

**PROPOSED GUIDANCE
ON
DOSE LIMITS FOR PERSONS EXPOSED
TO
TRANSURANIUM ELEMENTS
IN THE
GENERAL ENVIRONMENT**



**U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RADIATION PROGRAMS
CRITERIA AND STANDARDS DIVISION
WASHINGTON, D.C. 20460**

PROPOSED GUIDANCE
on
DOSE LIMITS FOR PERSONS EXPOSED TO TRANSURANIUM ELEMENTS
in the
GENERAL ENVIRONMENT

SUMMARY REPORT

September 1977

U. S. Environmental Protection Agency
Office of Radiation Programs
Criteria and Standards Division
Washington, D.C. 20460

Guidance on Dose Limits
for the Transuranium Elements in the General Environment

Table of Contents

	<u>Page</u>
1. Background Information on the Transuranium Elements	
1.1 Introduction	1
1.2 Current Sources of the Transuranium Elements	3
1.3 Movement Through the Biosphere	4
1.4 Potential Health Effects	7
2. Federal Guidance	
2.1 Agency Action and Authorities	11
2.2 Involvement of other Federal Agencies	12
2.3 Existing Standards and Guides	13
2.4 Criteria and Rationale for Proposed Guidance	14
2.5 Text of Proposed Guidance	20
2.5.1 Definitions	21
3. Implications of Guidance	
3.1 Scope of Sources and Population Groups	23
3.2 Risk Perspectives	24
3.3 Implementation	28
3.4 Remedial Measures and Economic Evaluation	30
References	32

Annex

- Annex I Transuranium Elements in the Environment
- Annex II Environmental Transport and Pathways
- Annex III The Dose and Risk to Health Due to the Inhalation and
Ingestion of Transuranium Elements
- Annex IV Risk Perspective
- Annex V Guidance Implementation
- Annex VI Environmental Assessment

Section 1

Background Information on the Transuranium Elements

1.1 Introduction

The transuranium elements include all of the elements with atomic number greater than that of uranium. Only extremely small quantities of these elements occur naturally and nearly all of the existing inventory has been created from other elements. Nuclides of the transuranium elements are radioactive and are considered to be toxic in humans. From the viewpoint of protecting the public health, the transuranium elements of primary interest are those which undergo slow radioactive decay with the emission of alpha particles and with half-lives greater than 1 year. When present in the environment, transuranium elements may be available for intake by humans over very long periods of time. Table 1.1 is a listing of such transuranium radio-nuclides of major interest, their mode of decay, half-life, and daughter products.

Plutonium is the most abundant, most studied, and most publicized of the transuranium elements. It is a metallic, radioactive element with atomic number 94, and was the first man-made element to be produced in relatively large quantities. Its primary use to date has been as fissile material in nuclear weapons, but an important potential use may be nuclear electric power generation. It is produced in all nuclear reactors and is a significant component of spent uranium fuels.

At present, americium and curium are the only other transuranium elements produced in sufficiently large quantities to be of interest as a potential environmental hazard.

Table 1.1

Nuclear Properties of Environmentally Significant Transuranium Nuclides			
Radionuclide	Mode of Decay	Physical Half-life	Daughter Product
Pu-238	α	87.4 y	U-234
Pu-239	α	2.4×10^4 y	U-235
Pu-240	α	6.6×10^3 y	U-236
Pu-241	β	14.3 y	Am-241
Pu-242	α	3.9×10^5 y	U-238
Am-241	α	433 y	Np-237
Am-243	α	7.4×10^3 y	Np-239
*Cm-242	α	0.45 y	Pu-238
Cm-244	α	18.1 y	Pu-240

* Included for reference because of potential quantities available for release. Not significant for environmental exposure considerations because of short persistence.

1.2 Current Sources of the Transuranium Elements

Present levels of the transuranium elements in the environment have resulted from several sources - regional and worldwide fallout from the testing of nuclear weapons in the atmosphere, accidents involving military and related operations, and local releases from nuclear facilities. The major portion of the transuranium elements in the environment is the result of surface and atmospheric nuclear weapons tests during the period 1945-1963. Atmospheric tests injected radioactivity into the stratosphere which has since then been slowly deposited more or less uniformly over the lands and oceans of the earth. As a result of these earlier weapons tests, the existing level of transuranium element contamination in soils of the United States is about 0.002 microcurie per square meter ($\mu\text{Ci}/\text{m}^2$). More recent weapon tests have not added significant amounts to this level. Isolated sites used by the United States to test nuclear devices, such as those in Nevada and the Bikini and Enewetak atolls, often have significantly higher amounts of the transuranium elements in small areas near the detonation point. Underground nuclear tests also have produced localized radioactive contamination, but this is contained below the earth's surface and is not expected to be readily available for uptake by humans.

Areas where there is substantial localized contamination above the general background level are well documented and extensive environmental analyses have been carried out at all these sites. Areas of highest contamination are, for the most part, on Federally owned property and therefore are under the direct control of the Federal government

and access to these areas may be restricted. Table 1.2 shows estimates of the amount of plutonium in the environment at known United States locations. More detailed information on the sources and current levels of the transuranium elements in the general environment is given in Annex I.

1.3 Movement through the Biosphere

Plutonium and other transuranium elements can move through the environment by a variety of transport mechanisms and pathways. These are determined by the chemical and physical form of the deposited material, the characteristics of the surface, local land use patterns, and other parameters such as wind or rainfall. Principal environmental pathways to humans are indicated in Fig. 1.

The principal modes of transport of these elements from a source to man are by direct airborne movement from the source (source-air-humans) or by resuspension of previously deposited small particles by the action of wind or other disturbance (soil-air-humans). Transuranium elements released to the environment may exist as discrete particles or they may become attached to soil particles. Resuspension is a complex phenomenon affected by a number of factors, including the characteristics of the surface, vegetative cover, meteorological conditions, and age of the deposit. The resuspension factor is defined as the ratio of the concentration in air ($\mu\text{Ci}/\text{m}^3$) at a given height above the surface to the average immediately adjacent surface contamination level ($\mu\text{Ci}/\text{m}^2$). Observed resuspension factor values range from about 10^{-5} to 10^{-11} per meter for a variety of conditions and sites. In

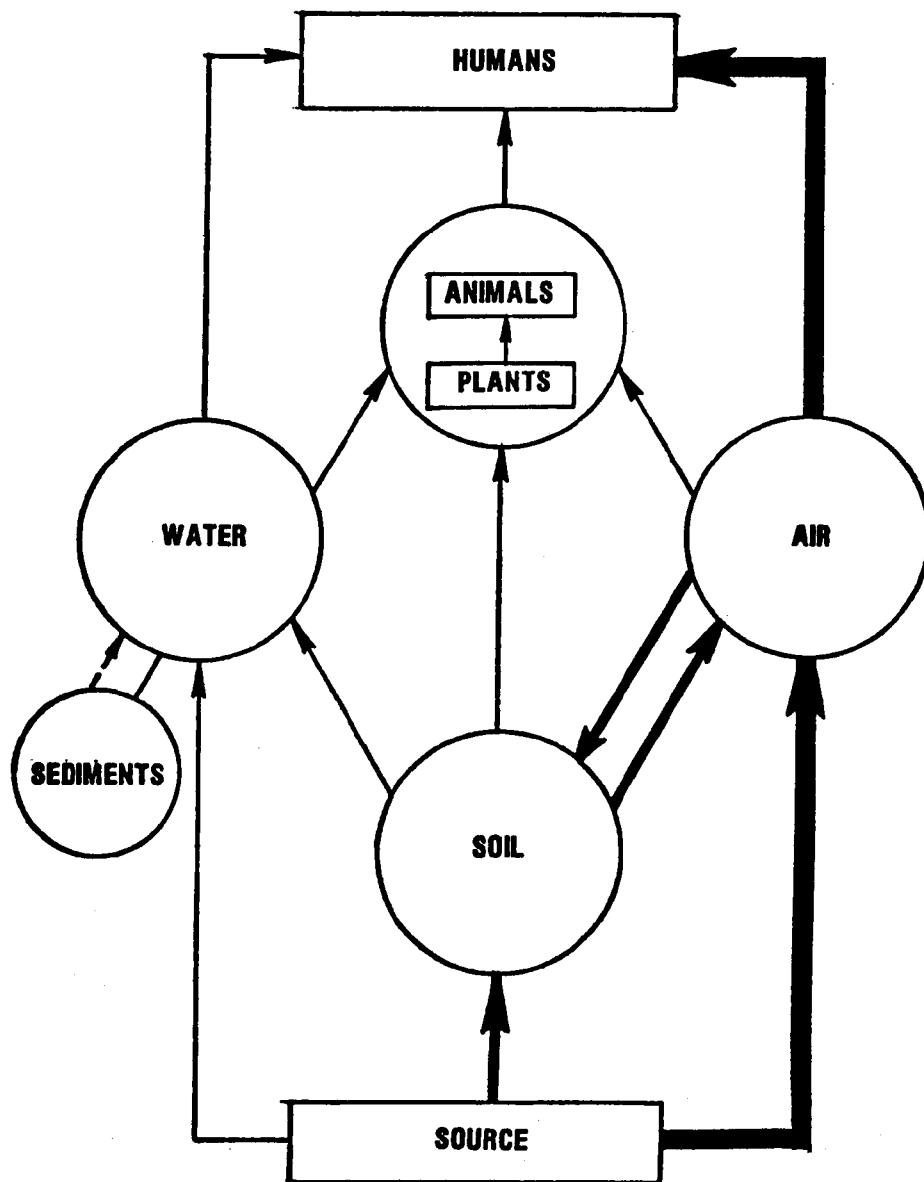
TABLE 1.2

Inventory and Levels of Plutonium for Selected U.S. Sites*

Location	Approximate Inventory	Soil Concentration** (above background)	Remarks
U.S. (Fallout)	~ 20,000 Ci	ave. = 0.06 pCi/g	
Nevada Test Site (near Las Vegas, NV)	> 155 Ci	Area 13: to 18,000 pCi/g Area 5: to 12,000 pCi/g off-site: 0.1-10 pCi/g	Surface and subsurface tests
Rocky Flats Plant (Jefferson County near Denver, CO)	8-10 Ci	on-site max > 1,000 pCi/g boundary: 0.1-4 pCi/g	Limited cleanup in progress
Mound Laboratory (Miamisburg near Dayton, Ohio)	Pu-238 = 5-6 Ci	off-site sediments max > 1000 pCi/g soils: 0.003-2 pCi/g	Sediments under water in canals
Savannah River Plant (SW part S. Carolina near Augusta, GA.).	3-5 Ci	N area: 30 pCi/g F area: 3 pCi/g perimeter: 0.02 pCi/g	Plutonium and higher isotopes production
Los Alamos Scientific Lab (NW of Santa Fe, NM)	1-2 Ci	on-site: 0.02-5 pCi/g off-site: 0.005-1 pCi/g	High local levels above 100 pCi/g in remote canyons
Hanford Site (Central Washington near Richland)	-	perimeter: 0.01-0.04 pCi/g	High levels in trenches on site
Oak Ridge Facilities (East Tennessee near Knoxville)	-	perimeter: 0.01-0.08 pCi/g	Research & Development facility
Trinity Site (near Alamogordo, NM)	~ 45 Ci	perimeter: ~ 55 pCi/g off-site: 0.1-4 pCi/g	Site of first atomic bomb test

* See Annex I for details and references

** 1 pCi/gram of soil = $0.015 \mu\text{Ci/m}^2$ for an assumed soil density of 1.5 g/cm^3 and a depth of 1 cm.



**PRINCIPAL PATHWAYS OF THE TRANSURANIUM ELEMENTS
THROUGH THE ENVIRONMENT TO MAN**

Figure 1

general, the resuspension factor will be relatively high immediately after initial deposition, gradually decrease with time, and approach a long-term constant within about one year after deposition.

Transport of plutonium and other transuranium elements through the food chain and subsequent ingestion is generally of lesser importance than the air pathway. Environmentally distributed transuranium elements may be deposited on plant surfaces or assimilated through the plant root system. The uptake by plants is relatively small and most animals, including humans, have a high discrimination factor against transfer of these elements into body tissues. The solubility of plutonium in water is very low and nearly all plutonium released into lakes and streams is ultimately deposited and sorbed onto sediments. Direct ingestion of contaminated soils and contamination of wounds are other possible routes of entry into humans, but are generally of minor importance relative to the inhalation and food pathways.

The environmental transport of the transuranium elements is discussed in more detail in Annex II.

1.4 Potential Health Effects

The potential health effects caused by a specific nuclide of a transuranium element are a function of several biological and physical parameters including the biological retention time in tissue, the type of radioactive emission, and the half-life of the nuclide. For the more important transuranium nuclides, such as Pu-238 or Pu-239, biological retention times are very long and radioactive decay occurs at such a

slow rate that uptake of these materials in the humans body will result in prolonged exposure of body organs. Many of the transuranium nuclides decay by emission of an alpha particle (ionized helium atom), in a manner similar to radium and other naturally occurring alpha emitting nuclides. Alpha particles are highly ionizing, and damaging, but their penetration in tissue is very small (about 40 μm). Thus, biological damage is limited to tissue in the immediate vicinity of the radioactive material, and a potential health hazard from transuranium elements in the environment can only result when these materials are inhaled or ingested into the body. Exposure of the unbroken skin from external alpha emitting sources is not a health problem.

Even though there is no direct evidence of cancer induction in humans due to the inhalation of transuranium elements, data from animal studies with these radionuclides and from human experience with other alpha-emitting elements such as radon daughters indicate that the inhalation of transuranium elements could cause lung cancer and other forms of cancer in humans. Cancer induction is not the inevitable result of such inhalation, but rather the result of this intake is an increase in the probability that a cancer may occur in the individual.

Inhaled particles are initially deposited in various regions of the respiratory tract, where they remain until either cleared or translocated to other body organs. Much of the material deposited in the lungs is cleared within a few days, but some of the smaller particles which diffuse into the pulmonary regions of the lung are removed much more slowly and have a biological half-life of a year or more. Estimates of

the risk of lung cancer due to the inhalation of transuranium elements are based on the alpha particle dose delivered to the pulmonary tissue, which is the portion of the lung receiving the highest dose as a result of inhalation. This is likely to overestimate the risk because the pulmonary region is not considered a probable site for induction of cancers in humans. In a recent study the National Academy of Sciences has concluded that the probability of cancer induction increases in proportion to the dose delivered to various lung tissues and that the incidence of cancer will not be underestimated by averaging the total alpha energy imparted over the mass of the pulmonary region (1).

Inhaled transuranium elements may also transfer and be retained in other body organs, and cause cancers of the bone and liver. For the less soluble transuranium compounds, such as plutonium oxide, this will contribute only marginally to the total risk for the inhalation pathway. Inhaled radioactivity is also concentrated in the respiratory lymph nodes. The dose delivered to the lymph nodes exceeds that to pulmonary tissues but, if a risk exists, animal studies indicate that it is small compared to the risk due to cancer in pulmonary tissue.

Ingestion of transuranium elements generally represents a smaller environmental risk to humans than inhalation. A relatively small fraction of any ingested transuranium element will be transferred by the bloodstream from the digestive tract and deposited in bone, liver, gonadal tissue, and other organs. In most cases, less than one-tenth of a percent of the ingested material is absorbed by the body, with the remainder excreted. The cancer risk to individuals as a result of

ingestion of transuranium elements is mainly due to potential bone and liver cancers.

Because of possible accumulation in gonadal tissues a potential risk of genetic damage to the progeny of exposed individuals exists as a result of exposure to transuranium elements. At the dose limits recommended in this guidance, this risk is very small compared to the natural incidence of genetic damage.

Preferred models for calculating the dose to pulmonary tissue, bone, gonadal tissue, and to the other organs as a result of inhalation or ingestion of transuranium elements are described in Annex III. In general, these dose models are based on publications prepared by the International Commission on Radiological Protection (ICRP) (2-4), supplemented by more recent data. In conjunction with estimates of the cancer risk due to alpha particle irradiation, these models are used to estimate the risk from radiation doses to specified body organs.

Section 2
Federal Guidance

2.1 Agency Action and Authorities

The Agency announced in the Federal Register on September 23, 1974, that it intended to review the current and projected environmental impact of the transuranium elements and consider whether guidelines or standards under its statutory authorities were needed to assure adequate protection of the health and safety of the general public (5). Subsequently, public hearings were held in Washington, D.C., on December 10-11, 1974, and in Denver, Colorado, on January 20, 1975, to permit interested citizens and organizations to present both technical evidence and opinions pertinent to this subject (6). Prior to this action the Agency had been considering the control of plutonium in soil as a result of a specific request during 1972 from the State of Colorado in regard to utilization of land in the vicinity of the ERDA Rocky Flats Plant. As an interim measure, Colorado adopted in 1973 a plutonium activity limit of 2 dpm/g in the top 1/8 inch of soil as a guide for protection of construction workers engaged in home building.

The Agency concluded that Federal Radiation Guidance was needed to control the potential health impact of plutonium and other alpha-emitting transuranium elements in the environment and that promulgating Federal Guides under the authority of the former Federal Radiation Council was

the appropriate procedure. This authority was transferred to the Administrator of the Environmental Protection Agency (EPA) by the President's Reorganization Plan No. 3 of 1970, (7) and included all the functions of the Federal Radiation Council, as specified in the Atomic Energy Act. Section 274(h) of this Act provides that "the Administrator shall advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation within States." Federal Guides developed under this authority and approved by the President are considered to be an adequate means to limit any problems of environmental contamination by the transuranium elements, since all sources of transuranium elements are under direct control of the Federal government. These Guides are therefore directed to all Federal agencies having regulatory or administrative control of transuranium elements. However, the Guides can also be viewed as advice by State and local governments.

2.2 Involvement of Other Federal Agencies

The implementation of these Federal recommendations is the responsibility of those agencies having regulatory and administrative responsibilities for the production, utilization, and control of transuranium elements, especially plutonium. Therefore, the Administrator established an Interagency Working Group to insure the availability of technical expertise and interagency coordination in the development of the Guides. This Interagency Working Group consisted of representatives from the Energy Research and Development Administration,

Nuclear Regulatory Commission, National Aeronautics and Space Administration, and the Departments of Defense, State, Commerce, Interior, and Health Education and Welfare. Although EPA has been primarily responsible for the development of these recommendations and supporting documents, this group has provided valuable assistance and has made available to the EPA both the expertise and viewpoints of these agencies.

