AD/A-003 413

SOURCE BOOK ON PLUTONIUM AND ITS DECONTAMINATION

FIELD COMMAND (DEFENSE NUCLEAR AGENCY)

24 SEPTEMBER 1973

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE	READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER: DNA 3272T	2. GOVT ACCESSION NO.
3. RECIPIENT'S CATALOG NUMBER: AD/A-003 443	
4. TITLE (and Subtitle): SOURCE BOOK ON PLUTONIUM AND ITS DECONTAMINATION	5. TYPE OF REPORT & PERIOD COVERED: Topical Report
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): Lt. Col. F. C. Cobb, USAF Lt. Col. R. L. Van Hemert, USAF	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS: Field Command Defense Nuclear Agency Kirtland AFB, New Mexico 87115	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS: Director Headquarters, Defense Nuclear Agency Washington, D.C. 20305	12. REPORT DATE: 24 September 1973
	13. NUMBER OF PAGES: 91
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)	15. SECURITY CLASS. (of this report): UNCLASSIFIED
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

PRICES SUBJECT TO CHANGE

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The effort expended by the study group in this undertaking was designed to accumulate as much information as possible that might provide a basis for a source book on plutonium, its deposition and retention in the human body, and methods to reduce the plutonium contamination hazard. The effort was divided between: (1) a literature search of past and current documents, manuals, regulations, etc., personal contact with knowledgeable persons in the field and by attendance at meetings and symposia that were fortuitously scheduled during the early months of the study, and (2)

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20. ABSTRACT (Continued)

development of the coupled differential equations, based on the 1965 and the proposed 1973 physiological lung models, that predict the deposition and translocation of inhaled plutonium in the human body. A historical review of plutonium and the general contamination situation, along with a review of early U.S. policy (e.g., in the days of the Manhattan Project), provide a good background for this problem. Next, the biology of plutonium contamination is discussed; past as well as current research in this field conducted by the DOD, AEC, and other agencies is reviewed. New concepts and ideas, such as lung lavage and the use of chelating agents, are introduced which are currently being used to reduce plutonium body burdens. That plutonium which remains in the body after inhalation (the most likely, as well as most dangerous, ingress method into the body) is translocated from the lung to the lymphatic system, blood, bone, liver, and gastrointestinal tract; the differential equations describing these phenomena are solved and with the best values available for the constants, graphs and tables are presented that give the burdens, doses, and dose rates to these different parts of the body. A very important conclusion reached, based on the 1965 physiological lung model and shown even more emphatically by the proposed 1973 lung model, is that the lung is the critical organ and not the lymph nodes or lymphatic system as lately proposed by a number of people. Foreign standards for plutonium contamination levels, as they appear in the literature available in this country, are given to give the reader an overall view. The last section of the report gives much useful information relating to the decontamination operation and includes decontamination methods and their efficiencies. The entire report is very rich in citing references to which the reader can refer; 101 references are cited, and some of these references refer to hundreds of others.

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INTRODUCTION

The effort expended by the study group in this undertaking was designed to accumulate as much information as possible that might provide a basis for a sourcebook on plutonium and its decontamination. The effort was divided between a literature search of past and current documents, manuals, regulations, etc., personal contact with knowledgeable persons in the field and by attendance at meetings and symposia that were fortuitously scheduled during the early months of the study. Our effort would have been more difficult but for the many persons who have helped the study group along the way. Doors were opened and comments and information were freely given at every stage of the effort. Although it would be impossible here to thank each contributor by name, we do want to take this opportunity to acknowledge, in this general way, the aid that was extended to us.

This study is divided into seven major sections. The contents of the sections are:

Section I presents a general statement of the plutonium hazard. It is included so that the reader can place himself on common ground with the information which follows in subsequent sections of the study.

Section II covers published policy or directives of the DOD, Atomic Energy Commission (AEC) and federal agencies relating to radiation decontamination, exposure limits and contamination limits.

Section III deals with DOD, AEC or other research that has contributed or may contribute to an understanding of the physical portion of the hazard model, that is, what happens from the initial accident until the plutonium aerosol is inhaled.

Section IV is a review of the research accomplished and planned to develop information concerning the biological hazard, that is, what happens to plutonium once it is in the body.

Section V presents the current 1965 lung model and develops the calculations which describe deposition and retention of respirated plutonium in the human body.

Section VI lists foreign standards for plutonium contamination levels as they appear in literature available in this country.

Section VII compiles information relating to the decontamination operation and includes some decontamination methods and expectations.

Appendix A uses the proposed 1973 lung model to update the work in Section V.

NOTE

References cited herein are current as of December 1972.

SECTION I

THE PLUTONIUM HAZARD

PURPOSE

This study has been designed as a primer for those personnel having responsibilities for plutonium bearing weapons or the decontamination of areas affected by weapons accident plutonium contamination. It has been prepared with the hope that a general background knowledge of plutonium and its hazards will result in better DOD operations and an enlightened personnel force.

THE GENERAL CONTAMINATION SITUATION

Experience indicates that plutonium contamination will result when a nuclear weapon is involved in a fire or impact situation sufficient to violate the weapon's containment. The plutonium may burn or the impact may cause detonation of the high explosive content of the weapon. In each case, plutonium, generally in the form of oxides, will be dispersed. The extent and character of the dispersed material is a function of many variables. Among these are the heat and duration of the fire, the containment material involved, the amount of high explosives and plutonium, aerodynamic variables of wind and stability and many more.

The plutonium, once on the ground or other surfaces, should not be considered as fixed in place. It is subject to resuspension into the air, adhesion to ground or airborne material, and settling deeper into the soil or other porous surfaces.

The primary plutonium hazard is considered to result from inhalation of the aerosol. (Aerosol is the generally accepted term for airborne particulate plutonium). The aerosol is present in the initial dispersal cloud and in material resuspended by natural or human activity in the area. Some of the aerosol is retained in the lung where it causes a radiological insult due to its alpha activity and long lung retention time. Transport to other body organs by body processes is of concern because of significant retention times at these other locations. Modes of entry into the body other than inhalation are considered to be less hazardous. These include absorption through the intact skin, deposition in open wounds and ingestion via foods grown or produced on contaminated ground.

Experience from various tests, previous accidents and theoretical calculations indicate that although a significant body burden of plutonium may be received during passage of the initial cloud, the greater danger to population appears to arise from long term occupancy of a contaminated area wherein the population is subjected to the resuspended radionuclide (Ref. 1). If the affected area is to be utilized after the accident, some decontamination effort must be undertaken.

SECTION II

REVIEW OF U.S. POLICY AND DIRECTION CONCERNING PLUTONIUM CONTAMINATION

THE EARLY YEARS

The development of current policy and direction concerning plutonium contamination had its genesis in the discovery of X-rays (1895) and radium (1898), their subsequent use and the inevitable incidents of biological damage which followed. The first organized efforts to establish protective criteria for these new tools were made 30 years later. In 1928 the Second International Congress of Radiology established the Committee on X-ray and Radium Protection. The U. S. Advisory Committee on X-rays and Radium Protection was organized in 1928 to provide U. S. input to the International Committee. These committees were the forerunners of the current International Commission on Radiation Protection (ICRP) and the U. S. National Council on Radiation Protection and Measurements (NCRP). Table I lists the early recommendations of these organizations for the employment of these ionizing radiations (Ref. 2).

TABLE I

EARLY RADIATION DOSE LIMITS

Source	Date	Tolerance Dose (R/day)	Applicability
U. S.	1931	0.2	X-ray
International	1934	0.2	X-ray
U. S.	1936	0.1	X-ray
U. S.	1938	0.1	X & γ-rays

It is of interest to note that these last recommendations are equivalent to 36.5 rem per year, more than seven times the currently permissible annual exposure dose for radiation workers.

In the early 1920's the harbinger for present plutonium standards emerged from a factory in New Jersey in the form of the deaths of a number of women employed as luminous watch face painters. Subsequent investigations over the period of several years pointed to the radium in the paint mix as the culprit (Ref. 3). By this time radium was also being employed in solutions as a tonic (Ref. 4). During the 1930's the work of Evans and his associates (Refs. 5, 6), in following radium cases and determining the biological parameters of uptake, elimination and body burdens, resulted in the recommendation that 0.1 μg of radium constitute a tolerance quantity and that 10^{-11} Ci/l of radon gas used as an air concentration limit (Ref. 7). Thus, in 1943 when the Manhattan District Plutonium Project was beginning there were only three tolerance values for occupational exposure to radiation and radioactive materials (Ref. 2).

1. 0.1 R/day for external X & γ-rays.
2. 0.1 μg of radium as a maximum body burden.
3. 10^{-11} Ci/l of radon as an occupational air concentration.

THE MANHATTAN PROJECT

The extraordinary safety record of our atomic industry is typified by events within the Manhattan District in early 1944. At a time when plutonium was available in only milligram amounts (much less for biological study), it was suggested by Dr. Hamilton at Crocker Radiation Laboratory in Berkeley, California, that plutonium might be less toxic than radium by a factor of 50 and that 5 μg (0.3 μCi) be used as an internal tolerance level. The estimate was probably based on equivalent alpha energy deposition in comparison with Ra-226 (Ref. 2). Based on this burden a two-year occupational air concentration of 2×10^{-15} g/cm^3 (1.2×10^{-4} μCi/m^3)* was proposed shortly thereafter. In April 1944 a one-year air tolerance of 5×10^{-10} μg/cm^3 (3×10^{-5} μCi/m^3) was suggested based on the 0.1 R/day limit for X and γ radiations and an assumption that alpha radiation is 10 times as damaging as X or γ. This last value remained as the air concentration guide for the remainder of the Plutonium Project. It is interesting to note that this value corresponds to a lung burden of 0.04 μCi, a value that is currently accepted as the non-occupational maximum permissible plutonium body burden (MPBB) for man. In March 1945 a safety factor of 5 was introduced to the MPBB because studies at Berkeley concluded that plutonium in bone was much less uniform than was radium. Hanford Operations authorities introduced a factor of 10 for their operations. In August 1945 Hanford established drinking water standards based on a maximum permissible body burden of 0.5 μg (Ref. 2).

Table II summarizes the plutonium standards situation as it existed at the termination of the Manhattan Project.

TABLE II

PLUTONIUM OCCUPATIONAL PROTECTION CRITERIA DERIVED DURING THE MANHATTAN DISTRICT'S PLUTONIUM PROJECT (1943-1946)(Ref. 2)

Maximum Allowable Body Burden		
Manhattan District	1.0 μg	(0.06 μCi)
Hanford Operations	0.5 μg	(0.03 μCi)
Air Tolerance Concentration		
1-2 year exposure	5×10^{-10} μg/cm^3	(3×10^{-5} μCi/m^3)
Water Tolerance Concentration		
Community	1×10^{-5} μg/cm^3	(0.6 μCi/m^3)
Plant	5×10^{-5} μg/cm^3	(3.0 μCi/m^3)

In summary we should note that all of the body burden standards were developed by comparison of plutonium with radium, with bone as the critical organ. The air concentration values are based on a lung burden producing 0.1 R/day. The plant water concentration value is based on a 30-year exposure, absorption of 0.05 percent and intake of 2 liters per day while the community value rests on a 60-year exposure and a 5-liter daily intake.

* For the remainder of this report we will use a specific activity for Pu-239 of 1.36×10^9 dpm/μg. Thus for Pu-239:

$$1 \text{ Ci} = 16.3 \text{ g} \qquad (1)$$

TO THE PRESENT

The period following World War II to the present has been one of continuing study of the plutonium problem. In September 1949 a tripartite conference on permissible dose was held at Chalk River, Ontario, Canada. Occupational protection criteria for plutonium were issued and are listed in Table III. The final selection of levels, derived in relation to radium, was made after considerable debate during and after the conference. United States AEC Operations proceeded using these values.

TABLE III

PERMISSIBLE PLUTONIUM LEVELS - CHALK RIVER FINAL REPORT (Ref. 2)

Maximum Permissible Amount in Body	0.5 μg (0.03 μCi)
Maximum Permissible Air Concentration (24 hr day)	2.5×10^{-5} μg/m^3 (1.5×10^{-6} μCi/m^3)
Maximum Permissible Drinking Water Concentration	20 μg/m^3 (1.2 μCi/m^3)

The value for the maximum body burden was adjusted to 0.04 μCi (0.6 μ g) soon thereafter and that burden is listed in the 1950 Recommendations of the ICRP (NBS Handbook 47) (Ref. 8).

We now enter a period when the biological research community increased their efforts to study plutonium as itself rather than plutonium in relation to radium. Biological half time and other metabolic constants were developed. These developments will be discussed more fully in Section IV of this study. It suffices for the present purpose to follow the results of this work as it applies to policy and direction. Table IV lists values adopted during the decade of the 1950's.

TABLE IV

PLUTONIUM STANDARDS ADOPTED DURING THE 1950's

	NCRP(Ref. 9) 1950	Harriman Conference(Ref. 3) 1953	ICRP(Ref. 10) 1955	ICRP(Ref. 11) 1960
"Soluble" Forms				
Maximum Permissible Body Burden	0.04 μCi	0.04 μCi	0.04 μCi	0.04 μCi
Maximum Permissible Concentration-Water	1.5 μCi/m^3	--	6.0 μCi/m^3	50 μCi/m^3
Maximum Permissible Concentration-Air	2.0×10^{-6} μCi/m^3	--	2.0×10^{-6} μCi/m^3	6.0×10^{-7} μCi/m^3

TABLE IV (Cont)

"Insoluble" Forms

Maximum Permissible Lung Burden	0.008 μCi	--	0.02 μCi	--
Maximum Permissible Concentration-Air	2.0×10^{-6} μCi/m^3	--	2.0×10^{-6} μCi/m^3	1.0×10^{-5} μCi/m^3

The 1960 ICRP values in Table IV are those currently recommended. The terms "soluble" and "insoluble" used in Table IV are relative and relate to plutonium's reaction to body fluids and processes.

The subject of maximum levels of surface contamination, although not treated in Table IV, was of concern to those working with plutonium. A level of 0.07 μCi/m^2 (1 μ g/m^2) was used during the 1950's as a suggested limit for laboratory working surfaces. This figure was never suggested as pertaining to the general environment. Rather, it represented the "then existing capability of a survey instrument" and was used as a "good house keeping" level (Ref. 1).

Incident to field testing operations in Nevada (OPERATION PLUMBBOB), a hazards analysis was made by Los Alamos Scientific Laboratory (LASL) personnel (Ref. 1). The analysis resulted in the earliest environmental surface contamination level which we have uncovered. The authors suggested a contamination level of 100μg/m^2 (6μCi/m^2) as being realistic for lifetime occupancy, with the lung being the organ at risk.

PRESENT STANDARDS

We have followed the development of standards during this century relating to the plutonium hazard. It remains to specify current standards as they exist within the several regulatory entities in the United States. Policies and directives of these organizations have generally resulted from two sources; first, those activities regulated or controlled by the AEC, and second, those activities of the Armed Forces dealing with weapons programs. In general, the doctrine of the latter have followed those of the former. It is reasonable, therefore, to begin with the policies of the AEC.

Atomic Energy Commission Policy

The AEC is the foremost regulator of the use of radioactive materials in this country. It derives this power from the Congress and furthers its responsibilities in two ways; first, through its licensing power and second, by regulation of the activities of its contractors.

The document of primary concern to these licensees and contractors in the area of contamination and radiation dose limits is Chapter 1, Title 10, Code of Federal Regulations, Part 20 (10 CFR 20), Standards for Protection Against Radiation. While a portion of Part 20 deals with administrative matters, much of it compiles the recommendation of the ICRP and NCRP and gives to them the weight of law. A list of those items of interest to the plutonium hazard follows (Ref. 12):

1. 10 CFR 20.101: Limits occupational dose to an individual's blood forming organs to 5 rem per year rated at 1.25 rem per quarter. The rate may be raised to 3.0 rem per quarter if the individual's accumulated whole body dose does not exceed 5 (N-18) rem, where N is the individual's age in years.

2. 10 CFR 20.103: This part establishes maximum concentrations of radioactive materials to which individuals in restricted areas may be exposed. For the present purpose we may refer to these as occupational concentrations. This part refers to a table of radionuclide concentrations which also appears in NBS Handbook 69 (Ref. 11). The table is based on a 40-hour week.

3. 10 CFR 20.104 (a): Restricts exposure of minors (less than 18 years) to 10 percent of the whole body limits specified in item 1 above, i.e., 0.5 rem per year whole body.

4. 10 CFR 20.104 (b): Restricts exposure of minors to lesser concentrations of radionuclides in restricted areas than is permitted for adults.

5. 10 CFR 20.105: Restricts the uses of radioactive material to those that limit the whole body exposure of non-occupational individuals to 0.5 rem per year, limited by a rate of 2.0 mrem per hour or 100 mrem per 7-day week.

6. 10 CFR 20.106: Restricts release of effluents to unrestricted areas to concentrations not greater than those referenced in item 4 for minors.

7. 10 CFR 20.303: Restricts release into sanitary sewer systems to certain tabularized quantities and to materials that are readily soluble or dispersible in water.

8. 10 CFR 20.304: Restricts dispersal by burial to certain tabularized quantities and modes of burial.

9. 10 CFR 20.305: Restricts disposal by incineration except as approved after application to the AEC.

The above directions and restrictions apply by law to activities licensed by the AEC. In general, they also apply to operations of AEC contractors. In many cases provisions are included permitting exceptions to rules upon an application which shows that the exception can be regulated with respect to safety to individuals and the population in general. As previously stated, this part is a compilation of the limits and concentrations recommended by the ICRP and NCRP. As such it is reasonable to use these rules as the basis of further discussions of the plutonium hazard. Table V summarizes the limits specified in 10 CFR 20.

TABLE V

10 CFR 20 RADIATION EXPOSURE AND PLUTONIUM LIMITS

Occupational exposure to whole body; head and trunk; lens of eyes; gonads; or active blood forming organs[1]		5.0 rem/year 1.25 rem/quarter or 3.0 rem/quarter if total exposure < 5(N-18) rem
^{239}Pu Maximum Permissible Occupational Concentration (MPC) in Air[2]	soluble insoluble	2×10^{-6} μCi/m^3 4×10^{-5} μCi/m
^{239}Pu Maximum Permissible Occupational Concentration in Water[2]	soluble insoluble	100 μCi/m^3 800 μCi/m^3
Non-occupational exposure to whole body or blood forming organs		0.5 rem/year limited to 2.0 mrem/hour or 100.0 mrem/week
^{239}Pu Non-occupational MPC in Air[3]	soluble insoluble	6×10^{-8} μCi/m^3 1×10^{-6} μCi/m^3
^{239}Pu Non-operational MPC in Water[3]	soluble insoluble	5.0 μCi/m^3 30.0 μCi/m^3
^{239}Pu release to sanitary sewer the larger of a. or b. limited by	soluble insoluble	a. 100 μCi/m^3 - day 800 μCi/m^3 - day b. 0.1 μCi/day 1.0 Ci/year
^{239}Pu for burial		10.0 μCi total

Notes: 1. Exposure to minors limited to 10 percent of these values.
2. Computed at 40 hrs per week, 50 weeks per year for 50 years exposure.
3. Computed at 168 hours per week, i.e., continuous. Also applies to minors in restricted areas.

It is useful at this point to present various limits for plutonium-239 listed in NBS Handbook 69 (Ref. 11), as is done in Table VI. Those values also appearing in 10 CFR 20 are inclosed by parenthesis. 10 CFR 20 values for the non-occupational case (168 hr week) are more restrictive by a factor of 10.

TABLE VI

1959 RECOMMENDATIONS OF THE NCRP FOR PLUTONIUM (Ref. 11)

	Organ	Maximum Permissible Body Burden (μCi)	Maximum Permissible Concentration ($\mu Ci/m^3$) For 40 hr week MPC Water	For 40 hr week MPC Air	For 168 hr week MPC Water	For 168 hr week MPC Air
Soluble	Bone	0.04	(100.0)*	(2.0×10^{-6})*	50.0	6.0×10^{-7}
	Liver	0.4	500.0	7.0×10^{-6}	200.0	2.0×10^{-6}
	Kidney	0.5	700.0	9.0×10^{-6}	200.0	3.2×10^{-6}
	G.I.	--	800.0	2×10^{-1}	300.0	6.0×10^{-2}
Total Body		0.4	1000.0	1.0×10^{-5}	300.0	5.0×10^{-6}
Insoluble	Lung	--	--	(4.0×10^{-5})	--	1.0×10^{-5}
	G.I.	--	(800.0)	2.0×10^{-1}	300.0	5.0×10^{-2}

*Parenthesis indicate values also appearing in 10 CFR 20.

Note that no limits have yet been set concerning maximum permissible surface contamination. Personal experience with AEC licensing indicates that where operations involve surface contamination, contamination densities must be shown on application to be such that the concentrations in air listed above will not be exceeded.

One other limit imposed by AEC regulations in 10 CFR 140 is of interest. That part deals with financial protection of licensees and indemnity agreements, and lists 0.35 $\mu Ci/m^2$ (Pu alpha) as a surface contamination level above which the AEC will find that a substantial discharge to offsite property has occurred (Ref. 12). This quantity of Pu-239 corresponds to contamination of approximately 6 $\mu g/m^2$.

Department of Defense Policy

Present DOD policy with regard to plutonium decontamination is contained in the joint AEC-DASA publication TP 20-5, "Plutonium Contamination Standards," 22 May 1968, with Change 1, 15 August 1969 (Ref. 13). TP 20-5 "prescribes joint DOD-AEC policy relative to decontamination requirements for terrain, structures, equipment, and other objects, contaminated by alpha emitting nuclear materials as a result of an accident involving United States (U. S.) nuclear weapons."

The decontamination standard prescribed is that areas contaminated to levels greater than 1000 $\mu g/m^2$ shall be decontaminated until surface readings are reduced to less than 1000 $\mu g/m^2$ where such reduction is possible and is consistent with reasonable cost and effort. This level corresponds to approximately 61 $\mu Ci/m^2$. The policy is not intended to represent an inflexible rule but represents a starting point for consideration.

Army Policy

Army policy is contained in FM 3-15, "Nuclear Accident Contamination Control," 17 June 1966 (Ref. 14) (now being revised). The Army designates Pu-239 contamination greater than 1000 $\mu g/m^2$ as being a "significant hazardous level," however, "any concentration higher than 10 $\mu g/m^2$ may produce a serious resuspension problem."

For decontamination purposes terrain contaminated above levels of 3,500 $\mu g/m^2$ is defined as a chronic hazard area and should receive first priority for consideration, i.e., immediate or deferred decontamination. Levels above 1000 $\mu g/m^2$ "should" be decontaminated depending upon area population, use, cost and similar factors. An additional statement is worth quoting; it is: "In general, populated areas should be decontaminated to essentially zero levels whenever possible."

The DOD policy level of 1000 $\mu g/m^2$ and the Army guides of 3500 $\mu g/m^2$ and 1000 $\mu g/m^2$ seem to be outgrowths of the same work done following OPERATION PLUMBBOB (Ref. 15). The level of 3500 $\mu g/m^2$ was arrived at using lifetime occupancy and the lung as the critical organ. This level was reduced by a factor of 3.5 and 1000 $\mu g/m^2$ was recommended as the level of concern in the accident situation.

Navy Policy

Navy policy relating to plutonium contamination is published in NAVMED P-5055, "Radiation Health Protection Manual," 1964, with Changes 1-6, the latest dated July 1970 (Ref. 16). Radioactive contamination is defined as "50 pCi for alpha activity as measured on a dry filter paper wiped over an area of 100 cm^2" (0.005 $\mu Ci/m^2$).

An airborne radiation area is defined as any area to which personnel may obtain limited access and in which airborne concentrations exist in excess of the occupational levels stated in 10 CFR 20. Although a plutonium body burden is not discussed per se, it is defined as one of a general case as, "A maximum permissible body burden shall be limited to that amount which results in an exposure not to exceed the biological equivalent of 0.1 μg of radium-226 in the bones, nor 15 rem per year or 5 rem per quarter for other organs."

It is of interest that rather than specifying 1.25 rem per quarter for whole body external exposure limits as does 10 CFR 20, the Navy omits that level and specifies the limit of 3.0 rem per quarter, an option of 10 CFR 20, limited by the lifetime dose formula, 5(N-18).

Decontamination, in NAVMED P-5055, is treated as a control measure, along with monitoring and isolation, "to protect personnel in the contaminated area (levels greater than 0.005 $\mu Ci/m^2$), and to stay within the limitation of maximum permissible concentrations of radionuclides....." of 10 CFR 20. This level is reasonable in the laboratory environment and agrees with those established for laboratories, e.g., LASL (Ref. 17).

The naval incident response team follows AEC-DASA publication TP 20-5 (Ref. 13) for case of environmental "real estate" contamination incidents. Thus, 1000 $\mu g/m^2$ (61 $\mu Ci/m^2$) is recognized as the level of concern.

Air Force Policy

Ultimate Air Force policy for decontamination following a nuclear weapon accident is provided by AF Manual 355-1, "Disaster Preparedness, Planning and Operations," with Change 1 dated December 1966 (Ref. 18). With reference to contamination levels paragraph 14-13 states, "The On-Scene Commander will have to evaluate the advice of the on-scene medical officer in the context of political, diplomatic, and other significant considerations."

