much of this plutonium is easily fissioned, it can be used as reactor fuel. Plutonium recycle is the nuclear industry-AEC proposal to recover the fissionable plutonium produced in LWRs, process it, and recycle it as fuel back into LWRs.

Several critical steps would be involved in recycling this plutonium. First, the used, or spent, fuel from the reactor must be shipped to a fuel-reprocessing plant where the plutonium would be removed.
It would then be shipped to fuel-fabricating and assembly plants for the next fuel-cycle stages. At the fabricating plants the plutonium oxide would be mixed with uranium to form fuel pellets; the pellets would be placed in fuel rods and the rods would be collected into fuel assemblies. These assemblies would then be sent to the reactors for use, thus completing the fuel cycle.

Plutonium recycle has not yet begun, and so far there is no major industrial commitment of resources

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This article is adapted from "The Plutonium Decision: A Report on the Risks of Plutonium Recycle," written in September, 1974, by J. Gustave Speth and physicists Arthur R. Tamplin and Thomas B. Cochran for the Natural Resources Defense Council, Inc.

to it. No major commercial plutonium fuel fabricating plants are in operation or under construction, and the three nuclear-fuel-reprocessing plants that have already been built do not represent a substantial investment in national terms. They may be needed, in any case, to prepare spent fuel for long-term storage. But if the plans of the AEC and the nuclear industry are carried out, a major plutonium industry will quickly develop. Such an industry could recover some 140 tons of plutonium from commercial reactors by 1985 and 1,700 tons by the year 2000. By the turn of the century the industry could involve hundreds of LWRs fueled with plutonium, perhaps a score of fuel-reprocessing and -fabricating plants, and thousands of interstate and international shipments containing hundreds of tons of plutonium.

The most pernicious product of the nuclear industry is plutonium. Microgram quantities in skin wounds cause cancer in experimental animals. Inside the body, plutonium is a bone seeker; once deposited there it can cause bone cancer. But plutonium is most dangerous when inhaled. In a recent article in the *Bulletin of the Atomic Scientists,* Donald Geesaman, a biophysicist formerly with the AEC and currently at the School of Public Affairs of the University of Minnesota, explains this hazard:

> Under a number of probable conditions plutonium forms aerosols of micron-sized particulates . . . if inhaled they are preferentially deposited in the deep lung tissue, where their long residence time and high alpha activity can result in a locally intense tissue exposure. The lung cancer risk associated with these radiologically unique aerosols is unknown to orders of magnitude . . . [but] under present standards, the permissible air concentrations are about one part per million billion . . . a commentary on plutonium's potential as a pollutant.

To determine the adequacy of the radiation protection standards for plutonium enforced by the AEC, two of the authors of the report from which this article was adapted, physicists Arthur R. Tamplin and Thomas B. Cochran, undertook a review of the biological evidence for the Natural Resources Defense Council. The conclusions of Tamplin and Cochran, found in their study "Radiation Standards for Hot Particles," are that insoluble plutonium particulates, or hot particles, are uniquely virulent carcinogens and that the current AEC radiation protection standards governing the amount of plutonium to which members of the public can be exposed are roughly 100,000 times too lax. The lung cancer risk associated with hot particles of plutonium, as estimated by Tamplin and Cochran, is comparable to the lethal dose of botulin toxin, a biological warfare agent. One would hope that this nation would give careful consideration to the risk involved and pursue all alternatives before implementing an energy policy based on such toxic materials.

Although the adequacy of present plutonium standards is a matter of considerable doubt and great controversy, the AEC's draft impact statement for plutonium recycle simply assumes that those standards are adequate. The entire risk analysis of the statement is based on the assumption that the use of plutonium, with plutonium recycle, is based on a premature and unexplained rejection of the hot particle hypothesis, which asserts that the intense local radiation emanating from minute hot particles is more carcinogenic than the same amount of radiation distributed over a larger tissue area. Yet despite the AEC's dismissal of this hypothesis in its impact statement, the commission concedes, in answer to a petition brought by the Natural Resources Defense Council in a separate proceeding, that the hypothesis "is being given careful consideration."

We submit that the AEC has no basis for concluding that plutonium recycle will not cause undue risk to the public health and safety until it has either satisfactorily resolved the doubts over current plutonium radiation protection standards or calculated the impacts of plutonium recycle on the assumption that hot particles are uniquely carcinogenic. The draft environmental impact statement for plutonium recycle does neither.

Some plutonium contamination of the environment has already occurred, principally as a result of the atomic weapons program. The leakage of plutonium from contaminated oil at the AEC's plutonium weapons plant at Rocky Flats, ten miles west of Denver, Colorado, in the late 1960s led to an uncontrolled source of plutonium that was much larger than the reported discharge from seventeen years of plant operation. Tens to hundreds of grams of plutonium went off site ten miles upwind from Denver.

The Nuclear Materials and Equipment Corporation, a facility in Apollo, Pennsylvania, that processes plutonium for energy research and development, was recently fined $13,720 for a sixteen-count violation of AEC regulations, ranging from failure to follow radiation monitoring procedures to failure to comply with certain safeguards requirements.

Production workers from the Nuclear Fuel Services facility in Erwin, Tennessee, which processes plutonium, met with AEC inspectors on August 13, 1974, to complain about the absence there of even the rudiments of accepted health physics practices. Occurrences such as these could multiply greatly if plutonium is made a major article of commerce.

As a fuel for power reactors, plutonium is expected to range in price from $3,000 to $15,000 per kilogram, roughly the equivalent of the street price of heroin. This same material might be hundreds of times more valuable to fanatics and desperadoes bent on obtaining power or wealth through the use of nuclear devices. A recent AEC study identified more than 400 incidents of international terrorism carried out by small groups during the past six years. In an age of bomb threats, aircraft hijackings, the kidnapping of diplomats, and the murder of Olympic athletes, the risks of nuclear theft, blackmail, and terrorism are not minimized even by some of the most ardent supporters of nuclear energy. Thus, Clarence Larson, former Atomic Energy Commissioner, has described the evolution of a plutonium black market:

> Once special nuclear material [plutonium] is successfully stolen in small and possibly

economically acceptable quantities, a supply-stimulated market for such illicit material is bound to develop. And such a market can surely be expected to grow once the source of supply has been identified. As the market grows, the number and size of thefts can be expected to grow with it.

The critical point here is that these tremendous risks will become real with the advent of plutonium recycle. Black marketeers or terrorists would find it too risky to steal plutonium that has not been made easier to handle by being separated from the highly penetrating radiation in the spent fuel of present-day reactors. Until irradiated fuel is reprocessed and the plutonium separated out, the possibilities of theft in the light-water reactor fuel cycle are accordingly minimal. But once the plutonium is reprocessed and recycled, the picture changes dramatically. Reasonable prudence, therefore, dictates that we have adequate answers to the problem of nuclear theft well in hand before plutonium recycling begins.

There is now widespread agreement, at least among those outside the nuclear industry, that present safeguards against plutonium theft are woefully inadequate. The AEC's own "Rosenbaum Report," an assessment of the adequacy of the agency's safeguards of special nuclear material made in 1974 by outside consultants, concluded:

> In recent years the factors which make safeguards [against plutonium theft] a real, imminent and vital issue have changed rapidly for the worse. Terrorist groups have increased their professional skills, intelligence networks, finances, and level of armaments throughout the world. . . . Not only do illicit nuclear weapons present a greater potential public hazard than the radiological dangers associated with power plant accidents, but . . . the relevant regulations are much less stringent.

The problem is not simply that the AEC has not implemented the necessary safeguards programs; rather the agency has not even developed an adequate program on paper.

On the subject of safeguards, the AEC's draft impact statement for plutonium recycle breezily concedes that the objective of keeping the risk of nuclear theft small "will not be fully met . . . by current safeguards measures." Among the several steps suggested by the AEC to correct inadequacies are the following: (1) Location of reprocessing and fabricating plants next to each other in order to minimize or eliminate the shipment of plutonium between them; (2) establishment of a new federal plutonium security police force to protect facilities and shipments; (3) creation of a sophisticated security clearance system for nuclear industry personnel.

These and other proposals are still under study; their content is not yet well defined. Some would necessitate substantial changes in the structure of the United States utility industry; others might require congressional action. And the initiation of a sophisticated safeguards program would pose a threat to civil liberties and personal privacy. Nevertheless the draft impact statement recommends that we proceed now with plutonium recycle because "the Commission has a high degree of confidence that through implementation of some combination of [its recommended steps] the safeguards general objective . . . can be met for plutonium recycle."

The commission's faith, unfortunately, is hardly reassuring. While it may be possible to devise an adequate safeguard system in theory, there is little reason to believe that such a system would be acceptable in practice. This is true for several reasons.

First, the problem is immense. The theft of plutonium is only one type of antisocial behavior a safeguards program must protect against. Terrorist acts against the reactors, fuel-reprocessing facilities, waste repositories, and shipments of radioactive wastes could result in serious releases of radioactivity. Moreover, a safeguards system would have to exist on a vast, worldwide basis. Some 1,000 nuclear reactors are projected for the United States by the year 2000, with hundreds of shipments of radioactive materials daily. Hundreds of tons of plutonium will be in the commercial sector of our economy by that date. Abroad, American firms are constructing nuclear reactors in countries that have little political stability and in others, such as Japan, that have not signed the nonproliferation treaty. Safeguarding nuclear bomb material would ultimately require a restructuring of sociopolitical institutions on a worldwide scale. The record of the United Nations unfortunately gives us little reason to believe that this is a practical reality.

Second, safeguards are strongly opposed by the nuclear industry. The degree to which the industry is sensitive to the hazards of theft, and likely to be an effective partner in the enforcement and implementation of safeguards programs; was apparent in its vociferous opposition to the modest strengthening of AEC safeguards, which was recently adopted.

Third, experience with present safeguards does not inspire confidence. Over several years of operation, the Nuclear Materials and Equipment Corporation was unable to account for 6 percent (100 kilograms) of the weapons-grade material it handled and, as already noted, was recently fined by the AEC, partly because of safeguards violations. At a recent safeguards symposium the director of the AEC's Office of Safeguards and Materials Management observed that "we have a long way to go to get into that happy land where one can measure scrap effluents, products, inputs, and discards to a 1 percent accuracy." This statement takes on particular significance when we realize that only 0.5 percent of the plutonium utilized by the commercial sector in the year 2000 would be enough to make hundreds of atomic bombs. The editors of the *Bulletin of the Atomic Scientists* have noted that the frequent misrouting of shipments of weapons-grade material highlights a key safeguards problem: hijacking. A spot check by investigators from the General Accounting Office at three AEC-licensed contractors showed that in some cases access to easily portable quantities of special nuclear material could be gained in less than a minute using the simplest of tools. At two of the three plants checked, the General Accounting Office found weak physical barriers, ineffective guard patrols, ineffective alarm systems, lack of automatic detection devices, and the absence of an "action plan" should material be stolen

or diverted. In contrast, the AEC's inspectors gave the same facilities good marks in virtually every security category.

Fourth, even if an effort is made to improve current safeguards, there is little reason to believe that the new system will operate with the virtual perfection that is essential. For example, the AEC supports the creation of a special federal police force to provide an immediate federal presence whenever the use of force may be needed to protect these incredibly dangerous materials from falling into the hands of would-be saboteurs and blackmailers. But is it plausible to believe that police would be effective at a level commensurate with the potential nuclear hazard? The New York City police department has shown itself incapable of maintaining security over confiscated heroin. Are similar losses of plutonium acceptable? The point is that our safeguards system must be essentially infallible; it must maintain what Alvin Weinberg, former director of the Oak Ridge National Laboratory, has called "unaccustomed vigilance" and "a continuing tradition of meticulous attention to detail." Yet our human institutions are far from infallible. Our experience indicates that rather than sustaining a high degree of esprit, vigilance, and meticulous attention to detail, our governmental bureaucracies instead become careless, rigid, defensive, and sometimes, corrupt. A basic question then is whether we want to entrust so demanding and unrelenting a technology as plutonium recycle to institutions that are often negligent of their own responsibilities and insensitive to the rights of others.

A final reason for believing that an adequate safeguards system would not be acceptable in practice is the tremendous social cost of such a system in terms of human freedom and privacy. Safeguards necessarily involve a large expansion of police powers. To deal with the terrorist threat, the AEC is considering surrounding us with what it calls a new "federal security system." In its draft environmental impact statement, the AEC decries court rulings "favorable to the protection of individual privacy" and calls for legislation to authorize expanded "background checks" and to create a federal plutonium police force. The plutonium police would meddle with our civil liberties on a vast scale. Surveillance would inevitably cover not only the millions seeking jobs in the plutonium economy but also those close to the job seekers and those in allied occupations.

The commercialization of plutonium will bring with it a major escalation of the risks now associated with nuclear power—risks that many already believe to be too great. And plutonium will further strain the weakened regulatory fabric of the nuclear industry.

Hannes Alfven, Nobel Laureate in Physics, has described the regulatory imperatives applicable to the nuclear industry in an article in the *Bulletin of the Atomic Scientists:*

> Fission energy is safe only if a number of critical devices work as they should, if a number of people in key positions follow all their instructions, if there is no sabotage, no hijacking of the transports, if no reactor fuel processing plant or reprocessing plant or repository anywhere in the world is situated in a region of riots or guerilla activity, and no revolution or war—even a "conventional one"—takes place in these regions. The enormous quantities of extremely dangerous material must not get into the hands of ignorant people or desperadoes. No acts of God can be permitted.

575

Writing in *Science* magazine, Alvin Weinberg suggests that "what is required is a cadre that, from now on, can be counted upon to understand nuclear technology, to control it, to prevent accidents, to prevent diversion" of plutonium to nonpeaceful purposes. The public and its decision makers must seriously question whether it will be possible to attract, train, and motivate the personnel required for these functions. These would have to be highly qualified persons able to maintain a tradition of "meticulous attention to detail" even when the glamorous aspects of a new technology become the commonplace operations of an established industry. We suggest that it is beyond human capability to develop such highly qualified groups generation after generation.

There is a sizable body of evidence at the present time to suggest that the fledgling nuclear industry is already unmanageable. Consider, for example, that 115,000 gallons of high-level radioactive wastes recently leaked from the tank at the AEC's reservation at Hanford, Washington, over a period of fifty-one days while no one monitored the tank. Radioactive releases from the Shippingport, Pennsylvania, reactor were higher than recorded. Executives of Consumers Power Corporation, a utility company in the Lake Michigan area, failed to notify the AEC that their radioactive gas holdup system was not functioning. Two reactors were half-completed before the AEC was informed that they were being constructed over an earthquake fault. The General Accounting Office found the security at plutonium storage areas totally inadequate after AEC inspectors had certified the facilities. Finally, one of the AEC's leading reactor safety experts recently quit in protest, stating that he was going to work with Ralph Nader and the Union of Concerned Scientists because the AEC has misled the public about the safety of nuclear reactors.

In view of this spotty record and considering the extreme toxicity of plutonium and the major difficulties of safeguarding it from theft, we believe that a decision at this time to proceed with plutonium recycle will escalate an already unmanageable situation to the crisis level.

An opportunity for a review of the decision has fortuitously appeared. In October of last year, President Ford signed a bill that splits the Atomic Energy Commission into two new agencies—one to regulate the nuclear industry, the other to develop energy technologies—both nuclear and nonnuclear. Critics of the AEC, which combined both functions under one roof, had long requested the government to take that action. The new regulatory agency is the Nuclear Regulatory Commission; the new promotional authority is the Energy Research and Development Administration. We cannot urge too strongly that upon assumption of office, the heads of both agencies take an independent and searching new look at the plutonium question before we start on such a risky and irrevocable course as plutonium recycle. □

R.R. 1
Carlock, Il. 61725
6 January 1975

Roger Strelow, Asst. Adm. for Air and Waste Mngt.
Office of Radiation Programs
U.S. Environmental Protection Agency
Washington, D.C. 20460

Dear Mr. Strelow:

I would like a transcript of the hearing and documents concerning Plutonium and Transuranium Elements.

In addition, I would like to comment that any plutonium produced is too much. Our radiation storage facilities have already proven to be inadequate (refer to Hanford, Wash.) plus incidents at on sight escapes to the environment have been recorded (refer to Miami R. in Ohio). Now I read that Plutomium is missing at a production unit of the Kerr-McGee Corporation in Crescent, Ok..(Wall Street Journal, 30 Dec. 1974).

Plutonium is among the most hazardous materials (potentially) known to man. Uncompromizing and only nothing short of perfect storage procedures must be devised which must last more than 500,000 years (approximately 20 half-lives). Human error, intentional sabotage plus unpredictable climatic and seismotic changes are too prevalent to take the risk of massive plutonium production for this great period of time. I believe the breeder reactor and plutonium production programs should cease immediately and direct our energy efforts toward perfecting solar power and deuterium fusion techniques.

Sincerely,

David N. Paddock

Jan. 9, 75

Mr. Carl Neiler

This letter is written in response to a notice in the Environmental Action Bulletin (Dec 28 74) that your office wishes to receive comments from concerned citizens regarding plutonium standards.

My opinion is based on a history of accidents, by those who handle radioactive materials, involving the release of hazardous radioactive materials to our air, water & food supplies; hazardous in that these materials are known to cause cancer, leukemia, birth defects, congenital defects etc.

I believe plutonium is one of the deadliest substances known and should not be used at all and that the standards for release should be set at zero especially for those who are in the

business of making money such as the Nuclear Power people.

I simply cannot condone the exchange of mere dollars for one more deformed baby, one more amputated limb or breast, or one more miserable human life.

Why must we bear the burden of Nuclear disturbance, genetic damage, hospital treatment expenses, surgical trauma, defective children etc. so that those in power can become even more powerful? Those people may be willing to take the risk to satisfy their greed for wealth & power but I am not.

Please — no more radioactive material of any kind. (I have to live with natural radiation however). Sincerely

R. H. Adolphson
927 Fillmore
Davenport, Iowa

WE SHOULD GO TO SOLAR OR OTHER FORMS OF POWER

(203) 693-4377

JUDITH C. FRIEDMAN
Lawton Road
Canton, Connecticut 06019

Dear Mr. Miller,

I am totally opposed to the use of plutonium in any amount for the purpose of nuclear energy.

My reasons are simple but very real and basic. The risk is too great because man is fallible.

Our new energy source

should be greatly increased

conservation, which is a concept

that has not even been touched

upon — seriously.

Thank you for considering

my opinion.

Sincerely,

Judith C. Friedman

ELOISE W. KAILIN, M. D.
RTE. 1 BOX 253
SEQUIM, WASHINGTON 98382

AREA CODE 206
TELEPHONE 683-6644

DIPLOMATE, AM. BD. OF
ALLERGY AND IMMUNOLOGY

1-10-75

Carl Miller
Director of Criteria and Standards Div.
Office of Radiation Protection, EPA
Washington, D.C. 20460

Dear Mr. Miller:

I can't attend your Denver hearing on Plutonium emissions from power plants but I can express my strong considered opinion that nothing less than a zero standard will be acceptable.

Please send me a copy of the EIS on this matter and a report on the hearings.

Eloise W Kailin M.D.

Dr. William A. Mills
Director of Criteria and Standards Division (AW-560)
Office of Radiation Program
EPA
Washington, D.C. 20460

January 10, 1975

Dear Dr. Mills:

I am writing to express my protest to the use of atomic reactors as a source of energy--plutonium being the deadly by-product of these reactors.

I have read a great deal of material on the subject, and feel the warnings made by authorities and scientists alike, warrant the immediate halting of further work and/or development on these reactors.

I am thoroughly disgusted with the people at the AEC, who know of these reactors' potential danger, and yet, refuse to believe the warnings of experts, preferring to sit on their hands instead.

These people are supposed to be working for us, and not against us. I believe the people at AEC are not earning their keep. They should be thrown out of their positions on their ears...unless they begin to take into account the safety of people and the future of the world, which is what they were hired to do.

It appears these people at AEC have run amuck and are under the illusion they are all-knowing and all-powerful. Need they be reminded they are working for the people of this country and that we are to be given the last word on what type of energy we will have? This one government agency is not to have the power of decision when there are so many risks involved.

Perhaps the AEC has not heard that there are several safe sources of energy available to us---solar, wind, ocean, and geothermal---to name a few.

If the AEC does not mind, I have made plans and am striving for a career. I would greatly appreciate having the opportunity to fulfill my hopes and live to a ripe old age--without the deadly danger of atomic reactors jeopordizing or thwarting my efforts.

Based on authoratative advice, stating that the use of atomic reactors could only mean further destruction of life in our already problem-riddled world, I demand that another source of energy be researched and used, so I and everyone else can live full lives.

For a change of pace, why don't the people serving us use a bit of foresight instead of the hindsight they are so very famous for?

Sincerely,

Theresa Ursulskis

Theresa Ursulskis

UNIVERSITY OF COLORADO
BOULDER, COLORADO 80302

DEPARTMENT OF SOCIOLOGY

AREA CODE 303
443-2211, EXT. 6427

January 13, 1975

W. D. Rowe
Criteria and Standards Division
Office of Radiation Programs (AW-560)
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Sirs,

I am submitting the enclosed report as part of written testimony for the EPA hearings on radiation standards held recently in Denver. It is an example of citizen research, one might say, since it was done by students at the University of Colorado. It reflects a growing concern on the part of Denver area citizens that the region be adequately protected from radiation risks from the Rocky Flats plant.

It may be that current standards are strict enough. I believe, however, that the EPA should be supportive of efforts of citizens to protect themselves through citizen inquiry, research and action. Any governmental organization such as the Atomic Energy Commission (AEC) should have counter-control organizations in the "citizen sector." Wise decisions are assured through such a checks-and-balance system.

I would hope that the EPA would concern itself with more than just radiation standards--and would encourage wherever possible citizen participation in projects that further their knowledge of the risks involved in nuclear energy development, and alternatives for protecting the health and welfare of their communities.

Sincerely,

Paul Wehr
Associate Professor
of Sociology

PW/gl

Enclosure

1.

This report is the product of the Action Research Group, one of the several student working parties in the Sociology of Peace-making course at the University of Colorado. Its research grew out of two convictions: 1.) that students are capable of and prefer to do social research on problems that seriously effect them, their society and their world; and 2.) that students can simultaneously learn the skills of research and contribute to the amelioration of a significant regional and global problem.

This research group passed through each phase of the research design: problem selection, data collection, analysis and interpretation of results. The group then concerned itself with a fourth phase which is an essentially equal component of reseach methodology; the uses to which its findings are put. Research utilization was a major concern of the research project and use of this document continues far beyond the dissolution of the research team.

The group defined the reseach problem as the existence and potential environmental impact of two war-related government installations in the Denver-Boulder area. Both the Rocky Mountain Arsenal- a repository for deadly nerve gas- and the Rocky Flats Plant of the Atomic Energy Commission have been in recent years charged with contaminating air, water, and soil resources in their environs. The Rocky Flats Plant has also been accused of possible radiation contamination of its employees and regional population.

The research team was concerned with what factual data could be gathered, how it could be used to shed new light on the problem and with suggesting citizen and decision-maker responsibility for

alternatives if the risks to local populations of operating these installations were as considerable as was charged.

The team was confronted by several inhibiting factors. Since both chemical and biological warfare (CBW) development and nuclear weapons are highly secretive operations, factual data on risk factors from the installations themselves are not readily accessible. Since those in charge of the installations feel threatened by increased public concern over their presence, the information provided voluntarily tends to be highly selective and plays down the potential risk. The public relations office of any bureaucratic organization, particularly those with "classified" status, tends not to be the most accurate source of complete and reliable data.

The limited research period (one academic semester) and the lack of financial support also inhibited the research. This report,then, is presented neither as exemplary of objective social scientific research nor as propaganda. It is, however, a piece of serious research which should raise the level of public awareness and lead to public debate and action based on the amount of accurate knowledge available.

HISTORICAL PERSPECTIVE

It might be of some value to briefly describe the historical and technological contexts which produced both the Rocky Mountain Arsenal CBW installation and the Rocky Flats nuclear weapons plant.

Rocky Mountain Arsenal (RMA)

RMA is located northeast of Denver in Adams County. Its southern perimeter is adjacent to 56th Avenue and Stapleton International Airport, and is 2 miles due north of Fitzsimons Army Hospital.

RMA was created at the onset of World War II, its primary function being the manufacture and storage of chemical munitions. Between 1951 and 1957, RMA manufactured the most lethal component of its current stockpile, the nerve gas "GB".