2.3 Existing Standards and Guides

Several specific standards or guides have been published (8-12) pertaining to the presence of the transuranium elements in soil, to the controlled release of such materials into the environment (e.g., the EPA standard for the uranium fuel cycle*), and the decontamination of equipment surfaces. With the exception of EPA's uranium fuel cycle standards, such guides are based for the most part, on recommendations on numerical limits by the International Commission on Radiological Protection (ICRP) and by the National Council on Radiation Protection and Measurements (NCRP). Recommendations by the ICRP and the NCRP include numerical limits on air and water concentrations applicable to individuals in the general population.

Department of Defense guidelines have been developed to deal with instances of accidental surface contamination and represent ad hoc guidance for accidents involving nuclear weapons. The decontamination

* Note: EPA published (F.R. 42:2858, Jan. 13, 1977) as title 40; Part 190, Environmental Standards for the Uranium Fuel Cycle, normal operations, a limit of 0.5 millicuries combined of plutonium-239 and other alpha-emitting transuranium radionuclides with half-lives greater than one year entering the environment as planned releases per gigawatt-year of electrical energy produced by the fuel cycle.

guidance prescribes that surface levels of less than $1000 \mu\text{g}/\text{m}^2$ plutonium (numerically equal to $61 \mu\text{Ci}/\text{m}^2$ for Pu-239) shall be achieved where such reduction is possible and is consistent with reasonable cost and effort. Each of the Armed Services has also developed more detailed implementation manuals.

For case of surface contamination of materials for shipment and of transport vehicles, the Department of Transportation specifies as "not significant", a level of $220 \text{ dpm}/100 \text{ cm}^2$ ($0.01 \mu\text{Ci}/\text{m}^2$) of removable alpha contamination. There is also a recommendation by the Nuclear Regulatory Commission that the average level of alpha contamination for reactor equipment to be released to the general public be no more than $100 \text{ dpm}/100 \text{ cm}^2$.

Recommendations have also been published by individuals for the specific situation of soil contamination by transuranium elements, but none of these have been adopted by government agencies (13-15).

2.4 Criteria and Rationale for the Proposed Guidance

The purpose of the proposed guidance is to establish maximum dose rates for persons in the general population who might receive radiation exposure to transuranium elements in the environment, which considers all possible pathways to humans and which the Agency judges to be protective of the public health. The two primary criteria used in determining the guidance recommendations were: that the added risk to an individual from exposure to the transuranium elements be very small, and that implementation of the guidance be feasible in terms of overall economic impacts. The Agency has also determined that the costs of remedial actions and the benefits of risk reduction differ so greatly

between sites, both for those where excess contamination already exists and for those where accidents might occur in the future, that generic guidance applicable to all sites cannot be provided by formal benefit-cost procedures.

The primary purpose of the guidance is to achieve a level of public health protection which will minimize the risk to exposed individuals. Radiation induced risks can be estimated in a number of ways, but the most prudent methods assume that there is some finite risk to humans no matter how small the amount of absorbed radiation might be and that this risk at any given dose level is directly proportional to the damage actually observed at much higher dose levels. Such a calculation, in the absence of data to the contrary, must be considered as the method appropriate for a regulatory or guidance function. On this basis, there is no level of radiation exposure which is absolutely safe and any radiation dose carries with it some degree of risk. Balanced against this is the realization that all persons are exposed to a large number of competing risks (including natural background radiation) and the reduction of a single risk must be viewed from the overall perspective of the costs and benefits to society.

The guidance recommendations are intended to achieve adequate health protection for the small fraction of the total population at greatest risk from exposure to transuranium nuclides in the environment, and will therefore offer much greater protection to the vast majority of the population at lesser risk. The numerical guidance can be related to a maximum risk level to an individual which is comparable to that used

for other carcinogens and lower than that for many other competing risks. The level of risk at the proposed guidance level, estimated by the above method, is less than one chance per million per year and less than ten chances per hundred thousand in a lifetime that an individual would develop a cancer from continuous exposure at the stated dose rates. Actual exposures and risks to individuals are expected to be well below this level. It must be recognized that these risk estimates are not precise but represent the best judgment of the Agency. There may be differences in reputable scientific opinions on their accuracy.

Other regulatory actions have considered lifetime risk levels for carcinogens but there is, at this time, no uniformity of approach to the regulation of carcinogens. The Food and Drug Administration (FDA) uses a lifetime risk level of one per million as "virtually safe," and has proposed to ban saccharin because, for an average consumption, the lifetime cancer risk was estimated to be 10/100,000. EPA has recommended an action level for kepone in fish for which the associated lifetime cancer risk is 30/100,000. Differences in the action levels reflect different regulatory requirements, constraints associated with remedial actions for a particular carcinogen, uncertainties in the calculations, differences in methods used to derive lifetime risks, and economic considerations. The proposed guidance is intended to be viewed from the perspective of providing a limit applicable to remedial and restorative actions, and must be distinguished from other risk levels

which provide for a routine level of acceptability.

The Agency has also considered the costs which may be involved in implementing the proposed guidance. These costs were estimated from soil contamination levels at existing sites of contamination. The magnitude of areas which might require remedial actions at each site has been estimated for a soil concentration contour which can with a very high probability be expected to result in an inhalation dose rate to an individual not to exceed 1 mrad/year, as well as for soil concentration contours higher and lower than the reference case by factors of ten. Changes by factors of ten are judged to represent the limit of precision available for such calculations and are considered sufficient for purposes of a generic assessment. However, in order to attempt to more closely define where costs of implementation would begin to rise rapidly, an intermediate contour of one-third the reference level was also considered. The costs of implementing the guidance can be expected to vary by location, contamination level, and other factors. A minimum cost of \$500 per acre has been assumed for estimating the costs which may be incurred in bringing all areas above the designated level into compliance. Such costs include dilution or removal of contamination by plowing or scraping as required and restorative actions needed to prevent erosion and assist ecological recovery. Costs necessarily increase as the difference between the existing contamination levels and those sought becomes greater. On this basis, costs of

remedial actions have been estimated for those few sites in the United States where some environmental contamination extends beyond the boundaries of the source from where it originated, and are shown in Table 2.1. It must be recognized that there are large uncertainties associated with both the areas involved and in the estimates of costs. Nonetheless, the calculations serve a useful purpose of comparison on a common basis. It can be concluded that the costs of implementing the guidance at the reference level would be reasonable and achievable, but that the cumulative costs increase rapidly when more restrictive limits are considered.

The Agency has recognized a difference between those sites presently contaminated and sites that may be accidentally contaminated in the future, and has considered whether separate numerical guides should be developed for these two cases. In the first case the judgment is between continued exposure to these elements at some given level and the cost of remedial action to reduce exposures. In the second case, the risk of possible future accidental contamination events must be taken into account in the initial decision to perform any activity where there is risk of accidental contamination, and include the entire cost of possible future remedial actions that might be required in event of an accidental release. However, no specific rationale is known that would justify different guidance based on health risk considerations. Therefore, the Agency recommends that the same numerical guide be used in both cases, but that all future contamination be cleaned up as soon as possible after occurrence to a level as low as reasonably

Table 2.1

Comparison of Costs of Remedial Actions At Various Sites
of Existing Plutonium Contamination For Several Possible
Levels of Maximum Soil Concentrations (Areas are Estimated
from Contour Maps and Costs Are Arbitrarily Assumed as \$500/acre)

	Reference Level $0.2 \mu\text{Ci}/\text{m}^2$		10 x Ref. Level $2 \mu\text{Ci}/\text{m}^2$		1/3 Ref. Level $0.07 \mu\text{Ci}/\text{m}^2$		1/10 x Ref. Level $0.02 \mu\text{Ci}/\text{m}^2$	
	Area	Cost	Area	Cost	Area	Cost	Area	Cost
Rocky Flats Plant	0	0	0	0	0.3 mi^2	100K	$\sim 1.6 \text{ mi}^2$	500K
Nevada Test Site	0	0	0	0	$<80 \text{ mi}^2$	25M	$< 165 \text{ mi}^2$	50M
Trinity Site	0	0	0	0	$<20 \text{ mi}^2$	$<6\text{M}$	$\sim 300 \text{ mi}^2$	100M
Mound Lab.	$\sim 0.01 \text{ mi}^2$	*	$\sim 0.01 \text{ mi}^2$	*	$\sim 0.01 \text{ mi}^2$	*	$\sim 0.01 \text{ mi}^2$	*

* Most of the existing contamination is in sediments of canals, and does not represent a hazard to humans. Costs of eventual remedial actions are indeterminate.

achievable and with the numerical guide as an upper limit.

In summary, the Agency has recognized that any radiation exposure is potentially harmful and has concluded that the guidance recommendations should limit the risk to those persons in the critical segment of the population to a level as low as reasonably achievable within the constraints of economic considerations. There is at present no uniformity of approach to the regulation of carcinogens among the various Federal agencies. It is the judgment of the Agency that for presently existing sites of contamination, the proposed guidance recommendations will provide the necessary protection of the public health and be achievable at reasonable costs. It is also the judgment of this Agency that, in some cases of accidental contamination of the environment by the transuranium elements, cost considerations will permit reduction of residual contamination to levels below those specified in this guidance. In all cases, the guidance recommendations represent a maximum value and further reduction should be sought when this can be accomplished at a cost judged to be reasonable in terms of alternatives available.

2.5 Text of Proposed Guidance

The Environmental Protection Agency proposes that the following recommendations be submitted by the Administrator to the President for issuance as Federal Radiation Guidance:

1. The annual alpha radiation dose rate to members of the critical segment of the exposed population as the result of exposure to

transuranium elements in the general environment should not exceed either:

- a. 1 millirad per year to the pulmonary lung, or
- b. 3 millirad per year to the bone.

2. For newly contaminated areas, control measures should be taken to minimize both residual levels and radiation exposures of the general public. The control measures are expected to result in levels well below those specified in paragraph one. Compliance with the guidance recommendations should be achieved within a reasonable period of time.

3. The recommendations are to be used only for guidance on possible remedial actions for the protection of the public health in instances of presently existing contamination or of possible future unplanned releases of transuranium elements. They are not to be used by Federal agencies as limits for planned releases of transuranium elements into the general environment.

2.5.1 Definitions

- a. "critical segment of the exposed population" means that group of persons within the exposed population receiving the highest radiation dose to the pulmonary region of the lung or to the bone.
- b. "rad" is the unit of absorbed dose, defined as the energy imparted to tissue due to ionizing radiation divided by the mass of the tissue. One rad is equal to the absorption of 100 ergs of radiation energy per gram of matter.
- c. "pulmonary lung" means that region of the lung consisting of respiratory bronchioles, alveolar ducts, atria, alveoli, and alveolar

sacs. The average total weight of this tissue, including the capillary blood, is assumed to be 570 gms.

d. "millirad per year to the pulmonary lung" means the equilibrium dose rate following chronic inhalation. This dose rate is calculated by dividing the alpha energy absorbed per year in the pulmonary lung by its mass.

e. "bone" means osseous tissue. The average total weight of this tissue is assumed to be 5000 gms.

f. "millirad per year to the bone" means the dose rate attained after 70 years of chronic exposure. This dose rate is calculated by dividing the alpha energy absorbed in the bone during the 70th year by the bone mass.

g. "general environment" means the total terrestrial, atmospheric and aquatic environments outside the boundaries of Federally licensed facilities or outside the boundaries of sites which are under the direct control of a Federal agency.

h. "curie (Ci)" is the basic unit to describe the intensity of radioactivity in a material. It is equal to 37 billion disintegrations per second.

$$1 \text{ millicurie (mCi)} = 10^{-3} \text{ Ci}$$

$$1 \text{ femtocurie (fCi)} = 10^{-15} \text{ Ci}$$

Section 3

Implications of Guidance

3.1 Scope of Sources and Population Groups Included

The objective of the proposed guidance is to assure that the public health and welfare will be adequately protected from the consequences of environmental contamination by the transuranium elements.

The proposed guidance is not intended to supersede existing radiation protection guides, such as the individual and population limits established by the Federal Radiation Council in 1960 (17), but rather to supplement these by specifying limits for one type of source and for one group of radionuclides within these broader limits. The scope of the proposed guidance includes all transuranium element contamination in the general environment from all sources. The recommendations are applicable to all individuals in the general population outside the boundaries of a Federal facility, Federally licenced facility, or other site under the direct control of a Federal agency.

The recommendations are expressed in terms of an annual limiting dose commitment to the pulmonary region of the lung or to the bone. It should be noted that, although the proposed guidance specifies dose rate limits to only certain organs, this also limits the potential accumulation in gonadal tissues and the attendant genetic risk. The limits apply to the critical segment of the exposed population, which is that group of persons in the general population who, because of residency, occupation, or other factors can on the average be expected to receive

the highest lifetime radiation dose from a specified source of transuranium elements.* The annual dose rate to the designated organs can be estimated from representative measurements of air concentrations or soil contamination levels, either by use of site-specific data or by use of calculations based on reasonable procedures and assumptions.

3.2 Risk Perspectives

Exposure to radiation may increase somatic risks, primarily that of cancer, as well as genetic risks to future generations. The somatic risks to persons caused by exposure to small amounts of radiation can best be viewed in terms of the probability of death to an individual when he is considered as a member of a large exposed population group. Although the genetic risks to humans as a result of the assimilation of transuranium elements cannot be quantified with exactness, these risks may be estimated in terms of the number of expected genetic defects per 100,000 live births where both parents are assumed to have accumulated a given gonadal dose. The development of this guidance is based on suitable models chosen by the Agency that relate environmental levels to the dose to internal organs. Health risks resulting from radiation exposure were estimated using models and recommendations of the Advisory Committee on the Biological Effects of Ionizing Radiation of the National Academy of Sciences (NAS-BEIR Committee) in its reports entitled "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation"

* The dose received during childhood, and any differences in radiosensitivity between children and adults assumed by the NAS, have been taken account of in the derivation of these guides. Because a lifetime radiation dose is, for the most part, received during maturity, adults are defined in this guidance as the critical population group.

(1972), and "Health Effects of the Alpha-Emitting Particles in the Respiratory Tract" (1976) as well as information in other technical reports.

The radiation risk due to inhalation of transuranium elements is primarily lung cancer. Additional risks may result from translocation of a small fraction of the transuranium elements from the lung to other body organs, especially to the liver, bone, and gonadal tissues. On the basis of models, it was estimated that, for a cohort of 100,000 persons* followed through their entire lifetimes, the continuous inhalation over their lifetimes of transuranium aerosols leading to an average annual dose rate to the pulmonary tissue of 1 mrad/year per person could potentially result in 10 premature deaths. For an average lifespan of 71 years, the annual risk to each person from lifetime exposure at this level is about 1×10^{-6} per year, and the estimated number of additional premature deaths would represent an increase of less than 0.1 percent of the current risk of death due to all cancers.

The lifetime risk due to ingestion of transuranium elements is due both to cancer mortality and an increased genetic risk. In a cohort of 100,000 persons, continuous ingestion over a lifetime of a transuranium radionuclide at a level causing an average skeletal (bone) dose rate of 1 mrad per year 70 years after the start of ingestion is estimated to result in less than 2 premature deaths from bone and liver cancer. In

* Epidemiological studies are generally based on results for a cohort of 100,000 persons. This number is arbitrarily large and bears no relation to the number of individuals expected to be exposed to the maximum recommended dose rates of this guidance.

order to approximate the same risk for the ingestion pathway as was estimated for the inhalation pathway, the equivalent bone dose rate limit would be about 6 mrad per year after 70 years. However, as noted below, ingestion at this rate entails a greater genetic risk than that resulting from the proposed inhalation limit. Therefore, in order to have more comparable risks for either pathway, the recommended limiting dose rate to bone is proposed as 3 mrad per year in the 70th year after the start of ingestion. This could potentially result in 5 premature cancer deaths in the cohort under consideration.

Genetic damage is possible as a result of assimilation of trans-uranium elements in gonadal tissue. There is considerable uncertainty in the estimated dose to gonadal tissue and resultant genetic risk. On the basis of very limited data it can be estimated that, for continuous ingestion at the limits set by this guidance, the total dose to gonadal tissue over 30 years of chronic exposure would be about 10 millirad. Such a gonadal dose to each of the parents is estimated to produce 1 to 20 genetic effects per 100,000 live births in the first generation. This number can be compared to the approximately 6000 congenital abnormalities normally observed in 100,000 live births. Potential genetic risk to succeeding generations is not well known but, if the guidance dose rate limit were maintained, may range as high as 5 to 150 impaired individuals per 100,000 live births. The dose rate to gonadal tissue and the resultant genetic risk from continuous inhalation leading to the guidance limit is estimated to be about six times smaller than that estimated for continuous ingestion.

In practice, very few, if any, individuals are expected to be subjected to the recommended guidance limits and the total number of individuals exposed above the level of worldwide fallout will be small. The Agency also considered the total impact on population groups but, because the exposed population groups are small and are likely to remain so, it was deemed unnecessary to separately consider the environmental radiation dose commitment in developing this guidance.

The estimated lifetime risk from exposure to transuranium elements at levels equivalent to the guidance recommendations can be put in perspective by comparison with other presently experienced risks of death. These comparative risks have been derived from Life Table for the U.S. Population for 1970 (18) and specific mortality rates as published in Vital Statistics of the United States (19), both published by the National Center for Health Statistics. Risk estimates are based on a hypothetical cohort of 100,000 individuals with the race and sex distribution of the U.S. population. Cohort members are subjected to the same age specific mortality rates as are observed in the U.S. population, and followed from birth until all members of the cohort are dead. Impacts are derived in terms of number of premature deaths and ages at death. The number of adverse health effects attributed to radiation doses from transuranium elements are projected from models which assume a linear non-threshold hypothesis for the dose effect relationship. Therefore, the calculated risks may be overestimated, and no deaths from exposure to transuranium elements in the environment have been identified. In contrast, the number of deaths resulting from

diseases are based on actual data taken from existing mortality records.

On the basis of these analyses, the Agency has concluded that the added risks imposed on those persons who might be exposed to environmental transuranium concentrations at the guidance recommendation levels over a prolonged period of time are of the same order of magnitude as those for relatively rare events, such as fatalities from bites and stings and from electric current in home wiring and appliances. On the basis of comparative life shortening, however, the potential risk from transuranium elements in the environment would be smaller. Complete results of life table models and data for other causes of death investigated are given in Annex IV.

3.3 Implementation

Implementation of this guidance will require measurement of the ambient concentration level of transuranium elements in air, soil, food, or water. In most cases the critical pathway for exposure to transuranium elements is through inhalation of airborne particulates derived from resuspension of soil particles and compliance with this dose guidance can be demonstrated by measurements of air and/or soil concentration. Air concentration may be related to the dose guidance using dosimetry models that include consideration of particle size distribution and other parameters appropriate to the specific site of contamination. Because of short-term and seasonal meteorological variations, results of air concentration measurements are best based on the average of consecutive weekly samples over a period of at least one year.

Air measurements may not indicate the specific source of contamination, nor provide data for particular land areas of concern.