Air Force Regulation 160-132, "Control of Radiological Health Hazards," December 1968 (Ref. 19) specifies permissible fixed alpha contamination limits for laboratory equipment and working surfaces at 900 dpm/100 cm^2 (0.04 $\mu Ci/m^2$). AFR 161-8 (Ref. 20) and AFP 160-6-7 (Ref. 21) also treat exposure limits and permissible concentrations. These publications are, in effect, 10 CFR 20 and NBS Handbook 69.

Thus we find that Air Force policy recognizes generally accepted limits for radiation exposure and airborne concentrations. In addition, the Air Force presents the On-Scene Commander certain latitude for decontamination of the nuclear weapons accident environment.

Policies of Other Federal Agencies

Literature search and personal contacts indicate that four other federal agencies currently specify contamination limits. All of these are specified in conjunction with packaging of materials for shipment. The agencies and reference documents are as follows:

1. Department of Transportation, 49 CFR 173.397 (Ref. 22).
2. Federal Aviation Agency, 14 CFR 103 (Ref. 23).
3. U. S. Coast Guard, DOT, 46 CFR 146.19-30 (Ref. 24).
4. U. S. Postal Service (Ref. 25).

The Department of Transportation specifies 220 dpm/100 cm^2 (0.01 $\mu Ci/m^2$) of removable alpha contamination as being "not significant", and specifies that level for reuse of previously contaminated transport vehicles.

Standards of the Department of Transportation are generally specified by the other three agencies in their directives. We note that these limits do not refer to large area environmental contamination. Table VII summarizes current U. S. federal standards.

TABLE VII

SUMMARY OF FEDERAL AGENCY PLUTONIUM

DECONTAMINATION STANDARDS

Environmental*

Agency	Standard $\mu g/m^2$	or ($\mu Ci/m^2$)
DOD/AEC (Weapons Accident)	1000	61
U. S. Army	1000	61
U. S. Navy	1000	61
U. S. Air Force	1000 or as directed by On-Scene Commander	61
AEC** (See note. Levels are inferred)	a. 5.6	0.34
	b. 0.96	0.06

Transportation

Dept. of Transportation	0.16	0.01
Federal Aviation Agency	0.16	0.01
U. S. Coast Guard, Dept of the Treasury	0.16	0.01
U. S. Postal Service	0.16	0.01

*These standards shown as "Environmental" pertain to wide area "real estate" contamination. Those listed under "Transportation" refer to conveyances and packages, and not to wide area situations. Many agencies have lower standards for laboratory use.

**AEC does not stipulate an environmental decontamination limit as such. Inferred level "a" is from 10 CFR 140 and concerns indemnity agreements. Inferred level "b" is based on 10 CFR 20 limits on effluent releases to uncontrolled areas and application of a resuspension factor of $10^{-6} m^{-1}$.

SECTION III

PHYSICAL PARAMETERS OF PLUTONIUM CONTAMINATION

This section of the study will deal with those factors involved during and after a weapons accident which lead to a resuspended plutonium airborne concentration that is potentially hazardous. The section immediately following will deal with information currently known concerning the disposition of inhaled plutonium in the body.

PHYSICAL PROPERTIES OF PLUTONIUM

Plutonium, with an atomic number of 94, is a heavy metal with a density of 16.5 - 19.7 g/cm^3 depending upon its crystalline phase. Although shiny when freshly worked, it quickly oxidizes when exposed to air taking on a dark brown to black appearance. It is pyrophoric and should be stored under oil when in fine form. It reacts with water and is potentially dangerous due to liberated hydrogen. There are many isotopic forms; however, those of interest in the accident situation are Pu-238 and Pu-239. These isotopes are alpha emitters and constitute an internal biological hazard. Plutonium-239 is of interest because of its abundance in weapons and Pu-238 because of its use in power sources. Half lives for the two isotopes are 24,360 years for Pu-239 and 89 years for Pu-238. The specific activity of Pu-239 is about 0.061 Ci/g. Plutonium-238 is about 270 times more active (Ref. 26).

DISPERSION OF HAZARDOUS MATERIAL

In the accident situation a wide spectrum of developments can ensue. These range, in the impact case, from the weapon remaining intact, through weapon case rupture to high explosive detonation. If the incident involves fire, damage may range from case charring, through component melting (to include melting of the plutonium), to high explosive burning or detonation. The hazards of interest result when the plutonium is involved in a fire or high explosive detonation.

In these last situations plutonium is released from the immediate area and is subject to trajectories dependent upon its physical and chemical forms. Some may be dispersed in chunk form and subsequently constitute a minimal hazard. Of much greater concern is that material which is dispersed in the form of respirable aerosols. The plutonium in these aerosols is in the form of the dioxide, a compound that is quite insoluble. Our interest then is to describe the physical parameters of this aerosol.

In the fire situation with no high explosive detonation, dispersal of the plutonium dioxide as to amount is quite variable, dependent upon the surface of plutonium available to the fire. Thus when the weapon case is intact or partially so, perhaps only a limited quantity of plutonium is at risk. When major case damage has occurred with component breakup and dispersal, a larger surface is at risk. In tests conducted in a flue system plutonium was subjected to oxidation at temperatures ranging from 500° C to 850° C, within the range of petroleum fires. In this case the entire surface of the plutonium sample was exposed. The sample tended to flake with only a small portion, about 2 percent, being liberated as an aerosol, all in the respirable range (Ref. 27). Other variables which must be considered are any surfaces or scavenging material in the immediate vicinity of the fire. Although attempts have been made to evaluate the action of scavenging and plating, the data available are sparse, especially with regard to particle size. It suffices to indicate that materials such as aircraft structure or storage bunkers will be relatively heavily contaminated (Ref. 28).

The situation following detonation of the associated high explosive is considerably different. In this case the plutonium is reduced to fine particles and entrained by the cloud of detonation gases (Ref. 29). With the exception of scavenging actions of nearby structures, virtually all of the plutonium involved may be available for dispersal to the atmosphere. Data from tests conducted during field experiments indicate that approximately 20 percent of the aerosol liberated in such situations falls within the respirable range of particle diameters (Refs. 30, 31).

The fate of the aerosol in both the fire and detonation cases is a function of many variables. In each case the material is carried aloft in the cloud associated with the fire or explosion. An empirical relationship has been developed for the explosion case (Ref. 32) and data is available from field testing which relates aerosol mass distributions within the cloud. The fate of aerosols in the fire situations should be similar. The absolute cloud height will, of course, depend on the intensity and area of the fire. Relative mass distribution in the cloud, however, should be similar to the explosion case (Ref. 29).

Once the cloud has formed, the aerosol, with its own aerodynamic characteristics, reacts with the prevailing atmospheric conditions. The most important of these are wind direction, speed and atmospheric turbulence. Computer codes have been developed which treat the hazard associated with passage of the plume (Ref. 29) and the fallout on the ground as the plume disperses (Ref. 33). The former is concerned with the acute hazard of inhalation as the plume passes, while the latter has among its options the development of isopleths (contours of equal contamination density).

The following tables present results obtained using the two codes. Table VIII indicates, as a function of distance and number of weapons, the percent of the time that meterological conditions would exist yielding an acute dose committment of 15 rem, a United Kingdom one-time emergency dose. Assumptions employed include:

1. Two percent of the plutonium involved in a fire is liberated as an aerosol and all of it is respirable.

2. Twenty percent of the plutonium liberated by an explosion is respirable.

3. Plume height for fire calculations is assumed to be the same as that for explosive releases. This is conservative if a jet fuel fire larger than about 100 to 200 ft^2 is involved.

TABLE VIII

PERCENT OF TIME A 15 REM COMMITTMENT IS EXCEEDED

DURING PLUME PASSAGE AT DISTANCE SHOWN (Ref. 30)

	Explosion			Fire		
#Weapon	10%	5%	1%	10%	5%	1%
1	-	0.2 mi	0.4 mi	-	-	-
2	-	0.9 mi	1.3 mi	-	-	-
3	-	1.0 mi	0.9 mi	-	-	-
4	1.0 mi	1.6 mi	2.5 mi	-	-	-

Eighty different combinations of meterological parameters were used in the calculations. The authors summarize stating, "The fire calculations for four weapons does not predict doses exceeding 15 rem anywhere (except possibly right in the fire); whereas, the detonation of even one weapon does."

Table IX presents a summary of deposition calculations. Predictions of fallout patterns are reported for cloud heights varying from 500 feet to 3000 feet for fires and 1000 feet for explosions. Winds at 5 and 10 mph, with and without shear, were considered. The data are presented in square kilometers and represent the prediction for incidents involving four weapons. The lower figure for each condition corresponds to the situation without wind shear while the larger represents that with wind shear.

TABLE IX

SUMMARY OF DEPOSITION CALCULATIONS (Ref. 29)

Level		Wind Speed	
		5 mph	10 mph
μ g/m^2	Case	(km^2)	(km^2)
0.1	Explosion	43-85*	43-64*
	Fire	0-10	0-5.2
2.0	Explosion	30-64*	32-47
	Fire	-	-
100	Explosion	4.4 - 5.0	5 - 5.2
	Fire	-	-
1000	Explosion	0.85 - 0.87	0.75 - 0.82
	Fire	-	-

* Areas exceed these values - isopleths did not close.

These data with respect to explosions are in general agreement with OPERATION ROLLER COASTER findings (Ref. 34).

RESUSPENSION OF THE HAZARD

Investigators are in general agreement that plutonium resting on the ground does not constitute a hazard to man. Concentration factors in plants and various food chain systems show large and successive reductions between stages.* (For example, see Ref. 35, p. 6). The hazard to man from plutonium contaminated surfaces is due to resuspension of the radioactive particles into the breathing zone. This resuspension is a function of many variables, few of which are understood to the degree necessary to form well founded conclusions.

* One exception to this situation is concentration in marine plants (Refs. 36,37). Concentrations by factors of 10^3 have been reported.

Consider the case of open terrain contamination and remember that our interest is with particles in the respirable range. We must trace the fate of particles falling to the surface that are in the respirable range and outside of it also. Particles in the fallout plume that are larger than the respirable range may later become respirable as a result of abrasion due to meterological conditions or use of the surface by animal and man. Particles that are within the respirable range may fall to earth and adhere to other particles, resulting in a combined particle that now falls beyond the respirable range. This adhesive force can be quite large. Laboratory tests using sonic vibration up to 90 G's failed to separate the adhered particles (Ref. 38).

Weathering alone tends to reduce detectable alpha contamination as the plutonium particles are mixed and covered by soil. Desert sands offer gigantic hiding places to the contaminant in the micron range. Resuspension from grassy surfaces tends to be greater than from soil-only surfaces, probably because of the contaminant being above the boundary layer between the surface and turbulent air (Ref. 39). Similarly, resuspension from buildings and other structures which are more subject to direct winds will differ from that of soil.

The term, resuspension factor, plays a significant role in the description of resuspended particulates. Resuspension factor is defined as the ratio of airborne concentration in units of activity or mass of material per unit volume, to the activity or mass of material per unit area of surface contamination. Its units thus become inverse length.

Experiments to determine resuspension factors under various conditions have been carried out in the laboratory and in field tests. Each is valid only for the conditions of the individual experiment.

Figure 1 summarizes the results of extensive laboratory tests conducted indoors with PuO_2 contaminant and several different types of surfaces (Ref. 40). The histograms are for different cases of personnel activity in the laboratory. The designators, K_2 and K_3, represent activities of 14 steps/min simulating detailed monitoring procedures, and 36 steps/min representing spot monitoring. Experiments to establish K_4 representing 200 steps/min and surface disturbance with blowers resulted in values which were not significantly different from those presented for K_3.

The data indicate that for these laboratory surfaces the resuspension factor will probably be in the range between 10^{-6} and 10^{-4} m^{-1}, and agree well with the resuspension factor of $4 \times 10^{-5} m^{-1}$ used to establish United Kingdom derived working limits for alpha contamination. The authors state in their conclusions that the resuspension factor in this series of experiments is probably larger than would generally be experienced.

Resuspension factor values from a number of United Kingdom field tests are available (Ref. 39). These values range from 10^{-4} m^{-1} for inclosed spaces and very dusty operations to 10^{-8} m^{-1} when no artifical disturbance was created. The author concludes that a representative value for an outdoor resuspension factor under quiescent conditions is $10^{-6} m^{-1}$. Under conditions of moderate activity this factor should be increased by a factor of 10. Results of OPERATION PLUMBBOB suggest that resuspension factors of 10^{-5} to 10^{-7} m^{-1} are reasonable.

Air sampling of resuspended particulates during OPERATION PLUMBBOB indicated that a majority of the particles lie within the respirable range with a mass median diameter of 1.5 - 2.0 μm. In addition, data are presented which show a general decline in air concentration with time. An effective half-life of 35 days for this decrease is suggested (Ref. 30).

There remain two interesting findings that deserve mention prior to summarizing. The first is a finding of OPERATION ROLLER COASTER resulting from a rather exhaustive particulate analysis

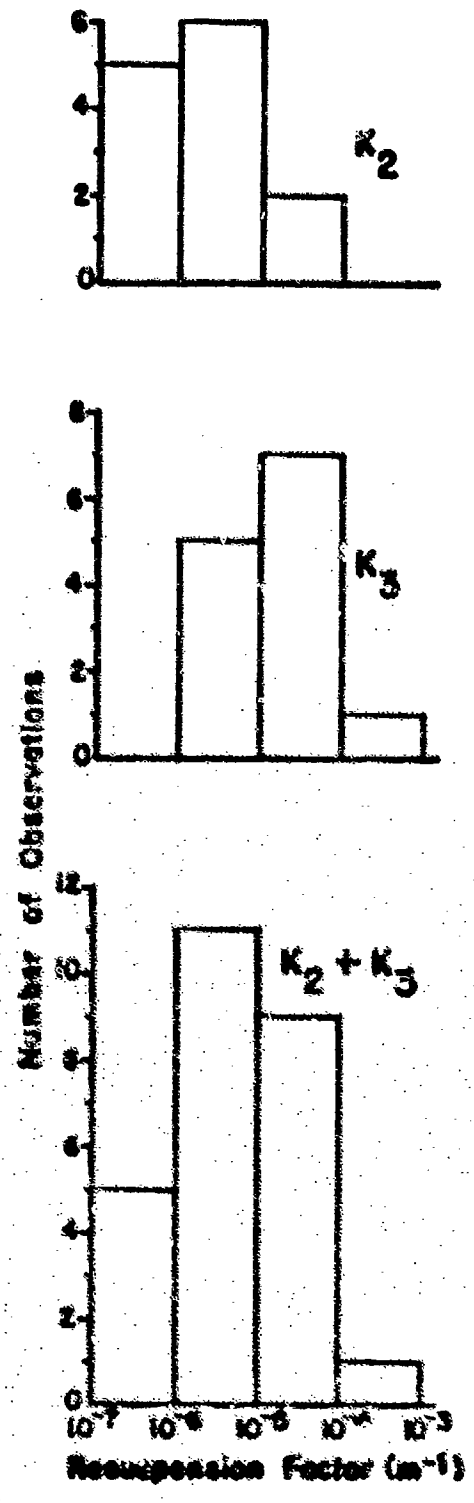

Figure 1. Summary of Laboratory Resuspension Tests (Ref. 40)

(Ref. 41). A large range of activity (radioactivity) was observed for a given particle size. While radiochemical analysis indicated that some particles of very high percentage of plutonium were formed, "the majority of particles had plutonium oxide contents of less than 20 percent...." in one test and less than 1 percent in another. Particle density estimates from less than 1 and up to 19 g/cm^3 were made, although the densities tended toward the higher range as particle size decreased. Thus, when drawing conclusions concerning inhalation hazard following a weapons plutonium contamination incident, it may be quite inaccurate to base the analysis on particle size alone. That is, a particle in the respiratory size range is not likely to be pure PuO_2. In addition, inhalation dynamics is a function of particle density. Analysis based on densities of the order of 10 g/cm^3 will be in error when particles of the range of densities cited make up the population. It is possible, however, that the variance may average out in the density case. No conclusion was made in the literature.

The second finding of interest is the general difficulty of determining an actual ground contamination level, especially when looking at relatively low levels of contamination. When surveying following an incident, movement of the water across adjacent areas can cause large changes in the surface contamination estimate. Additionally, radiochemical analysis of soil samples points up large variations of estimates of adjacent samples and substantial variance among different parts of the same sample due to sample inhomogeneity (Ref. 42). OPERATION PLUMBBOB isopleths determined by survey differed from those derived by soil sampling by factors of 2-4, the surveys being the higher of the two (Ref. 30).

We have pointed out that the variables associated with the description of the physical properties of plutonium contamination are many and poorly defined. We do not mean to imply that researchers do not recognize this fact. Indeed, such conclusions were constantly being drawn by knowledgeable investigators during our work on this study. Ongoing work is directed toward better definition of the parameters involved.

Perhaps the most notable current effort in this area is that being undertaken in a joint AEC/Environmental Protection Agency (AEC/EPA) interdisciplinary applied research program using the Nevada Test Site as the area of experiment. With a base of data developed from previous testing over the years, studies will be made covering all factors of the plutonium contamination hazard. Initial surveys and soil sampling are already underway. Work on determination of resuspension factor parameters started early in 1972. The programs presently envisioned will require several years for completion.

SUMMARY

The derivation of plutonium decontamination standards requires that the resuspension factor and description of the resuspended hazardous material be defined. In the case of the resuspension factor we have some values ranging over 9 orders of magnitude. Analysis of the literature and expert opinion suggests that a resuspension factor of

$$k = 10^{-6} m^{-1} \qquad (2)$$

be employed. Although we have cited some of the variables involved in describing the fate of the plutonium particulates, it seems most reasonable and properly conservative to assume that all of the material that is resuspended into the breathing zone falls into the respirable range. Additionally, any analysis performed at incident sites should be based on specific particulate activities and not rely on particle size for estimates of total activity.

SECTION IV

THE BIOLOGY OF PLUTONIUM CONTAMINATION

THE EARLY YEARS (Ref. 2)

The early days of the Manhattan Plutonium Project saw minimal amounts of plutonium being available for biological research. In January 1944 after concern was voiced as to its possible harmful effects, 11 mg was allocated to Berkeley for animal uptake, distribution and excretion studies. A record for speed in biological research may have been established when Dr. Hamilton reported about a month later,

> "Oral absorption of all valence states is less than 0.05 percent; lung retention high; absorbed material predominantly in skeleton; excretion very small in urine and feces."

Later in 1944 metabolism and toxicity studies were begun at the University of Chicago, along with studies to determine acute/subacute levels for plutonium. Inhalation and tracheal intubation studies were begun in June 1944. By early 1945 the data from animal excretion studies were applied to human urine assay for workers in plutonium laboratories. In April 1945 tracer studies in hopelessly ill human volunteers were being employed to further fix human excretion rates.

As mentioned in Section II of this study, the early permissible levels for plutonium contamination were based on comparisons of relative hazard with respect to radium. At this early time autoradiograph studies at Berkeley established that plutonium's distribution in the bone was much less uniform than was that of radium.

The aforementioned studies continued through the Forties. The work leading to the establishment of 0.04 μCi as a maximum permissible body burden was summarized by Bruce in April 1950. Relative toxicity ratios between radium and plutonium in terms of injected activity per kilogram of subject were:

	Ra:Pu
1. Acute toxicity to small animals	15:1
2. Chronic survival in small animals	10:1
3. Formation of bone tumors in rats and mice	15:1
4. Formation of bone tumors in rabbits	8:1
5. Bone fractures in rats and rabbits	10:1

Other studies during the period included delayed toxicity in dogs at the University of Utah School of Medicine and chronic inhalation in dogs at the University of Rochester School of Medicine and Dentistry.

In 1950 the ICRP recommendations previously cited incorporated concepts of a "standard man" and employed a lung model in its derivation of suggested standards. Recognition was given to previous experimental work which derived various constants for the metabolism of plutonium. The model used estimated that 10 percent of soluble compounds inhaled is absorbed with a biological half time of 10^{-4} days. The mean life for insoluble plutonium compounds in the lung was set at 200 days. The maximum permissible air concentration calculation for insoluble compounds led to a value over three times greater than that for soluble compounds. The lower figure was recommended as the limit for compounds in both categories, however, because of "the possibility of the transference of some of the insoluble material from the lungs to the skeleton" (Ref. 8).

The NCRP published a new lung model in 1953. Their suggested maximum permissible concentrations remained the same as those of the ICRP; however, some changes in metabolic constants were suggested. This model states that 75 percent of soluble inhaled material is deposited in the lung and two-thirds of this is scavenged in the upper respiratory tract and soon cleared through ciliary action and swallowed. The remaining one-third is retained in the lower respiratory tract. Fractions of both upper and lower tract quantities go to the blood and thence to organs fed by the blood. Twelve percent of insoluble aerosols are retained in the lower respiratory tract for long periods, with the lung being considered as the critical organ (Ref. 9).

National and international committees continued their work during the 1950's and early 1960's toward refining models and methods of treating plutonium in the human biological system. Concepts of absorbed dose, relative biological effectiveness, quality factors and the like were suggested, defined and redefined, all in an attempt to provide the investigator with better bases from which to draw conclusions. The current 1965 model is discussed in Section V of this study; the 1973 proposed model is discussed in Appendix A.

RECENT AND CURRENT RESEARCH

Researchers in biology have attacked the plutonium hazard in several ways. The research has involved soluble and insoluble compounds. Administration has encompassed laying in abrasions, injection in and to various body locations, intubation, implantation and inhalation. An important question for the present study is, "What portion of the literature is of interest in the weapons accident situation?" The answer is, "All, in varying degrees."

The argument has been repeated many times in the literature that the primary hazard to a population in the accident situation is the inhalation of the radioactive aerosol. The skin is a satisfactory barrier to external alpha radiations. The external gamma field is relatively insignificant following a nonnuclear detonation. Transport of insoluble material from contaminated abrasions is insignificant. Uptake through food chains is relatively low because of the large discrimination ratios involved, e.g., 10^{-4} to 10^{-6} (Refs. 43, 44, 45). One hazard of special interest in the accident situation - one of interest more to individuals than to the population - is that resulting from puncture wounds.

Thus, we are interested in the inhalation case. The fate of inhaled plutonium, however, includes uptake in organs other than the lung. The process involved in this uptake and in transport across deep lung barriers suggests that some solubilization or effectively similar process does occur. Langham in treating this situation suggests that the term "solubility" has not been well defined and "seems to relate more to the metabolic behavior of the material in the body than to the more familiar chemical concept of solubility" (Ref. 2). Additionally, a large portion of the aerosol that enters the lung is soon removed through ciliary action and ingested. The effect of these processes is not only to put the lung at risk, but also includes placing other organs at risk, some resulting from uptake from the blood and others resulting from direct radiation, e.g., the G.I. tract.

In summary, experiments involving inhaled PuO_2 aerosols are most directly applicable to our study. Those experiments employing injected soluble compounds give us information concerning metabolism of plutonium that has reached the blood. Implantation and intubation experiments provide an insight into biological mechanisms of insult and protection in the immediate area of particulates of high specific ionization potential.

One other source of information is of special interest. Through our years of experience with plutonium, accidents have occurred. A significant accumulation of data is available from this source and involves human subjects rather than laboratory animals.

Based on the early experimental work with plutonium there seemed little doubt that bone was the critical organ. Later work has cast some doubt on that finding, at least in some special cases. Experiments at Hanford on miniature swine have shown that after 2 years larger concentrations exist in the liver than in the bone. Earlier work was generally done at high dose levels. There is some evidence that a shift in critical organ may occur at lower dose levels (Ref. 46).

We should point out that the incident situation is concerned more with evidence gained from experiments resulting in low body accumulations than with that involved with high burdens. We are concerned with reducing the biological hazard to a minimum. The high dose experiments set the stage but the low dose experiments set the character of the play. The low dose experiment, however, is the more difficult and costly. Good statistics require large numbers of animals, both at risk and as controls. Lower doses are expected to require longer periods of latency before the insult is recognized.

Injection Studies

Our interest in plutonium administered by injection is to determine the fate of the radionuclide once it reaches the bloodstream. Injection was a favorite and reasonable delivery mode for the earlier studies whose emphasis was directed toward bone and other blood-fed organs, e.g., liver, kidney, spleen. The earlier studies were performed to determine gross uptake factors, metabolic rates and organ deposition rates. The injection experiments have continued and are now more sophisticated and are looking at specific biochemical processes and the differences that various forms of plutonium exhibit. Most of the work done to determine courses of therapy for the plutonium-burdened patient is also being done with injected solutions.

Uptake of plutonium from the blood to various organs is dependent upon its chemical form, aggregate size and blood chemistry of the experimental subject. Significant differences are seen at autopsy when graded solutions are administered. With decreasing particle size less plutonium is deposited in liver, spleen and bone marrow, while more is deposited in bone. With mice as the experimental animal, there is evidence that the pattern of deposition depends on a critical size of aggregated plutonium-containing particulates (Ref. 47). In a gross sense, investigations have found that twice as much polymeric as monomeric plutonium deposited in bone is required to produce equal incidence of bone tumors. The polymeric forms tend to deposit more in bone marrow than on surfaces as is the tendency for monomeric forms (Refs. 48, 49).

Plutonium initially deposited from blood on bone surfaces can also reach the marrow. The process is a function of subject age and is due to bone remodeling and osteoclastic resorption from surface to marrow (Refs. 50, 51).