US Army Field Manual 3-10 describes the effect of this gas:

> "Nerve Gas 'GB' Type- Causes muscles to contract. Breathing stops and death occurs. Paralysis of the nervous system."[1]
>
> "GB can be employed for immediate casualties in surprise dosage attack against targets containing unmasked dug-in troops..."[2] (emphasis added)

In addition to inhalation, a lethal dosage of nerve gas can be inflicted by absorption through the skin, eyes, and intestinal tract.[3] Failure to suffer immediate death or incapacitation after exposure to nerve gas does not necessarily suggest immunity. There may be gas-related and delayed complications. A study by the University of Colorado School of Medicine conducted under contract to the US Army describes the case of an RMA employee "who had a very mild exposure to nerve gas"[4] and was treated at the RMA hospital and released:

> "while driving home from work (he) suddenly pulled into a filling station and called for help. The attendant found him in a state of shock and called for an ambulance, and was admitted to the hospital where he died two days later of coronary thrombosis."[5]

Nerve gas disrupts the action of the cholinesterase enzyme, which is essential to the functioning of the nervous system; the immediate cause of death is usually respiratory or coronary failure.

Although the exact quantity of nerve gas stored at RMA has never been disclosed, in 1960, Representative Byron Johnson of Colorado told Congress that there was enough nerve gas stockpiled outside Denver "to kill every man, woman and child in the world."[6]

Some of the nerve gas is in a variety of bombs, rockets and underground storage tanks; but most of it is in one ton metal cylinders, clustered in neat rows throughout the Arsenal, above ground, exposed and unprotected, except against lightning, for which they are electrically grounded.[7]

Aside from GB gas, RMA also stores over 1,000 tons of obsolete phosgene gas;[8] and beginning in 1955, Shell Chemical Company contracted to manufacture commercial toxins at RMA, some with a similar chemical structure to nerve gas.[9] The Shell Pest Strip which hangs in many American homes is one commercial product from this plant.

Groundwater Contamination. Liquid wastes and contaminated water from the manufacturing process were disposed of in artificial lakes on RMA grounds. Toxins filtered downward, polluting underground water supplies which eventually spread beyond the perimeter of the RMA, a fact which did not become public until 1959.

From 1951, the first year of GB production, farmers in the RMA area complained of contaminated underground water supplies which caused crop failures and livestock deaths. Although suspicion was directed toward RMA, the nearest and most obvious source of contamination, RMA officials maintained that the source of the problem lay somewhere else. In 1955, however, the RMA installed an asphalt-lined waste disposal pit, which prevented further contamination but could not correct that which had occured previously, and which became a serious problem calling forth public outcry in 1959.[10]

In June of 1959, the Denver Post reported: "...livestock has died and thousands of acres of crops have been destroyed."[11]

Representative Byron Johnson wrote to the Secretary of Defense: "Already farms have been destroyed and the water supply serving more than 15,000 citizens is presently endangered."[12] The Post noted that the natural course of the groundwater flow from the RMA was toward the contaminated area, but RMA officials made no comment.[13]

In November of 1959, it was discovered that as early as 1954 the Army Chemical Corps began secret investigations in Adams County on the possibility that RMA wastes were contaminating underground water. At no time were the residents or the county health officials told that wastes from GB-type nerve gas might be threatening their water supply. When Army investigators who tested wells were asked the reason for testing they answered, " It's just routine" or "You'll be told later" or they refused comment.[14] Earlier, in 1957, the commanding officer of the RMA, Col. Ronald Martin, answered an inquiry from the Adams County supervisor of the Tri-County Health Department by saying the tests were secret but "there is nothing to worry about."[15]

In November of 1959, the United State Public Health Service, after conducting its own tests, concluded that RMA was directly responsible for the contamination. The Chemical Corps commented that their own years of testing were "inconclusive"[16] Yet the following day The Denver Post revealed that in 1956, the United States Geological Survey had submitted a report to the Chemical Corps which concluded that groundwater in the RMA area was "unsuitable for irrigation of crops" and warned that " the effects may become serious."[17] In addition, The Post revealed the exsistence of a report by the University of Colorado, made on contract with the Army between 1956 and 1959 which concluded that herbicides manufactured at RMA were present in shallow

wells near the RMA and that: 6.

> "it is quite likely that moving water from the original source of contamination will continue to carry toxic materials ... for years to come and that soil to which contaminated irrigation water has been applied will produce poor crops for a number of years.
>
> Furthermore, it may be expected that the front of toxic materials in the groundwater will continue to move toward the Northwest until the Platte River is reached; at which point groundwater migration will then carry the materials Northeastward down the valley."[18]

Both reports were classified and withheld from the public by the Army until public pressure forced their disclosure; thereafter, a third report, issued by the Army, suggested that a "Phyotoxicant has been introduced into the groundwater from GB (nerve gas) plant wastes commencing in 1953."[19]

After all the disclosures of 1959, the chief of the Army Chemical Corps, Major General Marshall Stubbs, was asked why residents had not been warned of the possibility of contamination of their underground water supplies. The Denver Post reported his answer:

> "The residents were not informed because the Chemical Corps was not aware of the probability that their domestic water supply was seriously contaminated."[20]

Waterfowl Deaths: In June of 1959, The Post commented;

> "The Denver Post has carried stories about the mysterious death of wild ducks that land on the 40 acre lake used by the arsenal as a filtration pond."[21]

And in November of 1959, in an aside to a story on water pollution from RMA wastes, The Denver Post noted that the United States Fish and Wildlife Service had been conducting studies in the RMA vicinity and had concluded that RMA liquid wastes were responsible for the death of waterfowl in the area.[22] This revelation failed to elicit much public concern and was followed by intermittent news stories

over the years. In January 1963, the RMA commander, Col. Charles McNary voiced objection to the publicity over the deaths of about 2,000 wildfowl per year due to dieldrin, a Shell Chemical Company toxin:

> "I wouldn't say there isn't something in the lakes that's harmful to ducks. But ducks can pick up the insecticide on a farm where crops have been sprayed, for example.
>
> Shell and other companies are making chemicals that go all over the world. Someone has to make them." [23]

Earthquakes. Beginning in 1962, a series of earthquakes occurred in Northeast Denver, in the vicinity of the RMA. Some geologists suggested that their cause was the operation of the RMA's newly-installed 12,000-foot waste disposal well, into which liquid wastes were pressure-injected. In November of 1965, the RMA commander said the well could not be blamed,[24] but in December of 1966, after a panel of geologists concluded that the pumping of wastes into the well lubricated the Derby Fault under the RMA, causing earthquakes, its use was suspended.[25]

At present, wastes produced at the RMA are transported to Nevada for disposal.[26] More recently efforts to gain permission to dump wastes in deep wells south of Denver were rejected by public agencies.

Detoxification. At present, detoxification of all nerve gas and detoxification or removal of all phosgene gas at RMA is underway. The projected completion date by the Army is late 1976 or 1977. Disposal of some 21, 224 M-34 cluster bombs began October 26, 1973, after Congress approved an environmental impact statement.

On November 9,1973, during detoxification, a bomblet containing GB, in an M-34 bomb exploded at 10:30 am. Public announcement of the accident was made at 2:30 pm. The bomb was the 60th to undergo detoxification.[27]

The actual impact on humans and other populations of RMA and its nerve gas production and storage was a constant focus of debate and study through the 1960's. Following are several accounts of the environmental dangers which have already occurred. Additional accounts are included in the appendix of this report. All quotes are from the Denver Post, except where indicated.

June 14, 1959:

"Cattle Deaths, Ruined Crops Linked to Arsenal Chemicals"

"...some livestock has died and thousands of acres of crops have been destroyed."

Representative Byron Johnson wrote to the Secretary of Defense: "Already farms have been destroyed and the water supply serving more than 15,000 citizens is presently endangered." Johnson said the contamination north of the Arsenal was noted more than six years ago as covering a strip of land about five miles long and almost two miles wide.

"There have been repeated attempts," he said,"to get a satisfactory answer from the government."

April 26, 1964:

"Poison-Loaded Arsenal Lakes Kill Hundreds of Waterfowl."

"In 1959, the Bureau of Sport, Fisheries and Wildlife reported that a Shell employee had been assigned the task of periodically gathering dead ducks and burying them."

"Heavy waterfowl losses have been occurring on the lakes since 1951, and reports have listed bird deaths as 2,000 a year."

December 13, 1966:

Use of the deep disposal well was suspended after a connection was established by geologists between pumping of wastes and Denver area earthquakes.

Rocky Flats Atomic Energy Plant (RF)

Rocky Flats is located about 16 miles northwest of Denver and six miles sotheast of Boulder in Jefferson County. The facility, completed in 1953, and employing 3,200 persons, is operated by the Dow Chemical Company under contract with the Atomic Energy Commission.[28] Plans to construct the Rocky Flats plant were announced by the United States Atomic Energy Commission on March 23, 1951. There was no citizen involvement in the decision to locate the plant. In 1953, the initial construction of Rocky Flats was completed at a cost of

approximately $44 million. Geographically, the rocky cow pasture 16 miles northwest of Denver was chosen as the site because it was close by Colorado University, skilled manpower in Denver and attractive recreational opportunities in the mountains. The selected area was also noted to be very adaptable for the stringent security procedures that the plant demanded.

The primary function of Rocky Flats is the production and manufacture of trigger mechanisms, which utilize radioactive plutonium, for thermonuclear weapons.

Plutonium is one of the most highly toxic substances known, and minute quantities of it inhaled or imbedded within the body can be lethal. Its radiation can cause permanent celluar damage leading to cancer, and the fact that plutonium oxidizes quickly makes it a serious fire threat.[29]

The refining process of plutonium and the resultant waste material has resulted in the contamination of RF and the surrounding area, despite elaborate safety precautions. Over 200 small fires have occurred at Rocky Flats, and in May 1969, a $50 million fire in two assembly buildings destroyed enough plutonium to build 77 Nagasaki-type bombs, although radioactive particles in the smoke were supposedly trapped by a special filtration system.[30]

Consequences for the external environment, should a fire fail to be contained, would be tragic. A study in inhalation exposure to plutonium at RF published in Health Physics describes the effects of a small fire confined to a plutonium production area:

> "Even though the fire was extinguished within a few minutes, airborne contamination spread quickly throughout the area. Air sampler filters indicated an activity...at the remote areas."[31]

At this time it is uncertain what level of contamination of the region, if any, has developed over the past years from accidents and strong winds. The environment, with certainty, has been contaminated through accidental wastage spills. In one case, a tank of contaminated oil being moved for disposal developed a leak, spilling oil over a mile length of the road. The decision was made to pave over the contaminated area with asphalt, which has a half-life far less than the 24,000 years of plutonium. When the paving wears away, then the radioactive material will again be exposed.

Normal procedure at RF had been to seal radioactive wastes in metal containers and bury them. Container leaks developed, contaminating surrounding water and soil. If disturbed, radioactive particles in the soil could become airborne, posing a threat to nearby populations. Today, no radioactive wastes are buried on the site; the plutonium and other radioactive waste that cannot be recovered in the plant's recovery facility is shipped to Idaho Falls, Idaho.[32]

Several things come through in an analysis of the development of both RMA and RF:

> 1) There has been no citizen participation in any of the decisions locating these installations or concerning any policies affecting their impact on the regional community.
>
> 2) Some serious doubts have been raised in the public mind about the existence or effectiveness of safeguards which would prevent the contamination of populations and environment.
>
> 3) Evidence of injurious effects upon local populations and/or their natural resources does now exist and the RMA and RF authorities with direct responsibility have

> concealed or attempted to minimize the importance of that evidence. Bill Colston, the Atomic Energy Commission's plant manager, in an interview concerning the federal agency's championing of the public's right to know attitude stated:"They didn't care about the public. They didn't believe the public had any right to know."[33]

POTENTIAL CURRENT and FUTURE RISKS to LOCAL POPULATIONS

While history helps us understand how these installations developed, their past effect on local population and environment, and how their latent conflict of interests with local populations became manifest, it tells us little about the level of current and future risk, if any, these installations pose for the people of the area. As was mentioned earlier in this report, the Rocky Mountain Arsenal is located adjacent to Stapleton International Airport. A heavy fog, a light snow, or perhaps a pilot's misjudgement could each set the stage for disaster.

Scenario:

> It is 5:30 pm, a weekday. A fully loaded DC-10, one of 300 flights daily at Stapleton International Airport, attempts to land; it crashes into a neat row of exposed, unprotected one ton nerve gas containers two miles west of the perimeter adjacent to RMA.
>
> Several tons of the nerve gas"GB" are released from the ruptured containers. A subsequent explosion in the nearly empty fuel tanks of the DC-10 has the same effect as a bomb of the type used to deliver the nerve gas: detonation causes the gas to form a toxic cloud; there is little fire to incinerate the gas.
>
> A moderate 20 mph northerly wind causes the cloud of gas to drift across the RMA south perimeter within 6 minutes. All the passengers who survived the initial crash are now dead. RMA personnel begin to die, some don protective clothing and masks, and medical treatment is fairly efficient.
>
> The cloud crosses Interstate-70 within 12 minutes, and motorists begin to die; traffic comes to a halt and will hinder emergency personnel when they are able to respond.
>
> Ironically, the two hospitals best equipped to treat nerve gas victims--RMA hospital and Fitzsimons Army Hospital-- are closest to spreading contamination. Hospital personnel and patients begin to die within 18 minutes, but the gas cannot penetrate every corridor and room, and many are spared. Panic breaks out; some personnel are aware that supplies of the antidote atropine, and gas masks, are stored somewhere, but few know where. Some personnel who don masks and administer aid in the contaminated wards begin to absorb the gas through the skin.

Not all will die. Of those who receive a less-than-lethal dosage, some will recover entirely, others will suffer brain damage, paralysis.

People begin to die at Stapleton Airport; maintenance crews on the tarmac die first, then passengers in the terminal complex. In the surrounding residential area people also die, but they are less vulnerable, small houses have even smaller openings for gas penetration, and the gas is beginning to dissipate.

There are the usual accidents attendant to any disaster: fires, explosions, and motor vehicle collisions.

After no more than half an hour, the toxic cloud dissipates entirely. No one south of Colfax Avenue dies--6 miles from the point of origin. Most of Denver still does not realize what has happened. Even though, the densely populated areas of the city have been spared, perhaps 50,000 are reported to have died. Emergency personnel have been slow to react, there are only 5,000 doses of atropine at Fitzsimons Hospital, and this is insufficient. Most of the affected people are by this time dead. The majority of casualties are those in the open; the highest proportion of death is, characteristically, among the very young, the very old and the physically weak.

With three hundred take-offs over the arsenal daily, an event such as the scenario describes is not impossible. Should such an accident actually happen, what might be the consequences for Metro-Denver residents? Since use of the nerve gas against humans has yet to be verified beyond doubt, it is difficult to describe the exact effects on humans. Extensive testing has been done however with lower animals and we have a quite sufficient estimate of its potential consequences for the human biosystem.

The Rocky Mountain Arsenal stores three types of gases: two types of nerve gas, "VX" and "GB" and one other type of lethal agent, mustard gas. Both of the nerve gases in their normal physical state are a colorless liquid. In their disseminated form the "GB" type is a vapor liquid and the "VX" type is a liquid aerosol. Although the nerve gases are usually used as harassing(small dose) or lethal(large dose) agents, they are also quick-kill agents of tremendous potency.

The onset of symptoms occurs up to ten minutes following inhalation, or up to a half hour following percutaneous absorption. The overall results of the two nerve gases are as follows:

NERVE GAS TYPE "GB"
Causes muscles to contract, breathing stops and death occurs. Paralysis of the nervous system.

NERVE GAS TYPE "VX"
Causes muscles to contract, breathing stops and death occurs. Much greater absorption through the eyes than the skin. Paralysis of the nervous system.

Stephen Rose's book, Chemical-Biological Warfare relates how nerve gas operations incapacitate an individual.

> Nerve gases are anti-cholinesterase agents, working by blocking the enzyme which the body uses to destroy one of its chemical nerve signals transmitters after it has done the job. This has two effects. One is that control is lost over the affected part of the nervous system. The other is that a large concentration of the chemical transmitter builds up in the body and that the chemical itself is a powerful poison. The body is at first incapacitated, and then forced to poison itself.
>
> The symptoms of nerve gas poison are diverse and spectacular. In a comparitively inactive man an exposure to "GB" of 15 mg-min/m^3 dims the vision, the eyes hurt and become hard to focus. This may last for a week or so. At 40 mg-min/m^3, the chest feels tight, breathing is difficult, and there is coughing, nausea, drooling, heartburn and a twitching of the muscles. At 55 mg-min/m^3, there is a strangling tightness and aching of the chest, vomiting, cramps, tremors and involuntary defecation or urination. At 70 mg-min/m^3, severe convulsions will set in followed closely by collapse, paraysis, and death.

Mustard gas is also stored at the Rocky Mountain Arsenal. In its normal physical state, mustard gas is a colorless-to-amber oily liquid, and in its disseminated form it can be either a vapor liquid or an aerosol. The symptoms of intoxication are:

HARASSING
Eyes: inflamation, photophobis, ulceration and blindness.

LETHAL
Coughing, retching, frothing at the mouth, asphyxia and pneumonia.

This agent injures every type of body tissue with which it comes into contact. The principal targets are the skin and the eyes which it burns and blisters. Because mustard gas is not a very volatile substance, it persists in the field for long periods and can be effective for days or even weeks after dissemination. It was used briefly in World War I and killed and blinded a number of combatants and perhaps civilians.

Preparedness. Should an air crash, a fire, an explosion, or some other unforeseen incident cause the release of nerve gas in Metro-Denver, how prepared would authorities and citizens be to respond effectively to the disaster?

The State Health Department informed us that Jefferson, Boulder and Denver counties have no immediate nerve gas contamination contingency plans the only ones being state-developed.* In interviews with staff personnel at specific health facilities the following questions were asked:

1. What are your contingency plans for a possible nerve gas leak?
2. How many people are trained to attend such an accident and how many can be treated?
3. Are there any decontaminants or antidotes available?
4. Will future generations be affected by any leaks?

A registered nurse who heads the emergency room at Boulder Memorial Hospital responded to the questions by saying that if such an accident occurred, the hospital would be able to handle it. Treatment for the nerve gas poisoning, according to her, would involve washing the affected area with soap and water and hoping for the best. Dr. Johnson of the Jefferson County Health Department replied to the questions by saying that plans for

* Interview with Mr. Montgomery, State Health Department.

treatment aren't practical since a person exposed to nerve gas would die anywhere from a few seconds to a minute after exposure.

Still more variety in official opinion among those responsible was reflected in interviews with Dr. Morris Gaon, a physician at the Rocky Mountain Arsenal, and Mr. Arthur Whitney, the public information officer at the Arsenal, who were questioned concerning the medical treatment of nerve gas exposure. Dr. Gaon stated that nerve gas exposure is treated with artificial respiration and atropine. Although he has treated more than 200 cases of nerve gas exposure, he declined to give the exact number for"reasons of security.

At the Army's Fitzsimons Hospital, Lt. Chafy, the public information officer, is the only person authorized to provide information on Fitzsimon's role in emergency contingency plans. The hospital has facilities to treat 1,600 people in an emergency and will treat civilian casualties. They have 5,000 doses of atropine in stock to treat nerve gas victims and there are 200 doctors, 300 nurses and 500 enlisted men trained in atropine injection.

Col. William Allen of Denver's Emergency Preparedness Office was interviewed about emergency plans for the city and the county of Denver. Allen remarked that any nerve gas from the Rocky Mountain Arsenal would have to be atomized by an explosion to be carried by winds and thus pose a serious threat to Denver. Since nerve gas is extremely heavy, according to Allen, if there were no explosion, the gas would remain concentrated on the ground. Furthermore, Allen noted that in order for the gas to reach Denver there would have to be northerly winds instead of the usual southwesterlies. Allen stated that the gas would have to travel two miles

to reach Denver and would most likely be too diluted by then to cause any harm.

According to Allen, gas masks are the only reliable protection against nerve gas exposure. He felt it improbable that the entire population of Denver would purchase or possess such masks. Since they are made to fit adult males, they would be useless for women or children.

Inspite of his belief that Denver would probably never face a nerve gas contamination problem. Allen did explain what emergency procedures would be initiated in case of a nerve gas emergency.

a. The arsenal commander would contact the Denver Emergency Preparedness Office and then the local media concerning the impending disaster.

b. Citizens would be instructed to: 1) find a place of safety inside their homes or other buildings; 2) close all windows and doors and shut off all air-circulation and air-conditioning equipment.

c. The fire department will move casualties to either Fitzsimons or the Arsenal Hospital.

After interviewing the various departments and organizations directly responsible for executing contingency plans in the case of a nerve gas emergency in the Denver area, one could hardly feel reassured that everything possible would be done expediently and efficiently. On the contrary, most offices which would handle such a crisis, (The Denver Emergency Preparedness office being an exception), were totally unprepared for any nerve gas disaster. The Adams County Civil Defense office, Denver General Hospital, and Jefferson, Boulder and Denver County authorities have no immediate contingency plans for nerve gas contamination. Many of the offices contacted refused to release the requested information stating that it was "classified."

The Colorado Civil Defense Office was unable to present a contingency plan because they didn't know whether the Rocky Mountain Arsenal was in Denver or Adams County. (The arsenal is located in Adams County.) What is perhaps even more disturbing is the lack of consistency of professional opinions concerning the medical treatment of the nerve gas victims. Prescribed treatment ranged from the simple washing of the affected parts of the body with soap and water, to a resignation that death is inevitable and treatment useless. Information on antidotes to treat nerve gas exposure also differed from source to source. Dr. Gaon of RMA stated that the drug pentamethonium and oximes were not used in nerve gas treatment while Lt. Chafy at the Fitzsimmons Army Hospital said pantamethonium was indeed used, although oximes were not. In contradiction to both their views is a United Nations report which recommends the use of oximes for nerve gas exposure.[34] The United Nations Report also contradicts Dr. Gaon, Lt. Chafy, and Col. Allen on the effectiveness of gas masks. According to the report, nerve gas can be absorbed by the skin, the eyes and intestinal tract. Gas masks then would provide insufficient protection.

Radioactive Contamination. Acute and rapid contamination of populations and environment by radioactive material is a constant possibility at the Rocky Flats plant. No scenario here is necessary to suggest the risk. A serious accident has already occurred at Rocky Flats in 1969, the immediate and long-term effects of which are still not publicly known. Should a similar or more serious fire or explosion occur in the future, the resulting oxidized plutonium could be spread over wide areas, depending on wind conditions and other meteorological factors.

Since the city of Denver is sixteen miles downwind from Rocky Flats,

if a prevailing south-west wind was blowing and oxidizing plutonium happened to leak from the plant, radioactive material would be blown across highly populated areas such as Westminster, Arvada, Broomfield and Central Denver. A worker who has been at Rocky Flats since the plant first opened created his own scenario about what would occur if the right wind conditions coupled with a plutonium leak should ever materialize.

> "... if some plutonium smoke leaked out we'd call all of the police agencies, tell them which way the smoke was going and tell them to move everybody out of its path. Afterwards, decontamination teams would have to scrape all the plutonium off everyones' roofs. It would take months! Then we'd have to bring all the people and put them through our one body counter (designed to measure radiation). It would be a hell of a mess."

Most of the immediate effects of a plutonium leak would be experienced by the employees at Rocky Flats. In addition to serious radiation burns, if particles of plutonium happened to enter a wound, amputation of the limb would probably be necessary. Particles of plutonium, breathed into the lungs, can result in death from radiation contamination. So far there has been one known contamination death at Rocky Flats.

In comparison with the inadequate plans that most Civil Defense offices would employ if faced with a nerve gas crisis many of these same offices have relatively well-organized contingency plans for an emergency involving Rocky Flats. According to a spokesman from the Colorado Civil Defense Office, each city and county is responsible for their own contingency plans in an emergency. The Jefferson County law enforcement agencies have many responsibilities in the Civil Defense

plan. The officers have received training and carry instructions on the procedures they are to follow if a crisis should occur. The agencies will also help monitor contaminated and suspected contaminated areas. The police departments will be responsible for evacuating rural areas.

According to the Jeffco Civil Defense, if the plutonium radiation were blown into a populated area, the people in the affected area would be told to stay in their homes, wait for the wind to die down, and then wash the radioactive dust off their home.

Dr. Johnston of the Jefferson County Health Department stated that in the case of such an emergency the department would close off the contaminated areas, organize decontamination centers staffed by local physicians, and would monitor the affected population for the long range effects of plutonium exposure. It would also impound all water downstream from the contaminated area. Dr. Johnston felt that the health department's most positive action would be in organizing local physicians in the decontamination centers and in providing emergency transportation for casualties.

Dave Berford of the Boulder County Civil Defense, Emergency, Preparedness Office explained Boulder's well-organized contingency plans. Berford pointed out that if an accident involving plutonium occurs at Rocky Flats, only alpha particles will be released. Since alpha rays cannot penetrate skin, the major danger of exposure to the plutonium will come from inhalation, exposure through an open wound, or eating contaminated food.