Therefore soil measurements may be more advantageous in certain cases. Such soil measurements are warranted when the air concentrations indicate that the dose guidance limits are exceeded. Air concentration measurements can be related to a corresponding soil contamination level by use of a resuspension factor, which is obtained either by experimental determination or by calculational techniques based on the mass-loading concept as described in Annex II.

For practical reasons of facilitating implementation of this guidance the Agency has derived a numerical value for a level of soil contamination which can reasonably be predicted to result in dose rates less than the guidance recommendations. On the basis of limited data available for several existing sites, the Agency suggests that a soil contamination level of $0.2 \mu\text{Ci}/\text{m}^2$, for samples collected at the surface to a depth of 1 cm and for particle sizes under 2 mm, would establish a reasonable "screening level" for this purpose. Use of such a numerical value can serve to reduce the land area requiring evaluation and minimize the number of measurements needed.

In some cases it may be prudent to specifically evaluate exposure from ingestion, although this is not normally expected to be the critical exposure pathway. The procedure suggested above does not explicitly consider such alternative pathways. However, if these are considered to be of sufficient importance from the viewpoint of human exposures, they must be included in the evaluation of a specific site.

Further information on implementation of these guides, and on the application of environmental measurements to demonstrate compliance with the recommendations, are discussed in Annex V.

3.4 Remedial Actions and Economic Evaluation

The recommendations of this guidance are primarily intended to serve as an indicator of the need for possible remedial actions to protect the public health. The guidance is stated in terms of a maximum allowable dose to a critical segment of the population occupying lands freely accessible to members of the general public. Remedial action may be required at any site that fails to meet these criteria. Economic evaluation is needed to determine the least cost method of achieving the recommendations.

Where a need for the use of remedial measures arises, two alternatives are available: 1) reduction or elimination of the source term or 2) establishing restricted access or use of the area in question. Remedial measures which reduce or remove existing surface contamination, in order to minimize the source term and subsequent transport of plutonium to humans, include:

1. In-place stabilization by the application of a relatively impermeable cover, such as oil, polymerized plastics, or asphalt
2. Dilution by plowing or other similar techniques
3. Disposal by removal of surface soils and burial either on-site or in a designated waste storage repository.

It can generally be expected that a variety of techniques can be used to achieve the guidance level at any site. The objective of an economic evaluation is to identify the technique or combination of techniques that attains the guidance level at the least total cost. Monetary costs, environmental costs, and other nonmonetary costs should

all be considered in the evaluation of each alternative combination of possible remedial actions.

Monetary costs include those for removal, stabilization and dilution of contaminated soils, radiological monitoring, protective measures for workers, and the maintenance of restricted areas if they are used. Generally these costs can be readily evaluated. Environmental costs, especially those of long-term degradation, must be evaluated to the extent possible. Nonmonetary costs, including such intangible factors as disruption of living patterns must be weighed in the evaluation. Whenever feasible, costs of alternative remedial actions should be evaluated monetarily or quantified in the best available nonmonetary units.

References

1. National Academy of Sciences - National Research Council: Health Effects of Alpha-Emitting Particles in the Respiratory Tract: Report of Ad Hoc Committee on "Hot Particles" of the Advisory Committee on the Biological Effects of Ionizing Radiation. Published by the Office of Radiation Programs, U.S. Environmental Protection Agency, Washington, D.C., Report No. EPA 520/4-76-013 (October 1976).
2. ICRU Report 25, 1976. Conceptual Basis for The Determination of Dose Equivalent, International Commission on Radiation Units and Measures, Washington, D.C.
3. ICRP Publication 19, 1972. The Metabolism of Compounds of Plutonium and Other Actinides, Pergamon Press, New York.
4. ICRP Publication 23, 1975. Report of the Task Group on Reference Man, Pergamon Press, New York.
5. U.S. Environmental Protection Agency: Plutonium and the Transuranium Elements - Contamination Limits: Intent to Review the Need for Establishing New Rules. Federal Register Vol. 38, p. 24098, Sept. 23, 1974.
6. U.S. Environmental Protection Agency - Office of Radiation Programs: Proceedings of Public Hearings: Plutonium and the Other Transuranium Elements (3 Volumes). ORP/CSD-75-1 (1975).
7. Presidential Documents. Federal Register Vol. 35, pp 15623-6 (Oct. 6, 1970).
8. International Commission on Radiological Protection: Report of Committee II on Permissible Dose for Internal Radiation - ICRP Publication 2 (1959).
9. National Committee on Radiation Protection and Measurements: Maximum Permissible Body Burdens and Maximum Permissible Concentrations of Radionuclides in Air and Water for Occupational Exposure - NCRP Report 22 (June 1959).
10. Atomic Energy Commission - Defense Atomic Support Agency: Plutonium Contamination Standards. AEC - DASA Publication TP 20-5 (May 22, 1968).
11. Department of Transportation: Chapter 49, Code of Federal Regulations 173.397 (1970).

12. U.S. Nuclear Regulatory Commission: Regulatory Guide 1.86: Termination of Operating Licences for Nuclear Reactors (June 1974).
13. C.E. Guthrie and J.P. Nichols: Theoretical Possibilities and Consequences of Major Accidents in U-233 and Pu-239 Fuel Fabrication and Radioisotope Processing Plants: Oak Ridge National Laboratory Document ORNL-3441 (April 1964).
14. R.L. Kathren: Toward Interim Acceptable Surface Contamination Levels for Environmental PuO_2 : Battelle Northwest Laboratories Document BNWL-SA-1510 (1968).
15. J.W. Healy: A Proposed Interim Standard for Plutonium in Soils: Los Alamos Laboratory Document LA-5483-MS (January 1974).
16. International Commission on Radiological Protection: Implications of Commission Recommendations that Doses be kept as Low as Readily Achievable. ICRP Publication 22 (April 1973).
17. Federal Radiation Council: Radiation Protection Guidance for Federal Agencies. Federal Register Vol. 25, pp 4402-3 (May 18, 1960).
18. U.S. Department of Health, Education, and Welfare, Public Health Service, U.S. Decennial Life Tables for 1969-71, Volume 1, Number 1, May 1975.
19. U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics. Excerpt from Vital Statistics of the United States 1969, Volume II-Mortality.

Annex 1

TRANSURANIUM ELEMENTS IN THE ENVIRONMENT

U. S. Environmental Protection Agency
Office Radiation Programs
Washington, D.C. 20460

Annex 1

Table of Contents

	<u>Page</u>
1. Introduction	1
2. The Nevada Test Site (NTS).....	9
3. Rocky Flats Plant (RFP).....	12
4. Mound Laboratory (ML).....	23
5. Savannah River Plant (SRP).....	28
6. Los Alamos Scientific Laboratory (LASL).....	38
7. The Trinity Site.....	42
8. Sites of Underground Nuclear Detonations.....	42
9. Enewetak Atoll.....	42
10. Other Sites.....	46
REFERENCES.....	58

Tables

A 1-1 Cumulative Deposit of Fallout Pu-239 at Selected Locations in the United States (3).....	3
A 1-2 Concentration of Fallout Pu-239 with Depth at North Eastham, Massachusetts (4).....	4
A 1-3 Fallout Pu-239 in New York City (6).....	5
A 1-4 Nuclear Properties of Environmentally Significant Transuranium Radionuclides (7).....	6
A 1-5 Inventories and Concentration Levels of Plutonium at Contaminated Sites.....	8
A 1-6 Estimated Inventory of Plutonium in Surface Soil (0-5 cm depth) at Specific Areas within the National Test Site and Tonopah Test Range (11).....	11
A 1-7 Pu-239 in Air Samples - Near the NTS (7).....	15
A 1-8 Plutonium Concentration in Ambient Air at Selective Locations - Rocky Flats Site (11).....	22
A 1-9 Concentrations of Plutonium and Americium in Water Supplies and in Finished Drinking Water - Rocky Flats Site (10).....	24
A 1-10 Concentration of Pu-238 in Environmental Media Mound Laboratory (10).....	29
A 1-11 Mound Laboratory U.S. Environmental Protection Agency 1974 Survey.....	33
A 1-12 Plutonium Concentration in Environmental Media Around the Savannah River Plant (10).....	37
A 1-13 Plutonium in Sediments in the Liquid Waste Receiving Canyons (20) - cy 1975.....	39
A 1-14 Plutonium and Americium in Environmental Media - LASL Site - (10) - CY 1975 -.....	40
A 1-15 Underground Testing Conducted Off the Nevada Test Site (10).....	44
A 1-16 Plutonium Concentration in Soil on Enewetak Atoll (22).....	47

	<u>Page</u>
A 1-17 Plutonium and Americium Concentration in Surface Air on Enewetak Atoll (14).....	48
A 1-18 Plutonium and Americium Concentrations in Various Environmental Media on Enewetak Atoll (14).....	49
A 1-19 Environmental Monitoring for the Transuranium Elements at the Pantex Plant Site.....	50
A 1-20 Environmental Monitoring for the Transuranium Elements at Argonne National Laboratory.....	52
A 1-21 Environmental Monitoring for the Transuranium Elements at Battelle-Columbus Laboratories (West Jefferson Site).....	53
A 1-22 Environmental Monitoring for the Transuranium Elements at the Idaho Engineering Laboratory.....	54
A 1-23 Environmental Monitoring for the Transuranium Elements at the Oak Ridge Facilities.....	55
A 1-24 Environmental Monitoring for the Transuranium Elements at Hanford.....	56
A 1-25 Environmental Monitoring for the Transuranium Elements at the Lawrence Livermore Laboratory.....	57

Figures

	<u>Page</u>
A 1-1 Population Distribution by Azimuth and Distance.....	10
A 1-2 Cumulative Deposit of Pu-239, 240 (mCi per km ²).....	13
A 1-3 Cumulative Global Fallout Deposit of Pu-239, 240 (mCi per km ²).....	14
A 1-4 Wind Rose for the Rocky Flats Site (10).....	16
A 1-5 Population Distribution Around Rocky Flats.....	18
A 1-6 Rocky Flats 1974 Plutonium Concentrations in Soil (Values in Picocuries Per Gram. (10).....	20
A 1-7 Rocky Flats Plutonium-239 Contours mCi/km ² (11).....	21
A 1-8 Population Distribution Around Mound Laboratory (0 to 10 km) LAT 39.6305 LONG 84.2897).....	25
A 1-9 Mound Laboratory Preliminary Estimate of Plutonium-238 Airborne Deposition (mCi/km ²) (10).....	27
A 1-10 Mound Laboratory.....	32
A 1-11 Savannah River Plant Plutonium Deposition (10).....	36
A 1-12 Trinity Site 1973-1974 Plutonium Soil Sampling Results (n Ci/m ²) (14).....	43

Annex 1

TRANSURANIUM ELEMENTS IN THE ENVIRONMENT

1. Introduction

Plutonium and other transuranium elements have been released into the general environment primarily from four sources. In order of quantities released from most to least, these are:

- a. Aboveground testing of nuclear weapons
- b. Accidents involving nuclear weapons and satellite power sources
- c. Accidents at nuclear facilities
- d. Planned discharges of effluents from nuclear facilities

The aboveground testing of nuclear weapons during 1945-1963 is responsible for a worldwide dispersal of plutonium and americium. For the most part, this radioactivity was injected into the stratosphere and has been redeposited more or less uniformly over the earth's lands and waters. This redeposited plutonium and americium is available to people through inhalation and ingestion pathways and exists as a ubiquitous source in the general environment upon which is superimposed the releases of transuranic elements from other sources.

Fallout plutonium is primarily a mixture of Pu-239 and Pu-240 with lesser amounts of Pu-238, Pu-241 and Pu-242. About 58% is Pu-239 and 39% Pu-240; because these two radionuclides are essentially identical with respect to chemical behavior and alpha energies, the sum of their activities in the environment is will be referred to in this Annex as "Pu-239" (1). The daughter of Pu-241 ($t_{1/2} = 12y$) is Am-241, so that as the Pu-241 continues to decay, the concentration of Am-241 in the

environment increases relative to the amount of Pu-239. The Am-241/Pu-239 activity ratio in soil is now about 0.25 and will eventually increase to 0.40 (2).

Aboveground nuclear weapons testing produced approximately 430 kilocuries of Pu-239 over the period 1945 to 1974. About 105 kilocuries deposited quickly near the various detonation sites. Of the 325 kilocuries injected into the stratosphere, 250 kilocuries have deposited in the mid latitudes of the northern hemisphere, 70 kilocuries have deposited in other latitudes and about 5 kilocuries still remained in the stratosphere as of 1974. This has led to cumulative depositions of Pu-239 on ground surfaces in the United States that range from 0.001 to 0.003 $\mu\text{Ci}/\text{m}^2$. Since 1967, sporadic aboveground nuclear tests have held the air concentration level of plutonium to relatively constant values, currently ranging from 0.01 to 0.1 fCi/m³ in ground level air. These levels are not believed to be the result of resuspension from soil surfaces (1, 3).

Table A 1-1 gives the cumulative deposition of fallout Pu-239 at selected locations in the United States (3). Table A 1-2 shows how it is distributed in soil with respect to depth at both an undisturbed site and a cultivated site (4,5). Table A 1-3 is a summary of fallout Pu-239 levels in New York City for both air and ground deposition as a function of time from 1954 to 1975 (6). Table A 1-4 is a listing of the nuclear properties of the more significant transuranium nuclides (7).

The total amount of Pu-238 injected into the stratosphere from aboveground nuclear tests is about 9 kilocuries (3). In addition, 17

Table A 1-1

Cumulative Deposit of Fallout Pu-239 at Selected
Locations in the United States (3)

<u>Approximate Location</u>	<u>Pu-239 Concentration (a)</u> <u>($\mu\text{Ci}/\text{m}^2$)</u>
Richland, Washington	0.0014
San Francisco, California	0.0009
Los Angeles, California	0.0007
National Test Site, Montana	0.0019
Rapid City, South Dakota	0.0025
Topeka, Kansas	0.0024
Tulsa, Oklahoma	0.0022
Corpus Christi, Texas	0.0010
Chicago, Illinois	0.0021
Augusta, Maine	0.0017
Cape Cod, Massachusetts	0.0023
Long Island, New York	0.0024
Raleigh, North Carolina	0.0024
Miami, Florida	<u>0.0010</u>
	Average ($\pm 2\sigma$) <u>0.0018</u> \pm 0.0006

(a) Top 30 cms of soil

Table A 1-2

Concentration of Fallout Pu-239 in Soil as a
Function of Depth at North Eastham, Massachusetts (4)

<u>Depth (Cm)</u>	<u>Undisturbed Site</u>	<u>% of Total</u>
	<u>Concentration of Pu-239</u> <u>($\mu\text{Ci}/\text{m}^2$)</u>	
0-2 (Includes Vegetation) (a)	0.91×10^{-3}	52 (a)
2-4	0.37	21
4-6	0.15	8
6-8	0.12	7
8-10	0.052	3
10-12	0.035	2
12-14	0.028	2
14-16	0.027	1
16-21	0.047	3
21-26	<0.004	<1

<u>Depth (Cm)</u>	<u>Cultivated Site⁽⁵⁾</u>	<u>% of Total</u>
	<u>Concentration of Pu-239</u> <u>($\mu\text{Ci}/\text{m}^2$)</u>	
0-5	0.42×10^{-3}	19
5-10	0.45	20
10-15	0.34	16
15-20	0.37	17
20-25	0.32	15
25-30	0.20	10
30-35	0.02 ± 0.03	0.01
35-40	0.02	0.01
40-45	0.02	0.01
45-50	0.02	0.01
50-55	0.02	0.01

(a) 12% of the total plutonium was associated with vegetation

Table A 1-3

Fallout Pu-239 in New York City (6)

Year	Deposition ($\mu\text{Ci}/\text{m}^2$)	Cumulative deposit ($\mu\text{Ci}/\text{m}^2$)	Surface air concentration (fCi/m^3)
1954	0.07×10^{-3}	0.00007	0.14
1955	0.09	0.00016	0.18
1956	0.12	0.00028	0.23
1957	0.12	0.00040	0.23
1958	0.16	0.00056	0.32
1959	0.23	0.00078	0.45
1960	0.04	0.00082	0.081
1961	0.06	0.00089	0.13
1962	0.32	0.0012	0.63
1963	0.62	0.0018	1.68
1964	0.41	0.0022	0.91
1965	0.14	0.0024	0.33
1966	0.05	0.00245	0.12
1967	0.04	0.0025	0.051
1968	0.04	0.0025	0.08
1969	0.06	0.0026	0.06
1970	0.03	0.0026	0.068
1971	0.03	0.0026	0.06
1972	0.02	0.0027	0.027
1973	0.01	0.0027	0.013
1974	0.02	0.0027	0.039
1975	0.01	0.0027	0.02

Table A 1-4

Nuclear Properties of Environmentally Significant Transuranium Radionuclides (7)

<u>Radionuclide</u>	<u>Radiological half-life (y)</u>	<u>Mode of Decay</u>	<u>Energy of Major Radiations (MeV)</u>	<u>Daughter Radionuclide</u>
Pu-238	87.4	alpha	5.50; 5.46	U-234
Pu-239	2.4×10^4	alpha	5.16; 5.11	U-235
Pu-240	6.6×10^3	alpha	5.17; 5.12	U-236
Pu-241	14.3	beta	0.021 (max)	Am-241
Pu-242	3.9×10^5	alpha	4.90; 4.86	U-238
9 Am-241	443.	alpha	5.49; 5.44	Np-237
Am-243	7.4×10^3	alpha	5.28; 5.23	Np-239
Cm-242	0.45	alpha	6.12; 6.07	Pu-238
Cm-244	18.1	alpha	5.81; 5.77	

kilocuries of Pu-238 were released in the high stratosphere of the southern hemisphere when a satellite containing a nuclear power source (SNAP-9A) failed to orbit and disintegrated (9). As a result, there are measurable amounts of Pu-238 in most environmental media. Also, an estimated 90 curies of curium (Cm-245 and Cm-246) have been produced as the result of weapons tests (1).

The principal additional potential source for future release of the transuranium elements to the general environment are operations associated with the Light-Water Reactor Fuel Cycle. About 250 kg of plutonium, which is inside the spent fuel rods, is removed per year from a 1000 Mw(e) light-water reactor. The isotopic composition of this plutonium is typically 59% Pu-239, 29% Pu-240, 11% Pu-241, 4% Pu-242 and 2% Pu-238 (9).

Sections that follow briefly discuss specific controlled sites and other areas in the general environment which have become contaminated with transuranium elements significantly above levels attributed to stratospheric fallout. There are several possible reasons: local fallout from aboveground nuclear tests, accidents at nuclear facilities or effluent releases from nuclear facilities. Data was selected to be representative of conditions at these sites as of 1973 to 1975; the years for which the most recent reports on environmental monitoring have been published. Much additional information is available in the documents that have been referenced.