Because of the historic link between plutonium and radium, there is a tendency to forget other organs at risk. The liver, spleen and kidney are also plutonium collectors and have also been studied with respect to plutonium uptake, retention and metabolic behavior. Work at the University of Utah has been underway for a considerable period. Some results of relatively low dose work are now being reported. Some effect of dose level is indicated. That is, splenic and renal retentions at lower doses are higher than would have been expected by extrapolating from higher dose studies. Beagles have received injections of from 0.016 to 2.8 μCi/kg of animal weight. This covers levels where life-shortening is negligible, through levels where osteosarcoma is induced to levels exhibiting extensive

skeletal and liver damage. Retention equations of decaying exponential form have been developed. The authors suggest that at low doses more attention should be placed on the aging mechanism rather than on sarcomas (Ref. 52).

In a "speculative" paper, investigators at the University of Utah have estimated probabilities of radiation induced tumors from a permissible body burden of plutonium. For a body burden of 0.04 μCi starting in the circulatory system with roughly half being deposited in bone and half in the liver, the authors find bone cancer probabilities of 0-5 percent and liver tumor probabilities of 0-10 percent. The range of probabilities is a function of the model employed in the calculation. Threshold, dose-rate and lifespan dose models were used. The threshold model is based on human experience with radium where no bone cancers have been observed below 1200 rads average bone dose. With conversion of 0.02 μCi plutonium organ burden to a 14 rad dose to bone and a 57 rad dose to the liver, this model suggests a vanishingly small probability. The dose rate model suggests that the risks to dog and man are linearly proportional to their dose rates averaged over long exposure times. At burdens of 0.016 μCi/kg, the lowest level at which neoplasms have been observed at Utah, skeletal dose rates of succumbing subjects averaged 8.8 rads/year. Probabilities by this model are 1 percent for bone and 2 percent for liver. The lifespan dose model suggests that for a fixed incidence of cancer among mammalian species, the required cumulative doses are rather similar. Probabilities for this model predict risks to the bone of 5 percent and 10 percent to the liver (Ref. 53). Although the authors admit that the risks estimated are uncertain, it is refreshing to see the estimates made. In many cases extrapolating to man from the various experimental animals is considered to be off-limits.

We do not imply that the preceding discussion is exhaustive with respect to injection experimentation. A comprehensive review would be impossible within the space available and the expertise of the authors. We do want to point out that much very fine work has been done and is ongoing at several locations. The low dose work that is underway will begin to provide answers in a few years to very nagging questions. Additionally, there has been a great deal of injection work done toward finding answers to accident conditions and therapy which we will discuss later.

Particulate Implants, Intubation and Injection

There is considerable interest among investigators concerning the relative risk to man of the homogeneous versus discrete distributions of hazardous material. In fixing maximum permissible organ burdens, the organ has historically been treated as a whole and doses have been averaged over the whole. In the case of highly radioactive particulates, a few particles can constitute a significant burden, because radiation doses in the immediate vicinity of the particles are high. Where most of the energy of decay is in the form of alpha particles (the case of plutonium), when we discuss a permissible burden we may be speaking of relatively few particles with limited volumes at risk. Investigators at LASL are studying the effects of small highly radioactive particles. In a theoretical approach, tumorigenic risk to skin and lung has been described (Ref. 54). With increasing dose, experimental evidence indicates that tumor incidence increases, finally reaching a maximum after which the cells at risk are damaged to the point where cell division can no longer occur. Thenceforth, the risk of tumorigenicity decreases with increasing dose. Currently 0.016 μCi of Pu-239 constitutes a lung burden.

Table X illustrates the strong dependence of lung tumor on particle size. The authors point out that there is as yet no experimental evidence for these probabilities which result from their use of a model of the alveolar region of the lung which maximizes the number of cells at risk.

TABLE X

TUMOR PROBABILITY FROM LUNG EXPOSURE TO 16 nCi OF $^{239}Pu\ O_2$ (Ref. 54)

Particle diameter	Number of Particles in 0.016 μCi Pu-239	Tumor Probability
0.1	5.61×10^{7}	2.2×10^{-4}
0.2	7.02×10^{6}	0.015
0.4	8.80×10^{5}	0.59
0.8	1.10×10^{5}	1.0
1.0	5.61×10^{4}	1.0
2.0	7.02×10^{3}	1.0
4.0	8.80×10^{2}	1.0

In the case of an exposure to a burden of 0.016 μCi Pu-239 for a period of 720 days, 3×10^{5} cells actually absorb over 95 percent of the total radiation for a dose of 1.6×10^{6} rad. A corresponding whole organ dose would be only 3.24 rads.

Experimental work at LASL employing injected PuO_2 microspheres is a follow-on to the theoretical study just discussed. (Ref. 55) A single microsphere was injected in the femoral vein of a rat. It was subsequently transported by blood until it lodged in the lung vasculature. These microspheres averaged 178 μm in diameter and were 81 percent Pu-238, 15 percent Pu-239. This mixture exhibits a considerably higher photon dose rate than would Pu-239 alone. In addition, some of the photons from Pu-238 are more energetic than those from Pu-239. The Pu-238 alpha energy averages about 5.5 MeV, slightly higher than Pu-239's 5.15 MeV. Considering only the alpha insult we must recognize that its energy is delivered over a limited volume. Its range is about 40 μm, but the dose rate falls rapidly beyond 25 μm. The authors estimate a cell population at risk of 3 to 6×10^{6} cells at 25 μm. This number seems large but is almost insignificant when one considers the organ cell population. The dose rate to the 40 μm sphere is estimated at 6×10^{8} rem/hr. Consider now the photon situation. Photon dose rates at the surface of the sphere run about 620 rads/hr, and decrease by factors of 10 and 100 at 200 and 700 μm respectively. In this experiment animals were sacrificed serially at from 1 to 600 days. What of the pathology? The authors concluded that these high dose rates and total doses caused surprisingly little change in the lung structure except in the immediate area of the particle. In addition, "The absence of severe biological response appears to be related to the number of cells at risk which, in turn, is a function of the number and size of particles making up the activity rather than to total activity."

An experiment was conducted at Colorado State University to determine the dynamics of lymph node accumulation from PuO_2 contaminated wound sites (Ref. 56). Results indicated that as much as 17 percent of the implanted activity was collected by the surrounding lymph node system at one year, with the majority of the material remaining at the implant site. The specific risk of lymph node irradiation is uncertain at this time. The situation is discussed in Section V of this study. These data do indicate, however, that a considerable burden of long-lived plutonium can reach the lymphatic system. Consideration should be given to these findings when considering wound site decontamination to include possible surgical removal.

Particulate Inhalation

We arrive in our discussion at the insult mode of the weapons and power source accident situation. Investigators are universal in their agreement that as long as the sleeping dog of plutonium contamination lies, it presents no hazard. But sleeping dogs awake, arise and travel the neighborhood. Therein lies the hazard-resuspended plutonium entering the breathing zone of the population.

Investigators using inhalation as the delivery mode have long recognized the importance of particle size in the response to lung deposition. Accurate determination of the particle size has been a continuing problem with these experiments. Work at Lovelace Foundation in the Fission Product Inhalation Program has attacked this problem and has resulted in excellent control of particle size samples. The latest publication of the ICRP Task Group on Lung Dynamics has recognized particle size dependence for lung deposition. Figure 2 presents their findings for man (Ref. 57, 58). The shaded area represents spread due to differences in lung tidal volume with the upper boundary at 2150 cm^3 and the lower at 750 cm^3.

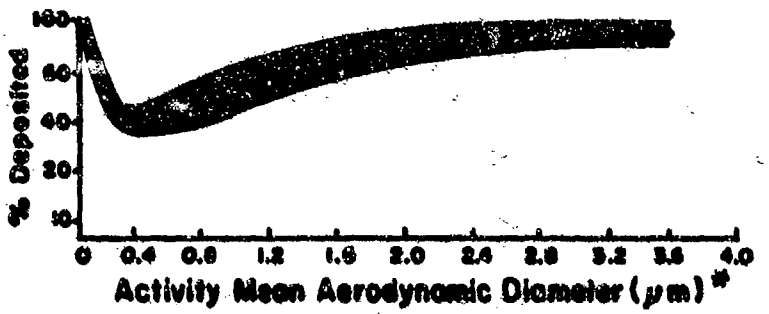

Figure 2. Total Deposition of Inhaled Particles (Ref. 57)

Small animals used in inhalation experiments do not exhibit the same deposition pattern as does man. Experiments have been done which point out the difference and we should consider that our knowledge of deposition is fairly good and will get better. Lovelace has developed a process for production of aerosols within tight bands of particle size. Their inhalation program using these aerosols is currently in progress (Ref. 59).

*

The term, "Activity Mean Aerodynamic Diameter" (AMAD) may require explanation. We can treat it by parts. "Aerodynamic diameter" is a term involved in a process which treats the physical properties (density, dimensions, etc.) of a particle and normalizes them to the aerodynamic properties of a particle of unit density. This normalization permits application of lung deposition models to particle populations of varying physical and aerodynamic properties. "Mean" applies to the arithmetic mean of the particle population. "Activity" denotes that the original measurements were made based on sample radioactivity levels.

Section V of this study treats the current ICRP lung model which includes data covering the stay times of plutonium particulates in the lung. (Appendix A does likewise for the proposed 1973 model). Work is underway at Battelle Northwest to determine the modes of lung storage and transport. We do know that the majority of particulates in the deep lung are very rapidly phagocytized (Ref 60). Similar phagocytic action has been reported after injection into the peritoneal cavity with over half being engulfed after 4 hours with subsequent translocation to visceral-abdominal tissues and out of the peritoneal cavity (Ref. 61). The Battelle Northwest work has found translocation to the alveolar epithelium (Ref. 60). Battelle work studying macrophagic action has led to several conclusions of interest (Ref. 62):

1. Plutonium particles are phagocytized rapidly reaching nearly 90 percent in the lung.

2. Particles are concentrated in macrophages creating hot spots of intense alpha activity.

3. The behavior of plutonium particles in the body seems to be largely determined in the early phases by the kinetic behavior of macrophages.

At this point we should treat an unknown. The ICRP lung model discussed in Section V and Appendix A of this study accounts for translocation of the contaminant from the pulmonary compartment of the lung to the blood and lymph. The process of this translocation is not well understood. At least two processes should be considered. The first involves transport of particulates into the alveolar wall structure as mentioned above and subsequent transfer to the bloodstream or lymphatic system. The second process involves solubilization of the "insoluble" particulate material followed by normal lung fluid transport processes. Both processes are being considered. There is some evidence to support both. The first has already been discussed. The second is based on relatively increased solubility of particulates as their surface to volume ratios increase. What part radioactivity of the material plays in supporting or hindering this transport is another unknown. Work with radionuclides other than plutonium also supports our investigation. Experiments carried out at the University of Rochester with chronic inhalation of UO_2 aerosols in the monkey, dog and rat are of interest (Ref. 63). Uranium is much less active than plutonium. However, the data are presented as a function of dose delivered (alpha energy of 4.5 MeV). Over periods of 5 years, the animals were subjected to concentrations of 5 mg U/m^3, 6 hours per day, 5 days per week. The lung and tracheobronchial lymph node burden accounted for over 90 percent of the U found in the body. After 5 years the integrated alpha dose to the lung of monkey and dog were 500 and 900 rads, respectively. The corresponding lymph doses are of the order of 10,000 rads. At 5 years, one dog in six exhibited necrosis and fibrosis of the lymph nodes. No other pathology was evident. In the monkey, lung and lymph node fibrosis appeared at 3.6 years at doses of 500 and 7000 rads respectively with similar findings at later times and larger doses. These histologic changes were described by the authors as being "minimal and present only in the tissues of a few animals." The trend was there, however, and histology of "similarly treated" animals clearly demonstrated radiation injury, at least sufficient for a recommendation for possible lowering of concentration limits in the uranium industry.

The inhalation work done at Battelle Northwest covers broad areas of interest and is perhaps the most comprehensive. This work can be covered here only in strict summary. We suggest Reference 62 for an expanded discussion of the effort.

First, we shall discuss mortality findings. Dogs have inhaled enough plutonium to cause death within a week at high doses. In this case the lung suffers total respiratory failure. At lower doses death at several months occurs from extensive and rapidly developing fibrosis. At still lower doses death occurs at from 1 to 5 years from progressively infiltrating fibrosis. At 3 to 6 years carcinogenic death occurs accompanied by some fibrosis. Death at lower doses and longer terms is still under study. Figure 3 is a summary of mortality experience. One plot represents inital lung deposition, the second is lung burden at death exhibiting two modes of death, pulmonary failure and tumor. Note that

extrapolation of lung burden at death based on pulmonary failure to a normal lifespan of the dog would indicate a "no life shortening" burden of about 2nCi/g. Extrapolating the other (tumor) component yields a burden of about 0.1 nCi/g.

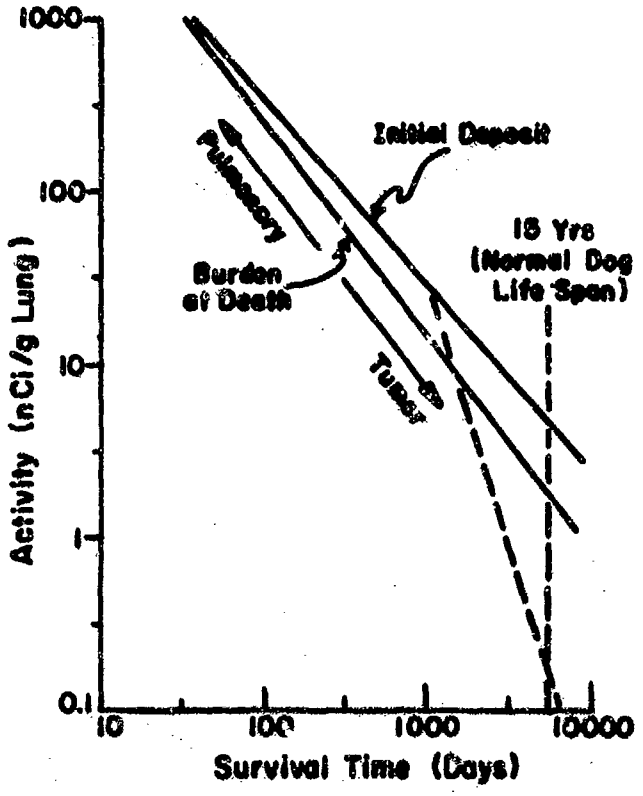

Figure 3. Initial plutonium deposit and burden at death for dogs (Ref. 62)

How does one extrapolate these data to man? Does one suggest that a similar concentration in man would have no life shortening effect? But man's lifetime is on the order of 5 times that of the dog. Should we decrease man's permissible burden by a factor of 5 to account for this difference in life expectancy? Is there perhaps a third component to this mortality burden curve that will not come to light until we see results of low dose experiments?

The burden based on tumor production (0.1 nCi/g) when extrapolated directly to man based on lung mass yields 100 nCi. The present permissible burden recommended or inferred by the ICRP and 10 CFR 20 is 16 nCi. We suppose the answer to all of these questions is, "We don't know yet." And, in truth, we have been unable to uncover striking evidence that present permissible lung burdens do not represent safe levels for man.

What differences does one see with the higher specific activity Pu-238 with respect to Pu-239? For the same activity deposits, Pu-238 particle concentrations are much lower than those of Pu-239. One would expect to see differences if only because a larger volume of the lung, i.e., more cells are at risk, in the case of Pu-239. Of course, this effect may be balanced to some unknown extent due to

the higher energy, longer range and higher specific activity photons emitted from Pu-238. Research at Battelle has found no significant difference in mortality of rats and dogs at early times between Pu-238 and Pu-239. Longer time experiments are not complete.

Dr. Bair's conclusions in his series of lectures in Japan (Ref. 62) can be our summation of the Battelle mortality studies. He stated, "In our studies, death caused by inhaled plutonium..... has always been due to irradiation of the pulmonary tissue and the resulting cardiopulmonary insufficiency and/or bronchiolo-alveolar carcinoma." Dr. Bair has lately found tumors in the lymphatic system draining the lungs. A few of Dr. Bair's dogs contracted cancer of the lymph nodes but all of these dogs died of respiratory failure. The only conclusions he would draw were expressed during conversation as we discussed contamination limits. When discussing our calculations based on the pulmonary lymphatic system, he suggested that it might be well to use the lymphatic system as the critical organ, if only because of the uncertainties involved in the whole field of man's response to plutonium inhalation.

Diagnosis and Treatment

We now turn to research which is aimed at diagnosis and treatment of plutonium incident victims. The questions are:

1. How do we determine if a subject has a plutonium burden? and
2. What can we do about it?

We point our discussion toward the weapons accident case although much of the data results from experience with manufacturing and processing plutonium in various forms.

Exposure can result from an accident situation or can be chronic. The accident situation alerts personnel to the probability of a hazardous deposition. Only good surveillance techniques guard against the chronic depositions. Basic research in this area goes back to early work at LASL done to establish health physics monitoring techniques for their workers in the Manhattan Project. Bio-assay in the form of urine and feces sampling was determined to be a reasonable surveillance technique. Data from animal experimentation yielded the equation (Ref. 64):

$$Y_u = 0.23\,X^{-0.77} \text{(standard error 32\%)} \qquad (3)$$

where Y_u is the daily urine assay expressed as a percent of initial deposition and X is the time of sampling in days past injection. These data are based on injected plutonium.

Adjustment based on urine sampling of laboratory personnel led to the expression:

$$Y_{ua} = 0.20\,X^{-0.74} \text{(standard error 42\%)} \qquad (4)$$

where Y_{ua} is the adjusted urine yield. Equation (4) represents data through 1750 days past deposition.

Fecal excretion data yielded:

$$Y = 0.63\,X^{-1.09} \text{(standard error 28\%)} \qquad (5)$$

where Y is fecal yield.

Analysis of continuing LASL data indicates high variance in estimated body burdens based on urine sampling and recommends true 24 hour samples, or series of such samples separated by several weeks be taken when important decisions must be reached concerning body burdens. (Ref. 65).

There is a considerable volume of excretion data based on injected radionuclides. For the inhalation case, however, we must look to other data, especially where the radionuclide inhaled is considered as "insoluble". In the inhalation case dog fecal sampling yields higher levels of plutonium excretion, generally by factors of over 100. We find that estimates of lung burden based on fecal sampling in the first few days is rather difficult. Most of the uncertainty is due to early clearance from the lung to the G. I. tract by ciliary action.

After 7-14 days fecal data show less variation sample to sample than does urinary data (Ref. 66). However, there are several reasons why fecal sampling is not considered to be a preferred method of assay. Generally, these reasons are related to difficulties in obtaining the sample and in its handling. There is considerable evidence that it should be employed where important decisions might be made as a result of more exact determination of lung burden. When estimates must be made in the first few days urine sampling is probably preferred if the sample is active enough to suggest good counting statistics.

The 1965 lung model of the ICRP (Ref. 58) and Langham's data covering human excretion experimentation (Ref. 64) are the basis for a theoretical approach to estimating initial lung burdens for inhalation cases. Figure 4 represents the findings of this approach in terms of urinary excretion of plutonium for an initial lung burden of 1.0 nCi as a function of time and particle size of the inhaled aerosol (Ref. 67). The authors suggest that urine sampling may be of inadequate sensitivity for smaller particle sizes but that fecal sampling may lead to an improvement in sensitivity in lung burden estimates.

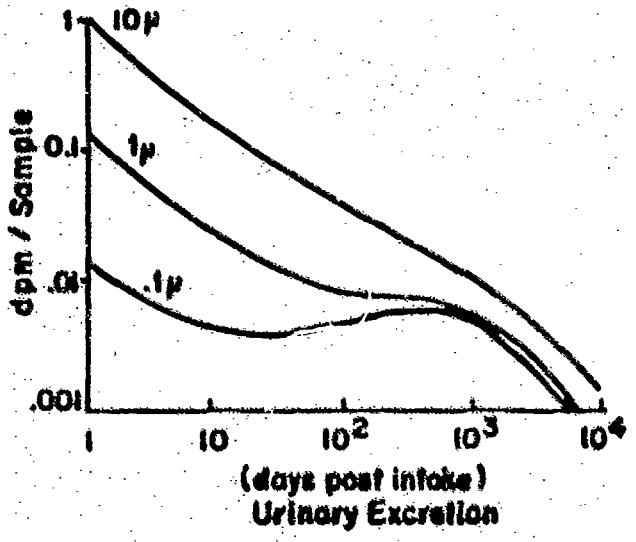

Figure 4. Theoretical Plutonium Urinary Excretion Curves. (Ref. 67)

One last method of lung burden estimation is in vivo counting. Due to the short range of alpha particles in tissue, the method depends on detection of the low energy photons from plutonium and americium-241, a decay product of Pu-241. The specific activity of americium is a function of the age of the batch of plutonium involved in the sample. Knowledge of the isotopic composition of the original plutonium batch is also required (Ref. 62). Counting for lung burden is made more difficult as the contaminant is biologically translocated to the lymphatic system. These counting systems are usually associated with fixed facilities and shielded rooms. Of interest to the accident situation is a portable (about 200 lbs) anti-coincidence system claiming capabilities in unshielded facilities for reasonable confidence at lung burdens of 5.3 nCi, about one-third of a permissible lung burden (Ref. 68). This level compares well with capabilities of fixed installation in three countries (Refs. 69, 70, 71).

Before proceeding to the subject of treatment of plutonium deposition we should discuss the case of contamination of wounds. In the accident situation, particularly during search and decontamination periods, there is a fair probability that some personnel will receive wound injury. If the area is contaminated, the wound will probably be contaminated also. Experience with wound cases has generally been from processing facilities and involve both soluble and insoluble compounds. Insoluble compounds exhibit little uptake in the systemic blood system. Soluble compounds react in the opposite direction. Early urine sampling, or fecal where possible, has been used to estimate the deposition (Ref. 72). A solid state device for wound assay has been developed (Ref. 73). The system offers such excellent resolution of the soft photons involved that ratios of photon peaks can be employed to estimate depth of deposition.

Reduction of the Body Burden

Once an estimate of body deposition has been made, what can we do to reduce that burden? Treatment, of course, depends on the estimate made of the total deposition. Treatment may be generally categorized into three different methods:

1. Chemotherapy
2. Lavage
3. Excision

Chemotherapy

Chemotherapy is represented by treatment with chelating agents, compounds which bond with metal ions. Chelating agents such as EDTA* and DTPA** have been employed with DTPA being the preferred agent (Ref. 74). DTPA may still be restricted to the experimental drug list. We understand that action is underway to relieve this restriction. The chelate is effective in binding with any free plutonium ions. The primary action, however, depends on the fate of plutonium in the systemic blood system. Iron in the blood is complexed with transferrin, a transport protein. The transferrin then distributes the iron to hemoglobin synthesis locations as needed. Excess iron is stored in the liver and spleen. Storage involves transfer from transferrin to ferritin and hemosiderin, two iron storage compounds. Transferrin also picks up plutonium that may have reached the systemic system. The plutonium is transported in the same manner as iron. If the plutonium reaches the bone it tends to transfer to the bone where it enjoys a long biological lifetime. DTPA chelating action removes the plutonium from transferrin with a bond sufficient to restrict bone uptake.

*EDTA is ethylenediaminetetraacetic acid.
**DTPA is diethylenetriaminepentaacetic acid.

The chelated plutonium then is excreted in the urine. Exchange of plutonium from transferrin to ferritin has been demonstrated (Ref. 75). DTPA also has indicated some effectiveness in reducing liver burden. Use of a reticulo-endothelial system stimulant, glucan*, has been successful in releasing plutonium from the liver. Subsequent uptake in bone by a similar amount was evident. Treatment with glucan and DTPA is successful in reducing both liver and bone burdens (Ref. 76). Plutonium once bound to bone cannot be chelated except during bone remodeling when osteoclasts act on resorbing surfaces. In general, DTPA treatment should be initiated as early as possible (within minutes to hours) after plutonium deposition for greatest effectiveness in reducing systemic burdens (Ref.77).

We should mention that there are contradictory views in the literature concerning DTPA. It is said to be nephrotoxic and a depleter of trace metals, most notably zinc. Additionally, some consider that DTPA treatment liberates plutonium to the systemic system with subsequent uptake in various organs. These views are a truly minority opinion (Ref. 78). As to nephrotoxicity, the agent probably should not be administered to subjects with existing renal complications. Complications that occur during DTPA therapy are reversible when treatment is stopped. Normal dietary control is sufficient to replace trace metals. The last fear seems to be overcome because plutonium is tightly bound to DTPA in the systemic blood until excreted. In summary, chelate treatment should follow evaluation by competent medical authority (Ref. 78). The U. S. Navy, alone of the three services, has a stated policy concerning the use of DTPA. That policy requires that the Bureau of Medicine and Surgery be consulted prior to use of the chelate (Ref. 79).

We repeat here that DTPA injections effect systemic plutonium. The chelate has also been used in aerosol form as an inhalant in an attempt to treat lung burden directly. The treatment was in conjunction with lung lavage. Total effect seemed to be the same as lavage and DTPA injection treatment separately. The chelate is absorbed from the lung rather rapidly and enters the systemic blood (Ref. 80).

Lavage

We very naturally enter discussion of lung lavage as a treatment for plutonium lung burden. We know of only one case; it was performed at Lovelace Clinic in 1971 on a plutonium inhalation case. The results are presently privileged medical information. The procedure was considered to be successful in lowering the lung burden (Ref. 80).

If lavage is to be most effective it should be accomplished as early as possible after the deposition, but not before initial ciliary removal of upper lung deposits has been completed. Lavage might interfere with ciliary action and even force the deposits to the pulmonary lung region. Thus, two to three days after exposure would be a good time to perform the lung lavage.