Unlike the Arsenal, the Rocky Flats plant is a crucial link

in a chain of emergency communications. If there is a major accident at RF, that would jeopardize the population of Jefferson County, the plant is supposed to immediately contact Jeffco Civil Defense which in turn will contact the Atomic Energy Commission. Rocky Flats is also responsible for notifying the State Department of Health, the State Office of Military Affairs, the Boulder Police, the Boulder Emergency Preparedness Office, the Jefferson County Sheriff's Department, the Atomic Energy Commission's radiological assistance team, and any other affected county. After a major accident, Rocky Flats will make the first press release. Subsequently, the Boulder County Health Officer will handle all press releases involving Boulder, which will be forwarded to the State Emergency Operations Center at Camp George West. Here the press releases will be coordinated with those from other agencies and counties in order to minimize confusion.

After word is received of a major accident at Rocky Flats, it is the responsibility of the State Health Department (SHD) to evaluate the situation and recommend the action to be taken. The SHD will also determine what areas are to be restricted. These secured areas will be monitored and the data obtained will be reported to the Boulder County Health Officer.

Both Lutheran and St. Anthony's Hospitals, in addition to other local hospitals, stated they are prepared to handle a radiation emergency.

Although contingency plans for handling an emergency occurring at Rocky Flats are better organized than those for handling an RMA accident, much improvement is still necessary to guarantee the safety

of all the people within the affected area.

The organization that seems the least prepared to handle an RF crisis is the Jefferson County Civil Defense. When interviewed about their contingency plans, its officials stated that warnings to the population would "probably" be by radio; sirens would not be used because they feel that the people would not know how to properly react to them. The endangered population "might be told to go out of the area". The Jeffco CD advises people caught in a plutonium contaminated area to wash any radioactive dust off their homes. Although the alpha particles released by the plutonium cannot penetrate skin, there is danger in the inhalation of the material. For this reason, nearly all other emergency organizations recommend that individuals keep as far away as possible from the contaminated area.

Possible Long Term Effects. Long term results of radiation contamination (i.e. leukemia, other cancer forms, and heredity alterations,)however, are more debatable and may be ultimately more dangerous than the risk of accident. The Ford report gives us the latest research findings on this.[35]

Plutonium, when it burns, becomes an oxide. This oxide is a source of alpha particles which in turn cause ionizing radiation. When high speed electrons take negatively-charged electrons from atoms and leave positively-charged ions, a chain reaction begins, setting electrons in motion causing still more ionization. Radiation also destroys chemical bonds between atoms. These processes cause various types of damage to body cells at random. Some cells become unable to reproduce while other cells are killed instantaneously. Certain cells are injured, not destroyed by the radiation, but when they reproduce, the injury is passed on to the new cells.

Although there are over one billion cells in every gram of cells in the human body, just one cell, injured in the proper way, can cause cancer or leukemia. Although the type of injury to a cell which could result in cancerous growth occurs rarely, the amount of cells that a dose of radiation affects greatly increases the chance of cancer incidence.[36]

It takes a while for leukemia and other forms of cancer to manifest themselves. From the time of radiation exposure to the appearance of the disease, there is an intervening latency period when the victim is unaware of his or her illness. Although the latency period for leukemia lasts about five years, other forms of cancer can remain latent for periods of up to twenty years. Because of this, people may be unaware of the dangerous effects Rocky Flats operations may be having on them and their families.

Plutonium radiation can also cause an affected individual to pass damaged chromosomes to his or her unborn offspring. Since it is the chromosomal structures within the nucleus of the cell that are primarily affected by ionizing radiation, if persons receive radiation in their reproductive organs and it resulted in mutated cells, their offspring will have every body cell mutated. The possible consequences of this are not unimaginable; children could be born with many physiological and genetic disabilities. We may soon have more definitive information on long term effects of exposure to plutonium. The AEC has been ordered to do a comprehensive health and death study of its plutonium workers before it moves ahead with plans for breeder reactor development. Rocky Flats

personnel will be the subjects for that study.

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DECISION-MAKING STRUCTURES

That RMA and RF were located in the Denver- Boulder area and currently pose a potential risk to regional populations are consequences of decisions within organizations. The risks created by these decisions can also be lessened or eliminated by decisions with citizen participation. Such participation requires citizen knowledge of the decision-making roles resposible for the current politicies.It also requires the knowledge of how they, as citizens might influence decision-making structures to bring about policy change.

Rocky Mountain Arsenal. The Rocky Mountain Arsenal is controlled by the United States Army and the Department of Defense. Presently, the commander of the arsenal is Col. Watson. Orders relayed from top officials in the Defense Department to Howard H. Callaway, Secretary of the Army, then passed down through the Chief of Staff to the United States Materiel Command. Functions of this command are research, development, product production, testing, evaluation, storage, maintenance and disposal of material. The Sixth U.S. Army under the command of Lt. Gen. Richard G. Stilwell is in charge of the Rocky Mountain and Western forces. The Rocky Mountain Arsenal is under the jurisdiction of the Sixth U.S. Army.(Appendix

Rocky Flats AEC Plant. As a result of the Atomic Energy Act of 1946, the newly formed United States Atomic Energy Commission took over the nation's energy program from the Army's Manhattan Engineer District on January 1, 1947. The Act established the primary mission of the Commission to be nuclear weapon research, development, testing, production and stockpile surveillance.

Albuquerque Operations is the name given to one of the eight operations in the United States organized by the Atomic Energy Commission. Other operations are located in Chicago, Idaho, Nevadà, Oak Ridge, Richland, San Francisco, and Savannah River.(Appendix B) The Albuquerque Operations of the AEC employs about 1,280 people, and its annual payroll is approximately 18 million dollars. Integrated operating contractors employ approximately 28,660 additional people. Some of these integrated and supportcontractors include: The University of California, Western Electric Company, Dow Chemical U.S.A., Monsanto Research Corporation, General Electric, Mason and Hanger Silas Mason Co. Inc., The Bendix Corp., and the Zia Company. Rocky Flats is an area office of Albuquerque Operations.

We might note here the extensive network of sub-contracting which spreads the economic impact of AEC operations into local communities. As with other sectors of the war industry, this wide distribution of contractors makes it difficult to build public opposition to the Rocky Flats operation or any other installation. There are always local vested economic interests.

The Rocky Flats Area Office, established July 1, 1952, administers the contract under which Dow Chemical, U.S.A. operates the Rocky Flats plant. Although the Rocky Flats Area Office employs only 51 people, Dow Chemical U.S.A. employs 3,480 people at the plant. The Rocky Flats Plant is a production facility, handling nuclear materials. Fabrication, manufacturing design and development, production engineering, and related activities are performed at the plant.

The Dow Chemical Corporation, which operates RF has six world operations: the U.S. area, Canada, Europe, Latin America and the Pacific area. (Appendix C) Another operation called Life Science is primarily responsibile for medical and technological research. Dow Chemical U.S.A. has several suboperations;

there are two in Texas, one each in Michigan, California, Louisiana and an Eastern division. Rocky Flats is one of these divisions. In addition to operating its nuclear weapon research and development laboratories, Dow is a leading manufacturer of such household products as HandiWrap, DowOven Cleaner, and Saran Wrap.

The decision-making process governing Rocky Flats is as follows; the manager of Albuquerque Operations, General Donnelly, is administratively resposible to the General Manager of the Atomic Energy Commission, Robert E. Hollingsworth. In accomplishing its assigned mission, Albuquerque Operations receives programmatic directions from various AEC headquarter divisions. General Donnelly, as manager of the Albuquerque division relays orders from the AEC to James Hanes, general manager of the Dow Chemical Corp. at Rocky Flats. Above Hanes is B.W. Colston, manager of the Rocky Flats area. Colston can appeal orders given to him to General Donnelly, who can contact the AEC. Hanes, as manager of Dow at Rocky Flats can appeal decisions to J.M. Leathers, executive Vice President of all U.S. Dow divisions. Above Leathers is Earl Barnes, President of the U.S.A. area operation of Dow Chemical. (Appendix C)

The relationship between government and private industry is part of the AEC's philosophy to utilize the resources and technical research of private and industrial firms.

Because of the complex organizational structure of Rocky Flats and the governmental and military restrictions at the RMA, it is very difficult for citizens to press these bureaucratic structures for complete and accurate information. Personnel at these two high-security

installations are extremely reluctant to answer any inquiries concerning long or short term risks that could result from their presence. Most public inquiries are handled by public relations officials who release only that information which has already been carefully examined and screened. For this reason, citizens must look more to sources of information outside the jurisdiction of RF and RMA.

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CONCLUSIONS

From the evidence it has gathered, the Action Research Group reached certain conclusions concerning potential hazards posed by RF and RMA and preparedness to respond adequately to such hazards.

1. A negative impact from both installations on both the human population of the area and its natural environment is suggested by the data. Even if the degree of impact is not measurable(or has not been revealed) to date, the potential risk is serious enough to warrant thorough investigation by local and state authorities and citizen's groups.

   There is an interesting linkage between the potential for disaster at the local and global levels. Denver's predicament is shared by the world. The two installations posing a clear and present danger to its limited ecosphere, manufacture and store the two most awesome weapons in the superpower arsenal, weapons that pose the same risk for the world's population as they do for Coloradans.

2. The disaster reponse and the contingency plans in Metro Denver and surrounding counties are totally inadequate given the dimensions of the potential risk. The capacity of emergency agencies to protect and care for the large numbers likely to be affected is minimal. The public is generally uninformed about facilities that do exist and what means of self-protection there are. Where contingency plans exist within counties and municipalities, they are poorly coordinated with other plans.

3. The people in decision-making roles who are responsible for the exsistence and operation of RMA and RF are not evil persons, but they do need to respond with candor and action to public concern and to discard the evasiveness and subterfuge of the past. They need to be persuaded that the health and safety of the local populations must come above all else.

4. Citizens must learn how to conflict creatively with forces that may or seem to threaten their present and future interests. The commonweal is protected only through various interest groups (in this case, citizen groups local, and state agencies, and these installations and the federal and industrial bureaucracies that operate them) engaging in creative conflict. Citizens must organize as conflict parties to investigate and challenge and to protect their health and that of their future generations. The RMA and RF impact problem is not unlike that raised by oil shale development and strip mining. The federal government is making decisions affecting the lives of Coloradans- who have minimal participation in those decisions, and who increasingly question that their best interests are served by the consequences of those decisions.

Our research has led us to questions as well as conclusions.

1. While a decision has been made to detoxify existing nerve gas supplies at RMA, the process is not scheduled for completion until some time in 1976. What is to be done in the interim to minimize the dangers of air disaster, toxic wastes and the like? By what means can citizens monitor the disposal process to insure faithful adherence to the schedule by RMA authorities and those higher in the Defense Department structure? The public has developed a healthy mistrust, during the past decade, of governments that misrepresent and manipulate in the "interest of national security" or some other ambiguously-defined objective.

2. What are the incontrovertible facts regarding the less visible long-term possibilites of radiation poisoning for populations surrounding nuclear installations, including nuclear power plants? This question becomes even more critical as concern over future energy supply pushes the nation forward toward crash programs in nuclear power plant and breeder reactor development.

POSSIBLE INTERVENTION STRATEGIES

The Action Research Group after completing its data-gathering turned its attention to the question of citizen intervention. If citizens are concerned about potential health and environmental hazards posed by the Arsenal and Rocky Flats, how might they intervene creatively? An analysis of the problem is certainly the place to begin, a process to which we hope this document contributes. We plan to disseminate it through several accessible communications networks including the mass media, public and private educational systems, environmental and other citizen groups. We would encourage other action groups to do more thorough research on different dimensions of the problem. Citizen action might lead in several directions.

1. Pressure for policy change through current state and federal political representatives, who could intervene at various levels in the two organizations. Don't expect overnight miracles! Very few congressmen are ready to take action on the subject of nuclear products. It takes time and an understanding of the problem to change opinions from:

a.) Not interested; "Nuclear plants are safe and clean."

to b.) Concerned; "Maybe there is a danger."

to c.) Determined; "I'll research and gather information."

to d.) Convinced; "Yes there is a danger."(or there is not.)

to e.) Ready for Action; "Its time to convince others."

Most members of Congress are somewhat between stage a. and b. It is expected that a member of Congress will move from stage b. to stage c. and d. whenever a significant number of home-state voters, groups, experts and newspapers voice their opinions loudly enough.

2. Influence upon candidates for national, state and local office. Make it an issue.

3. Pressure on local and state officials to investigate contamination possibilities more thoroughly.

4. Pressure on industrial forms involved. Dow Chemical(Rocky Flats), Campbell Soup (Nerve Gas), and Shell Oil (Insecticide Production at RMA) all have large individual consumer markets and are sensitive to public atitudes.

5. Develop educational programs in schools and civic associations.

6. Symbolic acts and actual disruption of the contamination process (eg. civil disobedience, vigils).

It is essential in intervening to bring about change, to have a reasonably clear set of alternative policy goals to present. In the intervention in question, the following might be among the conceivable alternatives.

Rocky Flats.

1. Shifting nuclear operations at Rocky Flats to an unpopulated area, or conversion of the plant to nonhazardous production. A shift would have serious shortterm economic consequences for the immediate area that would have to be dealt with.

2. Elimination of nuclear weapons production altogether. Our"overkill" capacity already assures us of nuclear "security".

Rocky Mountain Arsenal.

1. Increase the capacity of detoxification facilities to speed up the schedule.

2. Create underground storage space for nerve gas now in surface storage.

3. Prohibit any new (binary or other) toxic agents from being stored there.

4. Phase out the Arsenal and create a wildlife sanctuary on the site.

5. Press health and disaster agencies to develop and coordinate adequate protection programs.

Although, letter writing is often not as effective a means of

protest as are other forms of intervention it can often call attention to matters of citizen importance. The following agencies have shown a real concern over the presence of RF and RMA in the Denver Metro area, and could be of great help in planning strategies or simply for research information.

Environmental Protection Agency
1129 Twentieth St. N.W.
Washington,D.C. 20460

Environmental Action Commit
1100 14th
Denver, Colorado

The American Friends Service Committee
Colorado Area Office
2801 E. Colfax Ave.,#304
Denver, Co. 80203

David Massebaum
Clean Water Action Project
1325 Delaware St.
Denver, Co.

For those individuals who wish direct contact with persons involved in the decision-making process at Rocky Flats, the following addresses will be of help.

Atomic Energy Commission
Washington, D.C. 20545

Albuquerque Operations Office
Harold C. Donnelly
P.O. Box 5400
Albuquerque, New Mexico 87115

Regulatory Regional Office of the AEC-Denver
John W. Flora
10395 W. Colfax Ave.
Denver, Co 80215

Other individuals and groups that could provide information or assistance in dealing with the Rocky Mountain Arsenal and Rocky Flats are listed below.

Colorado Open Space Council

Sierra Club

Environmental Action Group

Colorado Committee for Environmental Information

Floyd Haskell, Colorado Senator

Ralph Nader

Ruth Correll, Member of Boulder City Council

Ed Mendillo, Denver Representative

Peter Metzger, Columnist for "Rocky Mountain News"

Dr. Arthur Tamplin and Dr. John Gofman, authors of the book Poisoned Power.*

THE PROJECT AS A LEARNING EXPERIENCE

Reflecting upon the value of this group research project, participants mentioned a number of definite advantages they saw in the experience.

The value of cooperative research was emphasized time and again. The sense of interdependence that developed among us during the semester was quite visible. This was a rather unique experience for most of us, having been socialized in a highly individualized and competitive system. In this project, shared learning was the major resource. The research tasks were divided and reports scheduled so that each was responsible for the others' learning and progress. If one of us failed to produce on time the others suffered. The importance of cooperation and humor in group research came through very clearly and participants expressed substantial satisfaction with the shared learning experience.

Participants spoke often of the interest and excitement generated

*Both Gofman and Tamplin have testified before governmental, public and professional groups to make Americans aware of the dangers of nuclear pollution

by doing research that would be utilized for and might make some difference in solving a critical real-world problem. We cannot overemphasize the value group members saw in the opportunity to benefit society as well as themselves in the process of creating knowledge.

Others felt that the project had helped to sharpen their knowledge of research methodology including library and action research skills. Having to pass through the problem selection, data-gathering, and analysis utilization phases and be forced to consider the third phase as you were moving through the others was helpful to a number of us. The distinction between committed research became quite clear and there was some discussion within the project as to whether or not "committed" research was scientific. The point is that values inevitably enter into research at both the problem selection and analysis utilization stages while data-gathering should remain as value free as possible.

The importance of data-gathering was another major realization emerging from the project. The essential nature of factual material on which to base conclusions and policy decisions is indisputable. While much of the learning produced in the group concerned process (i.e. how to get the factual material), group members did a good deal of content learning as well. They learned a great deal about weapons production and storage system, chemical/biological warfare, the military-industrial-academic complex and how it operates, and public attitudes and community leadership in surrounding areas. They also demystified the bureaucratic process as they learned how to "penetrate" bureaucratic organizations, how to persevere in the face of bureaucratic inertia, evasive reponses

and misrepresentation. They also learned how accepting and passive people generally are with respect to bureaucratic unresponsiveness to their problems. The "success feedback" most of the project members received from their persistence in gathering data proved to them that large bureaucratic organizations are vulnerable to good strategy and are not impenetrable. We should also note however, that some officials were both open and helpful and this should be encouraged through citizen dialogue with them.

The project group concluded that formal study in universities should and can be related to real-world problem solving. The group felt that there should be increasing concern for using the university curriculum, both graduate and undergraduate, to gather factual material on significant problems and make it available to citizen organizations. This facilitates the informed involvement of citizens in decision-making and action that affects their present and future.

Perhaps the most dramatic payoff the project had was the insight it afforded students into the sharp internal contradictions current weapons policies produce. One student remarked that one of the by-products of the production process at the Rocky Flats installation could well be a long-term serious increase in radioactivity-related morbidity in the surrounding area's population. The question must be asked, then, how can we protect ourselves from our own "defense" industries?

In the last group meeting, however, one student spoke of what must be the ultimate contradiction--a mix of the tragic and the absurd.

She spoke of signs posted inside the Rocky Flats plant that she had visited, a facility which produces plutonium trigger-devices for the nuclear weapons capable of destroying millions of people in seconds. The sign read:

LIFE IS FRAGILE

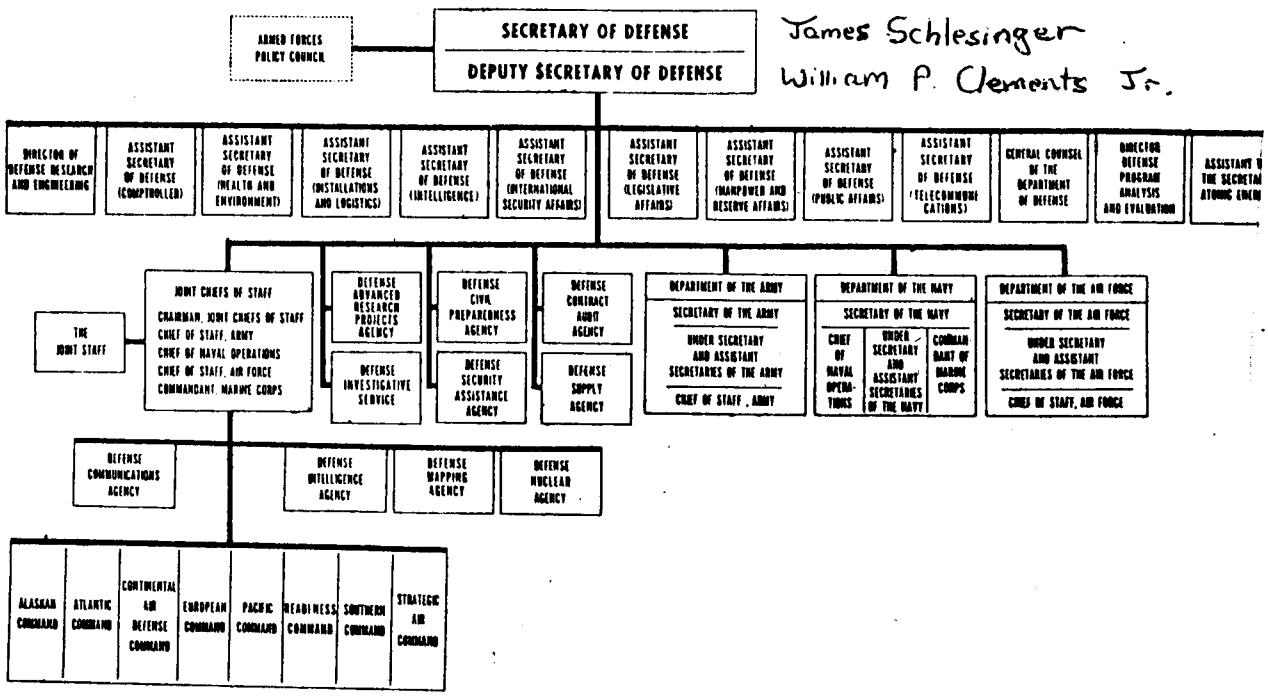
HANDLE WITH CARE

ACTION RESEARCH GROUP

Joyce Durol - Report Editor
Tom Esposito - Report Editor
Jeff Calvin
John Marvuglio
Marcee Orlin
Harvey Tevah
Dan Morimoto
Jeff Jones