Table A 1-5 provides a summary of these sites, their locations, inventories, and approximate onsite and offsite maximum soil concentration levels.

Table A 1-5

INVENTORIES AND CONCENTRATION LEVELS OF PLUTONIUM IN THE ENVIRONMENT

Location	Approximate Inventory of Plutonium released to Soils (curies)	Maximum Soil Concentration Levels		Comments	References
		Onsite	Offsite		
Nevada Test Site Nevada	> 155 (Pu-239)	6,000 $\mu\text{Ci}/\text{m}^2$	2×10^{-3} $\mu\text{Ci}/\text{m}^2$ 10 pCi/g	Onsite levels refer to small subregions of the site	1,11,12
Rocky Flats Plant Denver, Colorado	11 (Pu-239) 1 (Am-241)	> 1000 pCi/g ² > 10 $\mu\text{Ci}/\text{m}^2$	4 pCi/g	It is estimated that 8 curies of plutonium are onsite; 3 are offsite. Inventory of Am-241 will double due to ingrowth. Soil samples are taken 5 cm deep.	14,15,17
Mound Laboratory Miamisburg, Ohio	5 to 6 (Pu-238)	-	4,600 pCi/g	Most of the plutonium is associated with buried sediments in waterways adjacent to the site.	18,19
Savannah River Plant South Carolina	2 (Pu-239)	0.001 $\mu\text{Ci}/\text{m}^2$	0.01 $\mu\text{Ci}/\text{m}^2$	Offsite plutonium is located in a swamp area of the Savannah River in the top 8 cms of sediment.	1,10
Los Alamos Scientific Laboratory Los Alamos, New Mexico	> 1 (Pu-239 and Pu-238)	220 pCi/g	< 1 pCi/g	Plutonium is associated with soils in dry canyons (top 5 cms)	10
Trinity Site New Mexico	~ 45 (Pu-239)	-	0.1 $\mu\text{Ci}/\text{m}^2$		22
Enewetak Atoll Micronesia	Not published	530 pCi/g	0.3 pCi/gm 0.07 $\mu\text{Ci}/\text{m}^2$	Offsite value refers to islands most likely to be resettled (top 15 cm of soil)	23
Continental United States	16,000 (Pu-239) 4,000 (Pu-238)	0.001 to 0.003 $\mu\text{Ci}/\text{m}^2$		(top 30 cm of soil)	3

1. The Nevada Test Site (NTS)

The Nevada Test Site (10) is an area of about 3500 km² located in Nye County, Nevada, 90 kilometers northwest of Las Vegas. It is surrounded by an exclusion area 25 to 100 kms wide between the test site itself and public lands. The climate is for the most part arid, with insufficient rainfall (10 to 25 cm/y precipitation) to support trees or crops without irrigation. Winds blow primarily from the north, except for May through August when they are predominantly from the south-southwest. Fig. A 1-1 shows the population distribution around this site by azimuth and by distance.

Major programs conducted at NTS have included nuclear weapons tests, tests for peaceful uses of nuclear explosives, nuclear reactor engine development, basic high energy nuclear physics research, and seismic studies. As the result of these activities, the test site, exclusion area and, to a much lesser extent, large areas outside the exclusion areas have become contaminated with plutonium.

Although the total inventory of plutonium in soils within the NTS is not known, detailed surveys have been made of certain specific locations in the site and the Tonopah Test Range (TTR) which are believed to be the areas most highly contaminated with plutonium and americium (11). As shown in Table A 1-6, the inventory of plutonium in these areas is about 155 curies.

Estimates of offsite plutonium concentration in soil as the result of activities at NTS have been made (12). In units of mCi/km²

Table A 1-6

Estimated Inventory of Plutonium in Surface Soil at Specific
Areas within the National Test Site and Tonopah Test Range (11)

Area	Size of Area (m ²)	Estimated Inventory (curies Pu-239) ^a	Range of Soil Concentrations ^b (μ Ci/m ²)	Pu/Am Activity Ratios
13	3.6x10 ⁶	44 \pm 9	2 to 840	9
5 (GMX)	2.4x10 ⁵	3 \pm 0.3	3 to 530	10
Double Track	3.0x10 ⁵	5 \pm 1	7 to 2,800	22 to 26
Clean Slate 1	2.2x10 ⁵	5 \pm 2	15 to 120	
Clean Slate 2	7.9x10 ⁵	29 \pm 6	4 to 260	
Clean Slate 3	1.3x10 ⁵	30 \pm 5	12 to 370	
Plutonium Valley	4.8x10 ⁶	39 \pm 4	1 to 6,200	5 to 8
Total		155		

(a) Inventory as measured to 5 cm soil depth.

(b) Soil concentration range values refer to concentrations within sub regions of each site as selected by stratified random sampling.

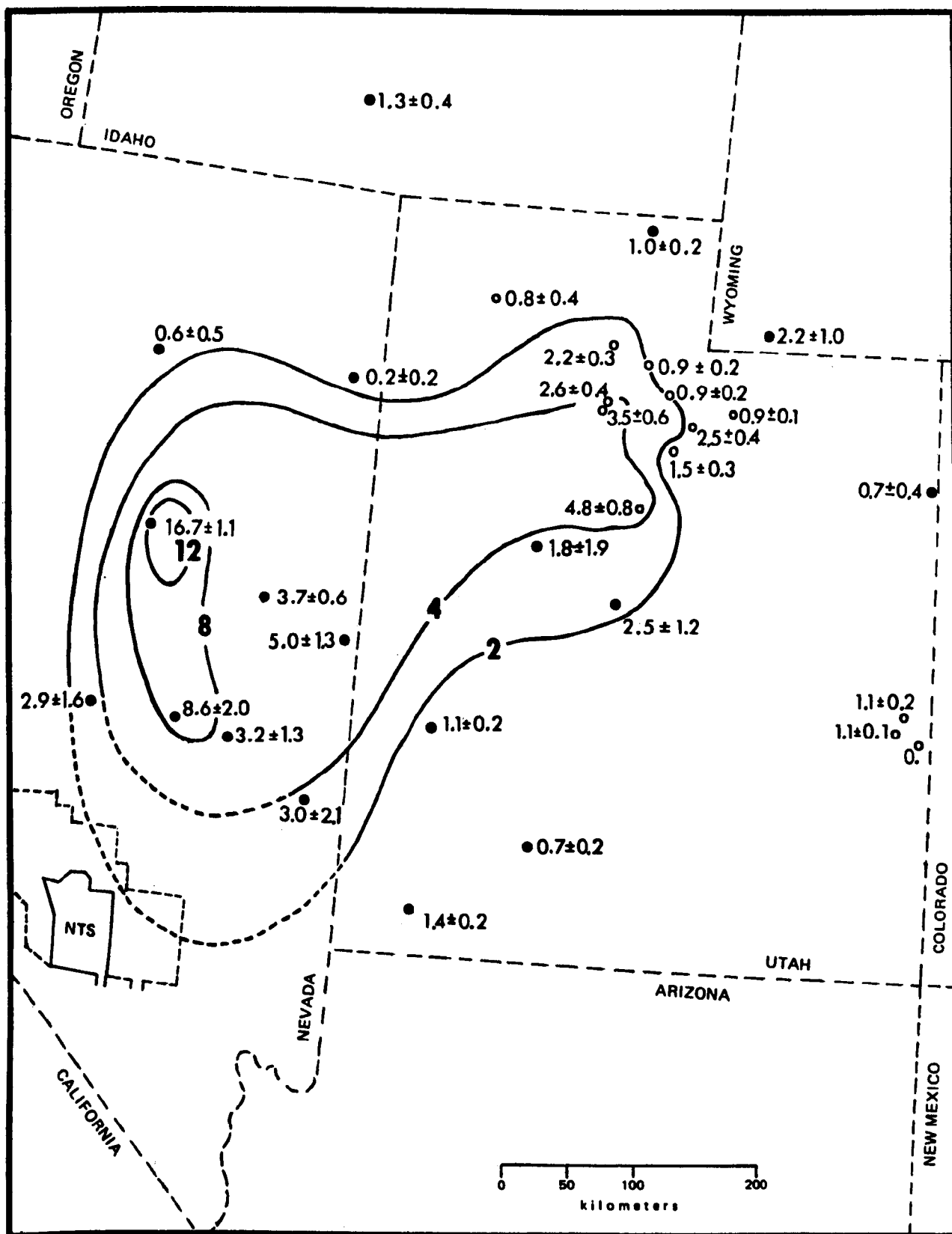
($1 \text{ mCi/km}^2 = 0.001 \text{ } \mu\text{Ci/m}^2$) Fig. A 1-2 shows isopleths for this material; Fig. A 1-3 shows, for comparison, the additional amounts of plutonium at the same locations due to fallout. Offsite levels of plutonium in soil are less than $0.1 \text{ } \mu\text{Ci/m}^2$, most areas being far lower.

A limited special study was made of plutonium concentrations in air at locations close to the NTS (13). Results are shown in Table A 1-7 and indicate that, while resuspended plutonium from NTS has probably been detected at three locations, the air concentration level has not exceeded 0.5 fCi/m^3 . Long term air surveillance from 1966 to 1972 at eight major centers of population in western states was performed for ambient plutonium levels in ground level air (13). Within annual cycles, these levels, which are ascribed entirely to fallout plutonium from the stratosphere, varied from 0.01 to 0.5 fCi/m^3 with mean annual values of approximately 0.1 fCi/m^3 .

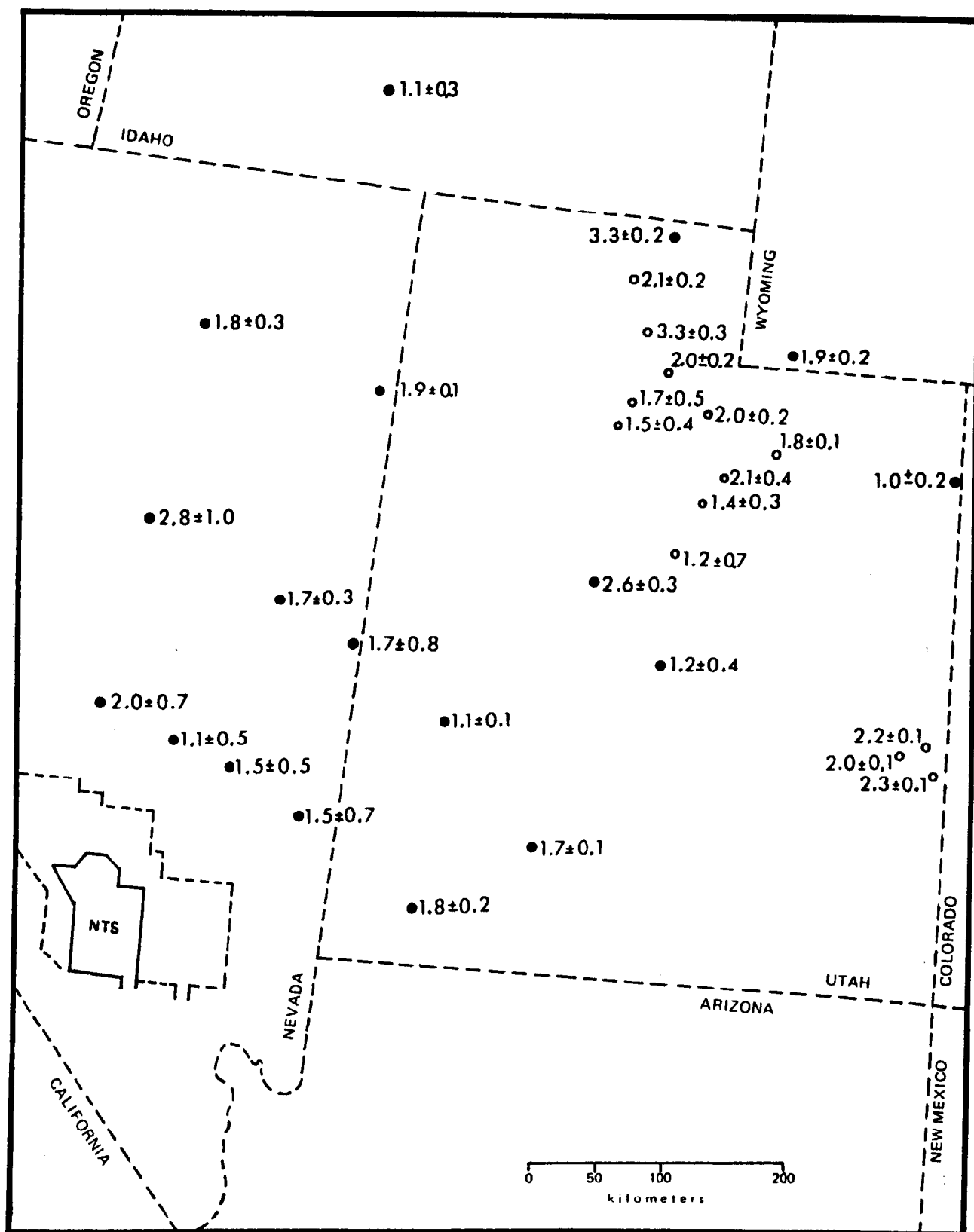
The milk surveillance network around the NTS does not analyze samples for plutonium. Water samples from wells both onsite and offsite in surrounding communities were analyzed for Pu-239 and Pu-238. Plutonium was not detected, indicating levels less than about 0.1 pCi/l Pu-239 or Pu-238.

3. Rocky Flats Plant (RFP)

The Rocky Flats Plant (10) is located in Jefferson County, Colorado, 26 kilometers northwest of Denver. Currently, the site consists of 6,500 acres of Federally owned land of which 385 acres is enclosed within a security fence. The area is arid (40 cm/y precipitation) with predominant winds from the northwest. Figure A 1-4 is a



CUMULATIVE NTS DEPOSIT OF Pu-239,240 (12)
(mCi per km²)
FIGURE A1-2



CUMULATIVE GLOBAL FALLOUT DEPOSIT OF Pu-239,240 (12)
(mCi per km²)
FIGURE A1-3

Table A 1-7 Pu-239 in Air Samples - Near the NTS (13)

Location	Date	Downwind Pu-239 Concentration (fCi/m ³)	Date	Upwind Pu-239 Concentration (fCi/m ³)	Pu-239 Concentration Downwind vs Upwind
Furnace Creek, CA	2/20/71	<0.05	4/20/71	0.20	No difference
	10/30/72	<0.04	12/7/72	0.051	
Death Valley Jct. CA	2/20/71	0.20	5/2/71	0.20	No difference
	3/31/71	0.12	5/3/71	0.20	
	10/24/72	0.10	10/2/72	<0.065	
	10/28/72	<0.07	12/6/72	<0.048	
	12/13/72	<0.03	12/7/72	0.055	
Beatty, NV	2/3/71	0.09	6/28/70	0.40	Significant difference
	2/25/71	0.08	4/20/71	0.17	
	3/1/71	<0.06	5/2/71	0.20	
	3/17/71	0.19	5/20/71	0.12	
	10/28/71	<0.08	10/16/71	0.20	
	4/25/72	0.088	4/16/72	0.075	
Diablo, NV	4/20/71	0.20	2/20/71	<0.06	No difference
	5/4/71	0.20	3/1/71	<0.07	
	5/26/71	0.40	3/31/71	0.30	
Hiko, NV	3/26/71	0.20	9/25/70	<0.07	Significant difference
	4/25/71	0.20	2/20/71	<0.07	
	6/26/71	0.20	3/18/71	0.09	
	9/25/71	<0.70	10/28/71	<0.06	
Indian Springs, NV	3/13/71	0.10	4/20/71	0.15	Probable difference
	4/25/72	0.087	4/16/72	0.19	
Lathrop Wells, NV	3/18/70	<0.06	4/20/71	0.20	No difference
	9/25/70	0.17	6/26/71	0.30	
	3/18/71	0.20	9/25/71	0.20	
	10/24/72	0.04	10/2/72	0.042	
Pahrump, NV	5/17/71	0.30	4/20/71	<0.10	No difference
	10/30/72	<0.12	10/2/72	<0.044	
Scotty's Jct., NV	2/19/70	0.15	4/20/71	0.17	No difference
	3/31/71	0.20	5/3/71	0.20	
			5/27/71	0.20	
	10/24/72	0.068	12/4/72	<0.036	
	10/29/72	0.15	12/6/72	0.056	
	12/13/72	<0.059	12/7/72	<0.042	
Warm Springs, NV	3/12/71	0.14	2/19/70	<0.09	No difference
	3/26/71	<0.08	3/18/70	0.23	
	4/25/71	0.13	3/31/70	0.10	
			2/3/71	<0.60	
			2/20/71	<0.60	
			3/17/71	0.20	
			3/31/71	0.12	

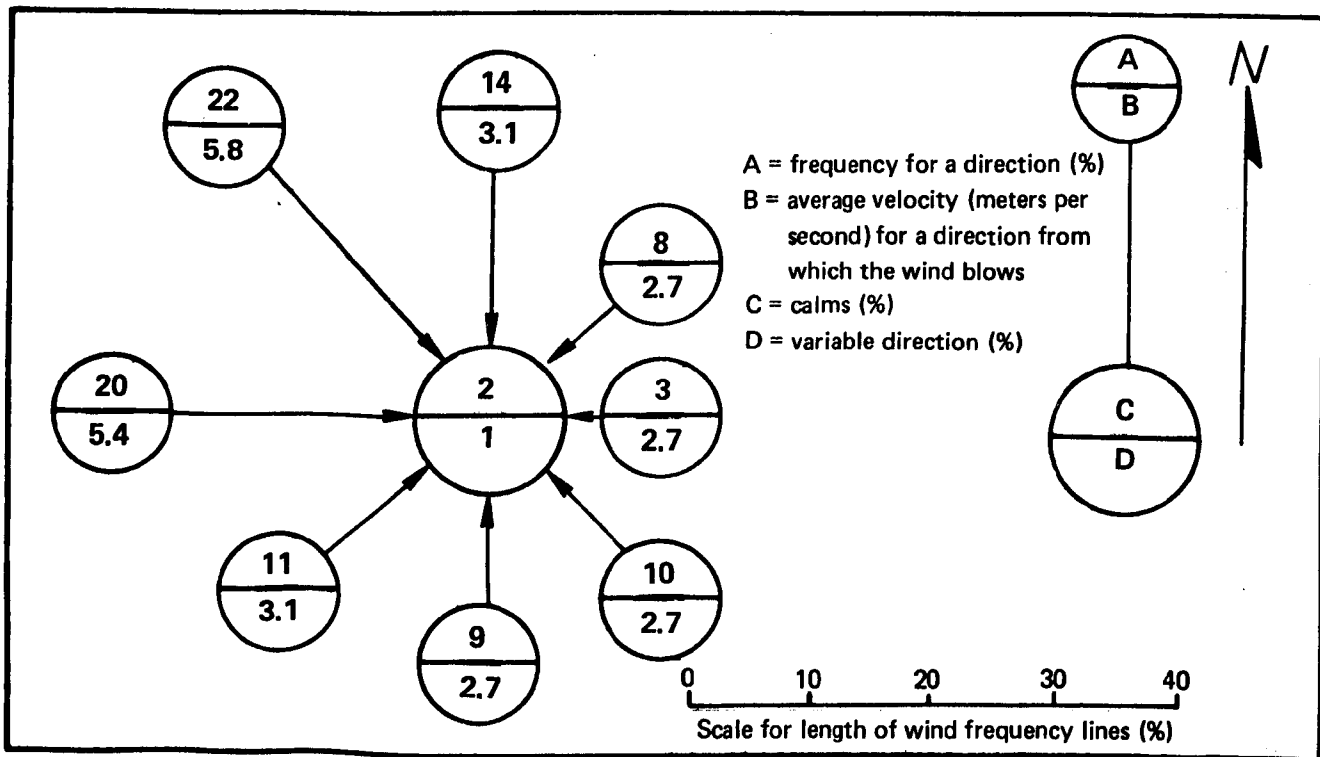


FIGURE A1-4
WIND ROSE FOR THE ROCKY FLATS SITE (10)

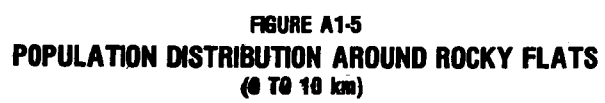
wind rose for the site; Fig. A 1-5 provides population densities around the site by azimuth and distance. Less than 10 μCi of plutonium was released from plant stacks and vents to the atmosphere in 1975 (10). Data on the total amount of plutonium released to surface waters from 1973 to 1975 have not been published.