One suggestion of a possible chemical prophylaxic against bone uptake of systemic plutonium was found in the literature (Ref. 81). The antibiotic, tetracycline, deposits on the bone, particularly in regions of active calcification. Its effect in reducing plutonium deposits at the same locations is evidently a blocking action or formation of a plutonium - tetracycline bond sufficiently stable to permit ultimate excretion.

*Glucan is a highly purified neutral polysaccharide fraction of yeast cell wall extract.

Excision

Surgical removal of contaminated tissues and particulate has been the prime treatment in wound cases. Of course, this treatment must be considered in balance with possible effects of the surgery involved and the projected insult of the plutonium deposition. Where used, excision has generally been successful. There is some evidence that early excision, particularly of insoluble compounds, may not be preferred. If one waits, the action of localized particulates on immediately surrounding tissue tends to result in incapsulation of the particulate. This volume can usually be removed with less total insult to surrounding tissue. Of course, monitoring of systemic burden should continue in the meantime in case serious uptake commences.

The treatments discussed all exhibit some negative aspects. These must be considered when contemplating action. In cases where the threat from the plutonium is sufficiently slight in terms of present knowledge, the preferred treatment may be no treatment at all. In this case some form of continued monitoring is indicated, perhaps in the form of periodic urine assay.

SUMMARY

Other than the impairment that may result from surgical excision, there is no reported case of death or significant debility as a result of plutonium insult to humans. There are known cases of significant plutonium burdens in humans. We are not able to draw conclusions regarding permissible burdens from the data in these cases because the subjects do not, as yet, show significant biological changes. The dosages which constitute their burdens have either been too small to cause significant damage, or the time over which the hazardous material has been acting has not been sufficient for damage to be manifested.

Conclusions pertinent to the present study must therefore rely on animal experimentation. We feel that we have gathered some interesting information from our study of the biological experimentation that has been or is being done. Certainly much of it is of general interest to this effort. We cannot conclude, however, that the research we have been able to review supports adoption of maximum permissible body or organ burdens differing from those presently in use in this country. Nor are we able to suggest that the lung is not the critical organ in inhalation incidents. To be sure, theory, animal experimentation and the discussion which follows in Section V of this study indicate that the pulmonary lymphatic system may be subjected to significantly higher lifetime concentrations than is the lung. There is insufficient evidence presently available, however, to conclude that the biological damage resulting from these higher concentrations has greater significant biological effect than associated smaller concentrations in the lung. Concern, however, is being voiced in the community concerning the implications of these relatively high concentrations in the lymphatic system. We will proceed, therefore, to consider lung and lymph in Section V.

SECTION V
THE DEPOSITION AND RETENTION OF INHALED PLUTONIUM IN THE HUMAN BODY

LUNG

The deposition and retention model for inhalation of a plutonium aerosol or plutonium-bearing resuspended dust is based on the physiological lung model proposed by the Task Group on Lung Dynamics for Committee II of the International Commission on Radiological Protection (Ref. 53). A schematic of this lung model is shown in Figure 5. In this model, D_1 through D_5 are the percent depositions of the amounts of dust in various respiratory volumes or areas, with the sum of D_2 through D_5 being equal to D_1, as

$$D_1 = 1.0 = D_2 + D_3 + D_4 + D_5 \qquad (6)$$

D_2 is the dust in the exhaled air, D_3 is the dust deposited in the nasopharynx region, D_4 is the dust deposited in the tracheobronchial region, and D_5 is the pulmonary dust deposition. Three other closely allied compartments are shown, the gastrointestinal tract, systemic blood, and pulmonary lymph.

The letters (a) through (j) indicate the different absorption and translocation processes which are associated with the clearance of various compartments. The values of the constants associated with these different pathways for the 1965 model are given in Table XI, while the values for the proposed 1975 model are given in Table XXI, Appendix A. The constants in Table XI indicate that the plutonium dust deposited in the nasopharynx and tracheobronchial compartments is cleared rapidly, whereas that deposited in the pulmonary region is cleared slowly. Because of this rapid clearance, the pathways (a) through (d) to the nasopharynx and tracheobronchial compartments will not be considered. The different absorption and translocation processes associated with the pulmonary compartment are discussed below:

(a) represents the direct translocation of dust from the pulmonary region to the blood.

(f) represents the relatively rapid clearance phase from the pulmonary region. Presumably, this route depends upon recruitable macrophages, which in turn is coupled to the ciliary-mucus transport process, and thus, the dust cleared goes to the G. I. tract via the tracheobronchial tree.

(g) represents a second pulmonary clearance process that is much slower than (f), but it still depends upon leukocytosis and ciliary-mucus transport, with the cleared dust again going via the tracheobronchial tree to the G. I. tract. The important distinction between (g) and (f) is that clearance via (g) is rate limited in the pulmonary region by the nature of the deposited dust, "per se".

(h) represents the slow removal of dust from the pulmonary compartment to the pulmonary lymphatic system. Qualitatively, this process is similar to (g), with the lymph transport in (h) performing the similar function as the ciliary-mucus transport in (g).

(i) represents the clearance process from the lymphatic system to the blood. Obviously, this pathway depends upon the ability of the dust to penetrate the lymph tissue, especially the lymph nodes. Penetrating the lymph tissue implies a partial or complete dissolution of the dust particles, but the turnover of the lymphocytes may contribute (Ref. 62).

(j) represents the collective absorption of cleared dust from the gastrointestinal tract by direct and indirect pathways. Quantitatively and temporally, (j) is coupled to processes (b, (c), (i), and (g).

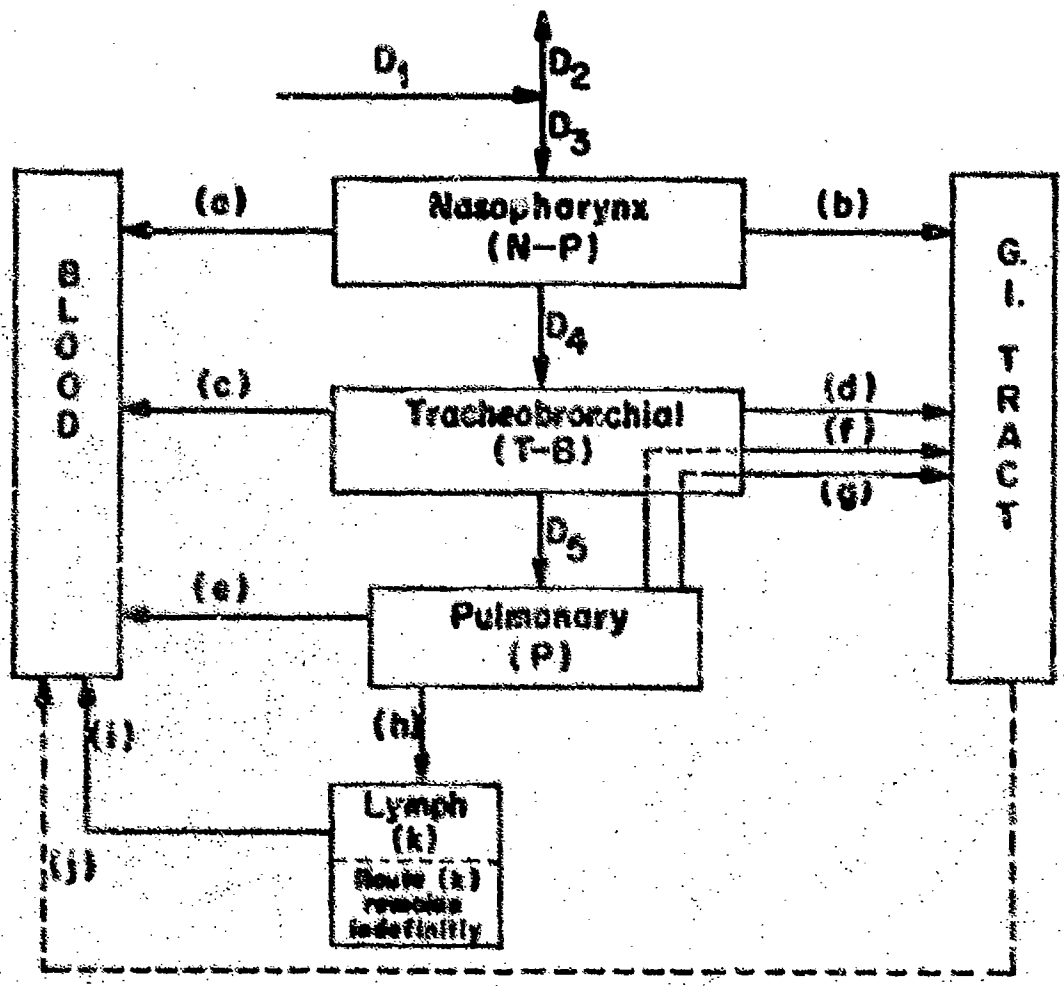

Figure 5. Schematic of physiological lung model for dust deposition sites and clearance processes.

TABLE XI

CONSTANTS FOR USE WITH THE 1965 LUNG CLEARANCE MODEL FOR $^{239}PuO_2$.

Region	Process	Biological Half-Time	Regional Fraction
N-P	a	4 minutes	0.01
	b	4 minutes	0.99
T-P	c	10 minutes	0.01
	d	10 minutes	0.99
P	e	500 days	0.05
	f	24 hours	0.40
	g	500 days	0.40
	h	500 days	0.15
Lymph	i	500 days	0.10
	k	∞ (infinity) (Subject only to radioactive decay)	0.90

No further attempt will be made to consider (j); however, a physiological model for gastrointestinal absorption has been made by Eve (Ref. 83):

(k) represents the portion of the dust that is never cleared from the lymphatic system. The biological half-time for this routine is infinity, so this process is not a clearance way; it is included for the sake of clarity.

The rate of change of the pulmonary lung burden for each of the four clearance processes can be expressed in terms of differential equations as follows:

Burden rate of change (in μCi/) = input - output (7)

$$\frac{dq_e}{dt} = RCD_5\, f_e - \lambda_e\, q_e \tag{8}$$

$$\frac{dq_f}{dt} = RCD_5\, f_f - \lambda_f\, q_f \tag{9}$$

$$\frac{dq_g}{dt} = RCD_5\, f_g - \lambda_g\, q_g \tag{10}$$

$$\frac{dq_h}{dt} = RCD_5\, f_h - \lambda_h\, q_h \tag{11}$$

where R = respiration rate, in m^3/day

C = air concentration at any time, in $\mu Ci/m^3$

q_e, q_f, q_g, q_h = the lung burdens associated with the respective clearance pathways, in μCi

λ_e, λ_f, λ_g, λ_h, = the elimination rates, in $days^{-1}$, for the data in Table XI, and are (ln 2)/(biological half-time)

f_e, f_f, f_g, f_h = the regional deposition fractions for the respective pathways and are given in Table XI.

The respiratory input is RC, where the air concentration, C, is a decaying exponential that is given by

$$C = C_o e^{-(\lambda_A + \lambda_{is})t} \tag{12}$$

where

λ_A = the air clearance rate constant = (ln 2)/(35 days) = 1.98×10^{-2} $days^{-1}$ (Ref. 30)

λ_{is} = the disintegration constant of the isotope being studied

C_o = the air concentration at t=0, i.e., the start of the inhalation exposure.

The half-life of ^{239}Pu is 24,360 years, so $\lambda_{is} = 7.90 \times 10^{-8}$ $days^{-1}$. For ^{239}Pu, $\lambda_A \gg \lambda_{is}$, so Eq. (12) reduces to $C = C_o e^{-\lambda_A t}$.

Eq. (11) will be integrated as a sample and the initial condition will be applied. The other equations can be solved in an analogous method. Eq. (11) can be rewritten, using Eq. (12) above, as

$$\frac{dq_h}{dt} + \lambda_h q_h = RC_o D_5 f_h e^{-\lambda_A t} \tag{13}$$

Eq. (13) can be multiplied throughout by the integrating factor of $e^{\lambda_h t}$ to give

$$e^{\lambda_h t}\left(\frac{dq_h}{dt} + \lambda_h q_h\right) = RC_o D_5 f_h e^{(\lambda_h - \lambda_A)t} \tag{14}$$

The left hand side of Eq. (14) is a perfect differential, as

$$\frac{d}{dt}\left(q_h e^{\lambda_h t}\right) = e^{\lambda_h t}\left(\frac{dq_h}{dt} + \lambda_h q_h\right) \tag{15}$$

Eq. (15) can be readily used to integrate Eq. (14) with the result of

$$\int \frac{d}{dt}\left(q_h e^{\lambda_h t}\right) dt = \int RC_o D_5 f_h e^{(\lambda_h - \lambda_A)t} dt \tag{16}$$

$$q_h e^{\lambda_h t} = \frac{RC_o D_5 f_h}{\lambda_h - \lambda_A} e^{(\lambda_h - \lambda_A)t} + \alpha \qquad (17)$$

where α is the constant of integration that will be determined from the initial condition of $q_h = 0$ when $t = 0$, giving

$$\alpha = -\frac{RC_o D_5 f_h}{\lambda_h - \lambda_A} \qquad (18)$$

Eq. (17) can then be multiplied by $e^{-\lambda_h t}$ to get q_h as a function of time as

$$q_h(t) = \frac{RC_o D_5 f_h}{\lambda_h - \lambda_A}\left(e^{-\lambda_A t} - e^{-\lambda_h t}\right) \qquad (19)$$

In a similar fashion, the other burdens can be obtained and are:

$$q_e(t) = \frac{RC_o D_5 f_e}{\lambda_e - \lambda_A}\left(e^{-\lambda_A t} - e^{-\lambda_e t}\right) \qquad (20)$$

$$q_f(t) = \frac{RC_o D_5 f_f}{\lambda_f - \lambda_A}\left(e^{-\lambda_A t} - e^{-\lambda_f t}\right) \qquad (21)$$

$$q_g(t) = \frac{RC_o D_5 f_g}{\lambda_g - \lambda_A}\left(e^{-\lambda_A t} - e^{-\lambda_g t}\right) \qquad (22)$$

The pulmonary lung burden, $q_p(t)$, is the sum of Eqs. (19), (20), (21) and (22) as

$$q_p(t) = q_e(t) + q_f(t) + q_g(t) + q_h(t) \qquad (23)$$

$$q_p(t) = RC_o D_5\left[\frac{f_e\left(e^{-\lambda_A t} - e^{-\lambda_e t}\right)}{\lambda_e - \lambda_A} + \frac{f_f\left(e^{-\lambda_A t} - e^{-\lambda_f t}\right)}{\lambda_f - \lambda_A} + \frac{f_g\left(e^{-\lambda_A t} - e^{-\lambda_g t}\right)}{\lambda_g - \lambda_A} + \frac{f_h\left(e^{-\lambda_A t} - e^{-\lambda_h t}\right)}{\lambda_h - \lambda_A}\right] \qquad (24)$$

Eq. (24) can be simplified by noting that the clearance rate constants for pathways (e), (g), and (h) are the same. Thus,

$$\lambda_e = \lambda_g = \lambda_h \equiv \lambda_1 = (\ln 2)/(500 \text{ days}) \qquad (25)$$

With the use of Eq. (25), the lung burden equation becomes

$$q_p(t) = RC_o D_5 \left[\frac{\left(f_e + f_g + f_h\right)\left(e^{-\lambda_A t} - e^{-\lambda_1 t}\right)}{\lambda_1 - \lambda_A} + \frac{f_f\left(e^{-\lambda_A t} - e^{-\lambda_f t}\right)}{\lambda_f - \lambda_A} \right] \tag{26}$$

The initial air concentration, C_o, is related to the inital surface concentration, S_o, through the resuspension factor, k, as

$$C_o = k S_o \tag{27}$$

where S_o is in $\mu Ci/m^2$ and k is in m^{-1}. The combination of Eqs. (26) and (27) yields

$$q_p(t) = R k S_o D_5 \left[\frac{\left(f_e + f_g + f_h\right)\left(e^{-\lambda_A t} - e^{-\lambda_1 t}\right)}{\lambda_1 - \lambda_A} + \frac{f_f\left(e^{-\lambda_A t} - e^{-\lambda_f t}\right)}{\lambda_f - \lambda_A} \right] \tag{28}$$

A graphical representation of Eq. (28) is shown in Figure 6, where on the left $q_p(t)/R\,k\,S_o\,D_5$ in days, and on the right $q_p(t)$, in $\mu Ci (S_o = 50\ \mu g/m^2)$, is plotted against the time, t, in days.* (Note that the time-varying part, i.e., the portion within the brackets, is plotted on the left versus the time so that the pulmonary burden q_p can be obtained readily by multiplying the curve value by the constants $R\,k\,S_o\,D_5$.) An important feature of this curve is the relatively long time scale. One-half of the maximum lung burden is attained in 26 days, the maximum lung burden is reached in 143 days, and the maximum burden has decayed to one-half its value in 700 days. The expontential tail follows closely a curve with a 500-day half-time. It is very important to note that the time available for clean-up operations is determined by the air clearance time (a value of 35 days was used in Fig. 6) and in order to significantly reduce the total lung burden, the clean-up must be done in the first few weeks or the higher concentrated area must be evacuated for a few months.

Evacuation of the contaminated area can greatly reduce the burden and dose to the body. For example, with the air clearance rate constant of $\lambda_A = \ln 2/35$ days, if the contaminated area is evacuated for the first 35 days from the time t = 0, then the burden and dose will be 1/2 of the amount graphically portrayed in this study. If the evacuation is for 70 days, the burden and dose will be 1/4 of the amount in this study. This halving of the burden and dose will continue for each additional half-time of 35 days that the individual is removed from the contaminated area. This burden and dose decrease is shown in Figure 7 where the relative burden and dose (as a fraction of the amount when there is no evacuation) is plotted versus the elapsed time in units of one half-time, T.

*The arbitary value of $S_o = 50\ \mu g/m^2$ has been used in all graphical and tabular presentations.

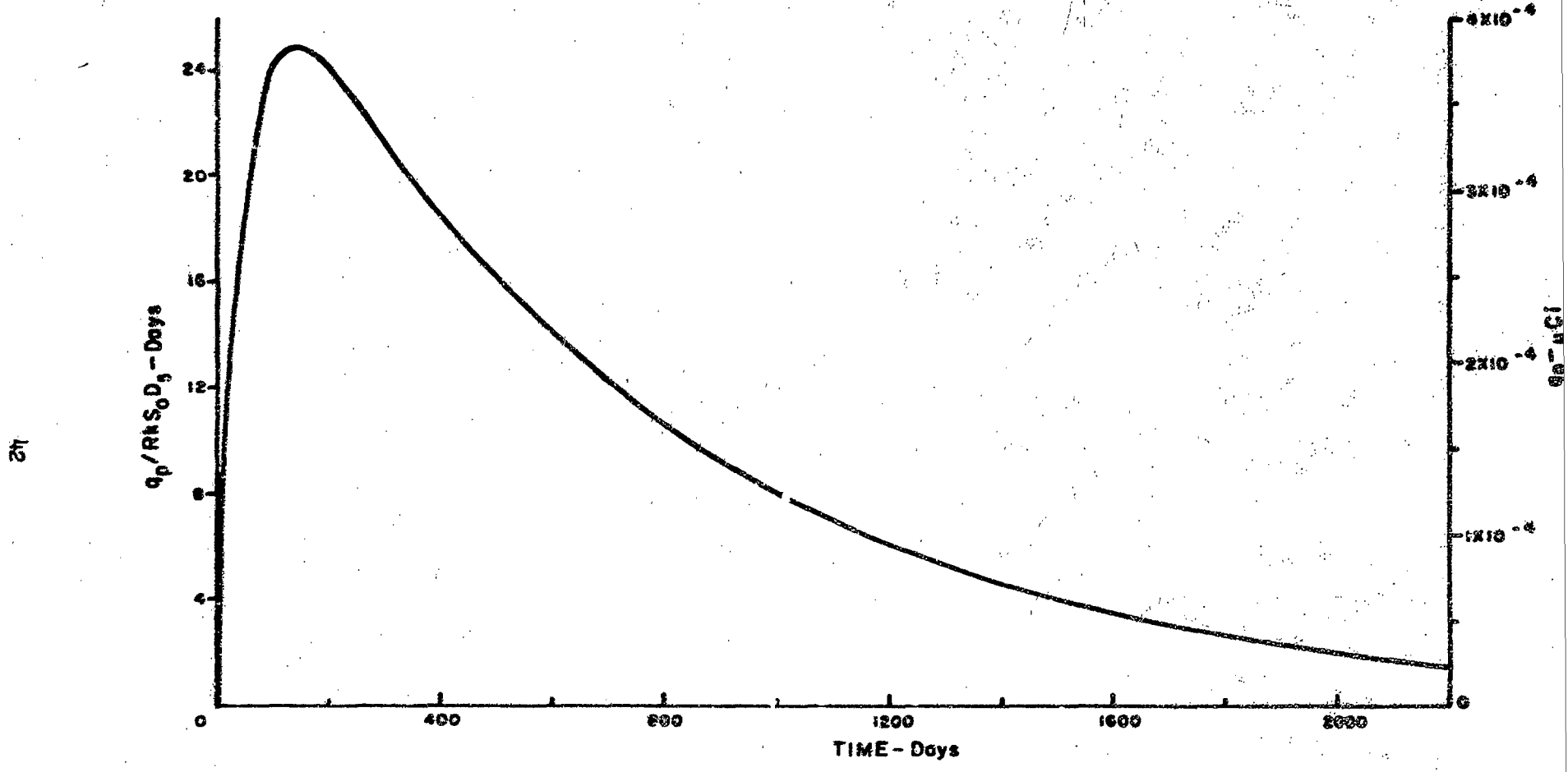

Figure 6. Lung burden following inhalation of $^{239}Pu\ O_2$ dust versus time of exposure in a contaminated area of $S_o = 50\ \mu g/m^2$. (Same graph for both the 1965 and proposed 1973 lung models)

The arbitrary value of $S_o = 50\ \mu g/m^2$ has been used in all graphical and tabular presentations.

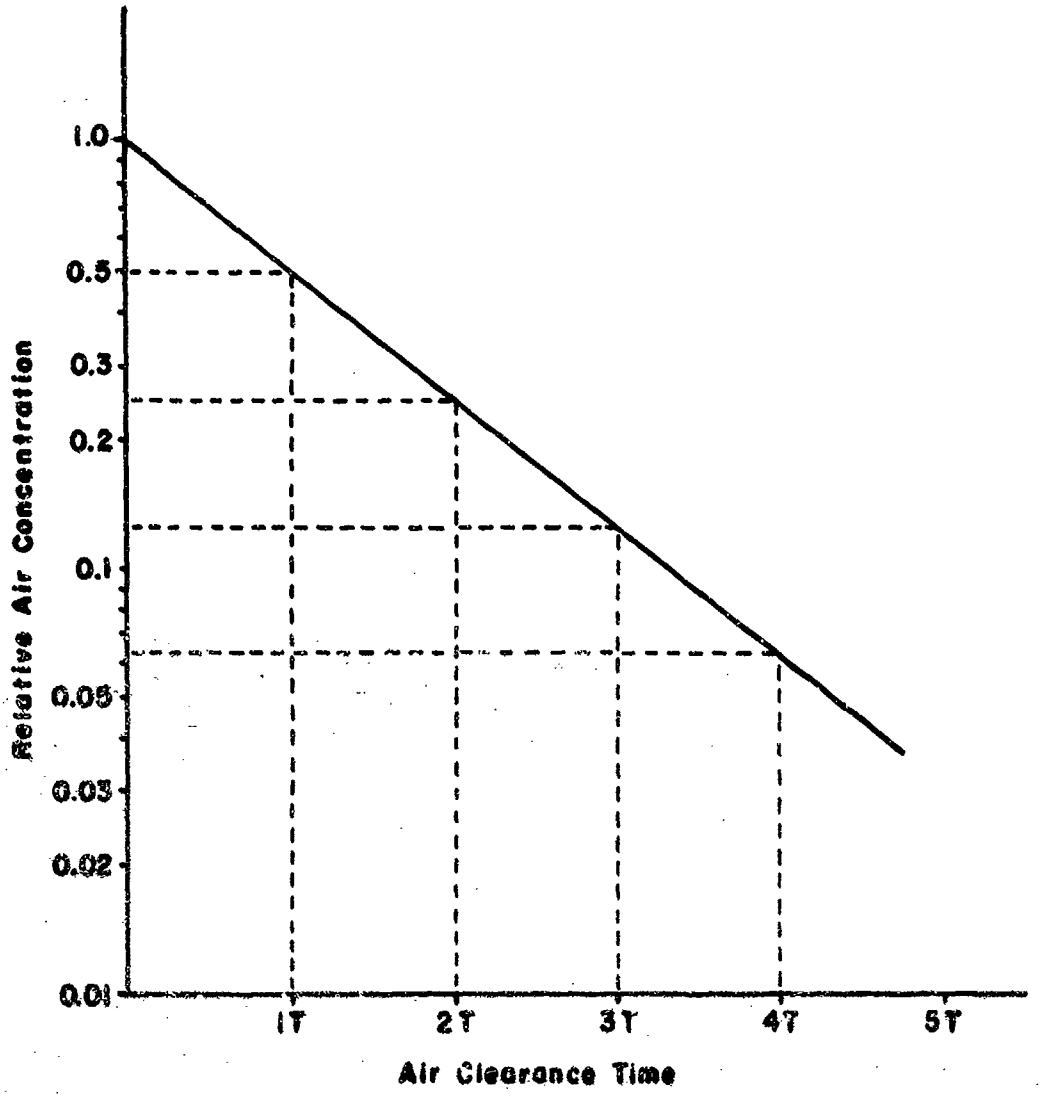

Figure 7. Relative Air Concentration as a Function of Air Clearance Time

The pulmonary lung dose, $D_{c,p}$, in μCi-days, is obtained by integrating Eq. (28) over time, and is

$$D_{c,p} = \int_0^t q_p(t)\,dt = RkS_0D_5 \left[\frac{(f_e + f_g + f_h)}{\lambda_1 - \lambda_A} \left\{ \frac{\left(e^{-\lambda_1 t} - 1\right)}{\lambda_1} - \frac{\left(e^{-\lambda_A t} - 1\right)}{\lambda_A} \right\} + \frac{f_f}{\lambda_f - \lambda_A} \left\{ \frac{\left(e^{-\lambda_f t} - 1\right)}{\lambda_f} - \frac{\left(e^{-\lambda_A t} - 1\right)}{\lambda_A} \right\} \right] \quad (29)$$

A lifetime in a contaminated area will be between 50 and 70 years. No significant error (less than a fraction of 1%) is introduced if this time is extended to infinity, i.e., let $t \rightarrow \infty$. If this time condition is imposed on Eq. (29), the infinite lifetime dose, $D_{c,p\infty}$, is found to be

$$D_{c,p\infty} = \frac{RkS_o D_5}{\lambda_A} \left[\frac{(f_e + f_f + f_g)}{\lambda_1} + \frac{f_f}{\lambda_f} \right] \tag{30}$$

A μCi-day of exposure can be converted readily into an exposure dose in rads, D_r, by using

$$D_r = D_c \frac{C_1 C_2 C_3 E}{C_4 m} = 51.1 \frac{D_c E}{m} \tag{31}$$

where C_1 = constant = 3.7×10^4 disintegrations/sec/μCi

C_2 = constant = 8.64×10^4 sec/day

C_3 = constant = 1.6×10^{-6} erg/MeV

C_4 = constant = 100 erg/g/rad

E = energy of particles, in MeV per disintegration

m = weight of organ, in g

For ^{239}Pu, the average alpha particle energy is 5.15 MeV, so Eq. (31) becomes

$$D_r = 2.63 \times 10^2 \frac{D_c}{m} \tag{32}$$

For the lung with a mass of 1000 g (this is the value of Standard Man from Ref. 2) the relation between rads and μCi - days is

$$D_{r,p} = 0.263 D_{c,p} \tag{33}$$

Eq. (30) expressed as a dose in rads for a 1,000 g lung is

$$D_{r,p\infty} = \frac{0.263 RkS_o D_5}{\lambda_A} \left[\frac{(f_e + f_g + f_h)}{\lambda_1} + \frac{f_f}{\lambda_f} \right] \tag{34}$$

The respiration rate, R, and the pulmonary fraction, D_5, are directly related parameters; in this study these values are taken from Ref. 2 and are $R = 20\ m^3$/day and $D_5 = 0.25$. D_5 is dependent upon the particle size of the aerosol dust; the AMAD value used in this study is 2 μm, based on the experimental data of OPERATION PLUMBBOB (Ref. 30).