Sociology 482, Fall 1973
University of Colorado
Professor Paul Wehr

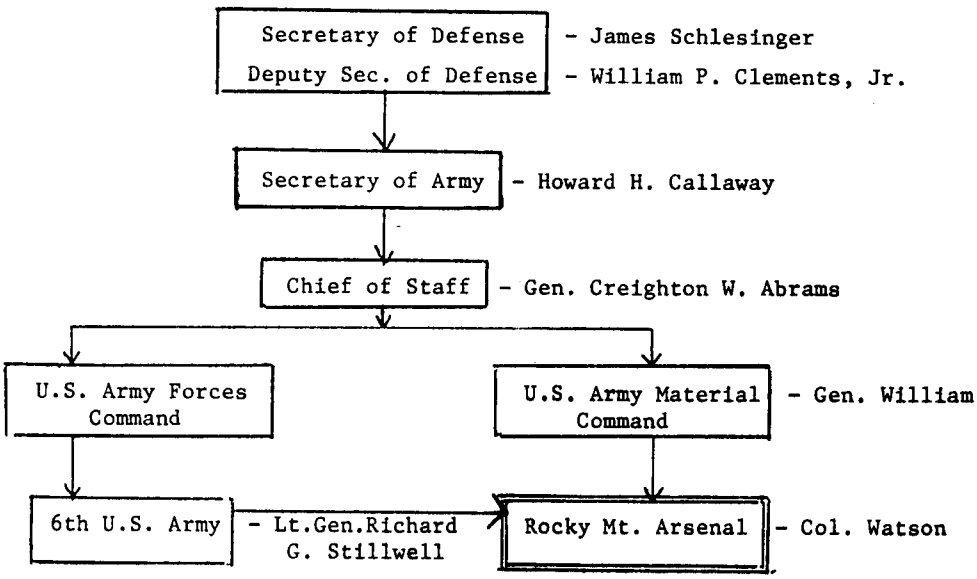
DEPARTMENT OF DEFENSE



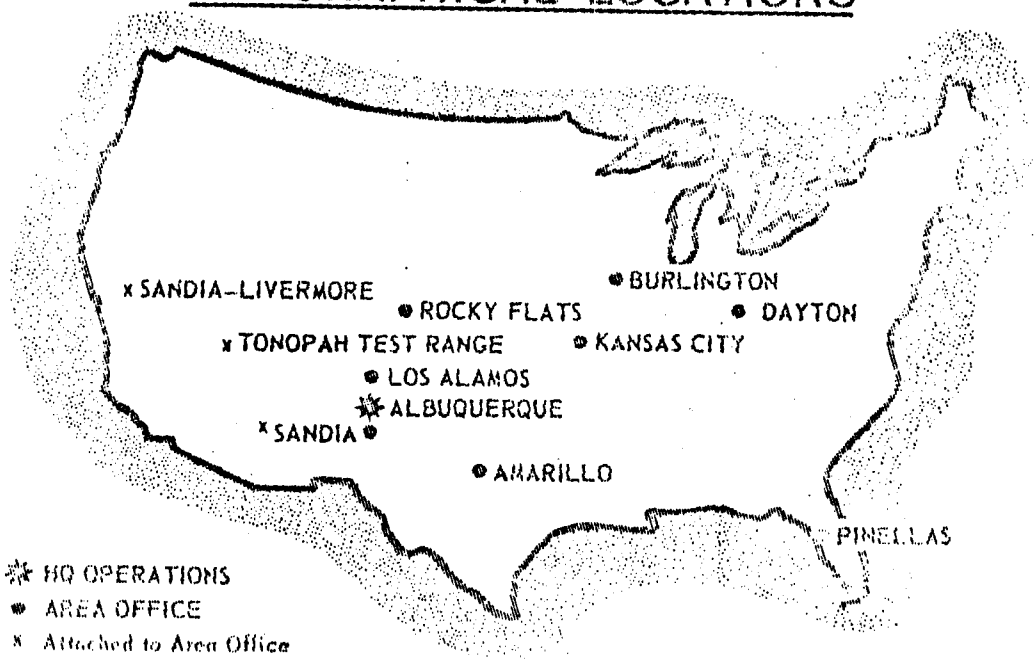
APPENDIX A - 2

Organizational Structure of the

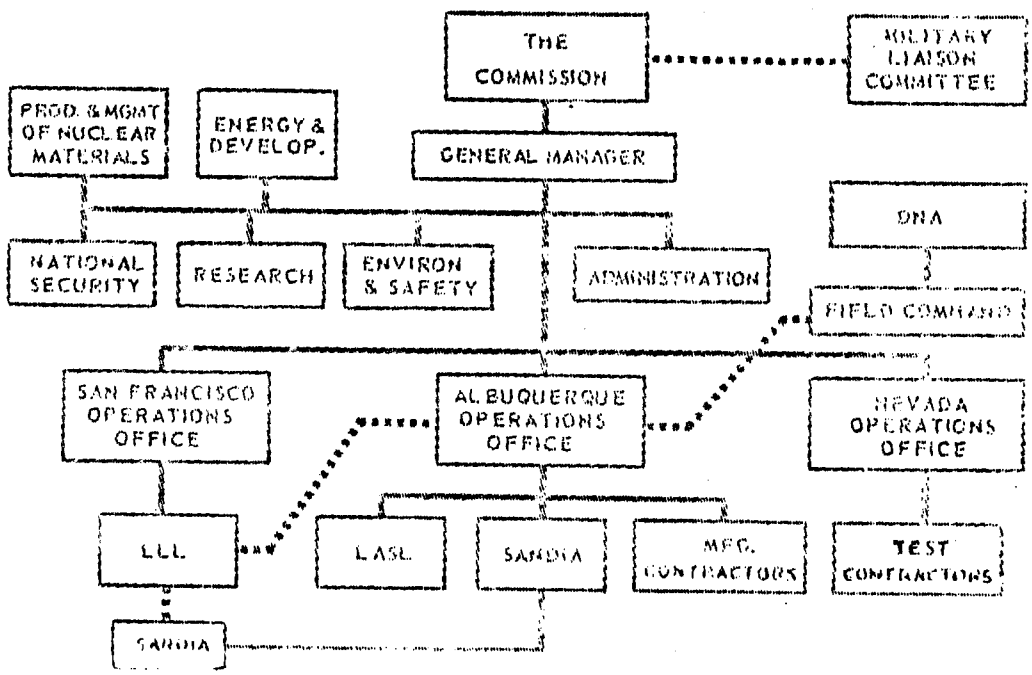
ROCKY MOUNTAIN ARSENAL



ALBUQUERQUE OPERATIONS
GEOGRAPHICAL LOCATIONS



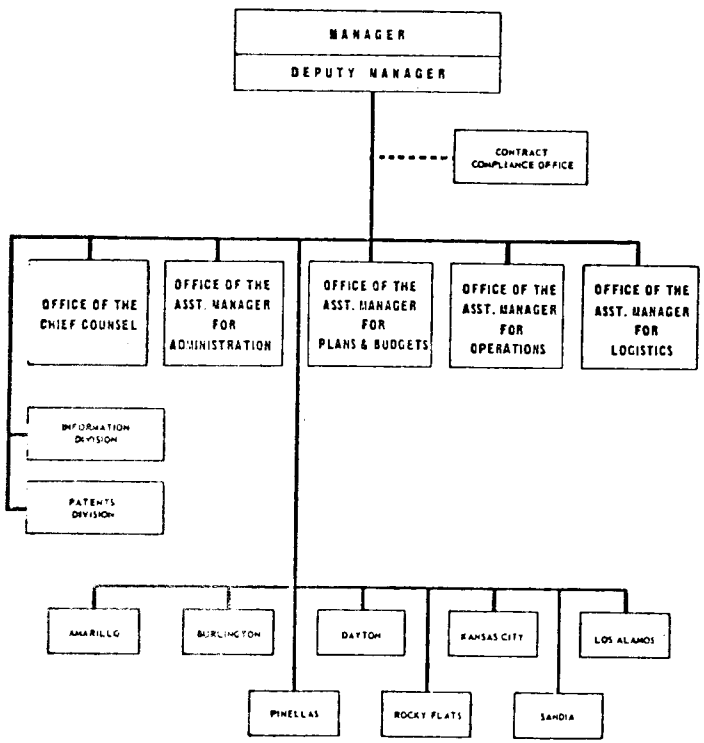
U.S. ATOMIC ENERGY COMMISSION



U.S. Atomic Energy Commission
ALBUQUERQUE OPERATIONS

ORGANIZATION CHART

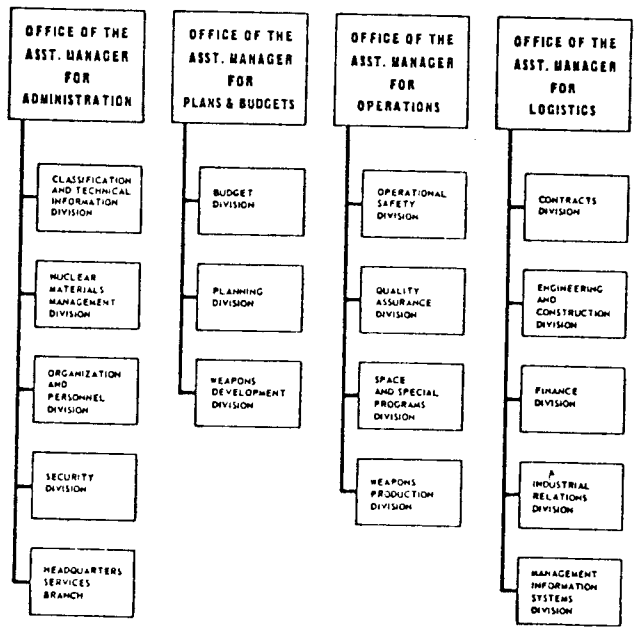
OFFICES REPORTING TO THE MANAGER

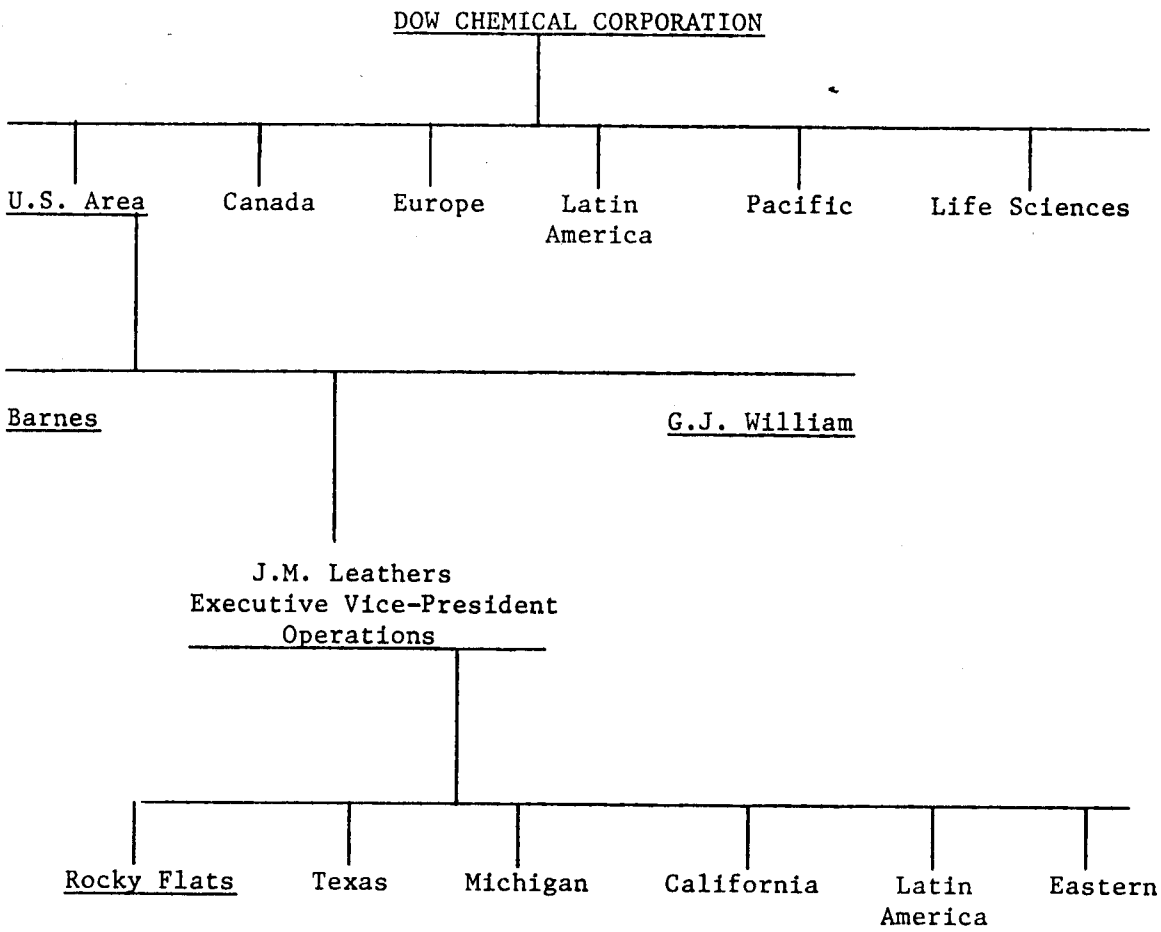


U.S. Atomic Energy Commission
ALBUQUERQUE OPERATIONS

ORGANIZATION CHART (Continued)

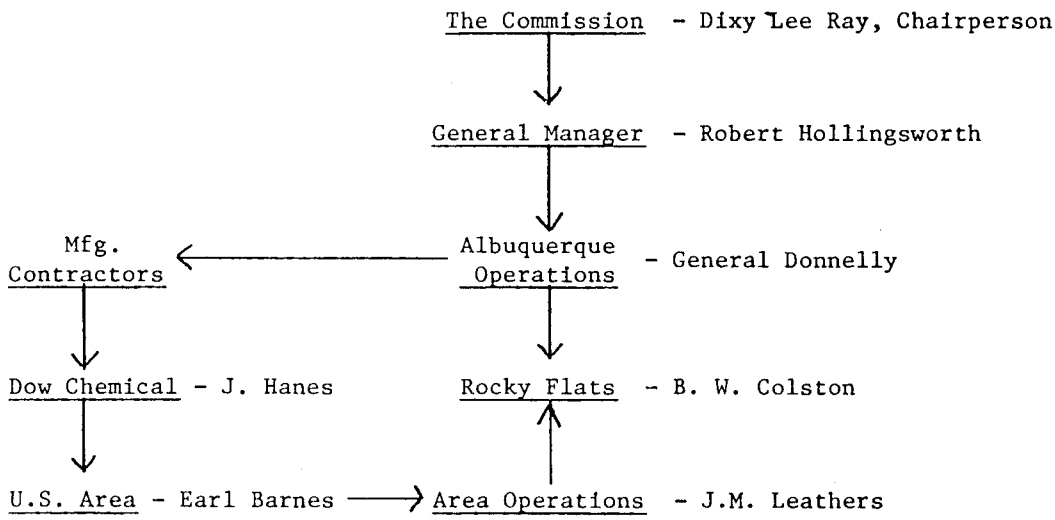
DIVISIONS REPORTING TO THE OFFICES OF THE ASSISTANT MANAGERS




DOW CHEMICAL CORPORATION
U.S. Area
Canada
Europe
Latin America
Pacific
Life Sciences
Barnes
G.J. William
J.M. Leathers
Executive Vice-President
Operations
Rocky Flats
Texas
Michigan
California
Latin America
Eastern

APPENDIX C - 2

ATOMIC ENERGY COMMISSION



ATOMIC ENERGY COMMISSION

Headquarters: Germantown, Md.
Mailing address: Washington, D.C. 20545
Phone, 301-973-1000

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Special Assistant to the Chairman	HARRY D. BRUNER.
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Special Assistant	MARY HALOW.
Commissioner	CLARENCE E. LARSON.
Special Assistant	JOHN A. GRIFFIN.
Commissioner	WILLIAM O. DOUB.
Special Assistant	THOMAS E. MURLEY.
Commissioner	WILLIAM E. KRIEGSMAN.
Special Assistant	(VACANCY).
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Chairman, Atomic Safety and Licensing Appeal Panel	ALAN S. ROSENTHAL.
Chairman, Atomic Safety and Licensing Board Panel	NATHANIEL H. GOODRICH.

FOOTNOTES

[1]"Employment of Chemical and Biological Agents," U.S. Army Field Manual, 3-10, Department of Army, Navy and the Air Force (March, 1966).

[2]Ibid.

[3]"Chemical and Bacteriological Weapons and the Effects of Their Possible Use," (NY: Ballantine, 1970; United Nations Report #E691.24).

[4]Kurt von Kaulla and Joseph H. Holmes, "Changes Following Anti-cholinesterase Exposure," Archives of Environmental Health, (Feb. 1961), pp. 168-177.

[5]Ibid.

[6]The Denver Post, Feb. 5, 1960, p. 3.

[7]The Denver Post, Nov. 3, 1965, p. 38.

[8]The Denver Post, Nov. 9, 1973, p. 27.

[9]The Denver Post, Dec. 6, 1973, p. 21; Shell Chemical Company now produces at Rocky Mountain Arsenal: Aldrin (pesticide), Azodrin (insecticide), Bidrin (insecticide), Ciodrin (insecticide), Nudrin (insecticide), Phosdrin (insecticide), Vapona (insecticide), Nenagon (soil fumigant), Planavin (herbicide

[10]The Denver Post, Nov. 12, 1959, p. 26.

[11]The Denver Post, June 14, 1959, p. 1A.

[12]Ibid.

[13]Ibid, p. 3A.

[14]The Denver Post, Nov. 13, 1959, p. 1.

[15]Ibid.

[16]Ibid.

[17]The Denver Post, Nov. 18, 1959, p. 3.

[18]The Denver Post, Nov. 25, 1959, p. 24.

[19] Ibid, p. 3

[20] Ibid, p. 24

[21] The Denver Post, June 14, 1959, p. 3A.

[22] The Denver Post, Nov. 17, 1959, p. 1.

[23] The Denver Post, Jan. 3, 1963, p. 2.

[24] The Denver Post, Nov. 3, 1965, p. 38.

[25] The Denver Post, Dec. 13, 1966, p. 3.

[26] The Denver Post, Dec. 6, 1973, p. 21.

[27] Denver Clarion, Nov. 16, 1973, p. 1.

[28] Roger Rapaport, "Secrecy and Safety at Rocky Flats, "The Los Angeles Times, 1969.

[29] Ibid.

[30] Ibid.

[31] J.R. Mann and R.A. Kirchner, "Evaluation of Lung Burden Following Acute Inhalation Exposure to Highly Insoluble PaO_2," Health Physics, Vol. 13, #8, (Aug., 1967), p. 877.

[32] The Colorado Daily, April 4, 1974.

[33] Ibid.

[34] op. cit.

[35] Report by the Energy Policy Project of the Ford Foundation, A Time to Choose: America's Energy Future, (Cambridge, Mass.: Ballinger, 1974).

[36] Edward A. Martell, "Radioactivity of Tobacco Trichomes and Insoluble Cigarette Smoke Particles," Nature, Vol 249, #5454, (May 17, 1974). A recent article by atmospheric chemist Edward Martell describes how this process occurs in tobacco smoking. It is similar to the bombardment of healthy cells by radioactive material such as plutonium oxide alpha particles.

BIBLIOGRAPHY

The Boulder Daily Camera. Nov. 29, 1952; Feb. 14, 1953; Sept. 16, 1961; Jan. 31, 1963; Aug. 15, 1968; Nov. 11, 13, 1969.

Boulder Eco-Center. "A Discussion of Objections to Nuclear Gas Stimulation." Boulder, Colo., Sept. 15, 1973. (Mimeo).

"Chemical and Bacteriological Weapons and the Effects of Their Possible Use." NY: Ballantine, 1970; United Nations Report #E69I.24.

The Colorado Daily. Apr. 4, 1974.

Colorado Department of Health, Division of Occupational and Radiological Health. "Emergency Handling of Radiation Incidents: A Handbook for Police Officers." n.d.

Colorado Department of Health, Division of Occupational and Radiological Health. "Emergency Handling of Radioactive and Metallic Fires: A Handbook for Fire Departments." n.d.

Colorado Department of Health, Division of Occupational and Radiological Health. "Handling the Radiation Accident Victim: A Guide for Hospital Personnel." n.d.

Colorado Department of Health, Division of Occupational and Radiological Health. "Radiological Response Plan." n.d.

Colorado Department of Health. "Radiation in the State of Colorado." 1972.

Denver Clarion. Nov. 16, 1973.

The Denver Post. June 14, Sept. 23, Oct. 17, Nov. 12, 13, 17, 18, 25, 29, 1959; Feb. 5, 1960; Jan. 3, 1963; Apr. 26, 1964; Nov. 3, 1965; Dec. 13, 14, 1966; Apr. 29, 1967; Nov. 1, 1971; Oct. 15, Nov. 9, Dec. 6, 1973.

Dow Chemical Company. "Dow Annual Report to Shareholders." Midland, Mich., 1972.

"Employment of Chemical and Biological Agents." U.S. Army Field Manual, Department of the Army, Navy and the Air Force, March 1966.

Gofman, John and Templin, Arthur. Poisoned Power. Eammaus, Pa.: Rodale Press, 1971.

Hersh, Seymour M. Chemical and Biological Warfare. NY: Doubleday, 1969.

Kaulla, Kurt von and Holmes, Joseph H. "Changes Following Anticholinesterase Exposure." Archives of Environmental Health, Feb. 1961.

Mann, J.R. and Kirchner, R.A. "Evaluation of Lung Burden Following Acute Inhalation Exposure to Highly Insoluble PaO_2." Health Physics, Vol. 13, #8, Aug. 1967.

Martell, Edward A. "Radioactivity of Tobacco Trichomes and Insoluble Cigarette Smoke Particles." Nature, Vol. 249, #5454, May 17, 1974.

McClintock, Michael, et.al. "Memorandum on the Possible Hazard to Denver of the Rocky Mountain Arsenal's Storage of Nerve Gases." Boulder, Colo., Aug. 15, 1968. (Typewritten).

Rapaport, Roger. "Secrecy and Safety at Rocky Flats." The Los Angeles Times, 1969.

Rapaport, Roger. The Great American Bomb Machine. NY: E.P. Dutton, 1971.

Report by the Energy Policy Project of the Ford Foundation. A Time to Choose: America's Energy Future. Cambridge, Mass.: Ballinger, 1974.

Rose, Stephen. CBW-Chemical and Biological Warfare. Boston: Beacon Press, 1968.

U.S. Atomic Energy Commission, Information Division. "The Story of Albuquerque Operations." Albuquerque, N.M., Revised March 1973.

Citizens Association for Safe Energy

•nuclear moratorium
•energy conservation
•safe energy research

27 Hastings Ave. Croton-on-Hudson, New York 10520 (914) 271-3629

631

January 14, 1974

Dr. W.D. Rowe
Radiation Programs, Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Rowe,

We are pleased to respond to your call for "viewpoints of concerned citizens as to the problems of plutonium in the environment."

The problems of plutonium in the environment can be quickly and easily reduced by STOPPING construction of all nuclear power plants and phasing out of operation those now in operation as quickly as possible.

To some such a course of action may seem unreasonable, but to informed citizens it is the only reasonable solution. Plutonium, with a radioactive half-life of 24,000 years, must be isolated for the next 500,000 years - more than 250 times the period between the birth of Christ and the present. Because each large nuclear power plant produces about 400 pounds of this poison each year, more than 99.999% of it must be contained if serious health effects (death) on the citizenry are to be avoided. For example, just three pounds of plutonium is enough, evenly distributed, to produce nine billion lung cancer cases (about three times the world's population).

The chances of successfully containing such large quantities of plutonium to 99.999% perfection are, to put it mildly, exceedingly remote. What other industry approaches that kind of accountability? Or even 99%? None that we know of.

We are pleased to report to you that over 147,000 American voters have consulted their common sense about nuclear power and petitioned their representatives in Government to keep nuclear fission out of their lives. More than 30,000 from New York alone have signed Clean Energy Petitions. If you doubt their authenticity, you are invited to inspect them in Washington, where they are kept filed according to Congressional District.

The only responsible action EPA can take is to oppose all further development of nuclear power plants in favor of energy from the sun which can be clean, safe, reliable, cheap and plutonium-free.

Sincerely,

Franklin L. Gage

Franklin L. Gage
Coordinator

cc:The Press

Jody Dittrich
301 East Virginia
Jamestown, New York
14701

Mr. Miller,

I am writing in response to your article in the December 28 issue of Rodales Environment Action Bulletin concerning the plutonium emission standards being set up by the Environmental Protection Agency.

I understand that plutonium is a very poisonous substance and feel that the job you are doing to rid this from our air is indeed commendable. With all that is wrong in the world today its good to know that there are people who have not given up. I think you would be wise in making your fight more public. I'm certain there are people who have never even heard of plutonium.

I am aware of the idea that plutoniuim can "live" for twenty four thousand years or longer. At this rate with more plutonium being put in the air every day one could begin forming concepts that we will be doomed by the effects of plutonium on the human body.

Once again I would like to comment on your efforts. Keep up the good work.

Sincerely,

Jody Lea Dittrich

UNITED STATES AIR FORCE

Dover AFB, DE.
January 17, 1975

Carl Miller
Director of Criteria and Standards Division (AW-560)
Office of Radiation Program, EPA.
Washington, D.C. 20460

Dear Mr. Miller:

This letter is in regards to the recent hearings on plutonium standards which took place in Denver. I'm not a scientist or an expert in radiology. I'm only an average citizen. But then again, that's what the hearings were all about, weren't they? Aren't public radiation exposure standards to be set by the outcome of these hearings? Regardless of whether they were or not, here is my opinion.

I don't like the idea of using nuclear power for anything. I feel the risks are much too great. There will always be radioactive leaks somewhere in the system. This is the greatest injustice we could possibly do to our environment. An environment that is already crumbling.

This is a virtually indestructable poison that lingers in our bodies, food, and other life supporting systems and slowly does its dirty work while we go about our lives totally unaware. There is always more to be added on top of that already received, too. It doesn't die quickly or easily in many cases, but rather it accumulates, stockpiling and slowly eating away at vital tissue. It slowly but surely kills the person cell by cell. Can you guarantee me that the standards about to be set will not cause one more birth defect, cancer or leukemia case, or bit of sorrow than what will naturally occur? When you can, then the standards

UNITED STATES AIR FORCE

are high (or should I say low) enough.

I'm nineteen and single. Could it be that my first child will be born deformed or retarded because of these standards? How about your next child? Or grandchild? Think about it, O.K.?

Sincerely yours,
Michael L. Grimes

Jan 17, 1975 635

Dear Carl Miller,

Even if plutonium were no more dangerous than a used refregerator I would still not want one once of it in the environment. Because of the CBS report on terrorist groups, who have said if they get plutonium they will build a bomb, it is to dangerous to have any where in the world. It goes from plants through airports where millions of Dollars of merchandise is stolen yearly. The amount of money to absolutely perfect plutonium from terrorist would be far to much to pay for the amount of electricity derived from it.

But this is not the worst hazard. Its danger to the environment is terrific and there is no way plutonium can be sealed off from the environment for ever with wars, earthquakes etc

Sincerely,

Michael P. Hurst
510 Andalusia
Hot Springs, Ark
71901

636

Wisconsin Ecological Society

P.O. Box 514

Green Bay, Wis. 54305

January 20, 1975

Dr. William A. Mills,
Director of Criteria & Standards Div. (AW-560)
Office of Radiation Program
U. S. Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

Our comments on environmental plutonium standards are brief:

1. There can be no minimum limits.

2. The world cannot sustain a plutonium economy with safety.

3. The breeder reactor is a disaster.

Cordially,

R. J. Barlament
Secretary

b

COSC
Colorado Open Space Council, Inc.
1325 Delaware Street, Denver, CO 80204

January 21, 1975

Director
Criteria and Standards Division (AW-560)
Office of Radiation Programs
U. S. Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

We originally applied to offer oral testimony at the plutonium and transuranium elements public hearing held in Denver on January 10, 1975. The representative for our group, Dwight Filley, was called away suddenly and was unable to appear before your panel. Unfortunately, he neglected to inform the panel or me until last week.

We would greatly appreciate it if you could enter the testimony as part of the written record at this time as a demonstration of our concern with the possible dangers of plutonium on the environment. I enclose two copies of the testimony for this purpose.