The plant produces components for nuclear weapons, which involves the processing of plutonium. As the result of leakage from barrels of plutonium-contaminated cutting oil, parts of the site and, to a lesser degree, the general environment around the site have been contaminated with plutonium and americium. The total amount of plutonium released to the environment is estimated to be 11 curies of which 3.4 ± 0.9 curies is estimated to be offsite (14). Of the approximately 8 curies onsite, more than half is believed to be stabilized by coverage with an asphalt pad and remedial measures are being taken to control the remainder to the extent practicable.

Figures A 1-6 and A 1-7 show values for Pu-239 concentrations in soils (5 cm depth) around the site (15, 16). Concentrations of Am-241 within the site boundaries are about 10% of Pu-239 values (17).

Table A 1-8 provides selected yearly average plutonium concentrations in ambient air within the plant boundary, at distances of 3 to 6 kilometers from the plant and in nearby communities (10).

Liquid effluents released from the plant may eventually reach the Great Western Reservoir, while storm water runoff from the site tends to collect both there and in Standley Lake. Both reservoirs are sources of



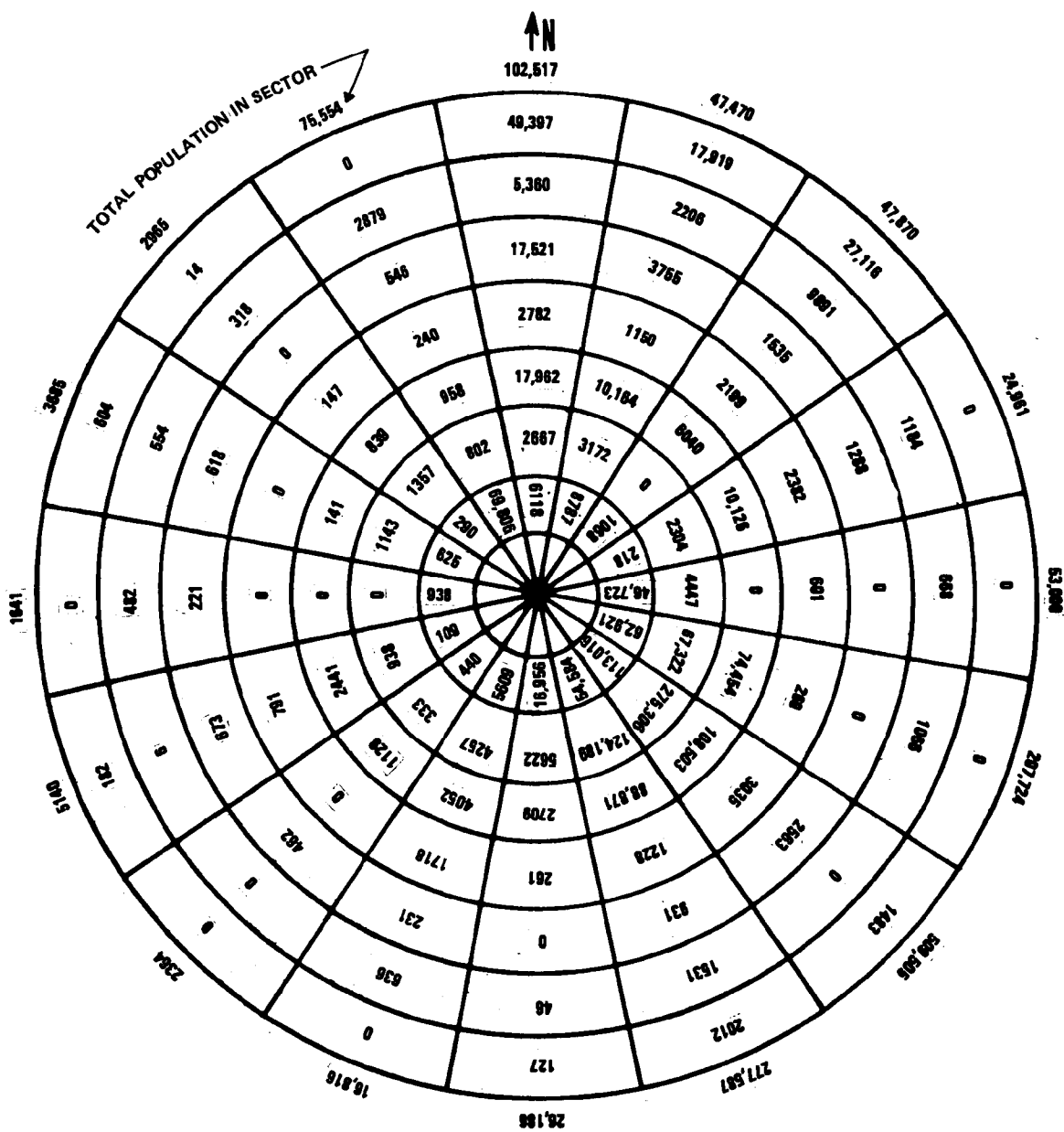


FIGURE A1-5
POPULATION DISTRIBUTION AROUND ROCKY FLATS
(10 TO 80 km)

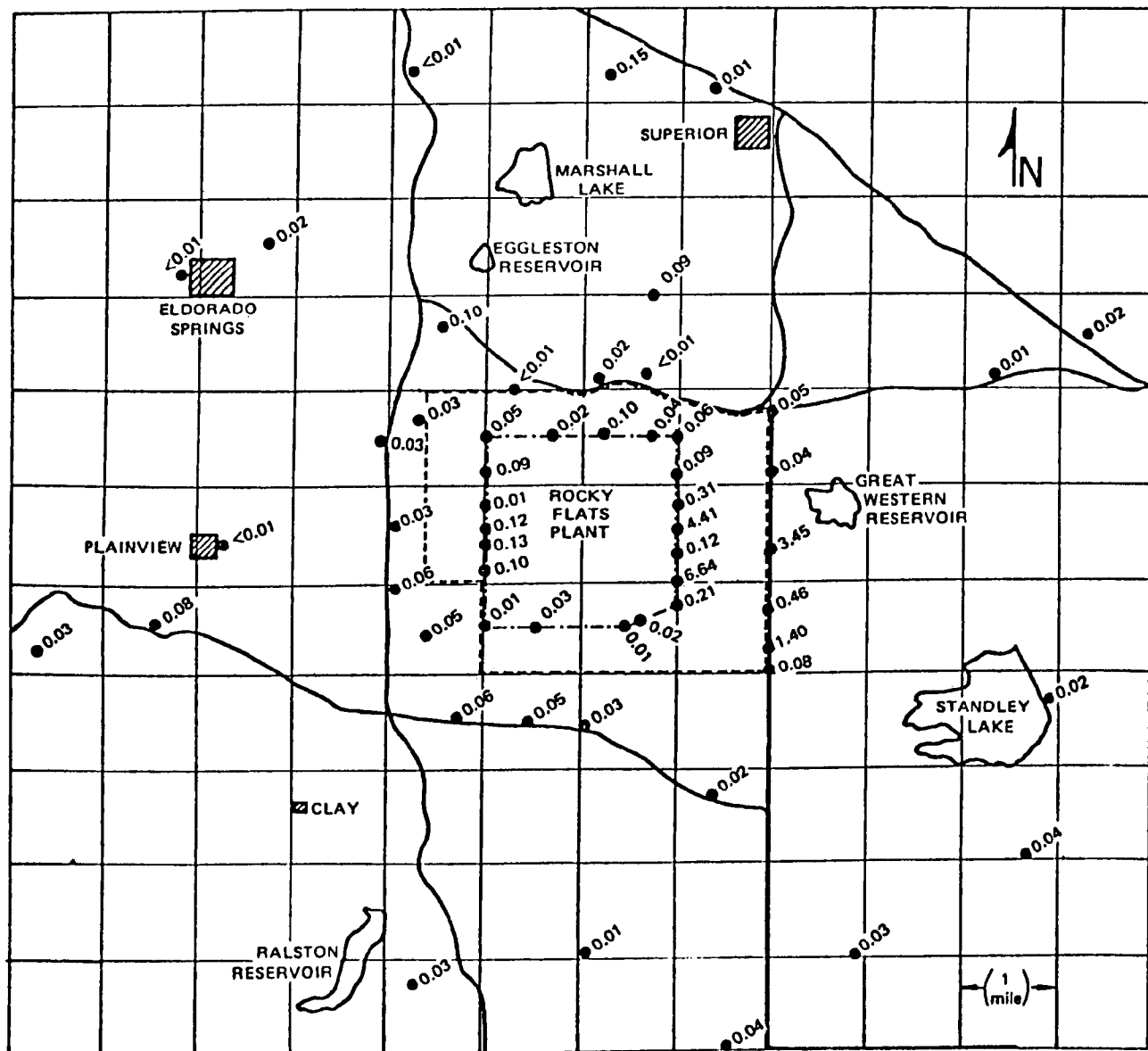


FIGURE A1-6
ROCKY FLATS 1974
PLUTONIUM CONCENTRATIONS IN SOIL.
(VALUES IN PICOCURIES PER GRAM. (15))

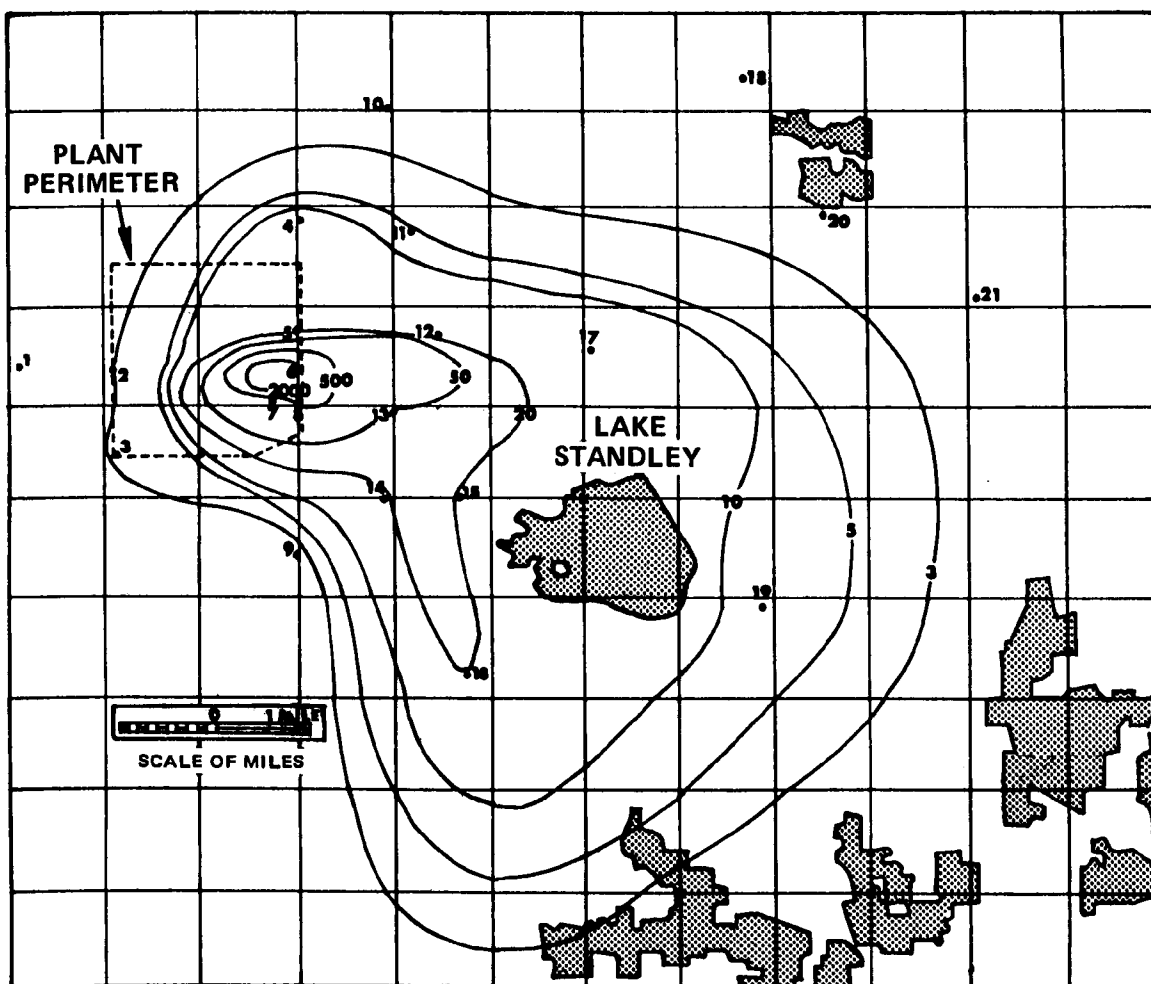


FIGURE A1-7
ROCKY FLATS
PLUTONIUM—239 CONTOURS mCi/km² (16).

Table A 1-8

Plutonium Concentration in Ambient Air at Selective
Locations - Rocky Flats Site, 1975 (10)

Location	Station	Average Plutonium Concentration (fCi/m ³)	Station Location with Respect to the Plant
Onsite	S-14	< 0.02	West
	S-16	< 0.06	Northwest
	S-4	0.1	North
	S-6	1.	East
	S-11	0.01	South
Three to six Kilometers Distant from Plant	S-31	< 0.03	West
	S-34 ^a	< 0.04	North
	S-37 ^a	0.06	East
	S-41	< 0.03	South
Boulder	-	< 0.03	Northwest
Marshall	-	< 0.03	North
Superior	-	< 0.04	North
Walnut Creek	-	< 0.03	East
Wagner	-	< 0.04	East
Leyden	-	0.04	South

(a) Site Boundary

drinking water. Table A 1-9 gives estimates of plutonium and americium concentrations in these water supplies as well as in finished drinking water for nearby communities (10). Modifications of the Rocky Flats plant operations have been proposed that will eventually halt all of its liquid effluent discharges.

In summary, in offsite areas around the Rocky Flats plant, the plutonium concentration in ambient air is $< 0.06 \text{ fCi/m}^3$, plutonium in finished drinking water is $< 0.03 \text{ pCi/l}$, and plutonium in soil is $< 0.1 \text{ } \mu\text{Ci/m}^2$ (5 cm deep samples) (17).

4. Mound Laboratory (ML)

Mound Laboratory (10) is located in Miamisburg, Ohio, 16 kilometers southwest of Dayton. The 180 acre site is within an industrialized river valley in a region that is predominantly agricultural. Corn and soy beans are major crops and livestock is pastured. Winds are predominantly from the south or west; average precipitation is 91 cm/yr. The population distribution around the site is given in Fig. A 1-8.

The mission of the laboratory includes research, development and production of components for the nuclear weapons program and fabrication of radioisotopic heat sources for medical applications and space operations. This latter operation involves processing large quantities of Pu-238 which has become the plutonium radionuclide of primary concern associated with this site.