The resuspension factor, k, varies more than any other parameter. As stated earlier, the most widely quoted values for k are 10^{-4} to $10^{-6}\ m^{-1}$. A very good discussion of this factor and a number of values for it are given in Ref. 39. Such a wide variance in values for the resuspension factor has made the necessity of choosing a number proposed by the world's experts in this field, the late

Dr. Wright H. Langham from the Los Alamos Scientific Laboratory, Los Alamos, New Mexico, and Dr. Ken Stewart from the Atomic Weapons Research Establishment, Aldermaston, Berkshire, England. Both experts state that the value of 10^{-6} m^{-1} is appropriate for accident situations, Langham in Ref. 35 and personal communications, and Stewart in Ref. 39 and a personal communication. Thus, we will use $k = 10^{-6}$ m^{-1}, as in Eq. (2).

With these constants used in Eq. (34), the result is

$$D_{r,p\infty} = 0.0288\, S_o \quad \left(S_o \text{ in } \mu Ci/m^2\right) \tag{35}$$

when S_o is in $\mu Ci/m^2$. With the use of Tinney's value as expressed in Eq. (1) (Ref. 84), where it was shown that 16.3 g of ^{239}Pu = 1 Ci, S_o can be expressed in $\mu g/m^2$ so that Eq. (35) becomes

$$D_{r,p\infty} = 0.00176\, S_o \quad \left(S_o \text{ in } \mu g/m^2\right) \tag{36}$$

Eq. (36) can be used to calculate the infinite lifetime dose to the lung when the surface contamination is known; values for these calculations are given in Table XII.

Eq. (33) can be used in Eq. (29) to obtain the time-varying dose to the lung. A plot of $D_{r,p}$, in rads on the left side and in rem on the right side, versus the time, t, in days is shown in Figure 8, where the value of $S_o = 50$ $\mu g/m^2$ was used. This graph shows that the lung dose increases rapidly, reaching one-half the maximum value in 551 days. The maximum dose of 0.089 rads is obtained in about 6,300 days and remains constant thereafter for the life of the individual.

LYMPHATIC SYSTEM

Another possibly critical organ is the pulmonary lymphatic system, for which the differential equations are, based on Figure 5:

$$\frac{dq_l}{dt} = If_l - \lambda_l q_l \tag{37}$$

$$\frac{dq_k}{dt} = If_k - \lambda_k q_k \tag{38}$$

where I = input from lung = $\lambda_h q_h$, with q_h from Eq. (19) and $\lambda_h = \lambda_1$ from Eq. (25)

λ_l = elimination rate for route (l) and is $\lambda_l = (\ln 2)/(500 \text{ days})$, so $\lambda_l = \lambda_1$ from Eq. (25)

f_l = regional fraction for route (l) = 0.10

λ_k = elimination rate for route (k) and since the biological half-time is infinity, $\lambda_k = 0$

f_k = regional fraction for route (k) = 0.90

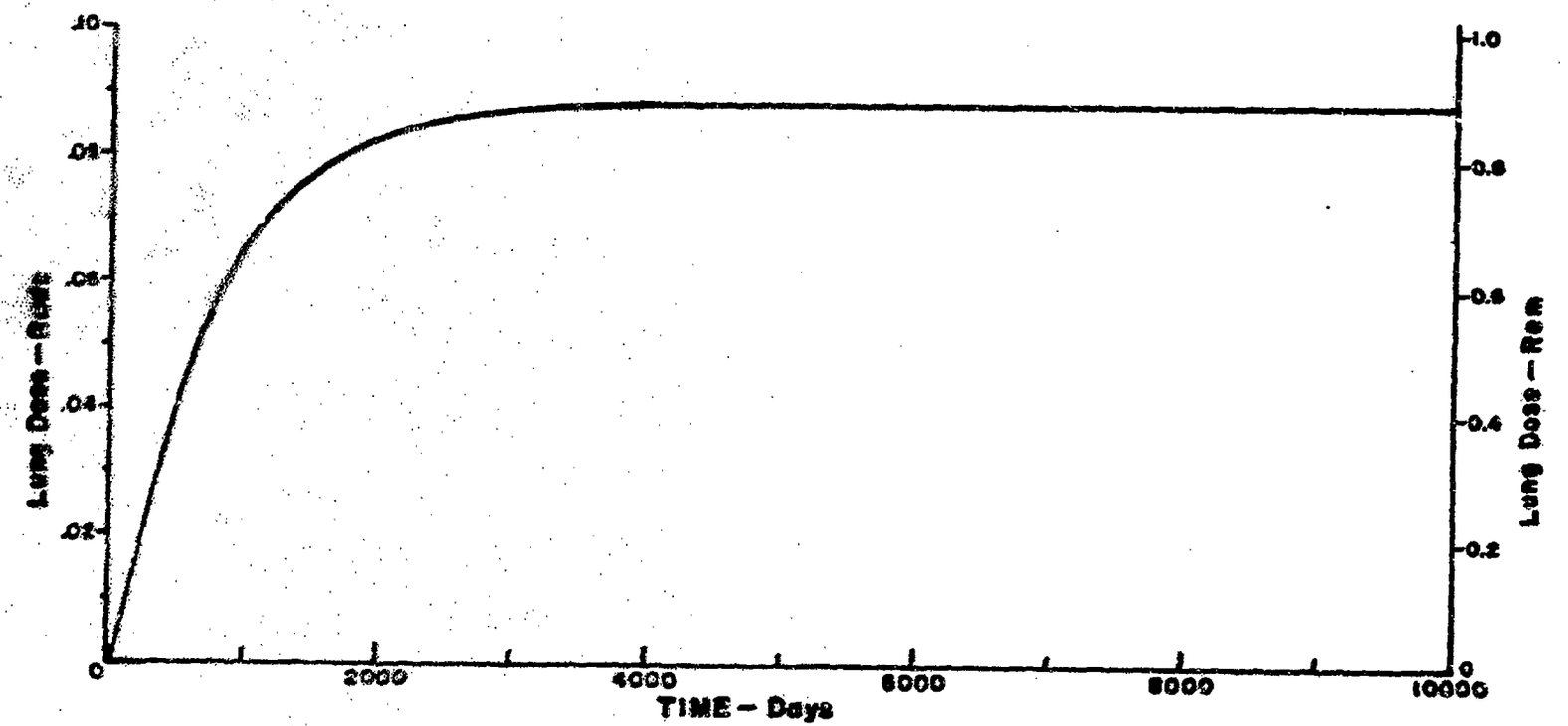

Figure 8. Lung dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50 \mu g/m^2$ (Same graph for both the 1965 and proposed 1973 Lung Models)

Eq. (37) can be readily integrated like Eqs. (8) through (11), and with the same initial condition of $q_1 = 0$ when $t = 0$, the result is

$$q_1(t) = \frac{\lambda_1 RC_o D_5 f_h f_l}{\lambda_1 - \lambda_A}\left[\frac{e^{-\lambda_A t} - e^{-\lambda_1 t}}{\lambda_1 - \lambda_A} - te^{-\lambda_1 t}\right] \tag{39}$$

Eq. (38) reduces to

$$\frac{dq_k}{dt} = If_k \tag{40}$$

since $\lambda_k = 0$. The integrated form of Eq. (40) is, with the same initial condition,

$$q_k(t) = \frac{RC_o D_5 f_h f_k}{\lambda_A}\left[\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1\right] \tag{41}$$

The total dose to these lymph nodes is q_n, which is the sum of Eqs. (39) and (41).

$$q_n(t) = q_1(t) + q_k(t) \tag{42}$$

$$q_n(t) = RC_o D_5 f_h\left[\frac{\lambda_1 f_l}{\lambda_1 - \lambda_A}\left\{\frac{e^{-\lambda_A t} - e^{-\lambda_1 t}}{\lambda_1 - \lambda_A} - te^{-\lambda_1 t}\right\} + \frac{f_k}{\lambda_A}\left\{\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1\right\}\right] \tag{43}$$

A plot of Eq. (43) is shown in Figure 9, where on the left $q_n(t)/RkS_o D_5 f_h$, in days, and on the right $q_n(t)$, in μCi ($S_o = 50\ \mu g/m^3$), is plotted against the time, t, in days. This curve has an even longer time scale than the lung burden curve. One-half of the maximum value is reached in 500 days, and the maximum burden is attained in about 4,100 days. Note that the burden in the pulmonary lymphatic system increases with time to a maximum value where it remains for the lifetime of the individual.

As time goes to infinity (about 4,100 days in Fig. 9), the lymphatic burden approaches the asymptotic value of

$$q_n(t \to \infty) = \frac{RC_o D_5 f_h f_k}{\lambda_A} = 34.1\, C_o\ \mu Ci \tag{44}$$

Eq. (44) gives the dose to the organ, but this dose must not exceed the value set forth by NCRP 39 (Ref. 85) and Ref. 12, which state that the exposure rate for the general population should not exceed 0.5 rem per year. This dose rate can be related to the energy absorbed by the organ by

$$Q = \frac{C_5 C_3 q \left[\sum \text{EF(RBE) N}\right]}{C_4 m} \tag{45}$$

where Q = allowable organ dose rate in rem/hr. This number is based on 0.5 rem/yr converted to rem/hr using 50 weeks per year and 168 hours per week.

C_5 = constant = 1.33×10^8 disintegrations/hr/μCi

C_3 and C_4 are constants defined by Eq. (31)

q = allowable dose to organ, in μCi

$\left[\sum \text{EF(RBE) N}\right]$ = effective absorbed energy per disintegration of ^{239}Pu = 53 MeV · RBE/dis for all organs except the bone, and = 265 MeV · RBE/dis for the bone (Ref. 86).

m = mass of organ, in g

Eq. (45) solved for q, with a limit of 0.5 rem/yr, is

$$\left.\begin{aligned} q &= 5.27 \times 10^{-7}\ m\ \mu\text{Ci for all organs except bone} \\ q &= 1.05 \times 10^{-7}\ m\ \mu\text{Ci for bone} \end{aligned}\right\} \tag{46}$$

The mass of the pulmonary lymph system is 20 g (Refs. 87 and 88), so the amount of plutonium in the pulmonary lymph nodes that will irradiate them with the maximum allowable radiation dose is

$$q = 1.05 \times 10^{-5}\ \mu\text{Ci} \tag{47}$$

Eq. (47) can be equated to Eq. (44), and with the use of Eq. (37), the surface contamination can be calculated to be

$$S_o = \frac{q\lambda_A}{Rk D_S \zeta_h \zeta_k}\ \mu\text{Ci/m}^2 \tag{48}$$

the result being

$$S_o = 0.31\ \mu\text{Ci/m}^2 \tag{49}$$

In terms of μg/m^2, Eq. (49), with the use of Eq. (1) becomes

$$S_o = 5.0\ \mu\text{g/m}^2 \tag{50}$$

The values of S_o given in Eqs. (49) and (50) are ultra-conservative numbers because the mass of the pulmonary lymphatic system has been used. Snyder points out that in considering dose to the lymphatic system, the dose should be averaged over the larger mass of the circulating lymphocytes of 1500 to 2000 g, since these cells will be irradiated as they pass through the hilar (pulmonary) lymph nodes and thus the energy is absorbed in a larger mass than that of the lymph nodes themselves (Ref. 89). With the lymphatic mass increased by a factor of 100, the value of q computed in Eq. (47)

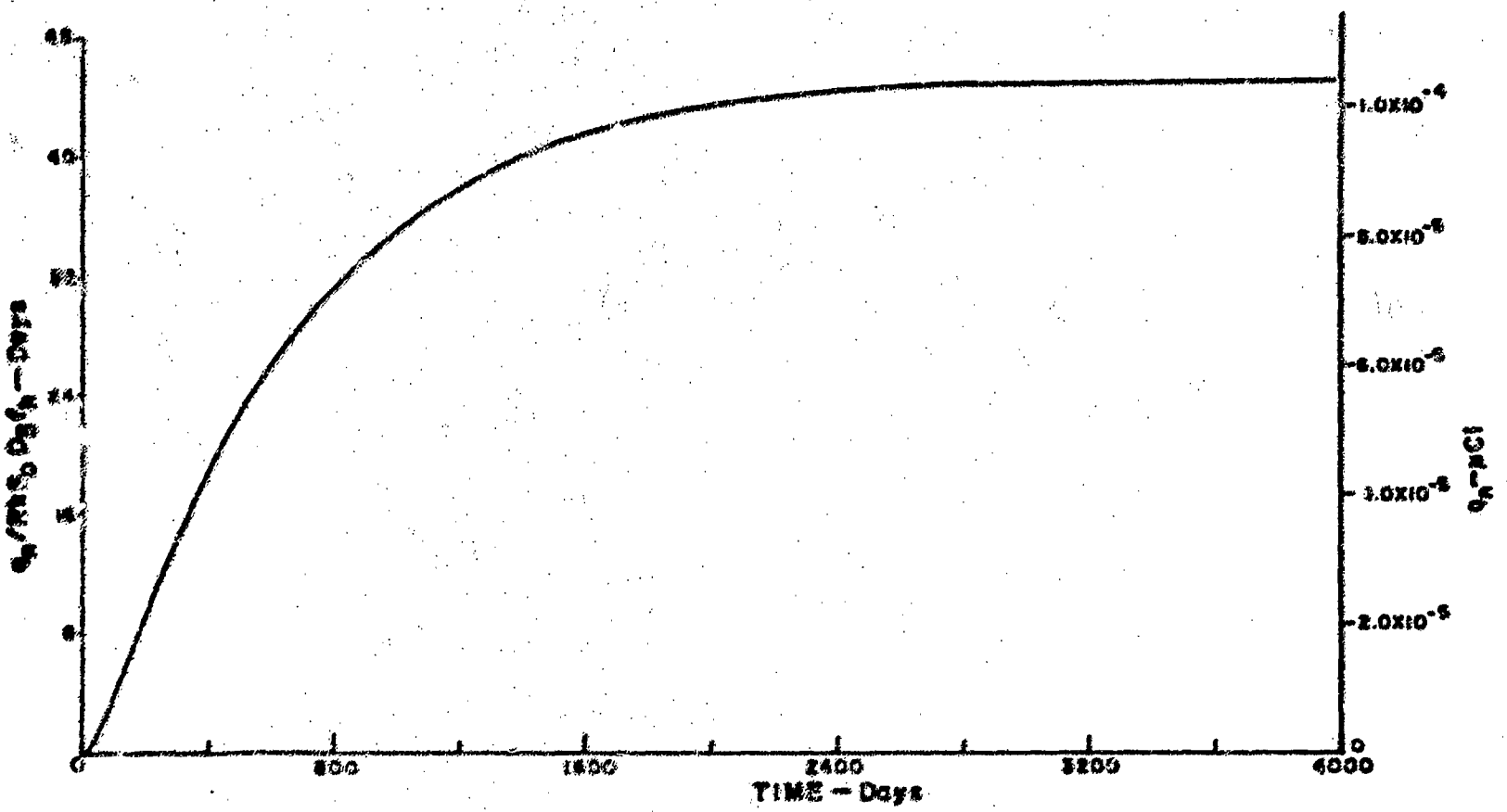

Figure 9. Lymphatic system burden following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50\mu g/m^2$. (1965 Lung Model).

is increased to 1.05×10^{-3} μCi, with the result that the computed value of S_o is now

$$S_o = 31\ \mu Ci/m^2 \text{ or } S_o = 500\ \mu g/m^2 \tag{51}$$

The time-varying dose to the lymphatic system, $D_{c,n}$, can be obtained by integrating Eq. (45) as

$$D_{c,n} = \int_o^t q_n(t)\,dt = RkS_o D_5 f_h \left[\frac{\lambda_1 f_1}{\lambda_1 - \lambda_A} \left\{ \frac{(e^{-\lambda_1 t} - 1)}{\lambda_1(\lambda_1 - \lambda_A)} - \frac{(e^{-\lambda_A t} - 1)}{\lambda_A(\lambda_1 - \lambda_A)} + \frac{te^{-\lambda_1 t}}{\lambda_1} + \frac{(e^{-\lambda_1 t} - 1)}{\lambda_1^2} \right\} + \frac{f_k}{\lambda_A} \left\{ \frac{\lambda_1 (e^{-\lambda_A t} - 1)}{\lambda_A(\lambda_1 - \lambda_A)} - \frac{\lambda_A (e^{-\lambda_1 t} - 1)}{\lambda_1(\lambda_1 - \lambda_A)} + t \right\} \right] \tag{52}$$

Eq. (32) for the lymphatic system, with a mass of 2,000 g, is

$$D_{r,n} = 0.132\, D_{c,n} \tag{53}$$

A plot of $D_{r,n}$, in rads on the left and in rems on the right, versus the time, t, in days, is shown in Figure 10, where the value of $S_o = 50\ \mu g/m^2$ was used. This graph shows that the lymphatic system dose rises very slowly and increases linearly with time beyond 4,100 days. One-half of the 70-year dose is obtained at 15,120 days (35.9 years). The 70-year dose is 0.34 rad.

Eq. (46) can be used to calculate the maximum dose that may be continuously delivered to any organ. If this is dose for the lung with a mass of 1,000 g, the result is $q = 5.27 \times 10^{-4}$ μCi. From the graph in Figure 6 the maximum dose to the lung is seen to be $q_p = 124 \times 10^{-6}\ S_o$ μCi. These latter expressions for q and q_p can be equated to solve for S_o, the result being

$$S_o = \frac{5.27 \times 10^{-4}}{1.24 \times 10^{-4}} = 4.25\ \mu Ci/m^2 = 68.5\ \mu g/m^2 \tag{54}$$

Note that the burden in the lung is around this maximum burden for only a few days, and since each organ can be irradiated above the maximum for a short period of time, the number computed in Eq. (54) is definitely conservative.

The calculation in Eq. (54) can be further substantiated by computing the "average" lung burden. Figure 6 shows that the lung reaches its maximum burden in 143 days and then exponentially tails off to zero. The total infinite lifetime dose calculated with Eq. (30) is $0.110\ S_o$ μCi-days. The graph in Figure 8 shows that one-half the infinite lifetime dose, or $0.055\ S_o$ μCi days, is reached at t = 551 days. The "average" dose then is $1.0 \times 10^{-4}\ S_o$ μCi, so the value of S_o is

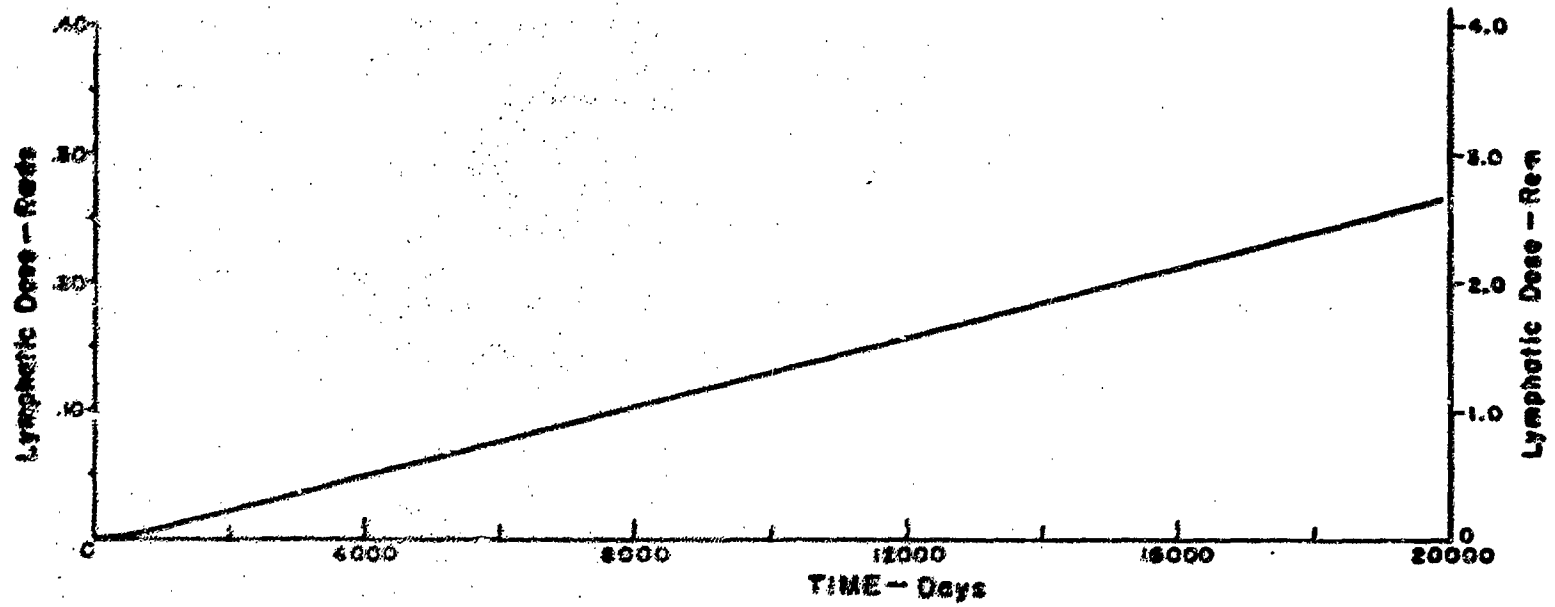

Figure 10. Lymphatic system dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of S_o = 50 $\mu g/m^2$ (1965 Lung Model).

$$S_o = \frac{5.27 \times 10^{-4}}{1.0 \times 10^{-4}} = 5.27\ \mu Ci/m^2 = 85.9\ \mu g/m^2 \qquad (55)$$

These calculations show that the lymph system is not as radiation critical as is the lung. This conclusion is supported by Morgan who states that the new Task Group on Lung Dynamics (this group will present the new physiological lung model in 1973 - see the Appendix for details of this model - which is similar to the one used in Fig. 5) has decided not to use the pulmonary lymph nodes as the critical body organ because animal studies and limited human experience do not suggest this is the more critical tissue in terms of carcinogenesis (Ref. 90). In the very extensive plutonium inhalation studies of Bair with Beagle dogs, no dog has ever died because of cancerous lymph nodes. A few dogs did develop cancer in the lymphatic system but all of these dogs died of respiratory failure, indicating that the lung is the most critical organ and not the lymph nodes (Ref. 62)*.