I wish to apologize for the Colorado Open Space Council for any inconvenience we may have caused. We shall make every effort that this does not happen again.

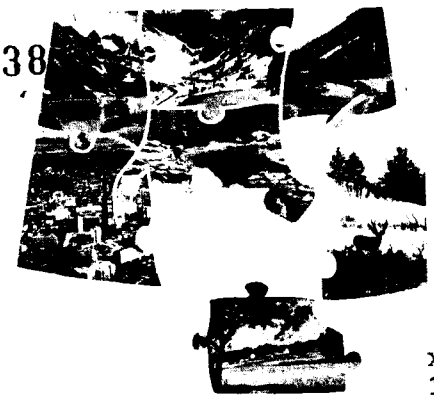
Yours very truly,

Bonnie F. McCune

Bonnie F. McCune
Administrator

Encs.

638



COSC

Colorado Open Space Council, Inc.

1325 Delaware St., Denver, CO 80204

January 10, 1975

STATEMENT OF DWIGHT FILLEY,
VICE PRESIDENT OF THE COLORADO OPEN SPACE COUNCIL,
FOR THE COUNCIL

My name is Dwight Filley, and I am the Vice President of the Colorado Open Space Council. The Council is state wide and is the largest coalition of environmental and conservation groups in Colorado.

My organization believes that Plutonium 239 is among the most hazardous substances, if not the most hazardous substance on earth. It is estimated that one pound of Plutonium 239, properly distributed, would result in 9 billion cases of lung cancer. There are not even ½ that number of people available for such a distribution.

But it is not our purpose to recite statistics. Rather we wish to convey the profound fear felt by a large number of informed citizens who have a general knowledge of the damage radiation causes to living tissue, both as a carcinogen and as a mutagen. There is also a general mistrust of reassurances that this or that technical problem is being "taken care of". Too often, when such reassurances are given, future developments

turn out to be other than expected.

We feel that the population is already sujected to multitudes of strange chemicals, ranging from food additives to atmospheric pollutants. Time after time we learn that some new technological inovation has an unexpected and harmful side effect. Examples are phosphates in the water and aerosol propellants in the ozone layers of the atmosphere. We therefore favor strong limits to further pollution.

We are proud that our state of Colorado has taken the lead on many environmental issues. The Colorado Open Space Council supports our state's standards for Plutonium contamination of the soil as a mimimum, and urge that the Federal Government protect all U. S. citizens as Colorado citizens are now protected.

Plutonium, because of it's intense pathogenic characteristics, as well as its flammability, require totally unprecidented control to prevent harmful releases. Such control may in fact be impossible to achieve. The record so far is not encouraging. There have been many accidental releases, either admitted by the A.E.C. or discovered by someone else. It has been estimated that with a fully developed nuclear industry, 99.999% containment is necessary to protect the public. It is doubtful that any substance has ever been accounted for so carefully; certainly Plutonium has not been thus far.

If we are ever to determine if such control can be achieved, standards must be set and compliance attempted. We urge that Colorado standards become minimum national standards. Thank you.

I will admit that Nuclear Power is vital. That Dr. Bethe, of Cornell University who received the Nobel Prize for discovering the thermonuclear process by which the sun develops energy. Is more informed about benefits of nuclear power than Ralph Nader.

But the fact remains. If the nuclear industry had to face a market test. They might not pass.

There was the Rasmussen report on nuclear power plans risks. But it was commissioned by the Atomic Energy Commission. Naturally they lean in their direction.

A accidental discovery at the largest nuclear plant in the world. Located at Brown's Ferry, near Athens, Ala. Saved the lives of people in a 75-square-mile area. The loss of lives from deadly radioactivity could not be even accurately counted.

A nuclear disaster could have occurred had the reactor been tested with the defective equipment in use.

Last spring a tornado passed directly through the site of a proposed nuclear plant near Dixon Springs, Tenn., another came within 10 miles of the plant in operation at Athens, Ala.

"Right now in 15 of the 16 most tornado-prone states there are 82 reactors planned, being constructed or in operation."

"One atomic power plant of the size they are now building has 932 times as much radioactive material as the atomic bomb that destroyed Hiroshima in World War 2."

The locations of A-Plants are important. Such as building in a water-short area. How are we going to keep getting water. Which is vital to life and which will be in short supply.

The federal government is unable to account for thousands of pounds of uranium and plutonium that could be used to manufacture nuclear bombs.

Small combined amounts of the two nuclear elements can be combined to fashion a crude nuclear weapon. Missing nuclear materials could fall into the hands of terrorist groups or hostile governments.

There will be commercial handling and transporting of plutonium-239. I question the credibility that plutonium-containment will be performed flawlessly.

Summary:

My plan would be to offset the building of all the A-Plants. Build one or a few to perfect them.

Build a solar energy plant also. To compare the differences technically and economically.

Let us sum up a decision. With the A-Plant we face A short water supply. Possible ~~and~~ radiation leaks. The elements and sometimes poor workmanship.

I think we should have a scale to determine where we stand. Human lives is a very high price. If there is a mistake.

Sincerely yours,

Jerry W. Stevens

Jerry W. Stevens
14423 Flallon St.
Norwalk, Calif. 90650

4300 Lakehaven Drive, N.E.
Atlanta, Georgia 30319
January 21, 1975

Mr. Russell E. Train
Administrator, Environmental Protection Agency
Rm. 1200 Waterside Mall Bldg.
4th & M Streets, S.W.
Washington, D. C. 20460

Dear Sir:

It is my understanding that the EPA recently held public hearings pertaining to establishment of emission standards for plutonium 239 by nuclear power plants. I would like to protest the lack of publicity given these hearings.

As a citizen who is unalterably opposed to nuclear fission power, I scan local newspapers, national news magazines and closely follow the broadcast media for news pertaining to nuclear energy. It seems incredible that permissible emissions for Pu239 would even be considered. I would appreciate any available information regarding positions stated at the hearings and any decisions reached.

It appears that nuclear fission is being rammed down the throat of the public. The licensing process for construction and operation of nuclear power plants is a tragic farce perpetrated by the utility industry and its Washington hearquarters, the AEC.

With our new vice-president being up to his armpits in Exxon I fear greatly for the survival of the EPA as guardian of the environment and the hope of posterity.

Sincerely,

Victor Skorapa, Jr.,M.D.

Hudson Valley Citizens' Watch on Nuclear Safety

P. O. Box 472, New Paltz, N. Y. 12561

January 21, 1975

Dr. W.D.Rowe
Radiation Programs
E.P.A.
Washington DC 20460

Dear Dr. Rowe,

At the suggestion of Franklin L. Gage, Washington coordinator of the Task Force against Nuclear Pollution I am sending you the enclosed article by Dr. Arthur Tamplin which deals with the problems of Plutonium in the environment.

I am sure you are familiar with other sources on the subject by Dr. Gofman and others.

Yours sincerely,

Ned Lehac

Ned Lehac
Secretary

NL:js
encl.

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The Nuclear Gamble: What Are the Odds?

No adequate system exists to regulate expanding industry

by Arthur R. Tamplin

AN unfortunate thing happened on our way to the present energy shortage—the Atomic Energy Commission had a vision. This vision was a program for the development of nuclear reactors for the generation of electricity. It was some 25 years ago when the AEC embarked upon this long-range reactor program. At the outset of this grand program, we were told by its high priests that they would develop a technology that would supply us an abundance of cheap power—so cheap that in most cases it would not be worthwhile to meter it.

Now some 25 years later we find that the cheapness prophecy was quite wrong and, equipped with today's hindsight, it is not at all clear that we should want an abundance of power to drive an ever-increasing gross national product with its attendant resource depletion and environmental pollution. Moreover, it is obvious today that the policy over the past 25 years has led to serious diseconomies. We put 85-90 percent of our energy research and development dollars into the fission reactor program and thereby preempted research on alternative sources of electrical energy that are both necessary and more acceptable. As a consequence, we now find ourselves confronted in an arrogant fashion by this bureaucratic *fait accompli:* we are told that we can have either clean nuclear or dirty fossil fuel plants. However, it is abundantly clear today that fossil fuels, primarily coal, will form the backbone of our generating capacity throughout this century and into the next. If we had spent our research dollars differently, we could have had clean fossil fuel plants years ago, and most likely we would be well on the way to obtaining all our energy needs from the sun.

Yet we find our political leaders and our bureaucracies proceeding under the same old guidelines of the past. Witness the following quotation that has now become a new commandment in the catechism of the AEC:

> In his June 4, 1971 Message on Energy to the Congress of the United States President Nixon presented a program which included ". . . A commitment to complete the successful demonstration of the liquid metal fast breeder reactor [LMFBR] by 1980." He further states that ". . . Our best hope today for meeting the Nation's growing demand for economical clean energy lies with the fast breeder reactor. Because of its highly efficient use of nuclear fuel, the breeder reactor could extend the life of our natural uranium fuel supply from decades to centuries, with far less impact on the environment than the power plants which are operating today."

The present generation of nuclear reactors, the light water reactors, are a technological cul-de-sac. The difficulty facing them derives from their use of uranium-235 as a fuel. According to the AEC the economically recoverable supply of this fuel will last only a few decades. The fast breeder reactor would utilize uranium-238, the predominant uranium isotope which is not fissionable under conditions present in reactors. In the fast breeder, however, it will be transmuted by nuclear process into plutonium-239 which can undergo fission in a reactor. The breeder will actually create more fissionable fuel than it will consume (it will create fuel or "breed"). By using uranium-238 in this manner the breeder reactor will, as the President said, "extend the life of our national uranium fuel supply from decades to centuries." With just this information as evidence, the President's decision to make the LMFBR our priority energy program would appear to be sound.

However, the serious nature of this nuclear energy decision was exposed by Dr. Alvin Weinberg, director of the Oak Ridge National Labora-

Drawing by Geri Lucas

646 tory and a proponent of nuclear power, when he wrote in the July 7, 1972, issue of *Science:*

> We nuclear people have made a Faustian bargain with society. On the one hand, we offer—in the catalytic nuclear burner—an inexhaustible source of energy. . . . But the price that we demand of society for this magical energy source is both a vigilance and a longevity of our social institutions that we are quite unaccustomed to.

Dr. Hannes Alfven, Nobel Laureate in Physics, had earlier described this "Faustian bargain" in the May, 1972, issue of *Bulletin of the Atomic Scientists:*

> Fission energy is safe only if a number of critical devices work as they should, if a number of people in key positions follow all their instructions, if there is no sabotage, no hijacking of the transports, if no reactor fuel processing plant or reprocessing plant or repository anywhere in the world is situated in a region of riots or guerrilla activity, and no revolution or war—even a "conventional one" —takes place in these regions. The enormous quantities of extremely dangerous material must not get into the hands of ignorant people or desperados. No acts of God can be permitted.

In his article in *Science* Dr. Weinberg stressed the need ". . . of creating a continuing tradition of meticulous attention to detail." He stated:

> In a sense, we have established a military priesthood which guards against inadvertent use of nuclear weapons, which maintains what *a priori* seems to be a precarious balance between readiness to go to war and vigilance against human errors that would precipitate war. Moreover, this is not something that will go away, at least not soon. The discovery of the bomb has imposed an additional demand on our social institutions. It has called forth this military priesthood upon which in a way we all depend for our survival. It seems to me (and in this I repeat some views expressed very well by Atomic Energy Commissioner Wilfred Johnson) that peaceful nuclear energy probably will make demands of the same sort on our society, and possibly of even longer duration.

The President's decision to guide us along the path to a nuclear future was a monumental decision. But was it the correct one? It is instructive to look at some of the highlights of past history of this nuclear priesthood which is the AEC.

A nuclear reactor offers the possibility of industrial accident far beyond anything past history has known. Such an accident could result from the meltdown of the reactor core with the consequent breaching of its containment structures and the release of vast quantities of lethal radioactivity into the atmosphere. An AEC study indicated that such an accident could cause the immediate death of 3,000 to 4,000 individuals as a result of exposure to radiation. Another 30,000 to 40,000 people would be exposed to levels of radiation sufficient to cause a substantial number to die prematurely from cancer. The genetic consequences of such an accident would affect many future generations. Expectable property damage losses were estimated at approximately $7 billion.

To minimize the risk of such accidents in the present light water reactors, the AEC and the industry are relying heavily on an Emergency Core Cooling System (ECCS) intended to prevent meltdown. The ECCS, however, has never been fully tested and the facilities for performing such tests are not even scheduled for completion until 1975. Six small-scale tests of the ECCS have been performed and the ECCS failed all six of these tests. There is thus a strong possibility that the ECCS will not work.

In 1971 a group of scientists from the universities in the Boston area (the Union of Concerned Scientists) investigated this overall problem of core meltdown and the possible effectiveness of the ECCS. In its published report the group concluded that the situation was so perilous as to warrant a halt to the construction of nuclear reactors.

In testimony presented to the Congressional Joint Committee on Atomic Energy, Ralph Nader and the Union of Concerned Scientists last January 29 made public a secret report by an AEC task force dated last October. On page 14 of that report was the following:

> Review of the operating history associated with 30 operating nuclear reactors indicated that during the period 1/1/72 - 5/30/73 approximately 850 abnormal occurrences were reported to the AEC. Many of the occurrences were significant and of a generic nature requiring followup investigations at other plants. Forty percent of the occurrences were traceable to some extent to design and/or fabrication related deficiencies. The remaining incidents were caused by operator error, improper maintenance, inadequate erection control, administrative deficiencies, random failure and combinations thereof.

Regarding these incidents, on page 16 the task force stated:

> The large number of reactor incidents, coupled with the fact that many of them had real safety significance, were generic in nature, and were not identified during the normal design, fabrication, erection, and preoperational testing phases, raises a serious question regarding the current review and inspection practices both on the part of the nuclear industry and the AEC.

There is good reason to suggest that, because of the meticulous attention to detail that is re-

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Dr. Arthur R. Tamplin, a biophysicist on the staff of the Atomic Energy Commission's Lawrence Radiation Laboratory at the University of California, is on leave working with the Natural Resources Defense Council on environmental questions concerning the AEC's liquid metal fast breeder reactor program. With Dr. John W. Gofman he wrote *Poisoned Power: The Case Against Nuclear Power Plants.*

quired at every step during construction and operation of nuclear reactors, it will be impossible to develop an adequate system of regulation and inspection. The expanding nuclear industry is most likely unmanageable.

But even after setting aside the technical arguments over engineered safety and the ECCS, it is important to recognize that in our troubled world terrorist activities are almost daily occurrences. Sabotage of a nuclear power plant is a real possibility. Aircraft hijackers have already made such a threat against the reactor at the Oak Ridge National Laboratory. The December 21, 1972 issue of *Nucleonics Week* carried a news item concerning threats against nuclear plants in England by the Scottish Nationalists and by the Irish Republican Army. An Associated Press dispatch dated March 27, 1973 from Buenos Aires stated:

> Troops and police searched yesterday for a team of guerrillas who seized an atomic plant under construction and made off with the weapons of its sentries.

The real possibility of a catastrophic accident was well recognized by the AEC and the Joint Committee on Atomic Energy in the fledgling years of the nuclear industry. Rather than push for the certainty of safety, they pushed to protect the industry, not the public, by sponsoring the Price-Anderson Act. This act limits the liability of a nuclear plant to only $560 million of the potential $7 billion of damage (seven cents on a dollar). And, Mr. or Ms. Citizen, you have no other recourse, because your homeowner's policy contains a nuclear exclusion clause. Eighty-five percent of this limited liability has to be underwritten by the United States government because of the timidity of the private insurance industry.

The operation of a nuclear reactor produces vast quantities of lethal radioactive fission products, the same materials that constituted the fallout from nuclear weapons tests. These radionuclides remain dangerous and must be isolated from the environment for a period of more than a thousand years. The Atomic Energy Commission's long-intended answer to the radioactive waste problem involved the use of abandoned salt mines at Lyons, Kansas, as a repository for the lethal fission products. In 1970 an investigation of the site by the Kansas Geological Survey found it to be the leakiest salt mine in the world. Their findings presented the strong possibility that radioactive wastes in the salt mines could contaminate ground water supplies and, accordingly, the AEC was forced to abandon its plans. The government's present interim plan is for above-ground engineered storage of the waste for an interim period of at least 25 years in the hope that by then some solution to this problem will have been found.

In May, 1966, the National Academy of Sciences' Committee on Geological Aspects of Radioactive Waste Disposal submitted a report to the AEC critical of various aspects of AEC practices. For example, the committee stated that *none* of the AEC radioactive waste storage sites was in a satisfactory geological location. The committee also criticized the practices related to low- and intermediate-level liquid wastes and all kinds of solid waste.

. . . it is important to recognize that in our troubled world terrorist activities are almost daily occurrences. Sabotage of a nuclear power plant is a real possibility. Aircraft hijackers have already made such a threat against the reactor at the Oak Ridge National Laboratory.

The AEC has done little in response to this critical report. In fact, this nuclear priesthood suppressed the report for three years until Senator Frank Church (D-Idaho) in a floor speech demanded that it be made public. Just last year, 115,000 gallons of high level radioactive wastes leaked into the ground at the AEC's Hanford, Washington, facility over a period of 51 days during which the staff failed to monitor the tanks. We are now some 30 years into the Atomic Age but have yet to devise a safe means for handling these radioactive wastes which must be guarded for a thousand years.

The most pernicious product of the nuclear industry is plutonium-239. This element is not native to Earth. The entire present-day inventory is man-made. Plutonium-239 will remain deadly not for 1,000 years but for some 500,000 years. Plutonium is also the most toxic material

known to man. One millionth of a gram (there are 28 grams to an ounce) of plutonium has been shown capable of producing cancer in animals. The biological evidence suggests that the radiation protection standards of the AEC for plutonium are far too lax. Last February 14 the Natural Resources Defense Council petitioned the AEC to reduce its present maximum permissible plutonium exposure levels by a factor of 115,000!

Besides being extremely hazardous because of its radioactivity, plutonium is also a substance from which atomic bombs can be made without elaborate technology. In the projected breeder reactor economy there will be some 10,000 tons of plutonium in the commercial sector of society and tons of plutonium would be in transit every day. Just some 10 pounds would be sufficient to fashion a crude atomic bomb in a basement workshop. The information required to fabricate such a bomb became unclassified years ago. In an age of aircraft hijacking and the ransom of diplomats and Olympic athletes the risks of such diversion, and consequent nuclear blackmail, are not minimized even by ardent nuclear energy supporters. Thus, Atomic Energy Commissioner Clarence Larson recently stated:

> Once special nuclear material is successfully stolen in small and possibly economically acceptable quantities, a supply-stimulated market for such illicit material is bound to develop. And such a market can surely be expected to grow once the source of supply has been identified. As the market grows, the number and size of thefts can be expected to grow with it, and I fear such growth would be extremely rapid once it begins. . . . Such theft would quickly lead to serious economic burdens to the industry, and a threat to the national security.

The breeder reactor decision is literally a decision for all men and all time. It is far too important to have been made with so little public and congressional awareness.

The history of the peaceful nuclear priesthood is certainly no formula for the future. Its present-day light water reactor safety program was allowed to be preempted by commercial interests and budgetary restrictions. The decision to commit this nation to the fast breeder reactor may prove to be the most significant technological decision since the Manhattan Project. Its importance notwithstanding, this commitment was not the end product of a rational and public decision-making process. Rather, it evolved over 25 years of increasing industrial and governmental effort to develop an inexpensive and unlimited source of energy. The commitment was made, moreover, in the face of mounting apprehension within the scientific community concerning the human and societal hazards of fission reactors, apprehension which would only be compounded by the liquid metal fast breeder reactor program.

As evidence of this apprehension among scientists, a statement of concern over the environment and world peace (the Menton Statement) was signed by 2,200 scientists. The statement included a call for an end to the proliferation of nuclear reactors. It was presented to U. N. Secretary General U. Thant and published in the *U. N. Courier* for July, 1971. Scientists from all nations at the 23rd Pugwash Conference on Science and World Affairs last September concluded:

> 1. Owing to potentially grave and as yet unresolved problems related to waste management, diversion of fissionable material, and major radioactivity releases arising from accidents, natural disasters, sabotage, or acts of war, the wisdom of a commitment to nuclear fission as a principal energy source for mankind must be seriously questioned at the present time.
>
> 2. Accordingly, research and development on alternative energy sources—particularly solar, geothermal and fusion energy, and cleaner technologies for fossil fuels—should be greatly accelerated.
>
> 3. Broadly based studies aimed at the assessment of the relation between genuine and sustainable energy needs, as opposed to projected demands, are required.

The problems associated with the present reactor program strongly suggest that we are only perpetuating and compounding a bureaucratic blunder by pursuing the LMFBR program. The breeder reactor decision is literally a decision for all men and all time. It is far too important to have been made with so little public and congressional awareness.

As a result of a lawsuit brought by the Natural Resources Defense Council on behalf of the Scientists' Institute for Public Information, the AEC is now preparing an environmental impact statement on the LMFBR program. This will undoubtedly increase the tempo of the growing national and international debate over nuclear reactors. It is to be hoped that at the very least this debate will result in much more substantial funding of research and development on the alternative energy sources mentioned by the Pugwash Scientists as well as on programs of energy conservation.

N. Y. TIMES EDITORIAL 1/10/75

TOXIC CARELESSNESS

In dealing with a substance as deadly as plutonium, it is not enough merely to minimize the risks. The Atomic Energy Commission and its successor agency, the Nuclear Regulatory Commission, have the urgent task of tightening controls over the use and disposition of this toxic by-products in the nation's installations.

The dangers from plutonium are infinite. Carelessly handled, even in minute quantities, it can cause death and disease. With only modest expertise, a few pounds of the lead-like element can be fashioned into a crude but highly explosive nuclear weapon. In the light of these potentialities, reports that thousands of pounds of highly enriched uranium and plutonium are missing from the inventories of the nuclear industry require something more than generalized denials. A more dozen pounds, which industry executives concede may have been unaccounted for from time to time in the past year, would be devastating in the hands of a terrorist group of hostile government.

Pressure is building up inside the nuclear industry for a Federal decision to authorize the use of plutonium as a fuel for civilian power plants to augment the dwindling supply of uranium fuel. It is inconceivable that responsible policymakers could permit wider dispersal of plutonium for any purpose as long as even marginal dangers of nuclear theft remain.

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1975/

January 21, 1975

232 Howland Canal
P.O. Box 249
Venice, California 90291

Dr. William A. Mills, Director of
Criteria & Standards Division (AW-560)
Office of Radiation Program
Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

Please make the following statement of my concern for plutonium emission standards a part of the official hearing record on these standards.

I am a former nuclear physicist who did some of the first work on the Liquid Metal (sodium) Fast Breeder Reactor while employed at Atomics International, Canoga Park, California, in the early 1960s. I am more aware than most of the terrible and intolerable dangers that even minute amounts of plutonium present to life on this planet, as we know it, when released into the planetary environment. Plutonium is one of the most toxic of all of the environmental poisons in minute amounts, and is extremely long lived by human time standards.

A very small AMOUNT of plutonium could fairly easily be fashioned into a crude fission device. It seems to me inevitable that someday in spite of all possible precautions, we will be faced with world wide nuclear terrorism if we continue to manufacture this substance.

I strongly urge that not only should EPA rule that a standard of ZERO plutonium emission be adopted, but that the entire LMFBR program be terminated because of the 100% probability of massive environmental damage in the future.

Thank you.

Yours cordially,

Ron Guenther

7500 g Terrace View Apts.
Blacksburg, Virginia 24060
January 22, 1975

Dr. William A. Mills
Director of Criteria & Standards
Division (A-W-560)
Environmental Protection Agency
Washington, D.C. 20460

Dear Dr. Mills:

I would like you to make the following comments to be part of the hearings on environmental plutonium standards which are being held by the E.P.A. in Denver, Colorado.

I would like to go on record as being opposed to Nuclear Fission plants in light of their potential dangers to the environment and mankind. From what I understand, I do feel that they are immune to the possibility of causing nuclear explosion, however my main concern deals with the dangers posed by their lethal wastes. Many questions come to my mind when considering the problem of transportation and disposal of such wastes. A substance which has a half life of 24,000 years and is as dangerous as Plutonium is too hazardous to fool around with. I know that I do not want this substance transported through my town via truck, train, or any other means of transportation which is subject to an accident. Nor do I want it to be disposed of anywhere near my place of residence, work or recreation.

It is my understanding that the number one cause of death among children under age fifteen is cancer. I just hope that the nuclear fission plants that have existed in the past are not a contributing factor to this statistic.