Pu-238 in airborne effluent discharges from the plant has, over the years, contaminated the site and, to a lesser degree, offsite areas. Figure A 1-9 shows preliminary estimates of the levels of Pu-238 in

Table A 1-9

Concentrations of Plutonium and Americium in Water Supplies
and in Finished Drinking Water - Rocky Flats Site, 1975 (10)

Location	Water Supply	Concentration	
		Plutonium (pCi/l)	Americium (pCi/l)
Great Western	Reservoir	< 0.1	< 0.03
Standley Lake	Reservoir	< 0.04	< 0.03
Boulder	Drinking Water	< 0.007	< 0.006
Broomfield	"	< 0.04	< 0.03
Denver	"	< 0.008	< 0.04
Golden	"	< 0.009	< 0.009
Lafayette	"	< 0.007	< 0.007
Westminister	"	< 0.04	< 0.03
(Walnut Creek at Indiana Street)	(Discharge to Great Western Reservoir)	(0.6)	(0.2)

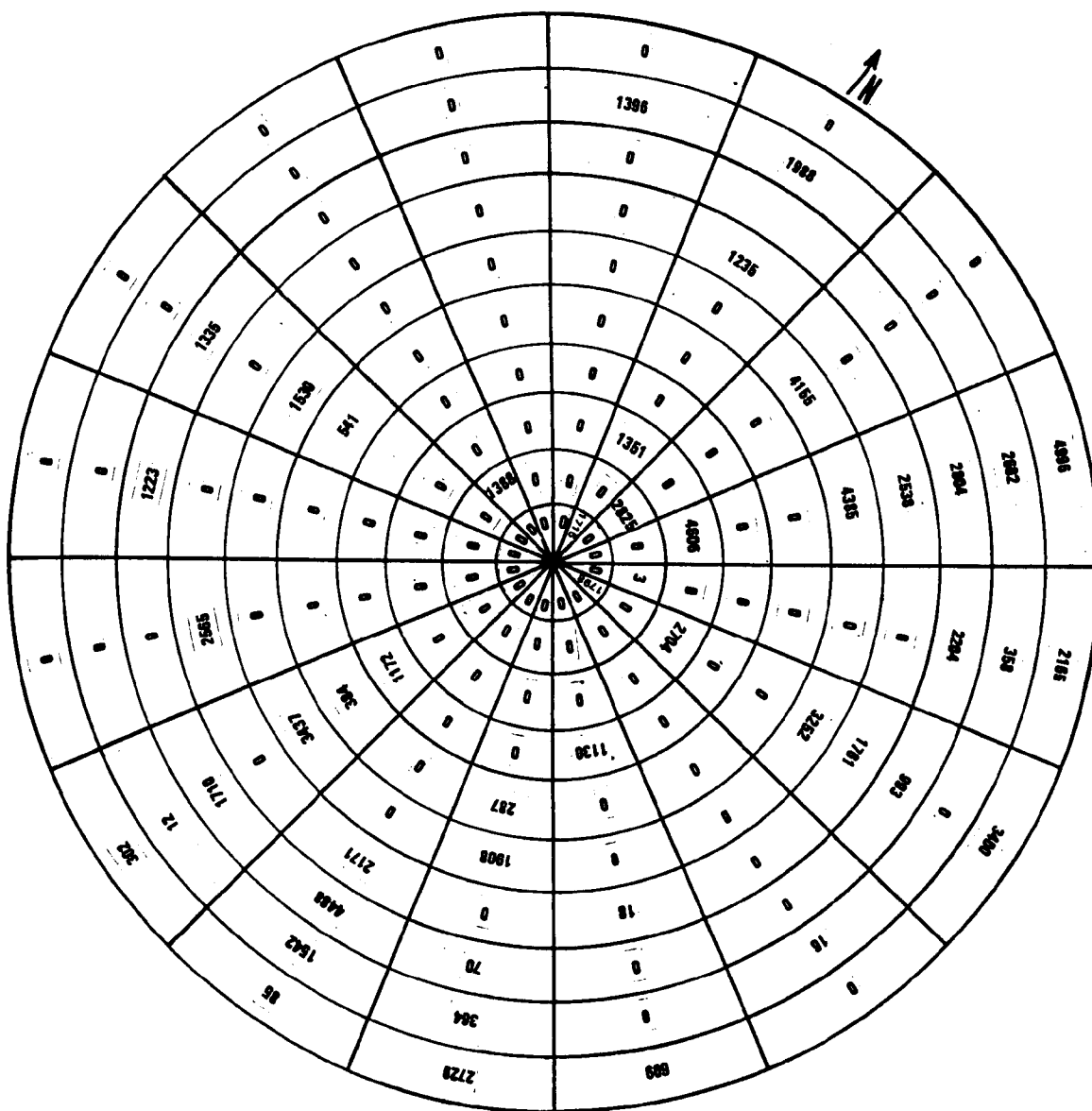


FIGURE A1-8
POPULATION DISTRIBUTION AROUND MOUND LABORATORY
 (0 to 10 km) (LAT 39.6305 LONG 84.2897)

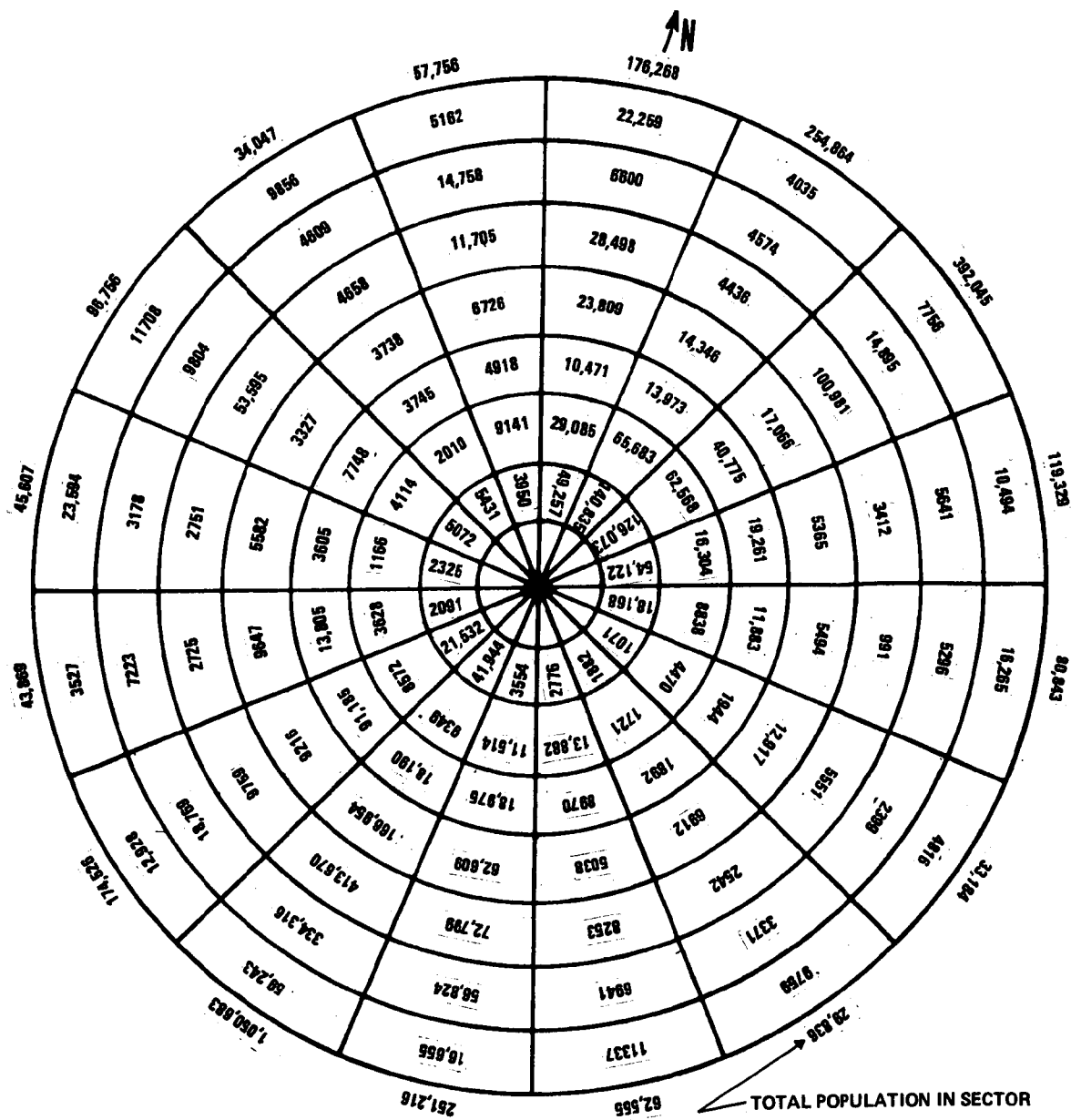


FIGURE A1-8
POPULATION DISTRIBUTION AROUND MOUND LABORATORY
 (10 to 80 km) LAT 39,6305 LONG 84,2897)
 TOTAL P = 2,903,384

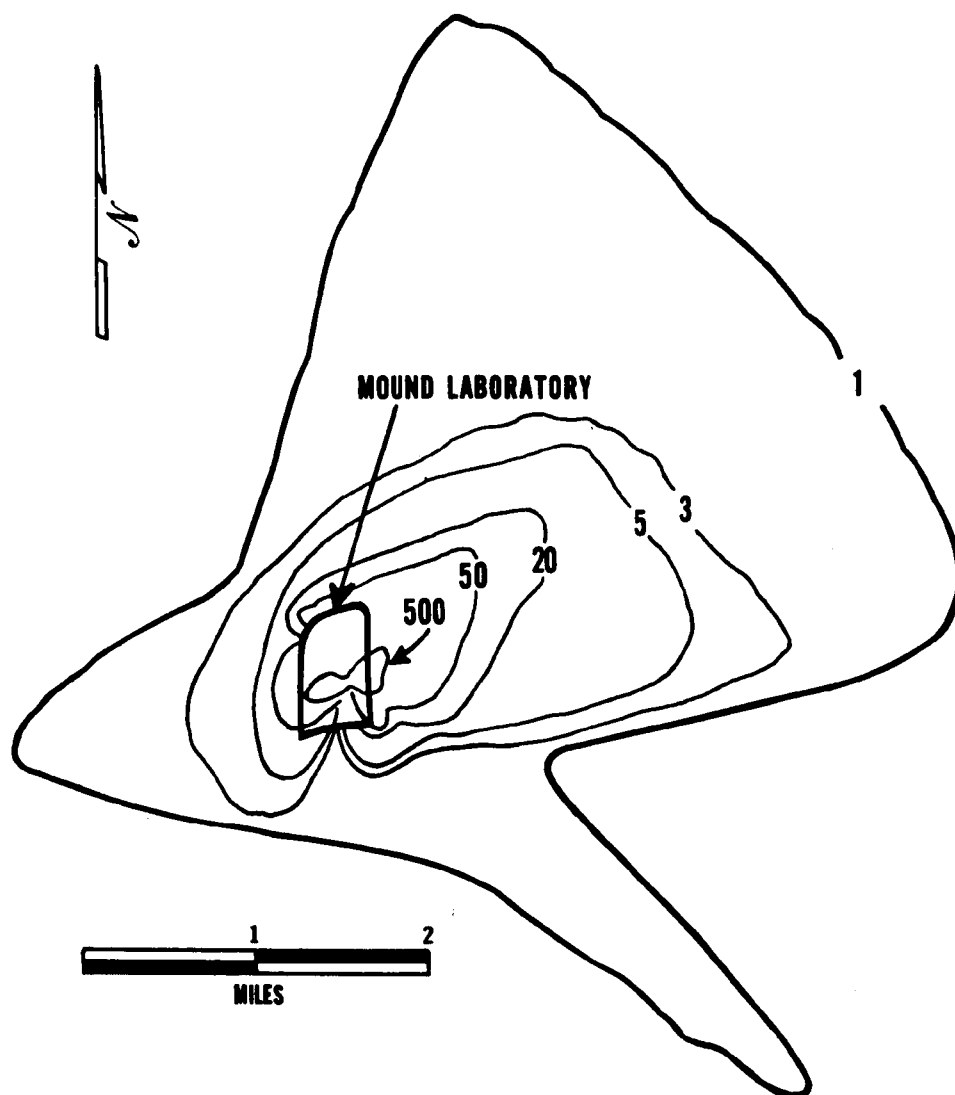


FIGURE A1-9

MOUND LABORATORY

PRELIMINARY ESTIMATE OF PLUTONIUM - 238 AIRBORNE DEPOSITION
(mCi/km^2) (10)

soil around the site. Approximately 0.5 curies of Pu-238 have been released to the offsite environment in this manner.

The concentration of Pu-238 in various environmental media around the Mound Laboratory is given in Table A 1-10 for 1975 (10). The annual average concentration of Pu-238 in offsite ambient air did not exceed 0.03 fCi/m^3 ; in surface waters it was as high as 1.4 pCi/l in an off-site pond but in water supplies it did not exceed 0.05 pCi/l .

In 1969, an underground pipe carrying acid radioactive waste solutions ruptured. During repair work on this pipe, heavy rains eroded the radioactive soil, and carried about 5 curies of Pu-238 off-site into waterways adjacent to the Laboratory. This plutonium now is in sediments that are mostly buried under approximately 1 to 3 feet of additional non-contaminated sediments added by normal processes later in time (18). A special study of this incident was also conducted by the U.S. Environmental Protection Agency; results are given in Fig. A 1-10 and Table A 1-11 (19).

5. Savannah River Plant (SRP)

The Savannah River Plant (10) is located on a 790 km^2 Federally owned site along the Savannah River in Aiken and Barnwell Counties, South Carolina, about 100 kilometers southwest of Columbia. The surrounding area is predominantly forested with some diversified farming, the main crops being cotton, soy beans, corn, and small grains, with the production of beef cattle. The climate is mild, with an annual rainfall of 115 cm/y . Population density around the site ranges from 10 to 400 people per square mile.

Table A 1-10

Concentration of Pu-238 in Environmental Media
Mound Laboratory (10)

Ambient Air

<u>Sample Location</u> <u>(Location Number)</u>	<u>Average Concentration of Pu-238</u> <u>(fCi/m³)</u>
onsite (211)	0.2
(212)	0.05
(213)	1.0
(214)	0.06
North of Plant (101)	0.02
East of Plant (103)	0.01
South of Plant (104)	0.01
West of Plant (105)	0.009
Miamisburg (122)	0.02
Dayton (108)	0.008
(National Average from Fallout)	(0.003)

Table A 1-10 (Continued)

<u>Sample Location</u> <u>(Location number)</u>	<u>Average Concentration of Pu-238</u> <u>(pCi/l)</u>
Great Miami River	
Above the Plant (1)	0.019 ± 0.0022
Below the Plant (4)	0.052 ± 0.004
Canal/pond area	
North Pond	0.22
South Pond	1.4
Miamisburg Drinking Water	0.043 ± 0.003
Private Well J	0.020 ± 0.002
Private Well B	0.006 ± 0.00003

Table A 1-10 (Continued)

Foodstuff Collected Close to the Plant

<u>Sample</u>	<u>Average Concentration of Pu-238 (pCi/g)</u>
Milk	2×10^{-4}
Fruits & Vegetables	$< 6 \times 10^{-4}$
Grass	1×10^{-2}
Field Crops	1×10^{-3}
Aquatic life	$< 3 \times 10^{-4}$

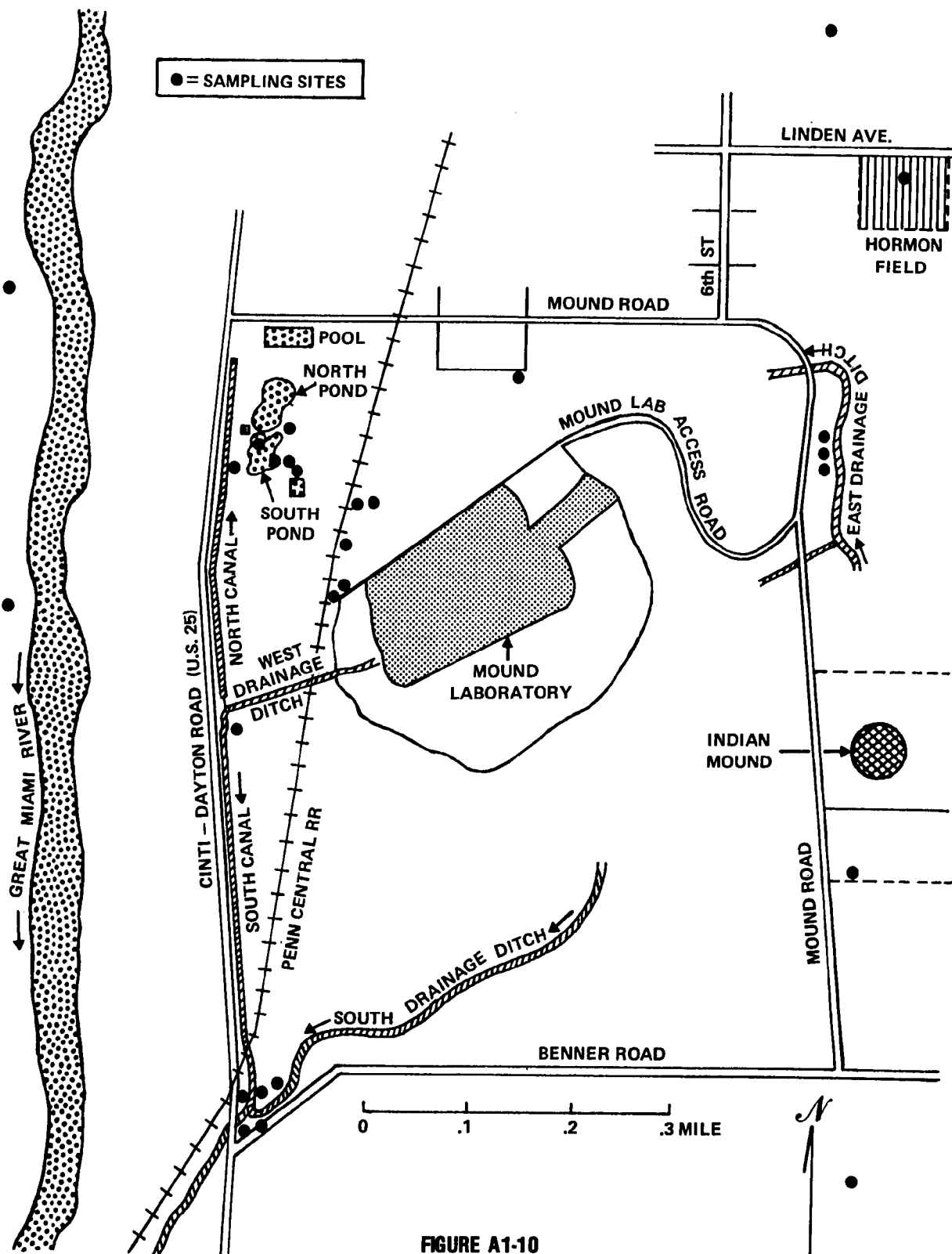


FIGURE A1-10
U.S. ENVIRONMENTAL PROTECTION AGENCY SAMPLING SITES
MOUND LABORATORY (19)

Table A 1-11
MOUND LABORATORY

U.S. ENVIRONMENTAL PROTECTION AGENCY
1974 SURVEY (19)

Plutonium in Samples from the Vicinity of Mound Laboratory

I.D. #	Location	Concentration	
		²³⁸ Pu	²³⁹ Pu
Core sediment samples collected by Mound Laboratory, pCi/g dried weight			
EA-1	North Canal at south end of South Pond	0.13	< 0.02
		0.09	< 0.02
EB-1	"	0.13	< 0.02
		0.11	< 0.02
EC-1	"	4.8	0.07
ED-1	"	1.1	< 0.02
EE-1	"	440	2.3
EF-1	"	1170	15.4
		1090	7.9
EG-1	"	10.8	0.18
EH-1	"	26	0.16
EI-1	"	0.98	0.06
EJ-1	"	0.89	0.05
		1.30	0.06
FA-1	North end of North Canal	8.9	0.51
		7.5	0.44
		9.1	
FE-1	"	16.9	0.18
		19.2	0.05
GA-1	North end of North Pond	0.48	< 0.02

Table A 1-11 (Continued)

I.D. #	Location	Concentration	
		^{238}Pu	^{239}Pu
HA-1	Middle of North Pond	5.1	0.07
IA-1	South end of North Pond	2.5	0.06
JA-1	North end of South Pond	0.70	0.02
KA-1	Middle of South Pond	27	0.27
LA-1	South end of South Pond	10.9	0.30
CE-1	South Canal at west drainage ditch	24	0.46
QE-1	South Canal where it crosses US 25	920	10.9
<u>Sediment samples, top 1 inch, pCi/g dried weight</u>			
EPA-1	South Canal at west drainage ditch	230	3.54
EPA-20	East drainage ditch, ~200 ft south of Mound Rd culvert	1.9	0.06
34 EPA-17	South drainage ditch, 15 ft from junction with South Canal	47	0.84
EPA-18	South Canal, 10 ft from junction with south drainage ditch	60	0.77
<u>Surface soil and mud samples, top 1 inch, pCi/g dried weight</u>			
EPA-2	Railroad cut south of control box	0.12	0.02
EPA-3	Railroad cut north of control box	0.39	0.13
EPA-6	Run-off hollow	3.8	0.11
EPA-7	At shelter house SE of South Pond	0.44	0.07
EPA-13	NE of Lab, at fence between tennis court and Harmon Field	0.10	0.02
EPA-14	SE of Lab, at SW corner of Mound Park	0.44	0.04
EPA-15	SW of Lab, at junction of US 25 with South Canal	0.96	0.04
EPA-12	NW of Lab, at alley south of Mound Rd	0.19 0.17	0.05 0.05

SRP produces plutonium, tritium and other special nuclear materials. Facilities include nuclear reactors, nuclear fuel and target fabrication plants, nuclear fuel reprocessing plants, a heavy water production plant and various supporting laboratories.