BLOOD (LIVER AND BONE)

Other critical organs such as the liver and bone will now be considered. The amount reaching these organs is dependent upon the total amount inhaled, q_I, which over an infinite lifetime is

$$q_I = RC = RC_o \int_0^\infty e^{-\lambda_A t}\, dt = \frac{RC_o}{\lambda_A} = 1010\ \mu Ci \qquad (56)$$

Of this total amount, the fraction D_3 goes into the nasopharynx region, D_4 into the tracheobronchial region, and D_5 into the pulmonary region. For the 2 μm particle under consideration, the graph in Figure 13 of Ref. 58 gives a value of $D_3 = 0.50$, the words in Ref. 58 state that $D_4 = 0.08$, and Figure 14 therein gives $D_5 = 0.25$. The clearance pathways (a) and (c) from the nasopharynx and tracheobronchial regions, respectively, transport 0.01 of the amounts deposited in these regions directly to the blood. However, these amounts transported to the blood by this means are not well known as there is doubt about the validity of the 0.01 numbers, as well as about D_3, which Snyder states is 0.25 (Ref. 89). Thus, these two routes into the blood will be neglected, since the maximum error introduced is less than 60 percent of the amount entering the blood from the pulmonary region (using the constants in Table XI, the amount that enters the blood from these two routes is $5.86\ C_o$ μCi while that which enters from the pulmonary region, shown in subsequent calculations, is $9.84\ C_o$ μCi). The amount that reaches the pulmonary compartment is $252\ C_o$ μCi, of which 0.40, or $101 C_o$ μCi, is transported immediately via (f) to the gastrointestinal tract. The remaining amount of $151 C_o$ μCi stays in the pulmonary region for a long period of time, as the biological half-times for the remaining three expulsion routes are all 500 days.

The differential equations that explain the blood input can readily be set up and are

$$\frac{dq_x}{dt} = \lambda_e q_e F \qquad (57)$$

$$\frac{dq_y}{dt} = \lambda_l q_l F \qquad (58)$$

* There remains the possibility that due to the longer lifetime of man in relation to the dog that the lymphatic system involvement might become a concern after long periods of relatively small initial lung burdens.

where q_x = burden entering the blood via route (e)

q_y = burden entering the blood via route (i)

F = fraction that remains in the pulmonary compartment for a 500-day biological half-time; $F = f_e + f_g + f_h = 0.60$

Note that there are no excretion routes for Eqs. (57) and (58), as the blood (in the liver and the bone) retains the plutonium indefinitely. Also, $\lambda_e = \lambda_1 = \lambda_1$ as these routes all have biological half-times of 500 days. The expressions for q_e and q_i are obtained from Eqs. (20) and (39), respectively. Eqs. (57) and (58) can be integrated like Eq. (40) with the results of:

$$q_x(t) = \frac{RC_o D_5 F f_e}{\lambda_A}\left[\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1\right] \tag{59}$$

$$q_y(t) = \frac{RC_o D_5 F f_h f_i}{\lambda_A}\left[\frac{\lambda_A \lambda_1^2}{\lambda_1 - \lambda_A}\left\{\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 \lambda_A (\lambda_1 - \lambda_A)} + \frac{te^{-\lambda_1 t}}{\lambda_1} + \frac{e^{-\lambda_1 t}}{\lambda_1^2}\right\} + 1\right] \tag{60}$$

The blood burden, $q_B(t)$, is the sum of Eqs. (59) and (60) and is

$$q_B(t) = \frac{RC_o D_5 F}{\lambda_A}\left[f_e\left\{\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1\right\} + \frac{f_h f_i \lambda_A \lambda_1^2}{\lambda_1 - \lambda_A}\left\{\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 \lambda_A (\lambda_1 - \lambda_A)} + \frac{te^{-\lambda_1 t}}{\lambda_1} + \frac{e^{-\lambda_1 t}}{\lambda_1^2}\right\} + f_h f_i\right] \tag{61}$$

In Eq. (61) $q_B = 0$ at $t = 0$, and as $t \to \infty$

$$q_B(t \to \infty) \to \frac{RC_o D_5 F}{\lambda_A}\left(f_e + f_h f_i\right) = 9.84\ \mu Ci \tag{62}$$

A plot of Eq. (61) is given in Figure 11, where on the left $q_B(t)\ \lambda_A / RkS_o D_5 F$, a dimensionless quantity, and on the right $q_B(t)$, in μCi ($S_o = 50 \mu g/m^2$) is plotted against the time, t, in days. This curve has a very long time scale similar to that of the pulmonary lymphatic system. One-half of the maximum value is reached in 683 days, and the maximum burden is reached in about 5,000 days. The blood burden, like the lymphatic burden, increases with time to a maximum value where it remains for the lifetime of the individual.

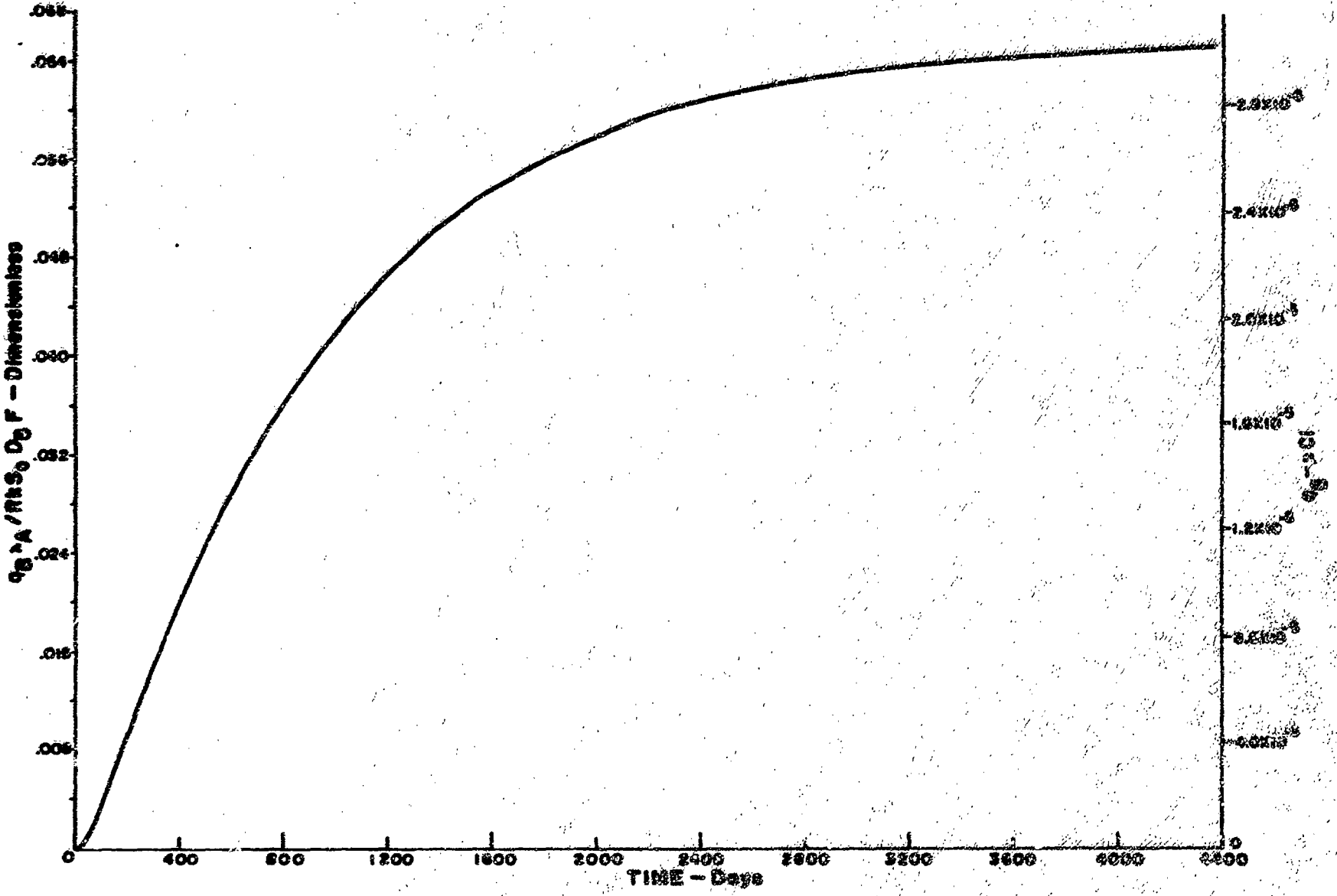

Figure 11. Blood burden following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50 \mu g/m^2$ (1965 Lung Model)

A commonly used assumption is that one-half of the amount of the blood burden goes to the liver and the other half goes to the bone, so each organ receives a maximum burden of 4.92 C_0 μCi. Both organs have negligible excretion of the plutonium (i.e., the biological half-time is infinity) and thus the plutonium will stay in these organs for a lifetime, which we will assume to be 70 years (2.56×10^4 days). The exposure to each of these organs is then about 1.26×10^5 C_0 μCi-days. For ^{239}Pu and with masses of the liver and bone (skeleton) being 1.7 kg and 10 kg (Ref. 35), respectively, Eq. (32) can be used to convert the exposure to these organs from μCi-days to rads, the results being

$$D_{r,l} = 0.155\, D_{c,l} \quad \text{(liver)} \tag{63}$$

$$D_{r,o} = 0.0263\, D_{c,o} \quad \text{(bone)} \tag{64}$$

Thus, the lifetime dose to these organs is 1.95×10^4 C_0 rads to the liver and 3.31×10^3 C_0 rads to the bone. With a resuspension factor of 10^{-6} m^{-1}, these exposures in terms of the initial surface concentration, S_0, are 1.95×10^{-2} S_0 rads for the liver and 3.31×10^{-3} S_0 rads for the bone, as compared with the exposure to the lung of 2.88×10^{-2} S_0 rads, where S_0 is in μCi/m^2. Eq. (1) can be used to express S_0 in μg/m^2 and then the organ exposures are 1.20×10^{-3} S_0 rads to the liver, 2.03×10^{-4} S_0 rads to the bone, and 1.76×10^{-3} S_0 rads to the lung.

The time-varying doses to the liver and bone can be obtained by integrating Eq. (61), and the results are, using Eqs. (63) and (64):

$$D_{r,l} = \frac{0.155\,(0.5)\,RkS_0D_5F}{\lambda_A}\left[\left(\frac{f_e}{\lambda_1-\lambda_A}\right)\left\{\frac{\lambda_1\left(e^{-\lambda_A t}-1\right)}{\lambda_A} - \frac{\lambda_A\left(e^{-\lambda_1 t}-1\right)}{\lambda_1}\right\} + f_e t\right.$$

$$+ \frac{f_h f_l \lambda_1}{(\lambda_1-\lambda_A)^2}\left\{\frac{\lambda_1\left(e^{-\lambda_A t}-1\right)}{\lambda_A} - \frac{\lambda_A\left(e^{-\lambda_1 t}-1\right)}{\lambda_1}\right\} - \frac{f_h f_l \lambda_A}{\lambda_1(\lambda_1-\lambda_A)}\left\{\lambda_1 t e^{-\lambda_1 t}\right.$$

$$\left.\left. + 2\left(e^{-\lambda_1 t}-1\right)\right\} + f_h f_l t\right] \tag{65}$$

$$D_{r,o} = \frac{0.0263\,(0.5)\,RkS_0D_5F}{\lambda_A}\left[\left(\frac{f_e}{\lambda_1-\lambda_A}\right)\left\{\frac{\lambda_1\left(e^{-\lambda_A t}-1\right)}{\lambda_A} - \frac{\lambda_A\left(e^{-\lambda_1 t}-1\right)}{\lambda_1}\right\}\right.$$

$$+ f_e t + \frac{f_h f_l \lambda_1}{(\lambda_1-\lambda_A)^2}\left\{\frac{\lambda_1\left(e^{-\lambda_A t}-1\right)}{\lambda_A} - \frac{\lambda_A\left(e^{-\lambda_1 t}-1\right)}{\lambda_1}\right\} - \frac{f_h f_l \lambda_A}{\lambda_1(\lambda_1-\lambda_A)}$$

$$\left. \times \left\{\lambda_1 t e^{-\lambda_1 t} + 2\left(e^{-\lambda_1 t}-1\right)\right\} + f_h f_l t\right] \tag{66}$$

The graphs of $D_{r,1}$ and $D_{r,0}$, in rads on the left and in rem on the right, versus the time, t, in days, are plotted in Figures 12 and 13, respectively, where the value of $S_0 = 50\ \mu g/m^2$ was used. The liver and bone doses rise very slowly and increase linearly with time beyond about 5,000 days. One-half of the 70-year dose is reached at 13,244 days (36.3 years). The 70-year dose for the liver is 0.058 rads while that for the bone is 0.0098 rads.

In comparing the organ dose curves, one should note that the dose to the liver, bone, and lymphatic system is delivered in 70 years, while the majority of the dose to the lung is delivered in less than 10 years.

The maximum allowable continuous dose to the liver and bone is, using Eqs. (45) and (46), 8.96×10^{-4} μCi for the liver and 1.05×10^{-3} μCi for the bone. The maximum burden in each of these organs is $4.92 \times 10^{-6} S_0$ μCi, so computed values for the allowable S_0 are 182 $\mu Ci/m^2$ (2970 $\mu g/m^2$) for the liver and 214 $\mu Ci/m^2$ (3,490 $\mu g/m^2$) for the bone. Obviously, these values for S_0 are so high that the plutonium burden received by these organs is very low in comparison to what doses these organs may safely receive.

In Table XII are tabulated the doses given to the lung, lymphatic system, liver, and bone for initial surface concentrations of 5, 10, 50, 100, 500, 1,000, 5,000 and 10,000 $\mu g/m^2$. Also given are the judgment values which are empirical numbers for which there is no expectation of biological consequences based on animal data and a small amount of human data. The NCRP reduced these judgment values by a factor of 10 to take care of uncertainties; judgment and NCRP values are not given for the lumphatic system. Even with a 60 percent increase in the liver and bone burdens, the liver becomes as critical as the lung but the bone is still the least critical of these three organs.

TABLE XII

LIFETIME ORGAN DOSES FOR AN AREA CONTAMINATED WITH $^{239}PuO_2$, PLUS JUDGMENT AND NCRP VALUES (1965 Lung Model)

Initial Surface Contamination, S_0 $\mu g/m^2$	Lifetime accumulated organ dose, rads			
	Lung	Lymphatic System	Liver	Bone
5	0.009	0.034	0.006	0.001
10	0.018	0.17	0.012	0.002
50	0.09	0.34	0.06	0.01
100	0.18	1.7	0.12	0.02
500	0.9	3.4	0.6	0.1
1,000	1.8	17	1.2	0.2
5,000	9	34	6	1
10,000	18	170	12	2

TABLE XII (Cont)

Initial Surface Contamination, S_0 $\mu g/m^2$	Lifetime accumulated organ dose, rads			
	Lung	Lymphatic System	Liver	Bone
*	100	-	100	50
**	10	-	10	5

* Judgment values for exposure to three of the critical organs for which there is no expectation of biological consequences based on animal and human data (Ref. 35)

** NCRP values are 0.1 those of the judgment values (Ref. 35).

Another important quantity that must be considered is the dose rate, in rem/yr, for each of the four organs. In Table XIII are listed the organ dose rates, for an initial surface contamination of $S_0 = 50\ \mu g/m^2$, based on the 1965 physiological lung model. Both NCRP and ICRP reference values are given. Note that the initial dose rate to the lung is the largest dose rate received by any organ at any time. When an "average" value of background radiation is added to the lung dose rate for $S_0 = 50\ \mu g/m^2$, the total exposure rate approaches the amount suggested by NCRP as the limit.

A parameter of great importance is the air clearance rate constant, λ_A. The only experimental data for a value of λ_A were obtained on OPERATION PLUMBBOB and OPERATION ROLLER COASTER, which value is applicable to the windy and arid terrain of the Nevada desert. No recent experimental data is available, but there may be different values for different climatic and soil conditions. To this end, a computer study was made to determine the effect on the body organs of different values of λ_A. In Table XIV are summarized the effects on the body organs for various values of λ_A. The results are self-explanatory.

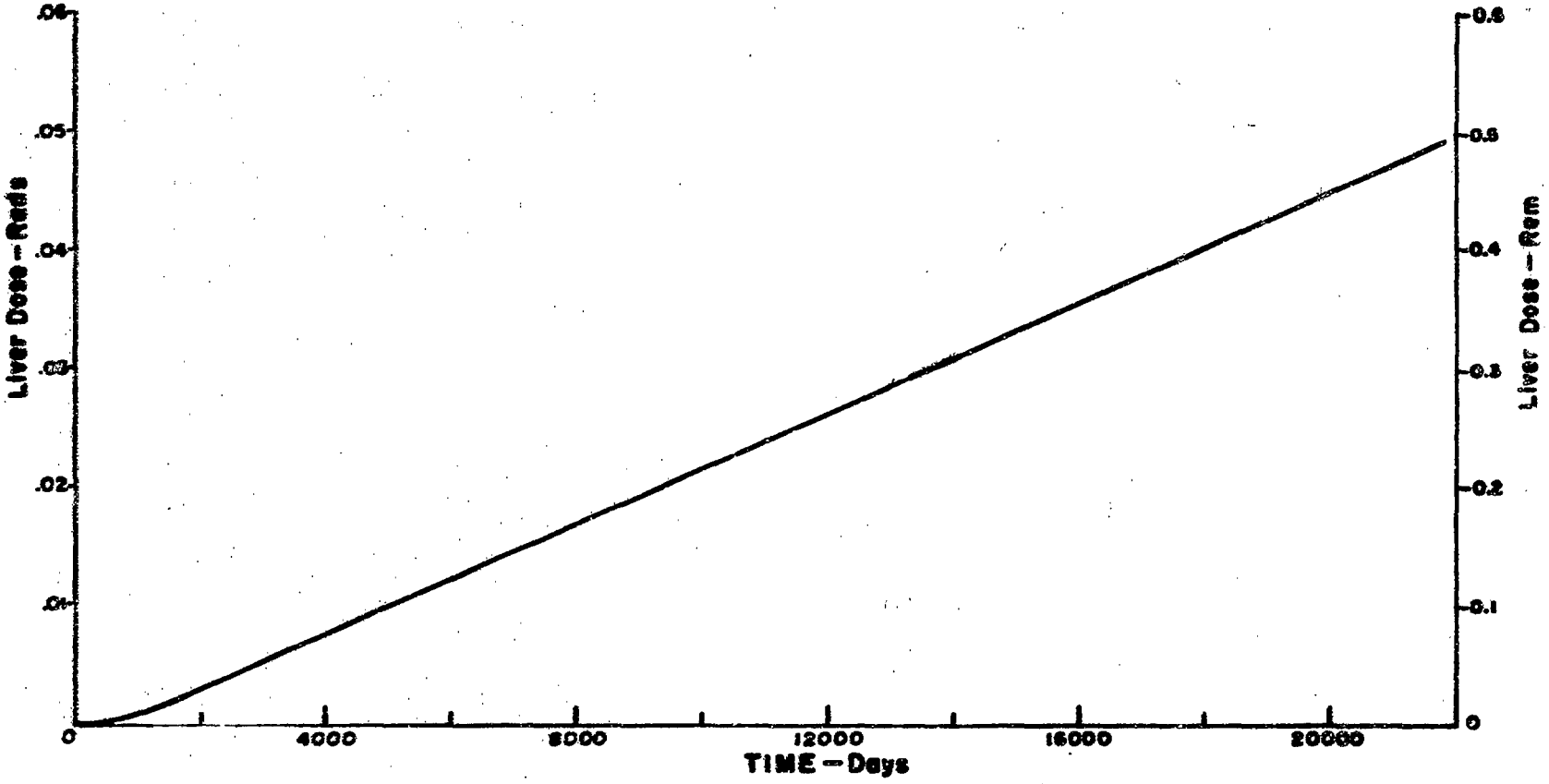

Figure 12. Liver dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50 \mu g/m^2$. (1965 Lung Model).

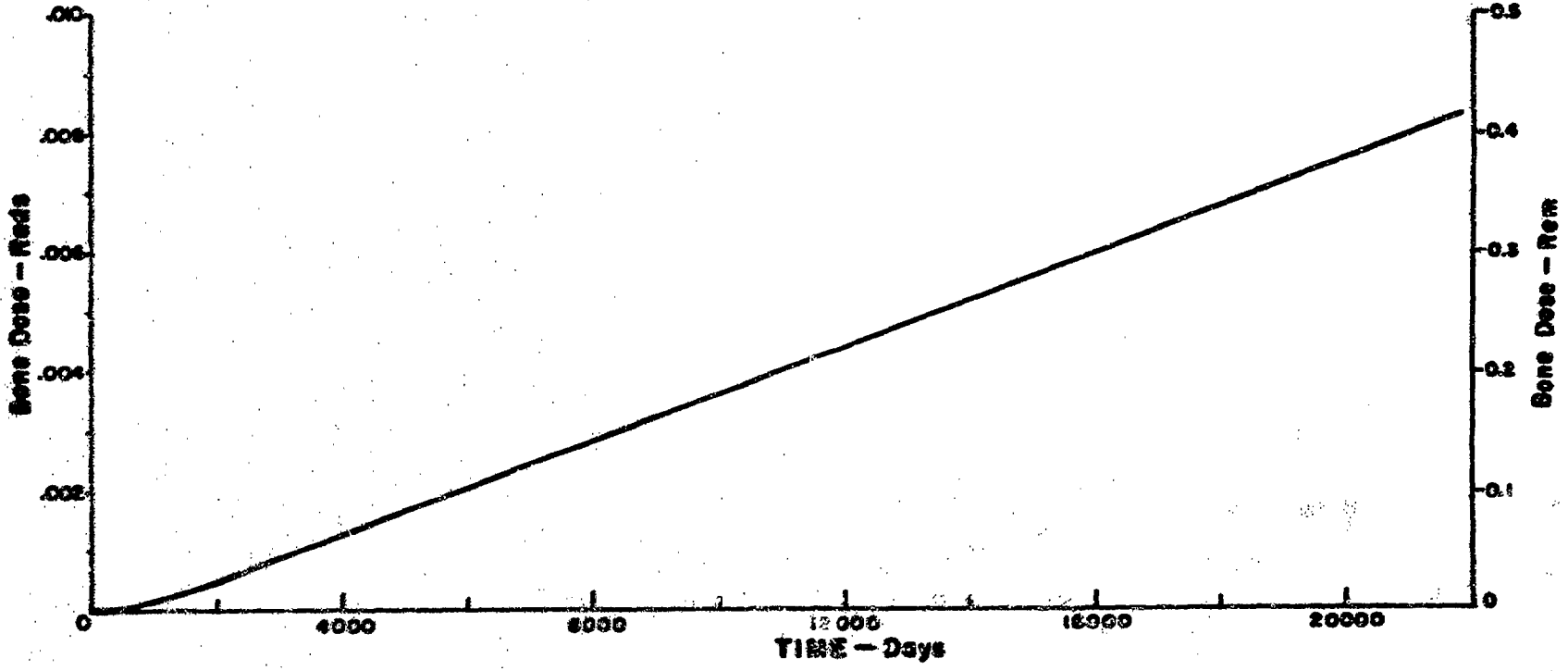

Figure 13. Bone dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50\ \mu g/m^2$ (1965 Lung Model).

TABLE XIII

ORGAN DOSE RATES OBTAINED FROM AN AREA CONTAMINATED WITH

$^{239}Pu\,O_2$ of $S_o = 50\,\mu g/m^2$,

PLUS NCRP AND ICRP VALUES (1965 Lung Model)

	Organ Dose Rate, Rem/Yr			
Year	Lung	Lymphatic System	Liver	Bone
0-1	0.31	0.0092	0.0011	0.0097
1-2	0.23	0.027	0.0035	0.0030
2-3	0.14	0.037	0.0052	0.0044
3-4	0.083	0.043	0.0064	0.0054
4-5	0.050	0.046	0.0071	0.0060
5-6	0.030	0.048	0.0076	0.0065
6-7	0.018	0.049	0.0079	0.0068
7-8	0.011	0.050	0.0082	0.0069
9-10	0.0066	0.050	0.0083	0.0071
10-11	0.0040	0.050	0.0084	0.0071
11-12	0.0024	0.050	0.0085	0.0072
12-13	0.0014	0.050	0.0085	0.0072
13-14	0.0009	0.050	0.0085	0.0072
14-15	0.0005	0.050	0.0085	0.0072
15-20	0.0003	0.25	0.043	0.036
20-25	0.0001	0.25	0.043	0.036
25-30	~0	0.25	0.043	0.036
30-40	~0	0.50	0.086	0.072
40-50	~0	0.50	0.086	0.072
50-60	~0	0.50	0.086	0.072
60-70	~0	0.50	0.086	0.072
NCRP 39 (Ref 85)	0.5	0.5	0.5	0.5
ICRP Pub. No. 9 (Ref. 91)	1.5	1.5	1.5	3.0

Good value for "average" worldwide background = 0.1 - 0.12 rem/year. (Ref. 89)

TABLE XIV

EFFECT ON THE BODY ORGANS FOR DIFFERENT AIR CLEARANCE RATES
(1965 Lung Model)

Body Organ	Item	Air Clearance Rate, λ_A - Days $^{-1}$			
		0.693/35	0.693/70	0.693/105	0.693/140
Pulmonary Lung	Max. burden - μCi	3.81×10^{-4}	6.76×10^{-4}	9.23×10^{-4}	1.14×10^{-3}
	Time to max. burden-days	143	230	298	356
	Time to 1/2 of max. burden-days	26	47	63	78
	70-yr burden - μCi	~0	~0	~0	~0
	Max. dose rate-rem/yr	0.312	0.531	0.804	1.18
	Yr of max. dose rate	0-1	0-1	1-2	1-2
	70-yr dose-rem	0.885	1.77	2.66	3.54
	Time to 1/2 of 70-yr dose-days	551	608	665	721
Lymphatic System	Max. burden - μCi	1.05×10^{-4}	2.09×10^{-4}	3.14×10^{-4}	4.19×10^{-4}
	Time to max. burden-days	4,100	5,600	6,200	5,100
	Time of 1/2 of max. burden-days	500	555	613	670
	70-yr burden - μCi	1.05×10^{-4}	2.09×10^{-4}	3.14×10^{-4}	4.19×10^{-4}
	Max. dose rate-rem/yr	0.0503	0.103	0.151	0.201
	Yr of max dose rate	12 thru 70	13thru 70	11 thru 70	11 thru 70
	70-yr. dose-rem	3.43	6.84	10.24	13.62
	Time to 1/2 of 70-yr dose-days	13,120	13,146	13,171	13,197
Blood	Max. burden - μCi	3.02×10^{-5}	6.05×10^{-5}	9.07×10^{-5}	1.21×10^{-4}
	Time to max. burden-days	5,000	6,800	6,400	5,000
	Time to 1/2 of max. burden-days	683	740	796	850
	70-yr burden - μCi	3.02×10^{-5}	6.05×10^{-5}	9.07×10^{-5}	1.21×10^{-4}
Liver	Max. dose rate-rem/yr	0.00855	0.0171	0.0256	0.0342
	Yr of max dose rate	20 thru 70	15 thru 70	14 thru 70	17 thru 70
	70-yr dose-rem	0.576	1.15	1.72	2.29
	Time to 1/2 of 70-yr dose-days	13,244	13,270	13,295	13,320
Bone	Max. dose rate -rem/yr	0.00737	0.0145	0.0216	0.0291
	Yr of max dose rate	20 thru 70	13 thru 70	15 thru 70	18 thru 70
	70-yr dose-rem	0.490	0.978	1.46	1.95
	Time to 1/2 of 70-yr dose-days	13,244	13,270	13,295	13,320

SECTION VI

FOREIGN PLUTONIUM DECONTAMINATION STANDARDS

Some knowledge of standards as they are established by foreign jurisdictions is of interest. The standards that are reported here are presented only as indications of what present foreign standards may be at this time.