Sincerely,

Vincent Pedicini

Vincent Pedicini

3667 Summit, Apartment 3
Kansas City, Missouri 64111
January 24, 1975

Dr. William A. Mills, Director of
Criteria and Standards Division (AW-560)
Office of Radiation Program
Environmental Protection Agency
Washington, D. C. 20460

Dear Sir:

As concerned citizens, we should like to submit the following comments on nuclear power plants and plutonium emission standards. We appreciate the opportunity to air our views, but we feel that the hearings on plutonium emission standards, and on other nuclear power issues, should be more widely publicized since nuclear power is a considerable threat to all Americans. The Environmental Protection Agency is the logical governmental agency to perform such a vital function. In the future, we hope that the EPA will live up to its information dissemination responsibilities.

Plutonium is the most toxic substance known to man. Atomic Energy Commission nuclear power plants have a well-deserved reputation for nuclear "accidents" involving plutonium emissions; Rocky Flats, Detroit's Fermi fast breeder, the Northeastern Utilities Connecticut Yankee plant, the Vermont Yankee plant, and the plant near Hanover, Washington, to name a few. A moratorium must be declared until nuclear technology has advanced to the point that plutonium emission accidents are no longer possibilities, much less probabilities.

The emergency core cooling system, the only system that might prevent a complete core meltdown, has never been effectively tested. With the advent of the liquid metal fast breeder reactor, which the Atomic Energy Commission is pushing so strongly, and the subsequent plutonium-recycling program, the possibilities of a core meltdown become truly horrifying. Even the AEC's damage estimates, when it concerns itself with the reality of a complete meltdown, concede that human and property losses will far exceed the limits established by the Price-Anderson Act. We feel that, if for no other reason, a moratorium should be established until full insurance coverage will be provided in the event of any loss-of-coolant accident and subsequent meltdown.

All of the factors involved in a possible accident can be expected to increase in potential, given the current goals of the AEC and the major utility companies. A core meltdown, with the right weather conditions, near a large city, could produce the worst casualty in the history of the United States.

We feel that, with plutonium, there are no acceptable emission standards.

Sincerely,

Robert D. Haun

Robert D. Haun

Tom Jennison

Tom Jennison

Diane Ezell

Diane Ezell

654

COLORADO DEPARTMENT OF HEALTH

4210 EAST 11TH AVENUE · DENVER, COLORADO 80220 · PHONE 388-6111

Edward G. Dreyfus, M.D., M.P.H. Executive Director

January 23, 1975

William Mills, Ph.D., Director
Standards and Criteria Branch (AW-56)
Office of Radiation Programs
U.S. Environmental Protective Agency
Washington, D.C. 20460

Dear Dr. Mills:

During the presentation of the Colorado Department of Health's testimony before the hearing panel in Denver, you requested that references be submitted regarding statements made in testimony. In this regard the following is submitted:

1) "The Problem of Large-Area Plutonium Contamination" from Selected Papers from the Bureau of Radiological Health Seminar Program. Seminar Paper No. 002 DHEW, PHS, CPEHS, ECA. (1968 paper of W. H. Langham, Ph.D.)

2) "Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads" Lawrence Radiation Laboratory publication UCRL-50639 (1969 paper of W. H. Langham, Ph.D.)

3) "Toward Interim Acceptable Surface Contamination Levels for Environmental Plutonium Oxide" by R. L. Kathren, April, 1968, BNWL-SA-1510.

4) "A proposed Interim Standard for Plutonium in Soils" by J. W. Healy, Los Alamos Scientific Laboratory LA-5483-MS Informal Report UC-41, January, 1974.

5) Transcript of hearing before the Colorado State Board of Health held February 14, 1973, and continued March 21, 1973.

As the representative from the city of Boulder and Dr. Carl Johnson, Jefferson County Health Department, were concerned with emergency response plans for the U.S.AEC's Rocky Flats Plant, please find enclosed a copy of a portion of the state plan for such a contingency. As this plan is under revision, a copy of the draft revision is also enclosed. Copies have been provided to these two individuals.

Questions were asked of me by Dr. Morgan and you relating to resuspension of the plutonium soil contamination. In this regard, the following is provided:

1) Tables of monthly average plutonium 239 and 240 air concentrations observed at CDH on-site sampling stations.

2) Maps locating air sampling stations on-site and in the Denver metropolitan area.

3) A graph depicting the annual average plutonium 239 and 240 air concentrations versus time observed at CDH on-site sampling stations.

The graph illustrates that revegetation and traffic curtailment have been effective in the D-3 and 4 area (immediately adjacent to the oil spill area where the approximate "average" soil concentration is 1 microcurie Pu per square meter soil surface). The highest contaminated areas are to be removed later this year. The levels observed over this time period at APC-56, D-1 and 2 identify that soil disturbances in this area (construction of waste water handling facilities in 1972), did indeed affect air concentrations.

Manipulation of the data provided in the tables identifies an air concentration half-life for this "old" deposition at station D-5 (0.1 microcurie Pu per square meter, soil surface) of between 4.4 years, for world-wide fallout Pu plus resuspension to 1.5 years, for resuspension only, using one concentration for world-wide fallout Pu for the entire period of time involved. The most reasonable air concentration half-life would approximate 2.7 years for resuspension only, using a 9.6 year half-time for world-wide fallout. From the above data a resuspension term, based on annual average air concentrations and the soil concentrations associated with stations D-3 and 4 and D-5, of 5×10^{-9} to 5×10^{-10}/M is identified.

The testimony provided by Mr. Earl Bean, USAEC-RFAO referenced world-wide fallout Pu levels for all their air sampling stations, which is what is being observed now (off-site). However, it must be pointed out that two stations located east of the plant site (SS-32 and SS-33) are located on "clean ground" in the wind-strewn field of the plutonium contamination. Station SS-32 is located at the intersection of Indiana Street and the Plant's east access road. The surrounding soil has been redistributed and reworked, and the major wind pattern corresponds to the layout of the east access road. Station SS-33 is located adjacent to a plowed agricultural field. Therefore, it would be expected that these stations would observe world-wide fallout levels. These stations cannot be justifiably used to illustrate that there is no resuspension of contaminated soil to an airborne hazard potential. These stations do illustrate that locations having equal or slightly greater than 2 disintegrations per gram of soil upon mixing (dilution of the soil) by plowing or land surface reworking will result in air concentrations equal to that of world-wide fallout.

Also enclosed is a retyped version of the Department's presentation of January 10, and a letter the Department received from Shelley Barthel.

Again, the Department wishes to express its appreciation for the bringing of the hearing to Denver and the privilege of participation. The efforts of EPA, both the Regional office and the Las Vegas facility, in identifying various situations at the Department's request is appreciated.

Should you require additional information or need clarification on items already provided, please advise.

Sincerely,

Albert J. Hazle
Director
Occupational and Radiological
Health Division

AJH:bs
Enclosures

cc: Paul B. Smith, EPA, Region VIII

239 + 240
Pu pCi/m^3

ON-SITE LOCATIONS

	1970	1971	1972	1973	1974	1975
			D-1			
JANUARY	---	.00020	.00020	.00119	.00037	
FEBRUARY	---	.00029	.00018	.00034	.00022	
MARCH	---	.00030	.00035	.00046	.00015	
APRIL	---	.00034	.00021	.00051	.00042	
MAY	.00042	.00045	.00177	.00101	.00058	
JUNE	.00039	.00051	.00277	.00322	.00035	
JULY	.00019	.00020	.00133	.00014		
AUGUST	.00008	.00022	.00189	.00059		
SEPTEMBER	.00024	.00038	.00351	.00017		
OCTOBER	.00020	.00019	.00258	.00059		
NOVEMBER	.00017	.00032	.00066	.00038		
DECEMBER	.00025	.00027	.00183	.00027		
AVERAGE	.00024	.00031	.00144	.00074		
			D-2			
JANUARY	---	.00010	.00020	.00053	.00007	
FEBRUARY	---	.00045	.00022	.00079	.00044	
MARCH	---	.00036	.00030	.00016	.00046	
APRIL	---	.00036	.00016	.00365	.00046	
MAY	.00091	.00063	.00024	.00026	.00071	
JUNE	.00042	.00048	.00047	.00038	.00269	
JULY	.00025	.00031	.00073	.00016		
AUGUST	.00024	.00032	.00175	.00012		
SEPTEMBER	.00021	.00019	.00126	.00049		
OCTOBER	.00019	.00027	.00178	.00062		
NOVEMBER	.00046	.00010	.00045	.00010		
DECEMBER	.00031	.00055	.00067	.00018		
AVERAGE	.00037	.00034	.00069	.00062		

239 + 240 Pu pCi/m^3

ON-SITE LOCATIONS

D-3

	1970	1971	1972	1973	1974	1975
JANUARY	---	.00689	.00240	.00203	.00010	
FEBRUARY	---	.02048	.00522	.00439	.00128	
MARCH	---	.00552	.00367	.00326	.00130	
APRIL	---	.00768	.00138	.00128	.00257	
MAY	.03300	.00844	.00245	.00210	.00748	
JUNE	.00530	.00592	.00655	.00522	.00442	
JULY	.00650	.03742	.00299	.00325		
AUGUST	.00160	.00418	.00458	.00477		
SEPTEMBER	.00100	.00316	.01410	.00317		
OCTOBER	.00097	.00534	.00652	.00154		
NOVEMBER	.00051	.01639	.00094	.00229		
DECEMBER	.00110	.00202	.00096	.00201		
AVERAGE	.00625	.01029	.00470	.00294		

APC-56

	1969	1970	1971	1972	1973	1974	1975
JANUARY	.00900	.00051	.00163	.00053	<.00008	.00003	
FEBRUARY	.00490	.00328	.00207	.00054	<.00008	.00015	
MARCH	.00299	.00049	.00388	.00353	.00090	.00015	
APRIL	.00310	.00076	.00069	.00039	NA	.00011	
MAY	.00650	.00079	.00043	.00037	.00030	.00048	
JUNE	.00124	.00330	.00092	.00040	.00047	.00036	
JULY	.00071	.00051	.00043	.00035	.00032		
AUGUST	.00166	.00086	<.00008	.01678	.00114		
SEPTEMBER	.00115	.00026	<.00008	.00128	.00020		
OCTOBER	.00037	.00102	.00859	.00230	.00011		
NOVEMBER	.00057	.00210	.00223	.00019	<.00008		
DECEMBER	.00057	.00058	.00025	.00071	<.00008		
AVERAGE	.000273	.00120	.00177	.00228	.00037		

239 + 240
Pu pCi/m^3

ON-SITE LOCATIONS

	1970	1971	1972	1973	1974	1975
			D-4			
JANUARY	---	.00076	.00211	.00263	.00044	
FEBRUARY	---	.01242	.00211	.00742	.00061	
MARCH	---	.00893	.00243	.00690	.00128	
APRIL	---	.00369	.00164	.00144	.00159	
MAY	.00440	.02758	.00152	.00220	.00338	
JUNE	.00082	.00419	.02256	.00428	.00223	
JULY	.00091	.00334	.00575	.00238		
AUGUST	.00660	.00506	.00784	.00391		
SEPTEMBER	.00107	.00731	.00613	.00191		
OCTOBER	.00067	.02485	.01378	.00197		
NOVEMBER	.00152	.00758	.00153	.00095		
DECEMBER	.00170	.00236	.00322	.00287		
AVERAGE:	.00221	.00734	.00588	.00324		

660

239 + 240 Pu pCi/m^3

OFF-SITE LOCATIONS

			D-5		
JANUARY	---	.00008	.00013	.00036	.00008
FEBRUARY	---	.00011	.00008	<.00002	.00003
MARCH	---	.00020	.00007	.00011	.00008
APRIL	---	.00015	.00007	.00010	.00013
MAY	---	.00024	.00006	.00010	.00019
JUNE	.00022	.00024	.00013	.00013	.00036
JULY	.00014	.00013	.00009	.00005	
AUGUST	---	.00012	.00015	.00017	
SEPTEMBER	---	.00016	.00010	.00010	
OCTOBER	---	.00009	.00047	<.00002	
NOVEMBER	---	<.00008	<.00002	.00003	
DECEMBER	.00011	.00005	.00009	<.00002	
AVERAGE	.00016	<.00013	<.00012	<.00010	

239 + 240 Pu pCi/m^3

OFF-SITE LOCATIONS

	1969	1970	1971	1972	1973	1974	1975
APC-2 DENVER (EPA DATA)							
QUARTERS							
FIRST	.00007	.00006	.00006	.00006	.00002	.00004	
SECOND	.00011	.00013	.00013	.00006	.00003		
THIRD	.00006	.00009	.00007	.00002	.00001		
FOURTH	.00004	.00003	.00002	.00002	.00001		
AVERAGE	.00007	.00008	.00007	.00004	.00002		
APC-15 ARVADA							
FIRST	---	.00018	.00006	.00003	<.00008	.00045	
SECOND	---	.00017	.00019	.00005	---		
THIRD	---	.00022	.00006	.00003	<.00002		
FOURTH	---	.00005	<.00002	.00012	.00014		
AVERAGE	---	.00016	<.00008	.00006	<.00006		
APC-16 GOLDEN							
FIRST	.00013	.00008	.00005	<.00002	<.00002	.00010	
SECOND	.00010	.00025	.00017	.00008	.00016	.00013	
THIRD	.00006	.00008	.00008	.00003	.00004		
FOURTH	.00004	<.00003	<.00002	.00012	<.00002		
AVERAGE	.00008	<.00011	.00008	<.00006	<.00006		

239 + 240
Pu pCi/m^3

OFF-SITE LOCATIONS

	1969	1970	1971	1972	1973	1974	1975
APC-19 BOULDER							
FIRST	---	.00012	.00005	<.00002	<.00002	.00010	
SECOND	---	.00018	.00013	.00005	.00005	.00009	
THIRD	---	.00013	.00007	<.00002	.00014		
FOURTH	---	.00010	<.00002	.00010	<.00002		
AVERAGE	---	.00013	<.00007	<.00005	<.00006		
APC-22 LONGMONT							
FIRST	.00006	.00007	.00003	<.00002	<.00002	.00010	
SECOND	.00007	.00020	.00014	---	.00015	.00024	
THIRD	.00010	.00015	.00007	<.00002	<.00002		
FOURTH	.00008	.00005	.00004	<.00002	<.00002		
AVERAGE	.00008	.00012	.00007	<.00002	<.00005		
APC-29 DURANGO							
FIRST	---	---	---	.00019	.00047	.0005	
SECOND	---	---	---	<.00002	.00030	.00015	
THIRD	---	---	---	<.00002	.00005		
FOURTH	---	---	---	.00012	<.00002		
AVERAGE	---	---	---	<.00012	<.00002		

$^{239+240}$Pu pCi/m^3

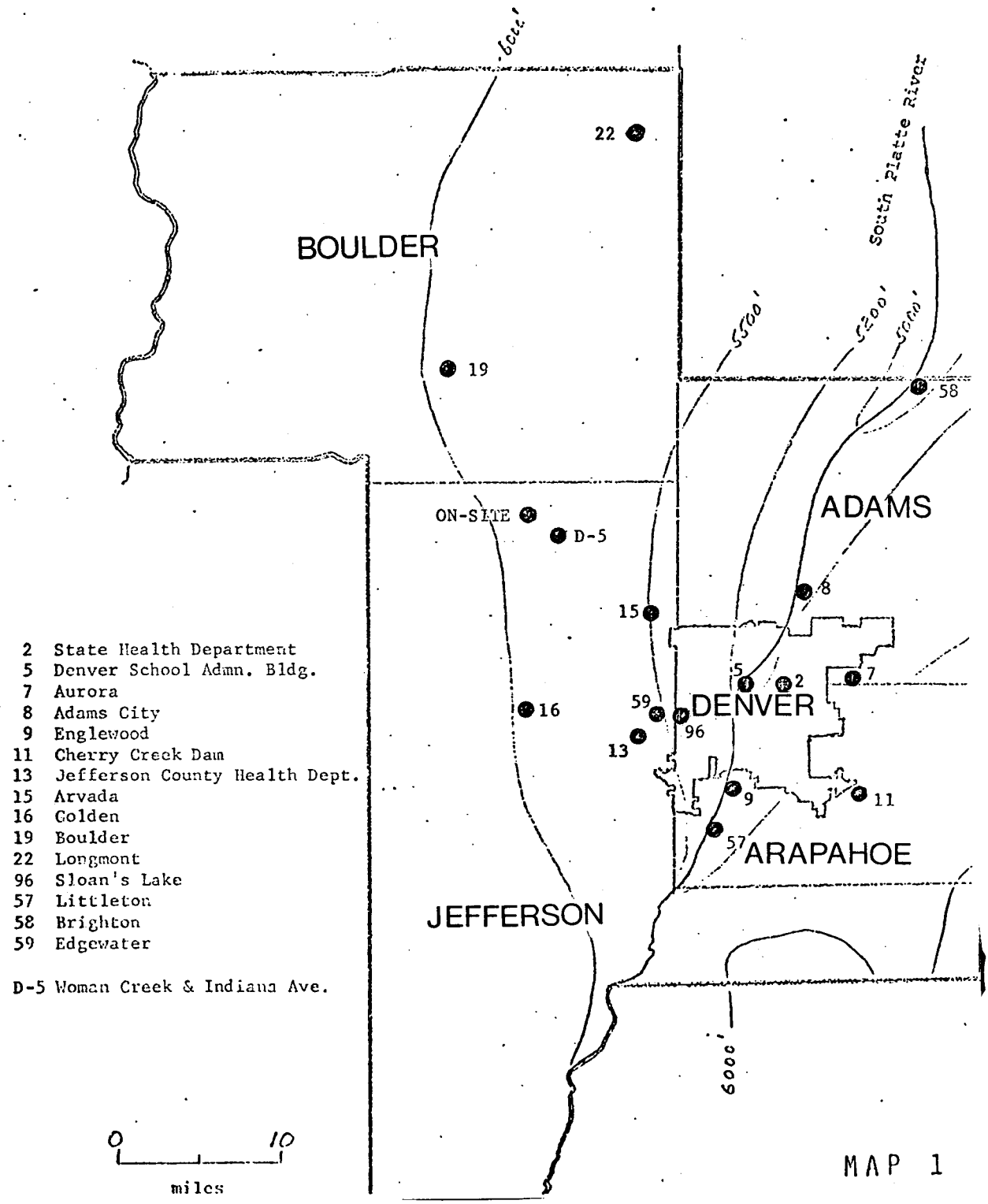
OFF-SITE LOCATIONS

	1969	1970	1971	1972	1973	1974	1975
APC-42 FT. COLLINS							
QUARTERS							
FIRST	---	---	---	<.00002	<.00002	.00015	
SECOND	---	---	---	.00004	.00006	.00015	
THIRD	---	---	---	.00007	<.00002		
FOURTH	---	---	---	<.00002	.00019		
AVERAGE	---	---	---	<.00004	<.00008		
APC-81 WALSENBURG							
FIRST	---	---	---	.00006	<.00002	.00014	
SECOND	---	---	---	.00011	.00074	.00017	
THIRD	---	---	---	.00008	.00003		
FOURTH	---	---	---	.00003	.00014		
AVERAGE	---	---	---	.00007	<.00006		
APC-108 RANGELY							
FIRST	---	---	---	.00006	<.00002	.00016	
SECOND	---	---	---	.00008	.00009	.00016	
THIRD	---	---	---	.00008	.00017		
FOURTH	---	---	---	.00020	<.00002		
AVERAGE	---	---	---	.00010	<.00008		

9.

1972

OFF-SITE AIR PARTICULATE
SAMPLING LOCATIONS

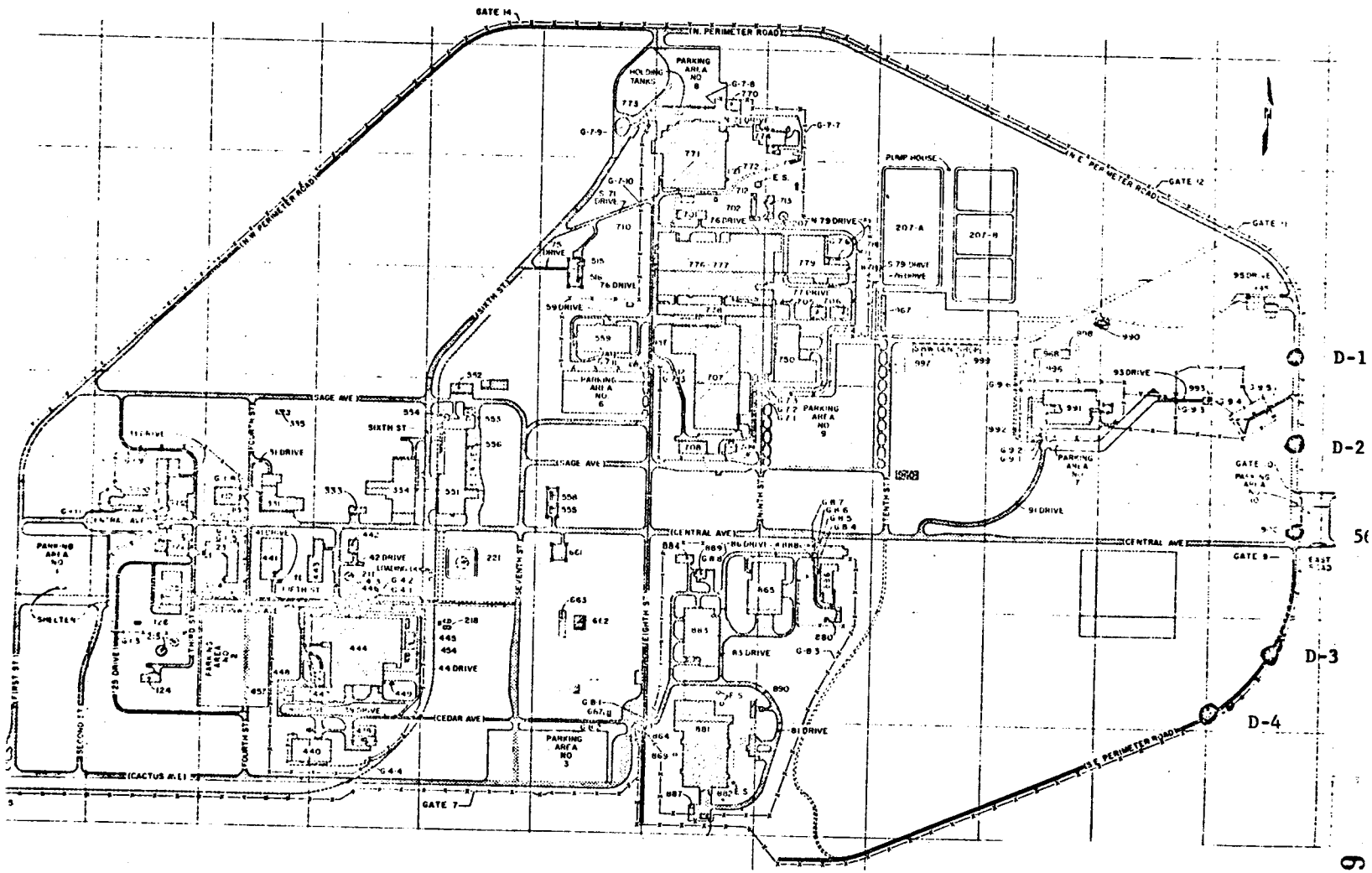


2 State Health Department
5 Denver School Admn. Bldg.
7 Aurora
8 Adams City
9 Englewood
11 Cherry Creek Dam
13 Jefferson County Health Dept.
15 Arvada
16 Golden
19 Boulder
22 Longmont
96 Sloan's Lake
57 Littleton
58 Brighton
59 Edgewater

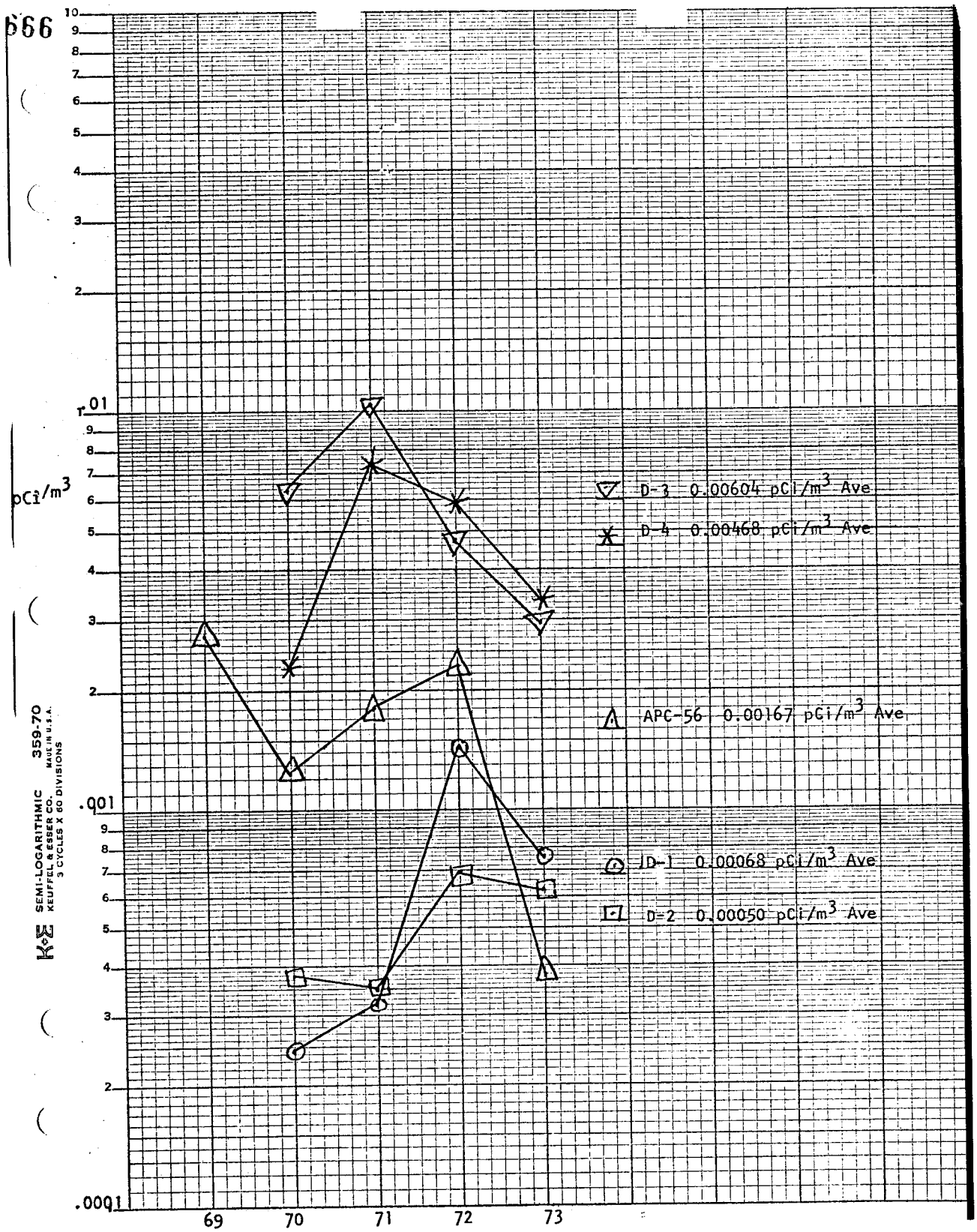
D-5 Woman Creek & Indiana Ave.