Two airborne releases, in 1955 and 1969, from fuel reprocessing plant operations are believed to have caused the detectable plutonium contamination of soil that is found within a 2 km radius around those facilities within the site perimeter. Approximately 1 curie of plutonium is estimated to be within the isopleths shown in Fig. A 1-11 (20).

During the 1960's, radioactive liquid effluents were released from SRP such that radioactive materials, including Cs-137, Co-60 and plutonium deposited in offsite swamp areas. In these areas plutonium concentrations range from 3 to $11 \times 10^{-3} \mu\text{Ci}/\text{m}^2$ Pu-239 and 0.3 to $6 \times 10^{-3} \mu\text{Ci}/\text{m}^2$ Pu-238. The amount attributed to fallout sources is approximately $1 \times 10^{-3} \mu\text{Ci}/\text{m}^2$ Pu-239 and $0.1 \times 10^{-3} \mu\text{Ci}/\text{m}^2$ Pu-238 (10).

Levels of plutonium in various environmental media in the general environment around SRP are given in Table A 1-12 for 1975 (10). Resuspended plutonium from the contaminated areas within the site was not detected in offsite ambient air. Values for plutonium concentration levels in ambient air at onsite locations have not been published. In 1975, the plant released 2 mCi Pu-238 and 0.5 mCi Pu-239 to the atmosphere; the plant released 8 mCi Np-239 and 19 mCi Pu-239 in liquid effluents.

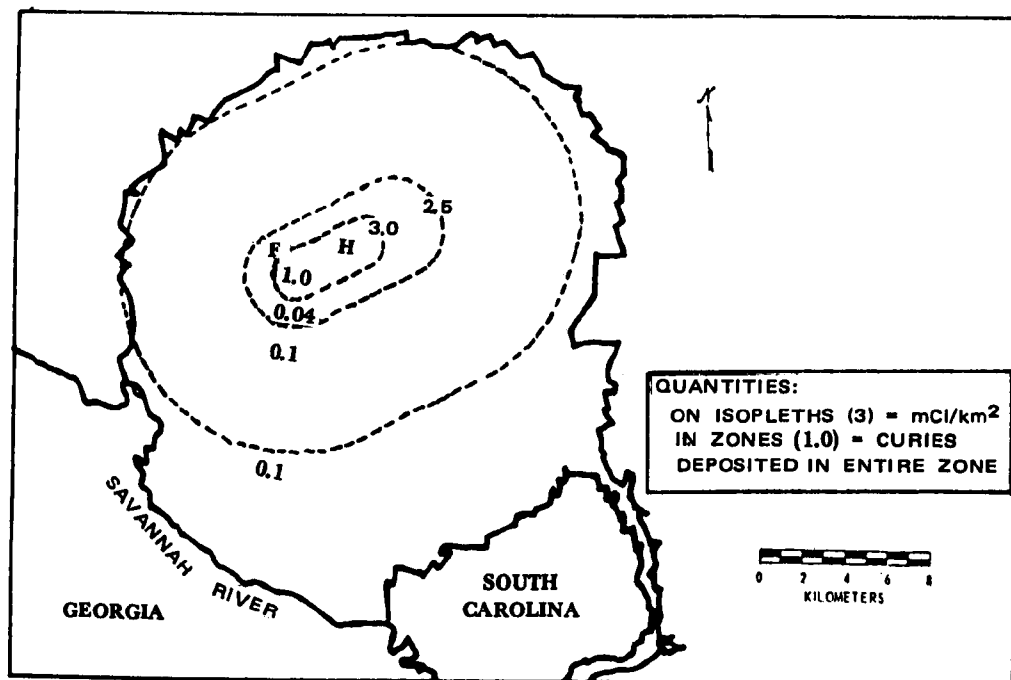


FIGURE A1-11
SAVANNAH RIVER PLANT PLUTONIUM DEPOSITION (10)

Table A 1-12

Plutonium Concentration in Environmental Media
Around the Savannah River Plant - 1975 (10)

Ambient Air

<u>Sample Location</u>	Average Plutonium Concentration	
	Pu-239 (fCi/m ³)	Pu-238 (fCi/m ³)
Plant Perimeter (Locations not specified)	0.02	0.001
25 mile radius (Locations not specified)	0.02	0.001

Rain Water

<u>Sample Location</u>	Average Plutonium Concentration	
	Pu-239 (pCi/m ²)	Pu-238 (pCi/m ²)
Plant Perimeter	2.2	0.11
25 mile radius	1.9	0.15

Soil (0-5 cm depth)

<u>Location</u>	Average Plutonium Concentration	
	Pu-239 (μ Ci/m ²)	Pu-238 (μ Ci/m ²)
Plant Perimeter		
NW quadrant	0.0009	5×10^{-5}
NE "	0.0014	8
SE "	0.0010	6
SW "	0.0012	8
Sprinfeld, SC	0.0003	4×10^{-5}
Aiken Airport, SC	0.0010	7
Clinton, SC	0.0008	3
Savannah, GA	0.0005	2

6. Los Alamos Scientific Laboratory (LASL)

The Los Alamos Scientific Laboratory (10) is located on a 110 km² site in Los Alamos County in North Central New Mexico about 40 kilometers northwest of Santa Fe. The site is on a series of mesas separated by canyons that run eastward from the Jemez Mountains to the Rio Grande Valley. The climate is semi-arid with rainfall of 46 cm/y. While the land around the site is undeveloped, about 16,000 people reside in the immediate area.

The primary mission of LASL is associated with nuclear weapons research and development. Industrial effluents from these operations have for some time been discharged onsite into canyons, where the transuranium nuclides in these effluents soon become attached to soil particles. It is estimated that less than 1 Ci of transuranic waste has been disposed of, in this fashion, to Pueblo, DP-Los Alamos, and Montandad canyons. Liquid effluents are usually absorbed in the soil so they do not flow beyond the site boundaries, but, during periods of heavy runoff, storm waters have carried detectable amounts of transuranium elements down the canyons and offsite. Plutonium concentrations in sediments in the canyons receiving liquid waste are given in Table A 1-13 (21).

Concentration levels of plutonium and americium in various environmental media around the LASL site are given in Table A 1-14 for 1975 (10). During the same year, the Laboratory discharged less than 0.3 mCi of plutonium to the air; the amount of transuranium elements discharged in liquid effluents was not published.

Table A 1-13

Plutonium in Sediments in the Liquid Waste Receiving
Canyons on the LASL Site - 1975 (20)

Distance from outfall (kms)	Average Plutonium Concentration in dry soil ^(a)		
	Acid Pueblo Canyon (pCi/g)	DP - Los Alamos Canyon (pCi/g)	Mortandad Canyon (pCi/g)
0	3	40	220
0.6	10	1	20
1.3	2	-	9
2.6	0.4	0.2	11
5.1	1	0.4	0.1
10.2	0.2	-	0.03
(Estimated Canyon Inventory)			(0.1 to 0.3 curies)
(Average Regional Plutonium Concentration in Dry Soil)		(0.01)	

(a) top 5 cm of soil

Table A 1-14

Plutonium and Americium Concentrations in Environmental
Media at the LASL Site - 1975 (10)

Ambient Air

Station Location (Station Number)	Average Radionuclide Concentration		
	Pu-239 (fCi/m ³)	Pu-238 (fCi/m ³)	Am-241 (fCi/m ³)
On Site 22	0.02	-	-
23	0.02	1x10 ⁻³	3x10 ⁻³
24	0.02	5x10 ⁻⁴	-
25	0.02	5x10 ⁻⁴	7x10 ⁻³
26	0.02	5x10 ⁻⁴	-
Perimeter 12	0.02	6x10 ⁻⁴	0.02
14	0.02	9x10 ⁻⁴	-
18	0.02	1x10 ⁻⁴	1x10 ⁻³
20	0.02	5x10 ⁻⁴	-
Off Site 1	0.02	6x10 ⁻⁴	-
4	0.02	7x10 ⁻⁴	4x10 ⁻³
8	0.02	9x10 ⁻⁴	4x10 ⁻³
(Santa Fe) 11	0.02	4x10 ⁻⁴	4x10 ⁻³

Surface Water and Water Supplies

Sample Location	Average Radionuclide Concentration	
	Pu-239 (pCi/l)	Pu-238 (pCi/l)
Regional Surface Waters	9x10 ⁻⁴	6x10 ⁻⁴
Perimeter Surface and Ground Waters	8x10 ⁻³	2x10 ⁻³
Los Alamos Water Supply ^(a)	-3x10 ⁻⁴	-3x10 ⁻⁴

(a) Negative values are due to statistical fluctuations in the measurement.

Table A 1-14 (Continued)

Soils

<u>Sample Location</u>	Average Radionuclide Concentration ^(a)	
	<u>Pu-239</u> <u>(pCi/g)</u>	<u>Pu-238</u> <u>(pCi/g)</u>
On site	40×10^{-3}	1×10^{-3}
Site Perimeter and Regional areas	12×10^{-3}	0.5×10^{-3}

(a) Top 5 cms of soil.

7. The Trinity Site

The first nuclear device was tested at the Trinity Site, 100 kilometers northwest of Alamogordo, New Mexico, on July 16, 1945. During 1973 and 1974, the site was surveyed by the U.S. Environmental Protection Agency to determine the extent of resulting plutonium contamination (22). Figure A 1-12 shows plutonium contours based on this study. Highest soil activity levels were 0.05, 0.09 and 0.02 $\mu\text{Ci}/\text{m}^2$ found along arcs 1A, 2 and 3, respectively. The total amount of plutonium estimated to be within the 3 nCi/m^2 contour ($1 \text{ nCi}/\text{m}^2 = 0.001 \mu\text{Ci}/\text{m}^2$) is approximately 45 curies.

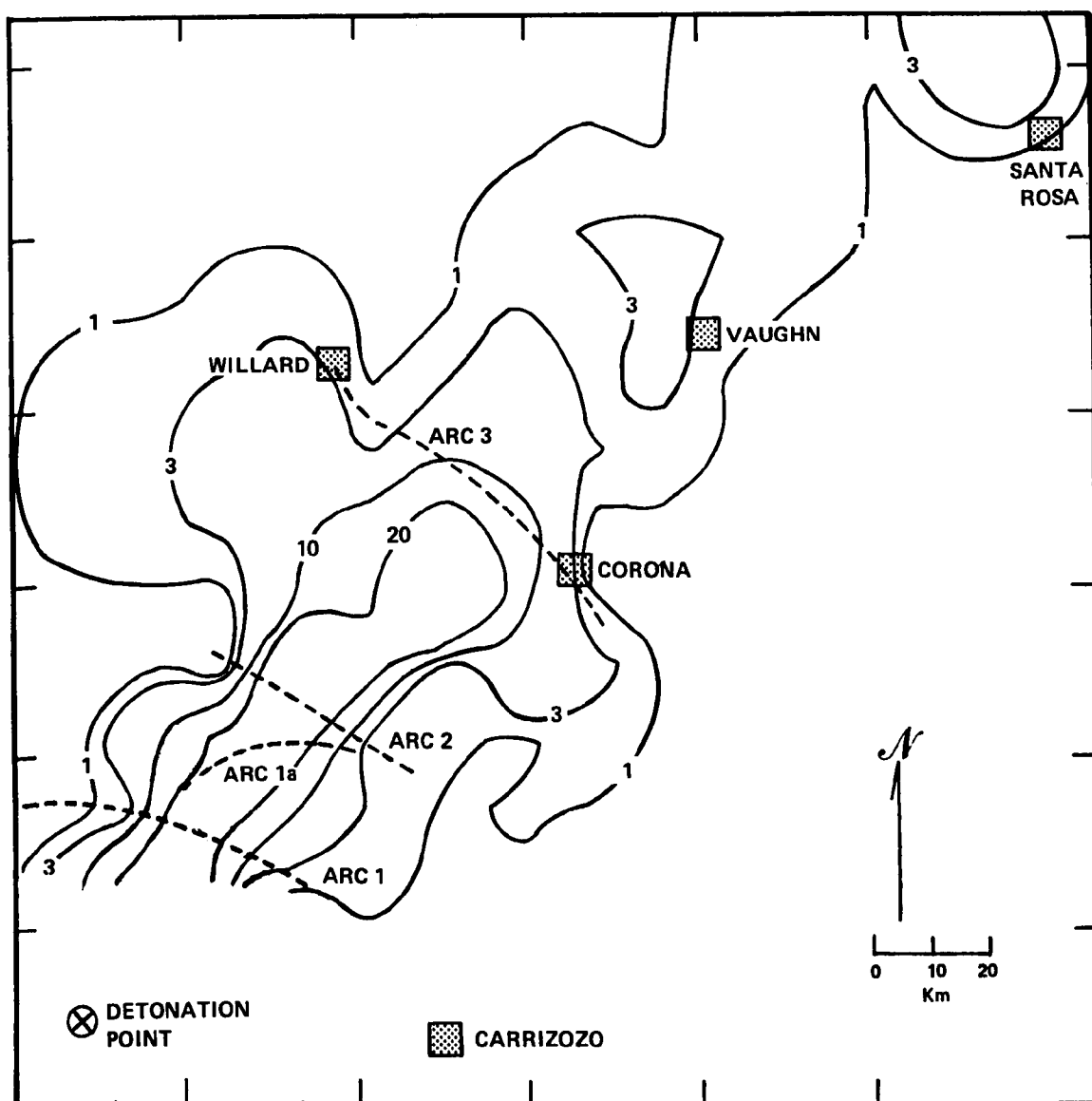
8. Sites of Underground Nuclear Detonations (4)

Table A 1-15 is a listing of underground tests conducted off the Nevada Test Site (10). Nuclear devices containing plutonium may have been used but all transuranium elements are believed to be contained.

9. Enewetak Atoll

The Enewetak atoll (23) consists of 40 islands on an elliptical coral reef about 3800 km southwest of Honolulu in the northern part of Micronesia. It was the site of 43 nuclear weapons tests during the period 1948-1958. The total land area is about 7 km^2 , the largest island of which is 1.5 km^2 with land heights above sea level of 3 to 5 meters. There are plans for future rehabilitation and resettlement of the atoll by the Enewetak people who were displaced in 1948.

The soil of many of the islands is highly contaminated with Sr-90, Cs-137, Co-60 and Pu-239. Plutonium concentrations vary considerably from island to island, depending upon the locations of the detonations



TRINITY SITE
1973-1974 PLUTONIUM
SOIL SAMPLING RESULTS
(nCi/m²) (14)
FIGURE A1-12

Table A 1-15

Underground Testing Conducted Off the Nevada Test Site (10)

Name of Test, Operation or Project	Date	Location	Yield ^d (kt)	Depth m (ft)	Purpose of the Event ^{d,e}
Project Gnome/ Coach ^a	12/10/61	48 km (30 mi) SE of Carlsbad, N.M.	3.1 ^f	360 (1184)	Multi-purpose experiment.
Project Shoal ^b	10/26/63	45 km (28 mi) SE of Fallon, Nev.	12	366 (1200)	Nuclear test detection re- search experi- ment
Project Dribble ^b (Salmon Event)	10/22/64	34 km (21 mi) SW of Hattiesburg, Miss.	5.3	823 (2700)	Nuclear test detection re- search experi- ment.
Operation Long Shot ^b	10/29/65	Amchitka Island, Alaska	~80	716 (2350)	DOD nuclear test detection experiment.
Project Dribble ^b (Sterling Event)	12/03/66	34 km (21 mi) SW of Hattiesburg, Miss.	0.38	823 (2700)	Nuclear test detection re- search experi- ment.
Project Gasbuggy ^a	12/10/67	88 km (55 mi) E of Farmington, N.M.	29	1292 (4240)	Joint Government- Industry gas stimulation ex- periment.
Faultless Event ^c	01/19/68	Central Nevada Test Area 96 km (60 mi) E of Tonopah, Nev.	200- 1000	914 (3000)	Calibration test.
Project Miracle Play (Diode Tube) ^b	02/02/69	34 km (21 mi) SW of Hattiesburg, Miss.	Non- nuclear explosion	823 (2700)	Detonated in Salmon/Sterling cavity. Seismic studies.
Project Rulison ^a	09/10/69	19 km (12 mi) SW of Rifle, Colorado	40	2568 (8425)	Gas stimulation experiment.
Operation Milrow ^c	10/02/69	Amchitka Island, Alaska	~1000	1219 (4000)	Calibration test.
Project Miracle Play (Humid Water) ^b	04/19/70	34 km (21 mi) SW of Hattiesburg, Miss.	Non- nuclear explosion	823 (2700)	Detonated in Salmon/Sterling cavity. Seismic studies.

Table A 1-15 (continued)

Name of Test, Operation or Project	Date	Location	Yield ^d (kt)	Depth m (ft)	Purpose of the Event ^{d,e}
Operation Cannikin ^c	11/06/71	Amchitka Island, Alaska	<5000	1829 (6000)	Test of war- head for Spartan missile.
Project Rio Blanco ^a	05/17/73	48 km (30 mi) SW of Meeker, Colorado	3x30	1780 to 2040 (5840 to 6690)	Gas stimula- tion experi- ment.