In the search for foreign standards, emphasis was placed on contacts within U. S. governmental agencies whose interests or concerns lie with contamination standards or policy for their agencies. These contacts were successful in providing leads concerning several references to foreign legislation and rules covering alpha and/or plutonium contamination (Ref. 91, 92, 93, 94). Table XV presents a summary of pertinent information from these references.

Note that most of the information presented represents contamination levels applicable to laboratory conditions and not to "environmental real estate." We have listed some levels under the heading "Environmental - Inactive Areas." We report these levels as being possible levels for the "environmental real estate" contamination situation. Our uncertainty is due to our inability to fully define the term, "inactive area," which appears in several of the references. The term is defined in Ref. 94 as an area that is not "active." "Active" areas include change rooms, offices, laboratories and process areas. Our uncertainty involves a feeling that the "inactive" area represents other spaces within boundaries of nuclear facilities. We have found no information covering the calculations employed to derive these standards. If an inactive area includes off-site environmental real estate, 0.01-0.1 pCi/m^3 corresponds to 0.163-1.63 µg/m^2, a level which would be masked by natural background in many areas of the world.

Each level presented in Table XV is accompanied by the date of the reference in which it was published. The spread in level shown under the heading "Laboratory-Workplace, Active Areas" represents the range of standards in that category for various equipment or facilities.

TABLE XV

SOME FOREIGN PLUTONIUM DECONTAMINATION STANDARDS

Country	Laboratory Workplaces, Active Areas	Skin	Environmental Inactive Areas
	Level ($\mu Ci/m^2$)		
Bulgaria	0.06 - 0.14 (64)	unk.	unk.
Canada	0.01 - 0.1 (66)	0.01 (65)	0.01 (65)
Czechoslovakia	0.011 (62)	unk.	unk.
France	0.1 - 1.0 (68)	0.05 (68)	0.02 (68)
East Germany	0.01 - 0.2 (64)	unk.	unk.
Poland	0.1 - 1.0 (62)	unk.	unk.
South Africa	1.0 (62)	0.1 (62)	0.1 (62)
Switzerland	0.1 - 1.0 (62)	0.05 (62)	unk.
United Kingdom	1.0 (63)	0.1 (67)	0.1 (67)
USSR	0.007 (60)	Background (62)	unk.

SECTION VII

REDUCTION OF PLUTONIUM CONTAMINATION HAZARD

INTRODUCTION

This section of the study deals with "happenings" after the occurrence of a plutonium contamination incident. We have used the term "reduction" in the title rather than "decontamination" because the former term is broader in scope, and the post-incident requirements are certainly broad, requiring the support of a number of specialties. Additionally, when we speak of contamination we must note that the term is relative, there being various levels of contamination. In any case, however, any contamination is contamination. In general one may reduce the level of contamination. We are seldom able to eliminate it entirely.

When the next plutonium incident occurs, there will be considerable pressure to take the contamination level to zero. Even if the level is reduced to one that is undetectable, there will be those who will remain unsatisfied. We take this space to point toward these factors because they effect the first important decision concerning decontamination of the incident environment. That is, what level of contamination will remain when the effort is complete. That decision will impact on most subsequent planning and operations. Reasonable decisions made early will reduce the effort and cost of the total operation.

Our discussions with various agencies and individuals focused on factors that affect the final decontamination level for the "next" incident. Several factors were mentioned often enough to be repeated here. The first factor, and one that was universally mentioned, was that no local authority would be satisfied with a level above that employed for previous incidents.

A second factor often mentioned was that negotiators, bystanders or news media may fix on the worst situation in an area and demand that the entire environment be treated as that worst case. For example, the hazard exists in that plutonium which is resuspended and can be inhaled. Plutonium under 10 inches of earth or under several coats of paint is not a hazard. The simple fact that it is there, however, may foster demands for total removal. Another example would be to require that the worst case of resuspension in a contaminated area be applied to the whole area. Or the "worst case" syndrome can operate in conjunction with soil sampling. We have already alluded to the inherent variability in soil samples. Table XVI shows results found in activity levels in seven different aliquots of the same soil sample taken at a follow-up study at Palomares (Ref. 95).

TABLE XVI

VARIABILITY OF SOIL SAMPLING DATA

Aliquot	1	2	3	4	5	6	7
dpm/gram (dry)	455	788	0	28	8	53	45
Average							195

Where it may be reasonable to use an average or to look at adjacent areas in defining that sample, the "worst case" zealot would select aliquot number 2 to define the sample results. Perhaps worse will be the news account which uses the highest figure to define the total situation. All of these worst case factors may operate singly or in conjunction, when the decontamination effort is underway.

DEFINING THE PROBLEM

When the fires are out and the EOD activities have been completed, surveys to determine the extent of radioactive contamination must be undertaken with credible haste. Some survey work will already have been accomplished, primarily to determine if fission products are involved in the incident and to provide health physics support to EOD and other early accident response personnel. The survey should first establish these areas of maximum contamination. Action should be taken as soon as possible to temporarily fix the contamination in these areas to their surfaces. In at least one previous incident, the final cleanup was made considerably more difficult because early fixing was not employed.

The agencies involved in negotiations will be very interested in survey methods and determinations. Their interest may include desire for participation in the survey. If satisfactory control can be maintained over this mixed survey force, the On-Scene Commander should consider operating in this way. Determinations tend to become more credible and negotiations less heated when one's own personnel are involved. Care must be exercised to assure that the joint survey, if employed, does not develop into a competition between agencies.

We have dwelled on determination of ground or surface contamination. We must remember airborne contamination is the hazard. Provision should be made for early placement of air sampling devices. Correct placement of these samplers is vital.

One last type of environmental sample is of importance to the total effort, that is, soil samples. The variance found in soil samples has already been discussed. The information gained from these samples, however, will probably constitute the prime determinator in establishing that the required decontamination effort has been completed. Samples should also be taken during initial survey work as a crosscheck.

There is one other type of survey that should be initiated as soon as arrangements can be made. Personnel in the area at the time of the incident may have accumulated significant plutonium burdens from cloud passage or later activity in the area. Prompt action in personnel surveys will permit early treatment when necessary and will support one's general knowledge of the extent of the early personnel hazard.

Experience with a number of incidents at Hanford indicate some correlation between the activity on nasal smears and in early fecal samples and in vivo lung counting data. This work has led to a set of criteria to be used in recognizing problem intakes and in follow up monitoring (Ref. 96). Table XVII shows these criteria.

Table XVIII (Ref. 96) indicates the type of data which are required to increase the reliability of plutonium burden estimates. Each of the items can add to the assurance of the estimate.

Soil sampling and bio-analysis results will not be available at the incident site for a considerable period. Normal workloads at Service assay laboratories require about one week for chemical determinations. Increased workloads will add several days to the figure. Additional time is consumed in sample preparation and transportation. A real problem exists in assuring that samples, once taken, are not contaminated further as a result of handling.

TABLE XVII

CRITERIA FOR SCHEDULING FECES SAMPLES

Schedule

1. Obtain five daily fecal samples within the first 7 days post intake if any of the following situations occur:

   a. Nasal smears exceed 500 dis/min,

   b. 100 < nasal smears < 500 dis/min and exposure duration > 5 min,

   c. 5 < nasal smears < 100 dis/min and exposure duration > 30 min,

   d. Exposure to fumes from a fire,

   e. Air sample results exceed 2×10^{-10} μCi/cc for an 8-hr period and exposure duration > 1 hr.

2. Obtain one fecal sample on the second day post intake if any of the following situations occur:

   a. Nasal smears are positive but do not meet criteria in Schedule 1,

   b. Any other person in the same incident meets the criteria in Schedule 1,

   c. Air sample results exceed 2×10^{-11} μCi/cc for an 8-hr period,

   d. Wide spread skin contamination in a dry form or facial contamination > 1000 dis/min,

   e. Clothing contamination in a dry form > 5000 dis/min,

   f. Possible plutonium inhalation is suspected for other reasons.

3. Obtain two fecal samples at periods > 10 days post incident:

   Obtain samples to coincide with positive lung counter examinations preferably following two days off of work on the 15th and 30th days post intake. Schedule at approximately monthly intervals thereafter provided that data useful for evaluation are obtained.

TABLE XVIII

TYPES OF DATA WHICH MAY BE COLLECTED FOR PLUTONIUM INHALATION INCIDENTS

1. Urine analysis.
2. Feces analysis.
3. In-vivo examination.
4. Isotopic composition and Pu alpha/Am -241 alpha ratio.
5. Chemical form of the aerosol.
6. Solubility of the aerosol.
7. Particle size (air samples-nasal smears).
8. Nasal and skin contamination activity.
9. Concentration of plutonium aerosol.
10. Duration of exposure.
11. Other details of incident.

CONTAMINATION CONTROL

The operations previously discussed are those carried out by trained personnel available in response teams. Their training has included not only that specific to particular instruments and operations, but also those many general operations and procedures concerning proper contamination control. Contamination control incorporates all those procedures and operations undertaken to keep the contamination within the original area of concern. Most of the personnel in the cleanup work force will not be trained in contamination control. Considerable effort must be expended initially and must continue during decontamination efforts to assure that this supplementary work force learns and follows contamination control procedures.

The incident response teams that form the decontamination force nucleus have been thoroughly trained in contamination control. The On-Scene Commander should rely on this cadre to train other personnel and monitor their actions. He must remember, however, that as personnel become more familiar with the hazards and procedures involved, they will probably become lax. He must be continually on the alert for signs of this laxness and take command action to keep it at a minimum.

The procedures involved in maintenance of the "Hot Line", the boundary of the contaminated area, and in dress-out and work force decontamination when leaving the contaminated area must be adhered to. They may appear to be time consuming and, in the later stages of the operation, less necessary. They are not. A considerable amount of contamination adheres to work clothing during the grubby work of area decontamination. Relaxation of contamination control and "Hot Line" procedures is the surest way to spread unwanted contamination and occasion a larger decontamination problem in the end. The problem, of course, increases if this spread should extend into previously uncontaminated and populated civilian areas.

PUBLIC INFORMATION

Public affairs guidance with respect to nuclear accidents is contained in DOD Instruction 5230.16 (Ref. 97). We will not dwell on these procedures here except to point out that the On-Scene Commander must recognize that requests and demands for information will be extensive in number and in depth. A Public Information Officer is generally included in the response force staff.

METHODS OF DECONTAMINATION

As noted previously, the faster that the clean-up operations are accomplished after the nuclear weapon accident, the less will be the maximum lung burden. This fact is graphically portrayed in Figure 6 which shows that the lung burden curve rises very rapidly and reaches half the maximum value in 26 days. Thus, fast and efficient decontamination methods will greatly benefit the well-being of individuals in the contaminated area.

Experience has shown when the decontamination is handled by experts that most contaminated surfaces can be restored to normal use. As great a variety of possible circumstances surround each contamination incident as there are methods to decontaminate the affected regions. In fact, many times a combination of decontamination methods are used. The reader is referred to Refs. 98 and 99 which give abstracts of hundreds of general decontamination methods. Some of these decontamination methods for hard surfaces and land areas with their respective efficiencies (these have all been taken from Langham in Ref. 35) are given below. Note that in all decontamination efforts, careful consideration must be given to proper disposal of the contaminated waste.

Hard Surfaces

Water, sandblasting, vacuum cleaning, and steam cleaning are some methods used to decontaminate hard surfaces. The efficiencies for these methods on various surfaces are given in Table XX.

Water - A water truck that can produce a water stream at a pressure of 200 to 400 psi is used for (1) plain water hosing, (2) water hosing and scrubbing, (3) hosing with 1 percent (by weight) commercial detergent and water solution, or (4) detergent solution hosing followed by scrubbing and rinse.

Sandblasting - In this operation the surface is removed, so sandblasting should be used only when other methods are unsuccessful. The loose residue created must be collected somehow, as by vacuum cleaning.

Vacuum Cleaning - In situations where water cannot be practically used, then vacuum cleaning may be suitable. In this method suitable filters must be placed over the exhaust to prevent resuspension of the contaminant blowing through the cleaner.

Steam Cleaning - Greasy or oily surfaces may be cleaned best with the use of steam cleaners.

Land Areas (See also Refs. 100 and 101)

Some methods for fixation and/or decontamination of land areas are plowing, scraping, oiling, oiling and scraping, wetting down with water and scraping, or flooding with water. The efficiencies for these methods are given in Table XX.

Plowing - Plowing to a depth of 12 inches will ensure adequate mixing and burial of the contaminant.

Scraping - The top 2 inches of the soil may be removed.

Oiling - An oil-distribution truck may be used to spread a rapid cure oil, like RC-O, over the decontaminated area. A semihardened surface is formed within 24 hours.

Oiling and Scraping - The oiling procedure outlined above can be followed by a scraping procedure whereby the hardened oil crust is scraped and removed.

Wetting Down with Water and Scraping - Wetting down with about 0.3 inch of water will tend to temporarily fix the contaminant and permit its removal by scraping without excessive resuspension of the contaminant.

Flooding with Water - Flooding with large amounts of water to 1 inch or more will accelerate the natural weathering action and tend to leach the contaminant into the soil, thus reducing the amount of contaminant available for resuspension into the air.

TABLE XIX

PERCENT EFFICIENCIES FOR VARIOUS HARD SURFACE DECONTAMINATION METHODS (Ref. 35)

	Method (% efficiency)						
Material	Vacuum	High pressure water	High pressure water with scrub	High pressure water and detergent	High pressure water and detergent with scrub	Sand blasting	Steam cleaning
Glass	98	99	97	100	99	100	97
Stucco	48	97	95	95	99	100	27
Painted wood	99	98	96	99	99	100	91
Unpainted wood	36	85	93	99	95	99	85
Aluminum	89	99	97	99	100	98	84
Plate steel	93	97	94	100	98	99	91
Asbestos shingles	61	99	98	96	99	100	63
Unpainted wood shingles	61	97	90	95	97	99	71
Brick	29	99	99	99	99	99	97
Tar paper	55	98	95	95	96	99	52
Corrugated galvanized roofing	69	99	97	99	99	100	85
Highway asphalt (2 ft^2)	32	99	96	99	99	99	44
Highway asphalt (10 ft^2)	72	92	94	98	96	92	22
Sealed asphalt (2 ft^2)	71	98	90	100	99	99	84
Sealed asphalt (10 ft^2)	64	90	82	96	97	90	48
Steel trowel concrete (2 ft^2)	74	98	-	96	99	100	-
Steel trowel concrete (10 ft^2)	-	78	97	-	98	98	27
Wood float concrete (2 ft^2)	-	98	92	100	97	100	65
Wood float concrete (10 ft^2)	56	97	-	98	98	98	85
Average of all surfaces	66	96	94	98	98	98	67

TABLE XX

LAND AREA FIXATION AND/OR DECONTAMINATION EFFICIENCIES (Ref. 35)

Method for ground decontamination	Activity present: Mean initial (dpm/m^3)	Mean final (dpm/m^3)	Efficiency (percent)
Plowing	8200	140	98
Oil and scrape	4200	80	98
Scrape	580	25	95
Water (0.3 in.) and scrape	1400	100	93
Oil (RC-O road oil)	1000	100	89
Flooding with water (1.0 in.)	1600	225	85
Water-$FeCl_3$ solution (0.3 in.)	4000	630	84
Disking	3000	730	76

SUMMARY

The information covered in this section is not intended to provide a check list concerning all the actions and procedures of a decontamination operation. We feel that each incident will have its peculiarities and that those must be attended to as they surface. We have pointed toward general problem areas that must be met and solved during most operations. We reiterate that the On-Scene Commander must be provided with a knowledgeable cadre of trained personnel. He must, in turn, rely on this cadre for expert advice and opinion as the operation proceeds.

We suggest that the Services develop criteria for bio-assay requirements in cases of wide area plutonium contamination. These criteria should be coordinated to facilitate inter-Service support as previously recommended in Section IV.

We recommend that DOD Instruction 5230.16 be reviewed toward fostering as open an information policy as possible.

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APPENDIX A

Dr. W. S. Snyder (Oak Ridge National Laboratory, Oak Ridge, Tennessee) and Dr. Paul Morrow (University of Rochester School of Medicine and Dentistry) have kindly consented to furnish this team with the values of the proposed constants for Class Y compounds (insoluble compounds with long retention times like plutonium) for the 1973 physiological lung model. These constants are given in Table XXI. Note that the constants for the pulmonary portion of the lung are the same as in the 1965 physiological lung model. There are slight changes in the half-times for the nasopharynx and tracheobronchial regions of the lung. The largest changes occur in the regional fractions for the pulmonary lymphatic systems, where in the 1973 model the values are $f_l = 0.90$ and $f_k = 0.10$, whereas in the 1965 model these values were $f_l = 0.10$ and $f_k = 0.90$; also the biological half-time for route l has been increased from 500 to 1,000 days.

The differential equations for the pulmonary region are the same as in the 1965 model previously discussed, and since the constants are the same, so also are the results.

TABLE XXI

CONSTANTS FOR USE WITH THE PROPOSED 1973 LUNG CLEARANCE MODEL FOR $^{239}PuO_2$

Region	Process	Biological Half-Time, Days	Regional Fraction
N-P	a	0.01	0.01
	b	0.01	0.99
T-P	c	0.01	0.01
	d	0.2	0.99
P	e	500	0.05
	f	1	0.40
	g	500	0.40
	h	500	0.15
Lymph	i	1,000	0.90
	k	∞ (Subject only to radioactive decay)	0.10

Since the biological half-time for route i has changed, the differential equation is also different and is

$$\frac{dq_i}{dt} = If_i - \lambda_2 q_i \tag{67}$$

where $\lambda_2 = \lambda_1 = 0.693/1000$ days^{-1}. The integrated result is

$$q_i(t) = \frac{\lambda_1 R\, k\, S_o D_5 f_h f_i}{\lambda_1 - \lambda_A}\left[\frac{e^{-\lambda_A t}}{\lambda_2 - \lambda_A} - \frac{e^{-\lambda_1 t}}{\lambda_2 - \lambda_1} + \frac{(\lambda_1 - \lambda_A)\, e^{-\lambda_2 t}}{(\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)}\right] \tag{68}$$

The differential equation for route k is the same as before, so $q_k(t)$ is given in Eq. (41) The total dose to the lymph nodes, q_n, is then

$$q_n(t) = R\, k\, S_o\, D_5\, f_h\left[\frac{\lambda_1 f_i}{\lambda_1 - \lambda_A}\left\{\frac{e^{-\lambda_A t}}{\lambda_2 - \lambda_A} - \frac{e^{-\lambda_1 t}}{\lambda_2 - \lambda_1} + \frac{(\lambda_1 - \lambda_A)e^{-\lambda_2 t}}{(\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)}\right\}\right.$$

$$\left. + \frac{f_k}{\lambda_A}\left\{\frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1\right\}\right] \tag{69}$$

A plot of Eq. (69) is shown in Figure 14. Note how the lymphatic system burden increases sharply to a maximum in 1150 days, then decays with about a 1,000 day half-time to an asymptotic value of

$$q_n(t \to \infty) = \frac{R\, k\, S_o\, D_5\, f_h\, f_i}{\lambda_A} = 3.79 C_o\ \mu\text{Ci} \tag{70}$$

This value in Eq. (70) is a factor of 9 less than that given in Eq. (44), which greatly reduces the lifetime burden in the lymphatic system, making the lymphatic system even a less critical organ than it was in the 1965 model.

The time-varying dose to the lymphatic system is

$$D_{r,n} = 0.1317\int_0^t q_n(t)\, dt = 0.1317\, R\, k\, S_o D_5 f_h\left[\frac{\lambda_1 f_i}{\lambda_1 - \lambda_A}\left\{\frac{(e^{-\lambda_1 t} - 1)}{\lambda_1(\lambda_2 - \lambda_1)} - \frac{(e^{-\lambda_A t} - 1)}{\lambda_A(\lambda_2 - \lambda_A)}\right.\right.$$

$$\left.\left. - \frac{(\lambda_1 - \lambda_A)(e^{-\lambda_2 t} - 1)}{\lambda_2(\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)}\right\} + \frac{f_k}{\lambda_A}\left\{\frac{\lambda_1(e^{-\lambda_A t} - 1)}{\lambda_A(\lambda_1 - \lambda_A)} - \frac{\lambda_A(e^{-\lambda_1 t} - 1)}{\lambda_1(\lambda_1 - \lambda_A)} + t\right\}\right] \tag{71}$$

A plot of Eq. (71) is shown in Figure 15, where the value of $S_o = 50\ \mu g/m^2$ was used. The time required to reach one-half of the 70-year dose is 6,893 days, which is about half the time of the 1965 model, but the 70-yr dose is 0.058 rad, a factor of about 6 less than with the 1965 model.