1972

ON - SITE AIR SAMPLING STATIONS



MAP 2


pCi/m3
10
9
8
7
6
5
4
3
2
.01
.001
.0001
69
70
71
72
73
D-3 0.00604 pCi/m3 Ave
D-4 0.00468 pCi/m3 Ave
APC-56 0.00167 pCi/m3 Ave
JD-1 0.00068 pCi/m3 Ave
D-2 0.00050 pCi/m3 Ave
K+E SEMI-LOGARITHMIC 359-70
KEUFFEL & ESSER CO. MADE IN U.S.A.
3 CYCLES X 60 DIVISIONS

1380 - 19th Street, #3
Boulder, Colorado 80302
January 8, 1975

Mr. Albert J. Hazle, Chief
Occupational and Radiological Health Division
Colorado Department of Health
4210 East 11th Avenue
Denver, Colorado 80220

Dear Mr. Hazle:

Because I must work, I am unable to attend the public hearing to be held Friday, January 10th regarding the Rocky Flats nuclear installation.

As a concerned citizen, I therefore respectfully request that the following brief statement be entered into the record on my behalf.

I am an eight year resident of Colorado, age 31. I am a certified elementary school teacher, planning to marry this year and to raise our children here.

I oppose and protest the philosophy which causes the existence of Rocky Flats. The United States does not need to produce or possess nuclear bombs ---especially since we intend never to use them. We already possess a more-than-adequate stockpile of conventional weapons and bombs for use as a deterrent. Nuclear bombs as deterrents are dangerous, and serve no rational purpose. The vast resources which are now wasted on these nuclear bombs should rather be spent toward solving our serious, present social and economic problems.

I appreciate the opportunity to express my opinion; and, hope that someday my rationale will form part of the basis for decisions made in the public's behalf. Thank you.

Sincerely,

Shelley Barthel

Shelley Barthel

Statement to be read at the Environmental Protection Agency Public Hearing Jan 10, 1975

The AEC has not effectively protected our environment - one example being the radioactive tailings found in some Grand Junction Houses. In the absence of provable facts as to harmful radiation levels, we therefore urge more stringent standards imposed at this time for nuclear weapons facilities and nuclear reactors than have been established in the past.

Guy Sink _ 4720 S. Ogden Englewood, Colo
Ellen L. Schwan 2750 So. Washington " "
Vera Mullhauser 11800 W. 29th Place, Lakewood.
Anna Lee Briggs 510 S. Franklin Denver 80209
Margo Sargent 2806 S. Paris St. Denver 80232
Cynthia Zellmer 2916 S. Paris St Denver 80232
William A. Zellmer 2916 S. Paris St Denver 80232
[illegible] 2806 S. Paris St., Denver, Colo

Byron J. Richards	1090 W. Oxford Ave.	Englewood 80110
Dick Richards	1090 W. Oxford Ave	Englewood 80110
Wyona H. Rowe	485 E. Bellwood Dr.	Eng 80110
Sandy L. Whitaker	3771 S. Clinton	Denver 80237
Ronell Hoppes	3801 E. Alexander Ave.	Littleton 80121
Marcella Stewart	2445 So. Dahlia St.	Denver 80222
Gertrude Alice Guiet	2160 S. St. Paul	Denver 80210
Jean C. Guiet	2160 So. St Paul	Denver 80210
Lucille Graham	6378 So Josephine Cy	Littleton 80121
Robert F. Shumaker	1201 RACE ST	DENVER 80206
Vernon L Tompkins	50 Odgen St	Denver 80218
Mack Curran	122 Stuart St	" 80211
E. R. Stoll	2886 So Corona	Englewood 80110
John C Yingst	8600 W Bowles Ave	Littleton 80123
Charlotte Krause	1403 S. Eudora	

Don Butterfield	4646 Montview	Denver	80207
John H. Bove	2555 S. Wolff	Denver	80219
H. Morgan	720 S. Hatfield	Denver	80209
Carolyn Morgan	720 S. Hatfield	"	80209
Robert M. Eddy	6594 So Clarkson	"	80121

Minister, 1st Universalist Church of Denver, 4101 E. Hampden Ave.

Irvin B. Appenzeller	2710 Wheeling	Aurora	80011
Louis Parry	999 So. Fillmore Way	Denver	80209
Edward White III	545 Circle Dr	Denver	80206
James White	545 circle drive	Denver	80206
Evelyn E White	4801 So Ogden	Englewood	80110
E. D. White, Jr.	480 S. Marion	Denver	80209
Mary Ann Dority	1639 S. Kearney	Denver	80222

take peace

Mr. & Mrs. C. A. Webber, Jr.
610 S. Elm Street
Greenville, N. C. 27834

Dr. W.D. Rowe
Radiation Programs
EPA
Washington, DC 20460

Dear Dr. Rowe:

It seems to me criminal that we should foul the world we live in, that we should risk leaving a world perhaps not even habitable by people.

The ironic thing is that we are endangering our own health and that of our descendants for things that are really undesirable. I'm quite comfortable in our house at 60° now — much more so than I'd be if the furnace were up at 80°. I am healthier for doing most of my errands by bicycle or on foot. And the extravagant use of energy by the military — in Indochina & Greece for example — buys repressive governments

Edith Webber

49 South Main Street
Southampton, NY 11968

January 27 1975

Dr W D Rowe
Radiation Programs
E P A
Washington, DC 20460

Dear Dr Rowe,

I learn from the Task Force Against Nuclear Pollution, Inc, that you are asking for "viewpoints of concerned citizens as they pertain to the problems of plutonium in the environment".

My viewpoint is that the most important challenge to civilization and to life on this earth is presented to everybody by the proposal of government and industry to build nuclear energy plants.

Dr Gerald A Drake, of Petoskey. MI, says, and is worth quoting, "If the tubercle bacilus, or virus of a potentially fatal form of encephalitis packed tremendous energy for fueling electrical plants, would it be rational to culture these organisms by the ton and ship them around the nation and the world for such a purpose? Hardly, and yet they are not nearly as deadly as plutonium 239."

We need energy. Well pretty soon we will need food. How about solving famine and the population explosion at one stroke by killing and canning the surplus population and shipping the meat to the famine striken areas of the world?

I think the U S and the whole world is at the mercy of and in the power of the insane.

The insane are,among others, but chiefly, the proponents of nuclear energy.

There is nothing left but hope.

Sincerely yours,

Fairfield Porter

Fairfield Porter

470 Windridge Drive
Racine, Wis. 53402
February 1, 1975

Dr. William A. Mills
Dir. of Criteria and Standards Div. (AW-560)
Office of Radiation Program
EPA
Washington, D.C. 20460

Dear Dr. Mills:

This letter is in response to requests for comments on the amount of Plutonium to be allowed in our environment.

For several years our group has been concerned with the environmental and health hazards of nuclear power plants. So me of our main concerns are those involving the use of Plutonium.

We feel that the use of this element should be stopped completely until all safety problems connected with its recycling and storage are solved. With the worldwide proliferation of nuclear plants, the possibilities for theft and blackmail become so numerous that it is only a question of time before terrorists carry out some tragedy. This could occur with a bomb or just by threatening to disperse a small amount of Plutonium into the air, causing cancer in millions of people.

Any substance as hazardous as Plutonium should not be put into our world for all future generations to guard. The possibility of armed fortresses and convoys necessary to guard the manufacture and transportation and storage of Plutonium should make any responsible informed person reject nuclear power plants and all they involve. It is morally wrong and incomprehensible to us that a supposedly civilized society is even debating the issue.

With any real commitment to alternate power sources and needed changes in our housing codes, air-conditioners, automobiles, and with recycling of our resources, there would be no need for these hazardous plants. The result of positive action on the above would help the people in this country regain some confidence in the future.

Yours truly,

Jean Reid

Jean Reid
Edee Sobel, Co-Chairmen
Racine-Kenosha Citizens for
the Environment

CC: President Gerald Ford
Nelson Rockefeller
Gaylord Nelson
William Proxmire
Les Aspin
Frank Zarb

674

NCRP National Council on Radiation Protection and Measurements
7910 WOODMONT AVENUE, SUITE 1016, WASHINGTON, D. C. 20014 AREA CODE (301) 657-2652

LAURISTON S. TAYLOR, *President*
E. DALE TROUT, *Vice President*
W. ROGER NEY, *Executive Director*

February 7, 1975

Dr. William A. Mills, Director
Division of Criteria and Standards
Radiation Office
Environmental Protection Agency
Waterside Mall E-635
401 M Street, S.W.
Washington, D.C. 20460

Dear Bill:

Referring to your telephone conversation the other day, I suggested the possibility that a study of the hot particle problem by the NCRP might be submitted for record relative to the EPA hearings which were completed in Denver on January 10th.

It now appears that this report may not be ready for release within the prescribed time for "open record".

As you know, any report prepared by a committee of the NCRP has to be reviewed by the total Council membership and all comments considered before the report is released. Our committee has completed a report entitled, "Alpha Emitting Particles in the Lung" and this is now under review by the Council. While some two-thirds of the members have approved the report and none have disapproved it, we still feel compelled to receive positive replies from the remaining members. For this reason it is impractical to have this in your hands by February 10th. We expect the report to be published soon after that date and we will, of course, make copies available to you.

You are no doubt aware that this report is one of several that we have in various stages of completion at the present time and dealing with certain problems of environmental and occupational exposure to radiation. Another report dealing with plutonium in occupational exposure situation is also under review, having been completed by the Scientific Committee that prepared it.

Other studies less far along are having to do with problems of surface contamination and removal of contaminated materials.

Since we cannot have the report in your hands by the required deadline, I would appreciate it very much if you could make this letter a part of the record so that there will be knowledge of the early availability of the other reports.

Sincerely yours,

Lauriston S. Taylor

STATEMENT RELATING TO THE PROBLEMS OF PLUTONIUM IN THE ENVIRONMENT

The United States is moving into an era wherein plutonium-239 is to be a major source of electrial energy in nuclear fission reactors. Thus plutonium-239 will be a major material of national and international commerce. As much as 200 million kilograms of Pu-239 is projected as moving through the nuclear fuel cycle over the next 45 years with many opportunities for accidental release of PuO_2 as aerosols occuring over that period of time.

This radioactive substance which is to be our major fuel is among the most potent of carcinogens. As little as 3 micrograms has consistently produced lung cancer in dogs. Plutonium has a half life of 24,400 yearsso that it must be isolated from the biosphere for literally a half million years.

Plutonium-239, the material of which nuclear weapons are made will be moving in progressively increasing volume by highway, rail and water. As such it will pose not only a severe public health hazzard but also portends of a threat to the survival of politiaal freedom. The necessity of stringent measures to prevent the diversion of plutonium and its destructive use against the public by extremists groups was discussed in the Senate by Senator Ribicoff on April 30, 74 in reporting the findings of an AEC study group reviewing the then current AEC safeguard system. The organization of a national police force has been discussed and recommended. This does not bode well for the democracy.

Dr. Alvin Weinberg, former head of the AEC's Oak Ridge National Laboratory, one of fission power's chief supporters has nevertheless acknowledged the serious social implications of accepting

nuclear fission technology. His reference to the "Faustian bargain" implied in national commitment to nuclear fission power is well known. Dr. Chauncy Starr, president of the Electric Power Research Institute, commenting on leaving future generations the burden of guarding high level radioactive waste for literally hundreds of thousands of years at the World Energy Conference in Detroit last September, acknowledged that this was a sociological problem. Yet it is incredible that most of the highly intelligent persons in the commercial and scientific communities who are promoting nuclear power, are unable or unwilling to percieve the great social problems which this technology entails. This (plus AEC ground rules pertaining to intervention) is a major factor in the lack of any significant public input into this question which so seriously affects its welfare. Indeed, the public has had very little education in the ramifications of commitment to nuclear power beyond the PR efforts of the electric utility industry and of the AEC in behalf of the utility industry.

This public hearing relating to the potential adverse environmental impact of release of plutonium and other trans-uranium elements with the objective of determining whether or not new and/or additional standards are required is a fragment in the regulatory process pertaining to nuclear fission technology. As such it is a worthy but quite insufficient effort. What is needed is an intense educational campaign relating to all phases of commitment to nuclear fission power preferably conducted by the EPA which hopefully has no vested interest in this technology. This should be

followed by a national referendum on the issue of whether or not the United States should commit itself to nuclear fission power, such as is to be held in Sweden later this year,

Victor Skorapa, Jr., M.D.
4300 Lakehaven Drive, N.E.
Atlanta, Georgia 30319

February 8, 1975

EMMA HARTZLER

202 South Sixth Street
Goshen, Indiana 46526

2-8-75

Dear Sir:

There is no safe level of plutonium emissions; plutonium emission standards should be zero.

Sincerely
Emma Hartzler

Paul B. Smith
Air and Hazardous Material Division
1860 Lincoln Street
Denver, Co. 80203

Sir,

I am writing this letter as a concerned Coloradoan, living between two potentially hazardous operations, one of which is located at Platteville and the other at Rocky Flats.

Being uneducated in the field of metallurgy and radiation, I can not communicate in technical terms, I can only ask why!

Why do we as American citizens have to be the recipients of the AEC's experimental blunders i.e. "accidental and programmed" releases of plutonium into our water, soil, and air at the Rocky Flats Plant, "unexpected problems" at the Rio Blanco site, and the "accidental loss" of containers having once carried radioactive materials, onto our main highway system, I 25!

There are too many of these "accidental happenings" in the name of nuclear progress and the AEC! Is the AEC oblivious of the future environment???? Is it because of national security and/or fattening the purse of many who seem to be unconcerned about our environment for future generations?

Please consider actions which will make it more difficult for the AEC to continue their present experimental programs with hazardous materials,

Sincerely,

A most concerned citizen

680 Form 365

UNIVERSITY OF COLORADO MEDICAL CENTER

Inter-office Communication

TO Robert Siek, Chairman
Lamm-Wirth Task Force on Rocky Flats

DATE January 14, 1975

FROM John C. Cobb, M.D., M.P.H.

SUBJECT: Plutonium in Dust at Rocky Flats

I urgently request that our Task Force make a detailed study of the circumstances surrounding the contamination of soil from leaking drums containing plutonium in cutting oil at Rocky Flats. I have heard from various sources that there may be even hundreds of curies of plutonium in the soil where the oil drums leaked and that the soil samples from the area immediately east of the spill have been recorded at levels as high as 40,000 disintegrations per minute per gram of soil. I also understand that the AEC is planning to dig up this contaminated soil for shipment elsewhere.

I am concerned about the present danger to employees at the plant and to citizens of Colorado from wind-blown dust containing plutonium coming from this source. I understand from reading the reports CER 71-72 RNM-FC-45 and CER 72-73 RNM-JAP-TGH-16, dated May 1972 and March 1973, "Wind Tunnel Site Analysis of Dow Chemical Facility at Rocky Flats," that the winds up there are frequently so strong that cars in the parking lot need protection "...from high velocity wind action assaulting vehicles with abrasive particles..." (Part II, p.ii). Wind tunnel studies were done to find ways of sheltering parked cars from this sand-blasting which had evidently become a problem.

Wind tunnel studies were also done to find out whether the monitoring stations around the perimeter of the Rocky Flats plant were spaced optimally. Conclusion #4 of this study (Part I, p. 37) reads as follows:

> "An array of monitoring devices arranged along the north-south road which exists to the east of the plant site should intercept plumes if they are placed at 500 ft. intervals or closer"

This implies that monitoring stations at intervals greater than 500 ft. along Indiana Street could miss detection of a plume of contaminated air coming from a point source in one of the buildings at Rocky Flats. From my study of the map published on pg. 29 of the Annual Environmental Monitoring Report of Rocky Flats Plant, (REP-ENV-73), dated April 26, 1974, I conclude that the four monitoring stations are placed approximately 5,000 to 7,000 ft. apart along this road. That is, according to the wind tunnel studies, there would need to be at least ten times as many monitoring stations as now exist in order to be sure to detect a plume of contaminated air coming from a point source. To be sure to detect a plume coming from a contaminated area approximately 500 ft. in diameter like the oil-spill area, one would need monitoring stations at least every 1000 ft. along Indiana Street.

What I conclude from this rough preliminary analysis is that a strong gust of wind from the west could pick up a lot of dust from the contaminated area and blow it past the monitoring stations in such a way that very little, if any, abnormal rise

in radioactivity would be detected. This is particularly true because the monitors, I understand, measure only the total amount of radio-activity accumulated over a 24-hour period. A brief gust or dust devil containing a large amount of plutonium and seriously contaminating the residential areas east of Rocky Flats might therefore escape detection completely by the present monitoring system.

It is my urgent recommendation that our task force should get the whole story on this from Rocky Flats. If the measurements we need have been classified, we must force them to be released because the health of the residents of this area is at stake. If the soil was indeed contaminated to the extent of 40,000 dpm/gm, as alleged, then a dangerous amount of plutonium has almost certainly been dispersed into the residential areas all the way from Boulder to Golden, including Denver, generally undetected by the monitoring system. We have no assurance that this is not continuing today. Some evidence suggests that it is.

The suggestion by Lauriston Taylor at the EPA Hearings on January 10th, that soil standards may not be needed because air standards are satisfactory protection, evidently does not apply under the particular conditions of strong gusty winds and inadequate air monitoring at Rocky Flats. At the same hearings, the questions by Prof. First regarding the relationship of contamination in the soil to inhalation of plutonium are pertinent. We need studies done on site at Rocky Flats under conditions of very high velocity gusty winds and twisters or dust devils in order to find out what is the actual situation regarding resuspension of plutonium from contaminated soil and how much of it is getting into the air breathed by residents of the area. When the winds are strong enough to sand-blast the paint and pit the windshields of cars at Rocky Flats, I suspect we have enough plutonium in the air to be dangerous to those who breathe it.

cc: Members of Lamm-Wirth Task Force (15)
Dr. Lauriston Taylor, President
National Council on Radiation Protection
Prof. Melvin First, Harvard School of Public Health
Dr. Paul Smith, EPA, Denver

COGMA

'75 JAN 15 AM 9:26
13787
EPA REGION VIII
CORRESPONDENCE CONTROL

COLORADO ORGANIC GROWERS AND MARKETERS ASSOCIATION
2555 W. 37th AVE
DENVER, COLORADO 80211
477-6291

A Petition

To Whom It May Concern:

Recent nuclear related accidents(?) involving Dow Chemical's Rocky Flats installation serve as a strong indicator that the facility threatens the lives of thousands in the surrounding area.

Broomfield and Westminster are now plagued with traces of Tritium in their H2O supplies as a result of Rocky Flats dumping radioactive effluents into Walnut Creek

The Colorado Public Health Department has recently deemed development lands in Arvada unsuitable building sites due to highly radioactive traces of plutonium oxide dust in the soil. NOW IS THE TIME TO TAKE ACTION rather than allow these atrocities to continue.

If nuclear plants are as safe as the Atomic Energy Commission (AEC) and utility companies would have us believe, then why do they need the protection of the Price-Anderson Act.

In the event of a nuclear catastrophe at Rocky Flats the AEC admits to losses as high as $280 billion. In accordance with the Price-Anderson Act the utilities and the AEC (us) would be responsible for a relatively small portion ($560million) of this loss while the remainder (?) of the burden would be taken out of the American taxpayer's pocket.

As citizens and taxpayers whose money and property is used in financing this insanity, we urge you to take action toward shutting down the Rocky Flats nuclear plant, a warhead trigger manufacturing installation and to petition against the reinstatement of the Price-Anderson Act when it comes up for reconsideration in early 1974.

NAME	ADDRESS/ZIP	PHONE
Sidney Wilson	[illegible] St. Denver [illegible]	[illegible]-2668
Becky Robertson	735 Clarkson Denver	831-8589
Marny Rosene	1250 Emerson #4 Denver 80218	
Philip Sappe	1409 Columbine #1 Denver, Co. 80206	355-8107
James Pessa	4680 Clay Denver, Colorado	
Angela P. Lewis	4680 Clay Denver Colo. 80211	
Christina Blume	4667 Clay Denver 80211	
Linda Greene	4675 Clay Denver 80211	388-8181

Labor Donated

COGMA

'75 JAN 15 AM 9:26

REGION VIII
CORRESPONDENCE CONTROL

COLORADO ORGANIC GROWERS AND MARKETERS ASSOCIATION

2555 W. 37th AVE.
DENVER, COLORADO 80211
477-6291

Petition

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If nuclear plants are as safe as the Atomic Energy Commission (AEC) and utility companies would have us believe, then why do they need the protection of the Price-Anderson Act.

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As citizens and taxpayers whose money and property is used in financing this insanity, we urge you to take action toward shutting down the Rocky Flats nuclear plant, a warhead trigger manufacturing installation and to petition against the reinstatement of the Price-Anderson Act when it comes up for reconsideation in early 1974.

NAME	ADDRESS/ZIP	PHONE
Barbara Baker	838 Bross Longmont 80501	776-0980
K. K. Wyckoff	88 Bross St Longmont Colo.	
Michelle E. Tuttle	838 Bross Longmont Colo. 776-0980	
Colin E. Rum	865 N. 6th Longmont, Colo.	
Rick Watson	838 Bross Longmont Colo. 776-0980	
Bonnie L. Peardon	1825 Lincoln Ave Longmont	

Labor Donated

684

COGMA

JAN 1 AM 9:26

COLORADO ORGANIC GROWERS AND MARKETERS ASSOCIATION

REGION VIII
CORRESPONDENCE CONTROL

2555 W. 37th AVE
DENVER, COLORADO 80211
477-6291

To Whom It May Concern:

Recent nuclear related accidents(?) involving Dow Chemical's Rocky Flats installation serve as a strong indicator that the facility threatens the lives of thousands in the surrounding area.

Broomfield and Westminster are now plagued with traces of Tritium in their H2O supplies as a result of Rocky Flats dumping radioactive effluents into Walnut Creek

The Colorado Public Health Department has recently deemed development lands in Arvada unsuitable building sites due to highly radioactive traces of plutonium oxide dust in the soil. NOW IS THE TIME TO TAKE ACTION rather than allow these atrocities to continue.

If nuclear plants are as safe as the Atomic Energy Commission (AEC) and utility companies would have us believe, then why do they need the protection of the Price-Anderson Act.

In the event of a nuclear catastrophe at Rocky Flats the AEC admits to losses as high as $200 billion. In accordance with the Price-Anderson Act the utilities and the AEC (us) would be responsible for a relatively small portion ($560million) of this loss while the remainder (?) of the burden would be taken out of the American taxpayer's pocket.

As citizens and taxpayers whose money and property is used in financing this insanity, we urge you to take action toward shutting down the Rocky Flats nuclear plant, a warhead trigger manufacturing installation and to petition against the reinstatement of the Price-Anderson Act when it comes up for reconsideration in early 1974.

NAME	ADDRESS/ZIP	PHONE
Mike Anderson	1452 Detroit #7 Denver 80206	399-0221
Leon Bechard	Star Rt Box 1063 Elizabeth, Co. 80107	646-4002
John E. Bertrand	3625 Raleigh St. Denver, Colo. 80212	(458-1193)
Mary Thompson	1623 Washington St., Denver, Colo.	832-1096
Jim Fowler	2555 W. 37 Ave Denver, Colo	477-0291

Labor Donated

Dear Sir,

I feel that some of us ordinary wage earning citizens, of this area, should have something to say about what happens with the Atomic Plant at Rocky Flats. The future operation of this plant seems to be continually threatened by people who either don't actually know much about the place or think that they know more than the people who work there. I believe that it is a known fact that there are many other places that would like to have this operation in their area, due to the potential employment, and other financial benefits derived from a plant like it. It seems possible that some of the effort to get this operation moved could be coming from other areas. It also seems pretty obvious that the people who are expending this

effort are not in the position to be adversely affected if we should lose this operation. I believe in protecting our environment in this area as much as anyone, since I intend to live here the rest of my life, but I donot believe that there is a need to get rid of the Rocky Flats operation. I do not believe that the Rocky Flats plant is any greater a threat to our health than many other places in this area. There are many places in this area that are potentially hazardous to our health, if the extremely unlikely should happen. Rocky Flats is one of them, so is the Bureau of Standards, the University of Colorado, Arapahoe Chemical, and many other places. I doubt if any of them have any better safety standards than Rocky Flats has. Some of them don't have anywhere near as good. I don't hear those other places mentioned, is it because they are in the back-yard, so to speak, of the ones doing all

of the hollering? It is obvious that a place like Rocky Flats must maintain rigid safety standards and this must be a joint effort by citizen groups as well as the A.E.C. or what ever governing body has the controll, to make sure that no one relaxes these standards. Dow did a pretty good job, over all, I believe, since they were more or less learning at first. They have done a verry good job these last few years and I believe that the next contractor will do even better, if the pressure is there. The Rocky Flats plant is, and has been for many years, a good source for employment for people in this area and to eliminate it, because of over reaction, on the part of some radical groups, could verry likely cause many people, undue hardships, and would verry likely set of some repercussions.

It took some real effort to get this plant located in our area and to my knowledge there is no real proof that anyone in this area has been adversly affected by it being here. I feel that I am pretty familiar with the conditions at Rocky Flats since I I worked there for over 12 years as a maintinance man in the sheet metal dept. and 1½ years as a construction sheet metal worker for Swinerton & Walberg at that plant. Most of my time there was spent in the so-called "Hot" area's and I am very familiar with the problems of containing contamination. I doubt that any professor or scientist in this area has more experience in that sort of thing than I do and very doubtfully any where near as much. I was active in the hourly workers safety comitee and have a lot of faith in the people that work there. The people who work in that plant are the best safe-guards you could get, if they are allowed to

have their say, in safety meetings, and if people in the right places will listen. It is really the people working at Rocky Flats whose lives and jobs are at stakes and they, in my opinion, will fight to protect both.
I find it hard to believe that cows, outside of the plant, could breath more contamination than people working inside would, unless they were purposely exposed to contamination, for some reason. I also am real suspicious of, and question the motives of those people who keep striving to have this plant moved. I hope I have said something that will help you see the other side of the picture and will keep trying if it is necessary.

Respectfully yours.
Dewey C. Poe.
1055 Miami Way
Boulder, Colo. 80303

690

THE DOW CHEMICAL COMPANY

ROCKY FLATS DIVISION
P. O. BOX 888
GOLDEN, COLORADO 80401

January 3, 1975

Mr. Paul B. Smith
Air and Hazardous Materials Division
Environmental Protection Agency
Region VIII, Suite 900
1860 Lincoln Street
Denver, CO 80203

Dear Mr. Smith:

Attached is a written comment which we would like to submit to the EPA Committee considering standards for plutonium.

M. A. Thompson/wj

M. A. Thompson
Manager of Environmental Sciences

cc:
E. W. Bean - RFAO, USAEC
H. E. Bowman - Dow, Rocky Flats

A prime contractor for the U. S. Atomic Energy Commission CONTRACT AT(29-1)-1106

STATEMENT FOR THE EPA COMMITTEE CONSIDERING STANDARDS FOR PLUTONIUM ON THE HISTORY AND PERFORMANCE OF ROCKY FLATS

Submitted by
M. A. Thompson
DOW CHEMICAL U. S. A.
Rocky Flats Division

Following is a brief summary of the history and performance of the Rocky Flats Plant as related to the handling and release of plutonium.

Announcement of the construction of the Rocky Flats Plant by the Atomic Energy Commission was made in March 1951 and initial construction was completed and operations started in 1953. By the end of fiscal year 1974, the acquisition costs for the plant have totaled 222 million dollars. This includes about 91 million dollars for equipment and 131 million dollars for land, buildings and facilities. Over 90 major structures containing approximately 1.7 million square feet of building area are included on the plant site. About 2900 people are employed at the plant. The plant is situated on about 2500 acres of government owned property approximately 15 miles north and east of Denver. The government is purchasing an additional 4000 acres around the perimeter of the currently owned federal land to act as a buffer zone. The site for the Rocky Flats Plant was selected because it best met the following criteria:

1. Located in Nebraska, Kansas, Oklahoma, Mississippi, Arkansas, Colorado, or the Texas Panhandle.

2. Area requirements of two miles by two miles square.

3. Supporting population of at least 25,000 no less than five or more than 25 mile distant.

4. Minimum displacement of homes.

5. Dry, moderate climate within 10 miles of a railroad, near good major highways, and a community airport served by north-south and east-west airlines.

6. Large military air field within 50 miles.

7. 12,000 KW electricity.

8. 1,000,000 gallons of water per day.

Since the initial plant construction, several major changes have occurred in the surrounding communities of significance to the plant operation. These include a significant increase in the population of the Denver area; population growth in the general direction of the plant; the establishment of the City of Broomfield approximately six miles northeast of the plant; the establishment of the Jefferson County Airport approximately four and a half miles northeast of the plant, and the acquisition of Great Western Reservoir in 1955 by the City of Broomfield for use as a community drinking water supply. The reservoir, which is on the water course carrying the Rocky Flats aqueous effluents, was originally built in 1903 and enlarged in 1958. Nonradioactive releases from the plant are controlled to conform with EPA regulations and discharge permits. Radioactive discharges are controlled according to AEC regulations.

The plant is part of the production complex of the Division of Military Applications of the Atomic Energy Commission. It has been operated since originally built by The Dow Chemical Company. The primary function of the plant includes research and development, metal working and chemical recovery. Plutonium is one of the primary materials handled at the plant. Since the original plant operation, extensive control techniques have been utilized to insure that plutonium in liquid and gaseous effluents leaving the plant site are below allowable standards and at the minimum practical level. Improvements have continually been made in these control techniques so that plutonium concentrations in air and water leaving the plant site have been reduced and have been significantly under currently accepted concentration guides.

Small amounts of plutonium have been released in the air and water due to normal plant operations and due to accidental releases. Two major fires have occurred at the plant site, in 1957 and 1969, in which small amounts of plutonium were released to the plant site, but no off-site releases were detected. Plutonium has been released from the plant site over a period from 1958 to 1968 because of leaking 55-gallon drums containing plutonium-contaminated lathe coolants. Winds which occasionally exceed 100 miles per hour at the plant site carried an estimated 0.47 curies (7.5 grams) of plutonium over 1390 acres outside the federally owned property. As a result, plutonium contamination exists in the soil outside the currently owned federal land and the land presently being purchased as a buffer zone in concentrations up to 10 d/m/g as compared to a background from fallout of about .05 d/m/g. Isolated spots may exceed the 10 d/m/g.

Since the original construction and operation of the plant, an extensive environmental monitoring program has been conducted to insure that all plant discharges were well within applicable standards. Air, water and soil are routinely analyzed for plutonium within the plant site, at the plant boundary, and in the community surrounding the plant. Background radiation measurements are made at various Colorado locations up to 20 miles from the plant. All water discharged from the plant is held in plant ponds and analyzed prior to release. In addition, vegetation and wildlife are also occasionally analyzed for plutonium. The results of the environmental monitoring are reviewed monthly with the Colorado Department of Health, the EPA, and other state and local officials. In general, plutonium concentrations in plant effluents have been less than 1% of the allowable standards--for instance, during 1974, the average plutonium concentration in water leaving the plant site was only .05% of the standard and the plutonium in the air at the downwind site of the buffer zone (which is currently being purchased) was less than .4% of the currently accepted standard. This compares to the maximum measured background concentrations of plutonium in water of .03% of the standard and plutonium in air of .4% of the standard. During 1974, plutonium in Great Western Reservoir, which is the drinking water supply for the City of Broomfield, averaged only .002% of the standard and plutonium in the Broomfield air averaged .4% of the standard. Both values are the same as found for background.

In addition to monitoring the surrounding community, an extensive monitoring program is conducted at the plant. This program includes monitoring the air in all of the working areas; multiple samplers and continuous alarms at gaseous release points; periodic soil, vegetation and animal analysis; monitoring of ground water through a series of wells and periodic employee health examinations. An extensive quality control program is included as part of all monitoring activities. Although a few employees have obtained a plutonium body burden or lung burden greater than the recommended amount, there is no indication that any employee has suffered any health effect because of this exposure or that there is any health hazard to the general population because of plant operations. It is recognized that our population is small and that sufficient time may not have elapsed for health effects to appear, but to date, the record has been excellent.

In addition to the routine monitoring activities, several long term research studies are under way to determine the movement and effect of plutonium in the ecosystem. Several studies are being conducted by the University of Colorado and Colorado State University to understand the behavior of plutonium in the aquatic and terrestrial environment. Other studies conducted by the USGS, AEC laboratories,

and private consultants have been concerned with resuspension and disposition of plutonium, the meteorology of the Rocky Flats area, and possible movement of plutonium through ground water and soil. Other long term studies are being conducted by Rocky Flats employees including the chemistry of plutonium in soil, methods of removing plutonium from soil by attrition scrubbing and high gradient magnetic field techniques, particle size characterization in air, water and soil by fission track techniques, and plutonium ecosystem modeling. The modeling includes an attempt to quantitatively determine the amount of plutonium in each environmental compartment, the rate of movement between compartments and the long term dose commitment to human beings as a result of the plutonium behavior. The difficulty in establishing this type of model is in simplifying the very complex ecosystem into meaningful compartments and assigning quantitative values to the various compartments and the intercompartmental rates. Results of all the studies to date have not identified any problem for man or biological systems in general in spite of the above-background concentrations of plutonium in the ecosystem near the plant.

In summary, the Rocky Flats Plant has been operated for about 22 years, during that time normal operations, two fires, and leaking drums containing waste liquids, have discharged small amounts of plutonium into the environment. There has been no demonstrated hazard from past or present operations based on currently accepted standards and the amount of plutonium in the air and water of nearby communities is the same as found in background measurements. Changes and improvements in operational procedures have continually reduced the amount of plutonium released from the Rocky Flats Plant due to normal operations and minimized the chance for further accidental release.

I believe that it is important to establish credible standards for plutonium because of the increasing concern of the public for possible hazards associated with plutonium. In establishing these standards, I believe the public must be assured that the standards which are adopted are adequate for their protection. I believe that, presently, nuclear materials are considered by the general public to be a particular hazard. It is important for the public to recognize that all of us are exposed to background radiation continuously and that with proper handling and adequate standards, releases from the nuclear industry will not appreciably increase the risk to their health and well being.

I believe you have a difficult and complex task; however, one that is exceedingly important to the nuclear industry and to the general population.

M. A. Thompson
1-3-75

CASE (Citizens Association for Sound Energy)
2125 W. Clarendon Drive
Dallas, Texas 75208

Dr. William A. Mills
Director of Criteria & Standards Division
Office of Radiation Program
Environmental Protection Agency
Washington, D. C. 20460

Gentlemen:

The following suggestions are submitted regarding setting of standards for the transuranium elements. The remarks are general in nature and apply to standard setting of all polutants for the most part as well as the transuranium elements specifically.

1) Set "basic" standards based on biological evidence. Present standards or recommendations[1] are the results of groups of scientists trying to balance societal needs against biological risks. A crisis of considerable proportion is evolving concerning the public's confidence in technology and scientists. This is largely due to past examples of a lack of professionalism and it is suggested that the group of scientists be charged simply with determining the biological risk which in turn is used in setting "basic" standards. After "basic" standards (including air, water and soil permissible concentrations) have been determined, a cost-benefit analysis may be employed to set "general" standards for a given segment of our society.

2) Establish a 95% confidence level that the risk does not exceed a specified value when evaluating experimental data. Much confusion has been caused in the past because of the lack of a rigorous approach when making statements about risks. Part of this confusion has arisen due to statements by Professor Sternglass.[2] If he and groups such as yours would employ a rigorous approach, much confusion would be removed. And if extrapolation of experimental data is required, it should be done in the most conservative manner and clearly labeled as an extrpolation (or guess).

3) Adopt a criteria based upon the most susceptible person in the general society when setting the "basic" standard. Sufficient research should be conducted to determine the possible dosage to the most susceptible person as a consequence of setting a given "basic" standard (and marginal consequences should be simultaneously determined). All modes of transport and accumulation (including water, air and soil) should be considered to determine where all possible sinks of pollution may exist. Sufficient research to determine the health effects (on all parts of the body[3]) of relevant dosages should also be conducted. The "basic" standard should be set such that the most susceptible person in the general society has a small chance of being affected deleteriously. All persons not employed in an associated industry in a manner which insures that they are entitled to full life and health insurance as well as retirement benefits should be considered to be persons in the general society. The most susceptible person concept does not eliminate a child, a fetus (unless lawfully declared not to be a person) or a transient from consideration.

4) The rate of allowable additions of pollutants to the environment should be limited in a manner consistent with the assumption that equilibrium conditions have been attained. The present ambient conditions should be evaluated in terms of both health effect and ecological (ability of the environment to feed and, in general, allow the most susceptible person to harmoniously exist) consequences. If additional releases are allowable, conditions which assume that the release rate under consideration has been taking place for a sufficiently long period of time to reach an equilibrium state should be adopted. This criteria would limit the yearly rate of release of PU-238 to approximately 1/125th of the allowable ambient and PU-239 to approximately 1/35000th of the allowable ambient. Other approaches would not be conservative.

5) Allowable release rates of each eligible portion of our society should be apportioned. When making these allotments, the possibility and consequences of accidents both on the plant site and in transit should be considered as well as normal releases in determining the relative demand of ecah segment of our society.[4] These allotments should be made by EPA and the possibility of including additional segments should be planned for by reserving an uncommitted portion for such contingencies.

6) A cost-benefit consideration should be utilized at this point - not before. If it is determined that the value to society of increasing the allowable release rate of a segment outweighs the risk to society, zoning of areas where the most susceptible persons are located should first be considered. If this approach does not yield a suitable solution by itself, an appropriate increase, which cannot be avoided by zoning, in the allowable release rate for that segment should be granted. It should be required that all analysis take into consideration health effects and/or ecological costs and that a common measuring base be used for all costs and/or benefits. A cross section of disciplines should be used to conduct the cost-benefit analysis under the auspices of the EPA. All cost-benefit decisions should be periodically reviewed to evaluate changes.

7) Application to EPA for point source releases should be required. The applicant should assume that all other segments are operating at their maximum allowable release rates when justifying his proposal. The applicant should show that his point source addition does not cause any deleterious effects to the most susceptible person - taking all spatial aspects into consideration with regard to all other point source emissions within the same segment which might interact with his emission to the detriment of the most susceptible person. The consequences of the proposed dosage to the most sensitive portions of the body should be evaluated and justifyed. The decision as to which point sources are the most desirable to society should be a joint decision between the EPA and the cognizant body of the associated segment (the NRC, AMA, or Department of Defense).

8) Timely reporting of all releases should be required. Criminal laws to protect the general public should make the user responsible to monitor all possible release paths and report in a timely manner all releases whether planned or unplanned - at the site or in transit. Similar laws should apply to individuals within governmental agencies. A criteria should be established whereby an accidental release rate which exceeds a predetermined value causes the user to be automatically penalized. An effective program of continuous govermental monitoring to assure compliance should be established. Until such time that proven sufficiently accurate measurement techniques and instruments become generally available,[5] EPA should monitor at the source where much higher concentrations exist.

References

1 National Academy of Sciences, Biological Effects of Atomic Radiation, Washington, D. C., 1956
National Academy of Sciences, Radiological Effects of Ionizing Radiation, Washington, D. C., 1972
National Council on Radiation Protection and Measurements, NCRP Report No. 39, 1971
International Commission on Radiological Protection, ICRP Publication 9

2 Hull, A. P. and Shore, F. J.,"Sternglass: A Case History", BNL 16613, March, 1972

3 Blair, W. J., Richmond, C. R. and Wachholz, B. W.,"A Radiobiological Assessment of the Spatial Distribution of Radiation Dose from Inhaled Plutonium", WASH-1320, Sept., 1974
Tamplin, A. R. and Cochran, T. B., "Radiation Standards for Hot Particles", Natural Resources Defense Council, Washington, D. C. 20036
Petkau, A., "Effect of $^{22}Na^{+}$ on a Phospholipid Membrane", Health Physics, Vol. 22, pp. 239-244, March, 1972

4 "Reactor Safety Study: An Assessment of Accident Risks in U. S. Commercial Nuclear Power Plants", WASH-1400, August, 1974
"Comments by the Environmental Protection Agency on Reactor Safety Study: An Assessment of Accident Risks in Commercial Nuclear Power Plants", EPA, November, 1974
Shappert, L. B., Brobst, W. A., Langhaar, J. W., and Sisler, J. A., "Probabilities and Consequences of Transportation Accidents Involving Radioactive-Material Shipments in the Nuclear Fuel Cycle", Nuclear Safety, Vol. 14, No. 6, Nov.-Dec., 1973
Rowe, W. D. and Holcomb, W. F., "The Hidden Commitment of Nuclear Wastes", Nuclear Technology, Dec., 1974
Willrich, M. and Taylor, T. B., "Nuclear Theft: Risks and Safeguards", Ballinger Publishing Co., Cambridge, Mass., 1974
"Environmental Analysis of the Uranium Fuel Cycle", U. S. Environmental Protection Agency, EPA-520/9-73-003, 1973

5 Jarvis, A. N. and Easterly, D. G., "Measuring Radioactivity in the Environment - The Quality of the Data", Nuclear Technology, Dec., 1974

Commonw lth Edison
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Chicago, Illinois 60690

699

February 10, 1975

Director
Criteria and Standards Division
Office of Radiation Programs (AW 560)
U.S. Environmental Protection Agency
401 Main Street S.W.
Washington, D.C. 20460

Dear Sir:

On September 23, 1974, the United States Environmental Protection Agency (EPA) published a notice of intention to evaluate the environmental impact of transuranic elements and to consider whether guidelines or standards were needed to assure adequate protection of the public health and the environment. (39 F.R. 34098) On October 24, 1974, EPA published a notice of public hearing on this subject. (39 F.R. 37810) Commonwealth Edison Company (Commonwealth) is in general agreement with the statement submitted and the oral presentations by the Atomic Industrial Forum and the United States Nuclear Regulatory Commission in response to these notices. However, Commonwealth believes that specific information should be added to the record which delineates the actual impact of plutonium and other transuranic elements which result from the operation of Commonwealth's seven light-water reactor units. The following comments are submitted by Commonwealth to provide this information.

Uranium fuel for light-water reactors usually consists of high strength uranium-dioxide ceramic pellets. At fuel fabricating plants, these pellets are hermetically encapsulated in zircaloy cladding tubes; thereafter, fixed arrays of these completed fuel rods are shipped, stored, and otherwise normally handled at individual light-water reactors as integral assemblies. Rigorous quality control and quality assurance procedures are conducted at each manufacturing stage by the fuel fabricators to ensure the leaktightness and integrity of the zircaloy cladding throughout the fuel's lifetime. In addition, Commonwealth conducts quality assurance audits of each of its fuel fabricators.

Prior to irradiation, normal uranium-dioxide fuel contains no transuranic isotopes. However, during irradiation of the uranium-dioxide fuel in the reactors, plutonium and other heavy elements

are produced through a variety of transmutation and radioactive decay schemes. These transuranic atoms remain within the ceramic fuel matrix and thus within the zircaloy cladding and under normal conditions are not released to the primary reactor cooling water. However, perforations develop in the zircaloy cladding during commercial operation of light-water reactors, and test reactors have been operated with intentionally defected fuel rods. The data from both of these types of operations indicate that fuel washout (the physical or chemical erosion of the fuel pellet and subsequent release into the primary cooling water) is insignificant in either uranium-dioxide or mixed-oxide fuel pellets. In fact, the excellent chemical stability of uranium-base oxide fuels in water coolant environments was an important consideration in their selection for use in light-water reactors. Should the zircaloy cladding perforate and fuel washout occur, transuranics could be released into the primary coolant. Our measurements, to date, show that Neptunium-239, a beta emitter, has been present in the primary coolant. When Neptunium-239 in the primary coolant is measurable, the concentration has ranged from 10^{-4} to 10^{-2} uCi/ml. However, the measurements of Neptunium-239 in the radwaste system storage tanks range from 10^{-4} to 10^{-6} uCi/ml. This reduction in concentration could be the result of dilution by additional water, decay of the Neptunium-239, the plating out on the reactor "internals" or retention in the demineralizer resins or other solid wastes from the radioactive waste treatment system.

The operational difficulties confronting a utility which proposes to use plutonium-recycled mixed-oxide fuels are basically the same as those associated with uranium-dioxide fuels. Criticality considerations and the safeguard regulations must be observed for both types of fuel. The basic difference is that the radiation levels, particularly neutron radiation, associated with new mixed-oxide fuel assemblies are increased, and thus, higher exposures to workers may result from handling recycled fuels containing plutonium as opposed to exposures resulting from handling uranium-dioxide fuel. Nevertheless, it is still possible to maintain personnel exposure well within acceptable levels. All experiments and

demonstrations to date indicate that utility operators can safely receive, store, and handle mixed-oxide fuels. Irradiation in a light-water reactor of either type of fuel assemblies causes the assemblies to become intensely radioactive and thus the same careful handling procedures are followed for both uranium dioxide and mixed-oxide fuel assemblies. In essence, both types of fuel assemblies are moved from the reactor core underwater by remote handling equipment to storage locations outside the reactor pressure vessel and the short lived radioactive isotopes are allowed to decay. Since facilities do not exist at Commonwealth Edison's nuclear power stations to reprocess or fabricate nuclear fuels, the complete assemblies are shipped from the stations in massive casks to a reprocessing plant where the plutonium and uranium can be recovered for recycling into new fuel assemblies.

While large amounts of potentially valuable plutonium can exist in depleted uranium fuel pellets, the exact amount depends on many factors, including the type and capacity of the reactor, the fuel design, and the extent of fuel depletion. At the beginning of a typical operating period of Commonwealth Edison's Dresden 1 reactor, 175 kilograms of plutonium would be present in the reactor core. By the end of a 12-month operating period, 250 kilograms of plutonium would exist in the reactor core. The fuel assemblies which would normally be removed from the core at that time would contain 75 kilograms of plutonium, and 72 per cent of this would be fissionable in thermal reactors. As another example, at the end of June, 1973, Commonwealth Edison's Dresden 2 nuclear unit, which is four times as large as Dresden 1, had 460 kilograms of plutonium in the reactor core. By the end of June, 1974, the reactor core contained 650 kilograms of plutonium and 80 per cent was thermally fissionable. Finally, within four or five years, each of Commonwealth Edison's new, larger, Zion units are expected to have fuel assemblies removed each year which contain 155 kilograms of fissionable plutonium. If Commonwealth Edison is allowed to recycle the fissionable plutonium, an equivalent fuel saving of more than 58 million gallons of oil or about 460,000 tons of coal can be obtained per unit per year.

The capability of utility nuclear plant operators to safely handle, store, and extract energy from nuclear fuels containing encapsulated plutonium in light-water reactors with no local impact on the environment has been demonstrated. This experience provides a reasonable basis for the continued use of uranium-dioxide fuel assemblies and the use of mixed-oxide fuel assemblies using the present standards for light-water reactors now set by the Nuclear Regulatory Commission in this area.

Very truly yours

Byron Lee, Jr.
Vice-President

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AW-560
U.S. ENVIRONMENTAL PROTECTION AGENCY
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