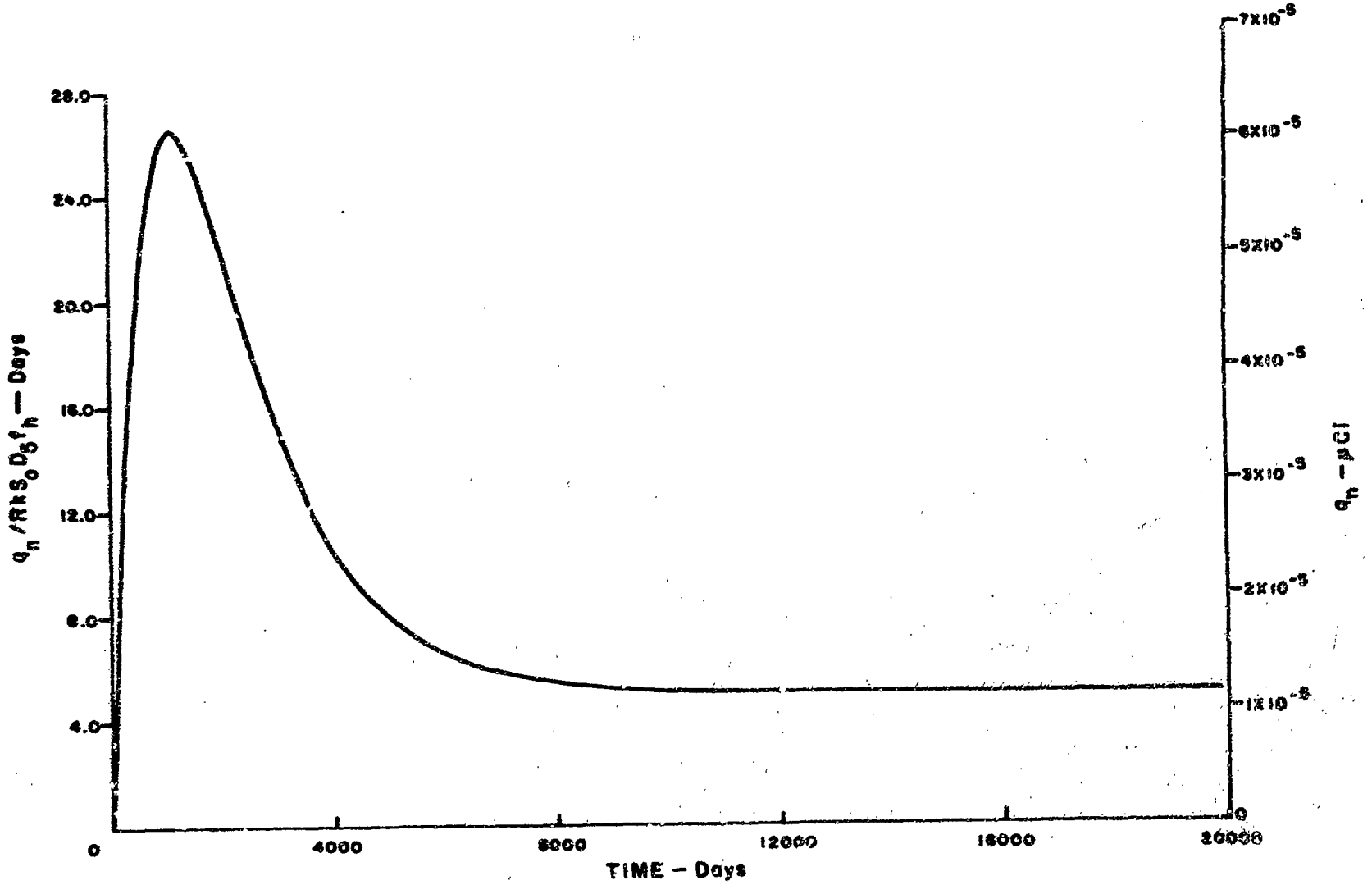

Figure 14. Lymphatic system burden following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50\mu g/m^2$. (Proposed 1973 Lung Model)

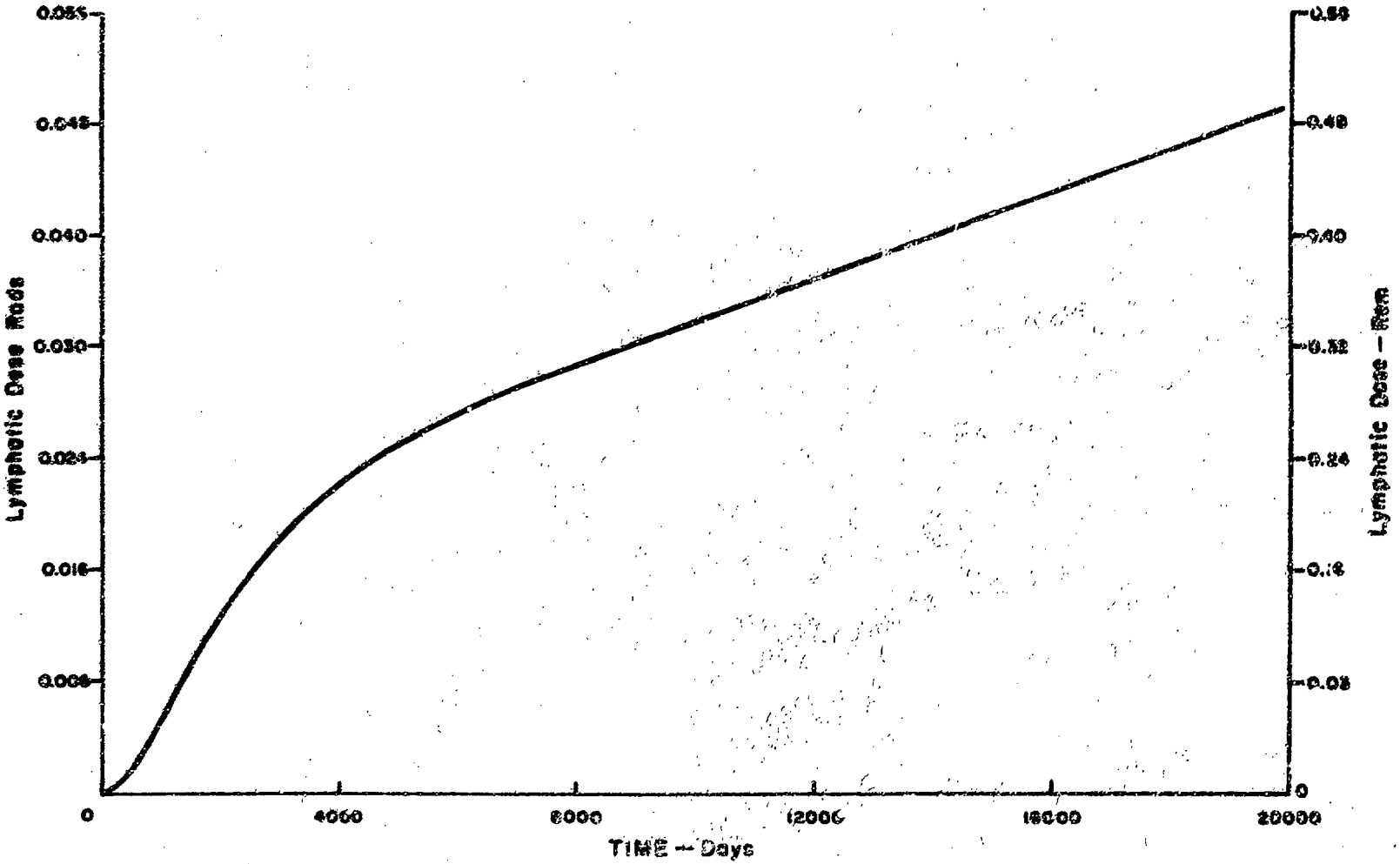

Figure 15. Lymphatic system dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_o = 50\mu g/m^2$. (Proposed 1973 Lung Model)

The blood input differential equations are Eq. (57) and

$$\frac{dq_y}{dt} = \lambda_1 q_1 F = \lambda_2 q_1 F \tag{72}$$

with the result of

$$q_y(t) = \frac{R k S_o D_5 f_h f_1 F \lambda_1 \lambda_2}{\lambda_1 - \lambda_A} \left[\frac{(e^{-\lambda_1 t} - 1)}{\lambda_1 (\lambda_2 - \lambda_1)} - \frac{(e^{-\lambda_A t} - 1)}{\lambda_A (\lambda_2 - \lambda_A)} - \frac{(\lambda_1 - \lambda_A)(e^{-\lambda_2 t} - 1)}{\lambda_2 (\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)} \right] \tag{73}$$

The blood input, $q_B(t)$, is then

$$q_B(t) = R k S_o D_5 F \left[\frac{f_e}{\lambda_A} \left\{ \frac{\lambda_A e^{-\lambda_1 t} - \lambda_1 e^{-\lambda_A t}}{\lambda_1 - \lambda_A} + 1 \right\} + \frac{f_h f_1 \lambda_1 \lambda_2}{\lambda_1 - \lambda_A} \left\{ \frac{(e^{-\lambda_1 t} - 1)}{\lambda_1 (\lambda_2 - \lambda_1)} + \frac{(e^{-\lambda_A t} - 1)}{\lambda_A (\lambda_2 - \lambda_A)} - \frac{(\lambda_1 - \lambda_A)(e^{-\lambda_2 t} - 1)}{\lambda_2 (\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)} \right\} \right] \tag{74}$$

The blood burden is plotted in Figure 16. The time to one-half the maximum burden is 1408 days, about twice as long as in the 1965 model. However, the 70-yr burden is about 3 times greater with the new model.

The time-varying doses to the liver and bone are:

$$D_{r,1} = 0.1549\,(0.5)\,R k S_o D_5 F \left[\frac{f_e}{\lambda_A} \left\{ \frac{\lambda_1^2 (e^{-\lambda_A t} - 1) - \lambda_A^2 (e^{-\lambda_1 t} - 1)}{\lambda_1 \lambda_A (\lambda_1 - \lambda_A)} + t \right\} + \frac{f_h f_1 \lambda_1 \lambda_2}{\lambda_1 - \lambda_A} \right.$$

$$\left. X \left\{ \frac{e^{-\lambda_A t} - 1 + \lambda_A t}{\lambda_A^2 (\lambda_2 - \lambda_A)} + \frac{(\lambda_1 - \lambda_A)(e^{-\lambda_2 t} - 1 + \lambda_2 t)}{\lambda_2^2 (\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)} - \frac{(e^{-\lambda_1 t} - 1 + \lambda_1 t)}{\lambda_1^2 (\lambda_2 - \lambda_1)} \right\} \right] \tag{75}$$

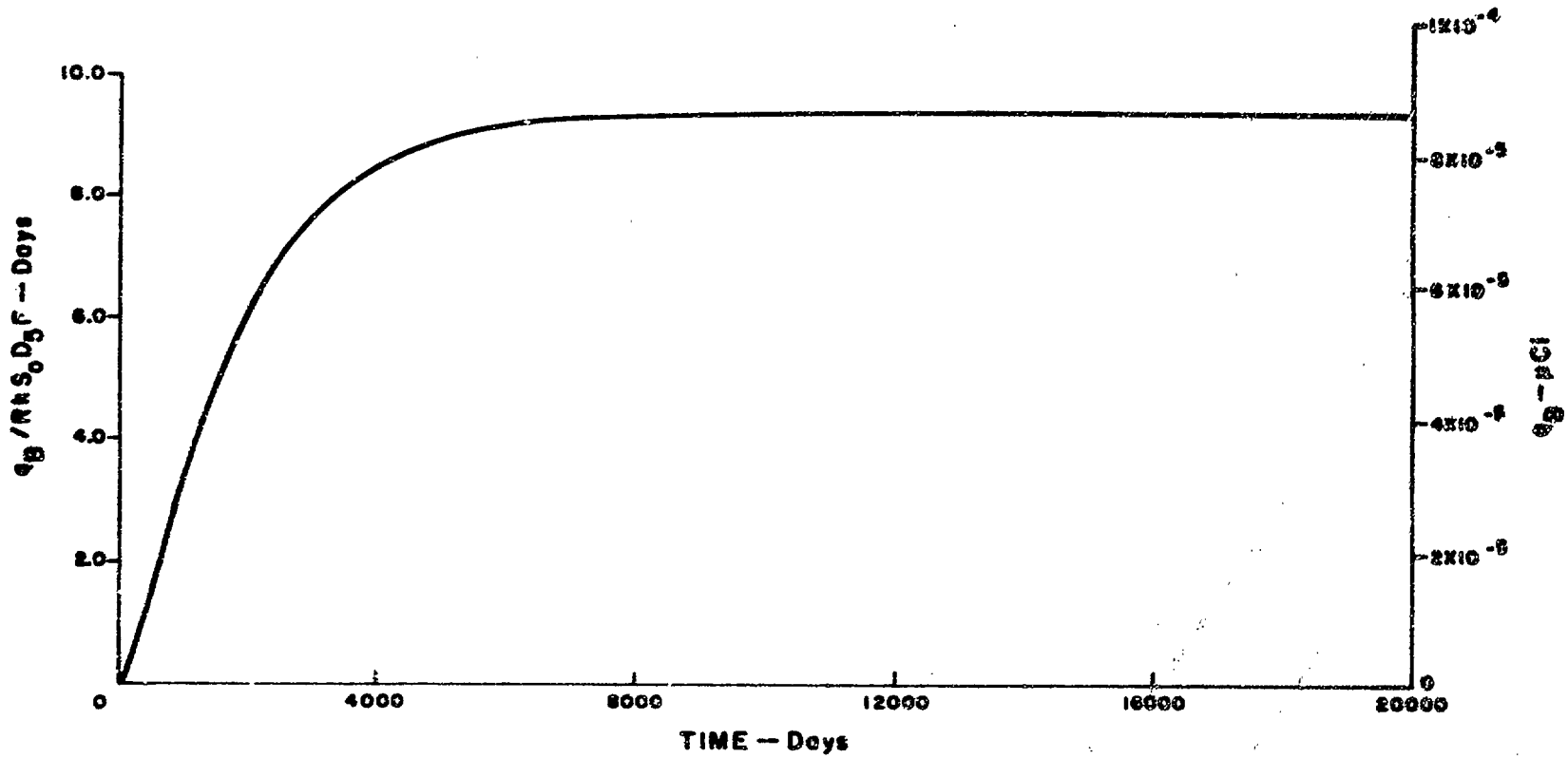

Figure 16. Blood burden following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_0 = 50 \mu g/m^2$ (Proposed 1973 Lung Model).

$$D_{2,o} = 0.02634(0.5) R k S_o D_3 F \left[\frac{f_e}{\lambda_A} \left\{ \frac{\lambda_1^2 (e^{-\lambda_A t} - 1) - \lambda_A^2 (e^{-\lambda_1 t} - 1)}{\lambda_1 \lambda_A (\lambda_1 - \lambda_A)} + t \right\} + \frac{f_h f_l \lambda_1 \lambda_2}{\lambda_1 - \lambda_A} \right.$$

$$\left. \times \left\{ \frac{e^{-\lambda_A t} - 1 + \lambda_A t}{\lambda_A^2 (\lambda_2 - \lambda_A)} + \frac{(\lambda_1 - \lambda_A)(e^{-\lambda_2 t} - 1 + \lambda_2 t)}{\lambda_2^2 (\lambda_2 - \lambda_1)(\lambda_2 - \lambda_A)} - \frac{(e^{-\lambda_1 t} - 1 + \lambda_1 t)}{\lambda_1^2 (\lambda_2 - \lambda_1)} \right\} \right] \qquad (76)$$

The doses to the liver and bone are given in Figures 17 and 18, where the value of $S_o = 50\ \mu g/m^2$ was used. The time to one-half of the 70-yr burden is reached in about the same time with the two models, but the 70-yr doses to the liver and bone of 0.16 and 0.027 rads, respectively, are about a factor of 3 higher with the 1973 model as with the 1965 model.

The organ dose rates, based on the 1973 model, are given in Table XXII. An inspection of the numbers clearly reveals that the lung is the most critical organ.

Table XXIII contains computer summarized information on how different air clearance rate constants affect the organs. These numbers can be compared with those in Table XIV which has the numbers generated with the 1965 model.

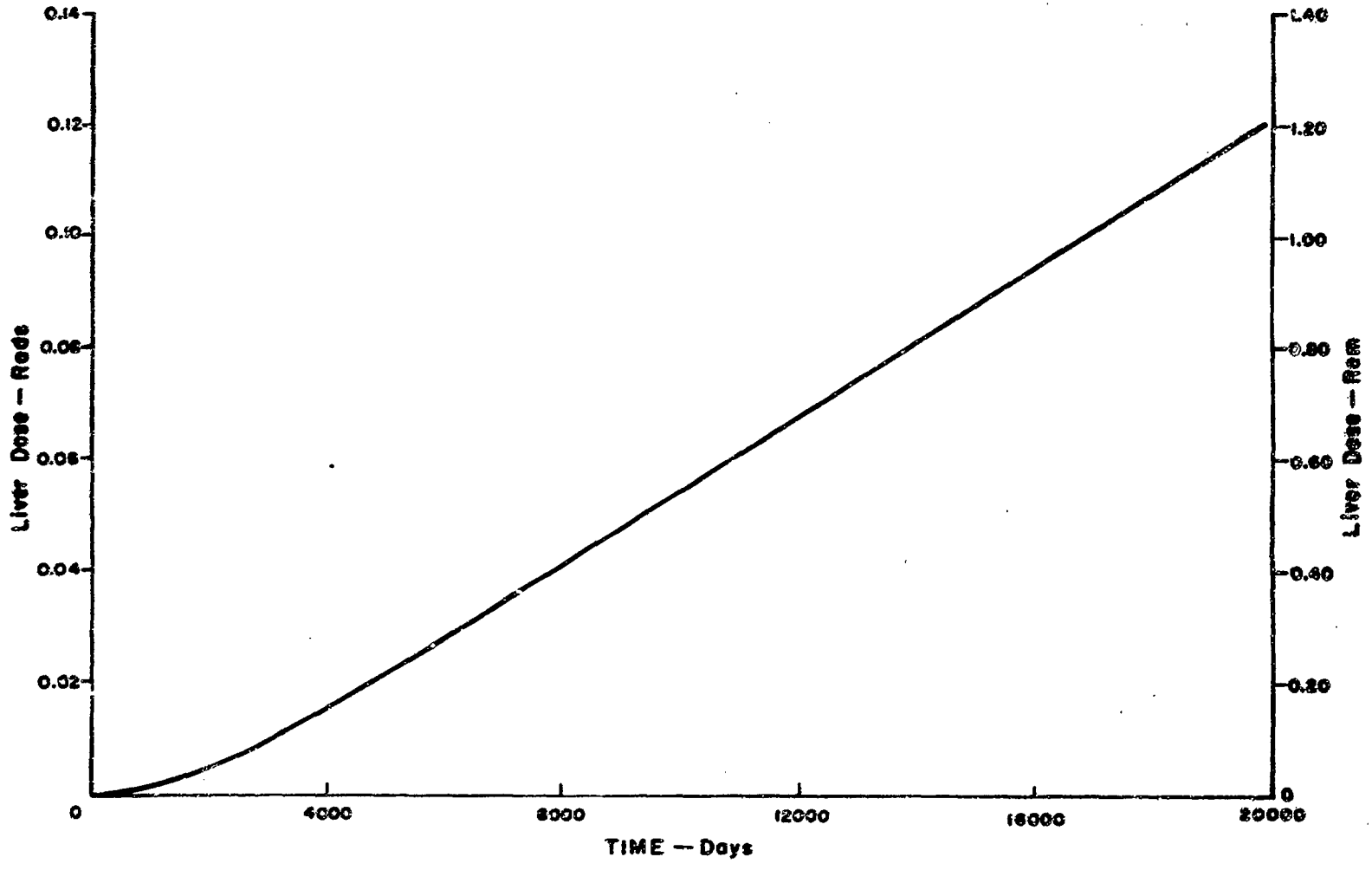

Figure 17. Liver dose following inhalation of $^{239}Pu($ ... versus time of exposure in a contaminated area of $S_o = 50\mu g/m^2$ ...oposed 1973 Lung Model)

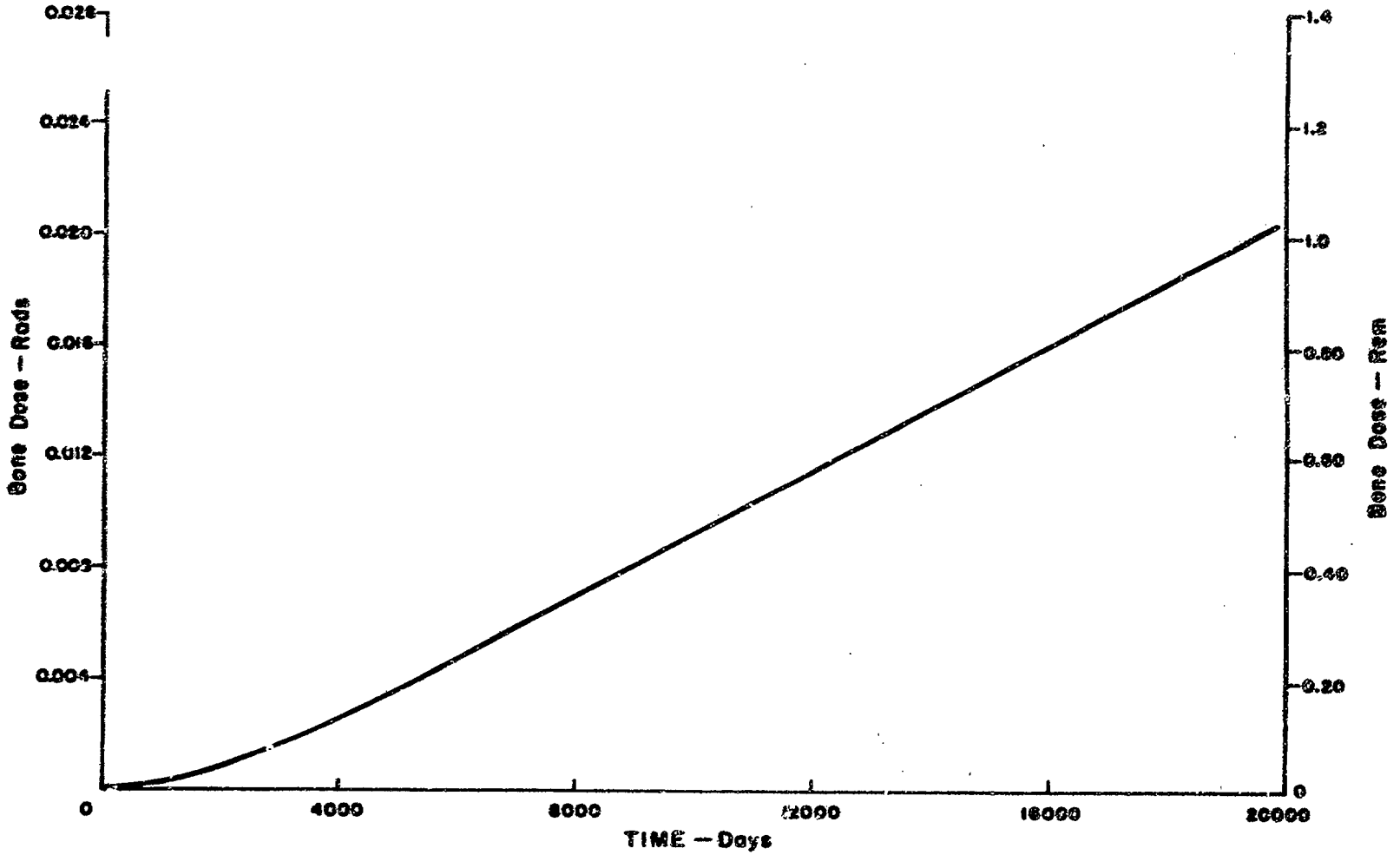

Figure 18. Bone dose following inhalation of $^{239}PuO_2$ dust versus time of exposure in a contaminated area of $S_0 = 50 \mu g/m^2$ (Proposed 1973 Lung Model).

TABLE XXII

ORGAN DOSE RATES FOR AN AREA CONTAMINATED WITH $^{239}PuO_2$ OF $S_o = 50 \mu g/m^2$, PLUS NCRP AND ICRP VALUES (PROPOSED 1973 LUNG MODEL).

Year	Organ Dose Rate, Rem/Yr			
	Lung*	Lymphatic System	Liver	Bone
0-1	0.31	0.0087	0.0013	0.0011
1-2	0.23	0.023	0.0048	0.0061
2-3	0.14	0.029	0.0082	0.0069
3-4	0.083	0.029	0.011	0.0095
4-5	0.050	0.027	0.014	0.012
5-6	0.030	0.024	0.016	0.014
6-7	0.018	0.021	0.018	0.015
7-8	0.011	0.019	0.019	0.016
8-9	0.0066	0.016	0.020	0.017
9-10	0.0040	0.014	0.021	0.018
10-11	0.0024	0.012	0.022	0.019
11-12	0.0014	0.011	0.022	0.019
12-13	0.0009	0.0098	0.023	0.019
13-14	0.0005	0.0089	0.023	0.020
14-15	0.0003	0.036	0.023	0.020
15-20	0.0001	0.050	0.12	0.10
20-25	~0	0.029	0.12	0.10
25-30	~0	0.028	0.12	0.10
30-40	~0	0.056	0.24	0.21
40-50	~0	0.056	0.24	0.21
50-60	~0	0.056	0.24	0.21
60-70	~0	0.056	0.24	0.21
NCRP 39 (Ref. 85)	0.5	0.5	0.5	0.5
NCRP Pub. No. 9 (Ref. 91)	1.5	1.5	1.5	3.0

* Same as for 1965 lung model.

TABLE XXIII

EFFECT ON THE BODY ORGANS FOR DIFFERENT AIR CLEARANCE RATES (1973 Lung Model)

Body Organ	Item	Air Clearance Rate, λ_A - Days^{-1}			
		0.0693/35	0.693/70	0.693/105	0.693/140
Pulmonary Lung	Same as for 1965 Lung Model (Given in Table XIV)				
Lymphatic System	Max. burden -μCi	6.11×10^{-5}	1.22×10^{-4}	1.82×10^{-4}	2.40×10^{-4}
	Time to max. burden-days	1150	1200	1250	1350
	Time to 1/2 of max. burden-days	294	347	395	406
	70-yr burden -μCi	1.16×10^{-5}	2.33×10^{-5}	3.49×10^{-5}	4.65×10^{-5}
	Max. dose rate -rem/yr	0.0291	0.0582	0.0869	0.114
	Yr of max dose rate	3-4	3-4	3-4	3-4
	70-yr dose-rem	0.578	1.16	1.73	2.30
	Time to 1/2 of 70-yr dose-days	6893	6922	6952	6981
Blood	Max. burden - μCi	8.61×10^{-5}	1.72×10^{-4}	2.58×10^{-4}	3.44×10^{-4}
	Time to max. burden-days	15,500	9000	9500	10,000
	Time to 1/2 of max. burden-days	1408	1455	1513	1565
	70-yr burden - μCi	8.61×10^{-5}	1.72×10^{-4}	2.58×10^{-4}	3.44×10^{-4}
Liver	Max. dose rate-rem/yr	0.0243	0.0487	0.0730	0.0973
	Yr of max. dose rate	24 thru 70	41 thru 70	34 thru 70	33 thru 70
	70-yr dose - rem	1.58	3.16	4.72	6.28
	Time to 1/2 of 70-yr dose-days	13,687	13,713	13,738	13,763
Bone	Max. dose rate-rem/yr	0.0207	0.0414	0.0620	0.0827
	Yr of max. dose rate	28 thru 70	34 thru 70	28 thru 70	30 thru 70
	70-yr dose-rem	1.34	2.68	4.02	5.34
	Time to 1/2 of 70-yr dose-days	13,687	13,713	13,738	13,763