^aPlowshare Events

^bVela Uniform Events

^cWeapons Tests

^dInformation from "Revised Nuclear Test Statistics," distributed on September 20, 1974, by David G. Jackson, Director, Office of Information Services, U.S. Atomic Energy Commission, Las Vegas, Nevada.

^eNews release AL-62-50, AEC Albuquerque Operations Office, Albuquerque, New Mexico. December 1, 1961

^f"The Effects of Nuclear Weapons" Rev. Ed. 1964.

and the fallout patterns that followed. Table A 1-16 gives data on Enewetak soils (23); the islands most likely to be reoccupied are Fred, Elmer, David, and eventually Janet. The average plutonium contamination level in surface soils on Fred, Elmer, and David which were not as heavily contaminated as the northern islands, is $9 \times 10^{-3} \mu\text{Ci}/\text{m}^2$, with a range of 0.9 to $70 \times 10^{-3} \mu\text{Ci}/\text{m}^2$; on Janet the average is $2 \mu\text{Ci}/\text{m}^2$, with a range of 0.2 to $4 \mu\text{Ci}/\text{m}^2$.

A limited number of measurements have been made on plutonium and americium concentrations in surface air and in other environmental media. These are given in Tables A 1-17 and A 1-18 (23).

10. Other Sites

The following tables show levels of the transuranium elements in the general environment around other facilities known to use the transuranium elements in their operations. At this time, the total amount of the transuranium radionuclides in soils both on and off such nuclear facility sites is believed to be less than 1 curie.

Table A 1-16

Plutonium Concentration in Soil on Enewetak atoll (23)

Island (U.S. Occupational Designation)	Pu-239 in top 15 cm of soil	
	Mean (pCi/g) ^b	Range (pCi/g)
Alice	12	4-68
Belle dense ^a	26	7-130
light ^a	11	6-26
Clara	22	4-88
Daisy dense	41	22-98
light	15	4-33
Edna	18	13-24
Irene	11	2-280
Janet	9	0.08-170
Kate dense	17	9-50
light	2	0.2-14
Lucy	8	2-22
Mary	8	2-5
Nancy	9	2-28
Percy	4	2-23
Olive dense	8	2-30
light	3	2-4
Pearl hot spot	51	15-530
remainder	11	1-100
Ruby	7	3-24
Sally	4	0.2-130
Tilda dense	8	1-17
light	3	1-34
Ursula	1	0.3-7
Vera	3	0.6-25
Wilma	1	0.1-5
Yvonne southern	3.2	0.02-50
northern beaches	3	0.3-18
David, Elmer, Fred	0.04	0.004-0.3
Leroy	0.6	0.02-2
All others	0.07	0.004-1.1

a. "dense" and "light" refer to vegetation cover

b. 1 pCi/g in the top 15 cm of soil is approximately equivalent to 0.23 $\mu\text{Ci}/\text{m}^2$ or 0.045 $\mu\text{Ci}/\text{m}^2$ if only the top 1 cm of soil is considered and 20% of the total activity is assumed to be in the top 1 cm of soil.

Table A 1-17

Plutonium and Americium Concentration in Surface Air
on Enewetak Atoll (23)

<u>Radionuclide</u>	<u>Location</u>	<u>Concentration</u> <u>fCi/m³</u>
Pu-239	Runit (Yvonne)	0.03-3
	Other islands	0.001-0.03
Pu-238	Reunit (Yvonne)	0.04-0.13
	Other islands	0.003-0.008
Am-241	Runit (Yvonne)	< 0.3-0.3
	Other islands	Not Detected

Table A 1-18

Plutonium and Americium Concentrations in Various
Environmental Media on Enewetak Atoll (23)

<u>Media</u>	<u>Location</u>	<u>Radionuclide</u>	<u>Activity</u>
Sediments	Lagoon	Pu-239	460 mCi/km ²
		Am-241	170 mCi/km ²
Surface Waters	Lagoon	Pu-239	9-40 fCi/l
	Ocean (East)	Pu-239	0.3 fCi/l
Coconuts	As Found	Pu-239	< 0.022 pCi/g ^a
Birds	As Found	Pu-239	
Muscle			0.001-0.1 pCi/g ^a
Liver			0.004-0.07 pCi/g ^a
Eggs			0.0005-0.02 pCi/g ^a
Coconut Crabs	As Found	Pu-239	0.001-0.01 pCi/g ^a

(a) dry weight

Table A1-19

Environmental Monitoring for the Transuranium Elements
at the Pantex Plant Site

Site: Pantex Plant (10,13,24)
 Location: 25 kilometers northeast of Amarillo, Texas
 Mission: Atomic Weapons Assembly involving significant quantities
 of uranium, plutonium, tritium

Transuranium Elements Released to the Environment

No releases during the period 1973-1975

<u>Media</u>	<u>Location of Sample Collection</u>	<u>Sample Station</u>	<u>Average Plutonium Concentrations - CY 1975</u>
Air ^(a)	10 kilometers from plant in various directions	1	0.03 fCi/m ³
		2	0.07
		3	0.03
		4	0.02
		5	0.00 ± 0.01
		6	0.01
		8	0.4
		9	0.00 ± 0.01
		10	0.09
25 kilometers from plant		11	0.5 (1 sample only)
		12	0.00 (1 sample only)
Soil ^(b)	Offsite in various directions from the plant	31	0.00 ± 0.02
		Different Stations	to 0.05 ± 0.02 pCi/g
Jackrabbits	Onsite	11 samples	0.00 ± 0.02 pCi/g (wet) in kidney, liver, lung, flesh, and bone

Table A1-19 (continued)

- (a) Average air concentrations of plutonium in 1973 ranged from 0.4 to 2 fCi/m³ (10), which is higher than any other site. These high levels are believed to have been caused by analytical errors.
- (b) Soil samples collected to a depth of 5 cm.

Table A1-20

Environmental Monitoring for the Transuranium Elements
at Argonne National Laboratory

Site: Argonne National Laboratory (10,13,24)
 Location: DuPage County, Illinois, 43 kilometers southwest of Chicago
 Mission: Research and Development including chemical and
 metallurgical plutonium laboratories

Transuranium Elements Released to the Environment (CY 1975)

To air - not published
 To surface waters (Sawmill Creek) - 0.1 mCi Pu-239;
 0.5 mCi Np-237;
 0.05 mCi Am-241;
 <0.05 mCi Curium and
 Californium

<u>Media</u>	<u>Location of Sample Collection</u>	<u>Number of Stations</u>	<u>Average Plutonium Concentrations - CY 1975</u>
Air	Site Perimeter Offsite	Av. of 2 Stations 1 station	0.02 fCi/m ³ 0.02
Surface Water (Sawmill Creek)	Downstream from Outfall		$< 5 \times 10^{-4}$ pCi/l Pu-239 $< 3 \times 10^{-3}$ Pu-238 4×10^{-3} Am-241 4×10^{-2} Np-237 $< 1 \times 10^{-3}$ Cu-242 5×10^{-3} Cu-244
Sawmill Creek	Upstream from Outfall		2.4×10^{-5} pCi/g Pu-239
Phytoplankton	Downstream from Outfall		1.5×10^{-5} Pu-239
Des Plains River	Upstream from Sawmill Creek		5×10^{-4} pCi/l Pu-239
	Downstream from Sawmill Creek		8×10^{-4} Pu-239
Illinois River	McKinley Woods State Park		2×10^{-4} pCi/l Pu-239
	Below Dresden Power Station		3×10^{-4} Pu-239
Soil ^(a)	Site Perimeter	Av. of 10 Locations	2×10^{-3} μ Ci/m ² Pu-239 1×10^{-4} Pu-238
	Offsite	Av. of 10 Locations	2×10^{-3} Pu-239 2×10^{-4} Pu-238

(a) Soil samples collected to depth of 30 cm.

Table A 1-21

Environmental Monitoring for the Transuranium Elements at
Battelle-Columbus Laboratories (West Jefferson Site)

Site: Battelle Laboratories (West Jefferson Site) (10,13,24)
Location: Columbus, Ohio
Mission: Reactor Fuel Research (Plutonium Laboratory)

Transuranium Elements Released to the Environment (CY 1975)

To air - 1.5 μCi Pu-239
To surface waters - not published

<u>Media</u>	<u>Sample Collection Location</u>	<u>Average Plutonium Concentrations - CY 1975</u>
Air	(Site Boundary concentration as calculated using atmospheric dispersion equations)	$(4 \times 10^{-3} \text{ fCi/m}^3)$
Silt	Above and Below Outfall	$< 2 \times 10^{-2} \text{ pCi/g (dry)}$
Grass	Onsite and Various Locations Onsite, 3-8 kilometers	$< 2 \times 10^{-2} \text{ pCi/g (dry)}$
Food Crops	Corn, Soybeans, Rye, Vegetables 0.4 to 8 kilometers in Various Directions around Site	$< 2 \times 10^{-2} \text{ pCi/g (dry)}$

Table A1-22

Environmental Monitoring for the Transuranium Elements at
the Idaho National Engineering Laboratory

<u>Site</u>	Idaho National Engineering Laboratory (10,13,24)
<u>Location</u>	Southeastern Idaho; 35 kilometers west of Idaho Falls
<u>Mission</u>	Includes - Fuel reprocessing, calcining liquid radio-active waste, and storage and surveillance of solid transuranic waste

Transuranium Elements Released to the Environment (CY 1975)

To air	- 2 mCi Pu-238, Pu-239, and Np-237
To disposal well	- "very small amounts"

<u>Media</u>	<u>Sample Collection Location</u>	<u>Average Plutonium Concentrations-CY 1975</u>
Air	Boundary Stations	0.02 fCi/m ³ Pu-239 0.01 fCi/m ³ Am-241
Surface Soils	Boundary Stations 18 Samples	$2 \pm 2 \times 10^{-2}$ pCi/g (a)
	Distant Location 12 Samples	$3 \pm 4 \times 10^{-2}$ pCi/g (a)

(a) Soil Samples Collected to Depth of 5 cm.

Table A1-23

Environmental Monitoring for the Transuranium Elements
at the Oak Ridge Facilities

<u>Site</u>	Oak Ridge Facilities (10,13,23)
<u>Location</u>	Oak Ridge, Tennessee
<u>Mission</u>	Multipurpose Research Laboratory, Gaseous Diffusion Plant, and Nuclear Weapons Operations (Y-12 Plant)

Transuranium Elements Released to the Environment (CY 1975)

To air	- 4 μ Ci	sum of all transuranium elements
To Clinch River	- 20 mCi	sum of all transuranium elements (CY 1973 - 80 mCi; CY 1974 - 20 mCi)

<u>Media</u>	<u>Sample Collection Location</u>	<u>Average Plutonium Concentrations-CY 1975</u>
Air	Perimeter Stations	0.014 fCi/m ³ Pu-239 < 0.001 fCi/m ³ Pu-238
	Remote Stations	0.013 fCi/m ³ Pu-239 < 0.001 fCi/m ³ Pu-238
Soil	Perimeter Stations (a)	4x10 ⁻² pCi/g (a) Pu-239
Water	White Oak Creek	Not published
	Clinch River	Not published

(a) Soil Samples Collected to Depth of 1 cm.

Table A1-24

Environmental Monitoring for the Transuranium Elements
at Hanford

<u>Site</u>	Hanford
<u>Location</u>	Southeastern Washington, 320 kilometers east of Portland, Oregon
<u>Mission</u>	Includes Fuel fabrication, liquid waste solidification and radioactive waste burial. Originally, plutonium for nuclear weapons was produced here.

Transuranium Elements Released to the Environment (CY 1975)

To air	1 mCi sum of all plutonium elements
To surface waters -	0.9 mCi sum of all plutonium elements

<u>Media</u>	<u>Sample Collection Location</u>	<u>Average Plutonium Concentrations-CY 1975</u>
Air	Perimeter Stations	<0.03 fCi/m ³ Total Pu
	Distant Stations	<0.04 Total Pu
Soil	Perimeter Stations	<7x10 ⁻³ pCi/g (dry) Pu-239
		<4x10 ⁻⁴ Pu-238
Water	Columbia River-Upstream	<0.03 pCi/l Pu-239
	-Downstream	<0.02 Pu-239
Vegetation	Perimeter Stations	<2x10 ⁻³ pCi/g (dry) Pu-239

Table A 1-25

Environmental Monitoring for the Transuranium Elements at
the Lawrence Livermore Laboratory

<u>Site</u>	Lawrence Livermore Laboratory
<u>Location</u>	Alameda County, California, 64 kilometers east of San Francisco
<u>Mission</u>	Research and development on nuclear weapons

Transuranium Elements Released to the Environment (CY 1975)

to air - not published
To surface waters - not published

Media	<u>Sample Collection Locations</u>	<u>Average Plutonium Concentrations-CY 1975</u>	
	Site 300	0.28 fCi/m ³ 9.0x15 ⁴ fCi/m ³	Pu-239 Pu-238
Air	Perimeter Locations (6)	0.019-0.034 fCi/m ³ 1.9-9.5x10 ⁻⁴	Pu-239 Pu-238
Soil ^(a)	Site 300 (11)	0.001-0.03 pCi/g (dry)	Pu-239
	Livermore Valley (20)	0.001-0.1	Pu-239
Water	Reclamation Plant Effluents	0.6 pCi/l	Pu-239

(a) Soil Samples Collected to Depth of 1 cm.

Annex 1

References

1. Wrenn, McD. E., "Environmental Levels of Plutonium and the Transuranium Elements", in Proceeding of Public Hearings: Plutonium and the Other Transuranium Elements, Vol 1 (ORP/CSD-75-2), U.S. Environmental Protection Agency, Office of Radiation Programs, Washington, D.C. (December 1974).
2. Krey, P.W. et.al., "Mass Istopopic Composition of Global Fall-Out Plutonium In Soil", IAEA-SM/199-39, International Atomic Energy Agency, Vienna, (1976).
3. Hardy Jr., E. R., "Worldwide Distribution of Plutonium", in Proceedings of Public Hearings: Plutonium and the Other Transuranium Elements, Vol 1 (ORP/CSD-75-2), U.S. Environmental Protection Agency, Office of Radiation Programs, Washington, D.C. (December 1974).
4. Hardy, E., "Depth Distribution of Global Fallout Sr-90, Cs-137 and Pu-239 (240) in Sandy Loan Soil" in Fallout Program Quarterly Summary Report (HASL-286) U.S. Atomic Energy Commission, New York, N.Y. (October 1974).
5. Bennett, B. G. HASL, U.S. Energy Research and Development Administration, New York, N.Y., Personnel Communication, (December 1976).
6. Bennett, Burton G., "Transfer of Plutonium From the Environment to Man" in Transuranium Nuclides in the Environment (IAEA-SM-199/40). International Atomic Energy Agency, Vienna (1976).
7. Chart of the Nuclides. Knolls Atomic Power Laboratory Naval Reactor, U.S. AEC (operated by the General Electric Company) 11th Ed, Revised to April 1972.
8. Krey, P. "Atmospheric Burn-up of a Plutonium-238 Generator, Science, 158 No. 3802 pp. 769771 (Nov. 10, 1967).
9. Erdman, C. A. and A. B. Regnolds, Nuclear Safety 16 43 (1975).
10. "Environmental Monitoring at Major U.S. Energy Research and Development Administration Contractor Sites - Calendar Year 1975" (ERDA-76-104) Energy Research and Development Administration, Division of Safety, Standards and Compliance, Washington, D. C. (August 1976) 2 Vols.

11. Gilbert, R. O. et. al., "Statistical Analysis of Pu-239(240) and Am-241 Contamination of Soil and Vegetation on NAEG Study Sites" in the Radioecology of Plutonium and Other Transuranics in Desert Environments (NVO-153) U.S. Energy Research and Development Administration, Nevada Operation Office, Las Vegas, Nevada (June 1975).
12. Hardy, E., "Plutonium in Soil Northeast of the Nevada Test Site", in Health and Safety Laboratory - Environmental Quarterly, (HASL-306) Energy Research and Development Administration, New York, N.Y. (July 1976).
13. "Environmental Monitoring Report for the Nevada Test Site and Other Test Areas Used for Underground Nuclear Detonations - Jan. through Dec. 1973" in Environmental Monitoring at Major U.S. Atomic Energy Commission Contractor Sites - Calendar Year 1973, (WASH-1259 (73) U.S. Atomic Energy Commission, Division of Operational Safety, Washington, D.C. (June 1973).
14. Krey, P. W. "Remote Plutonium Contamination and Total Inventories from Rocky Flats", Health Physics 30, 209 (1976).
15. "Environmental Monitoring at Major U.S. Energy Research and Development Administration Contractor Sites - Calendar Year 1974" (ERDA-54) U.S. ERDA, Division of Operational Safety, Washington, D.C. (August 1975).
16. Krey, P. W. and Hardy, E. P., "Plutonium in Soil around the Rocky Flats Plant in Fallout Program Quarterly Summary Report (HASL 235) U.S. Atomic Energy Commission, New York, N.Y. (1974).
17. Werkema, G. J. and M. A. Thompson, "Annual Environmental Monitoring Report Rocky Flats Plant" in "Proceedings of Public Hearings: Plutonium and the Other Transuranic Element, Vol. 2", ORP/CSD-75-1, U.S. Environmental Protection Agency, Office of Radiation Programs, Washington, D. C. (Jan. 10, 1975).
18. Rogers, D. R., Mound Laboratory Environmental Plutonium Study-1974, (MLM-2249) Mound Laboratory, Miamisburg, Ohio (September 1975).
19. U.S. Environmental Protection Agency, National Environmental Research Center, Cincinnati, Ohio. Letter to Mr. Gary Bramble, State of Ohio, Environmental Protection Agency, from Bernd Kahn (Oct. 1, 1974).
20. McLandon, H. R., "Soil Monitoring for Plutonium at the Savannah River Plant," Health Physics 28, 347 (1975).
21. "Annual Report of the Biomedical and Environmental Research Program of the LASL Health Division - Jan. through Dec. 1974" (LA-5883-PR) Los Alamos Scientific Laboratory, Los Alamos, New Mexico (Feb. 1975).

22. Douglas, R. L. (Report in Press) U.S. Environmental Protection Agency, Office of Radiation Programs, Las Vegas, Nevada.
23. "Cleanup, Rehabilitation, Resettlement of Enewetak Atoll - Marshall Islands (Final EIS) Defense Nuclear Agency, Washington, D. C., 4 Vols. (April 1975).
24. "Environmental Monitoring at Major U.S. Atomic Energy Commission Contractor Sites - Calendar Year 1973" (WASH-1259) U.S. Atomic Energy Commission, Division of Operational Safety, Washington, D.C. (June 1973).

ANNEX II

ENVIRONMENTAL TRANSPORT AND PATHWAYS

U. S. Environmental Protection Agency
Office of Radiation Programs
Washington, D.C. 20460

Annex II

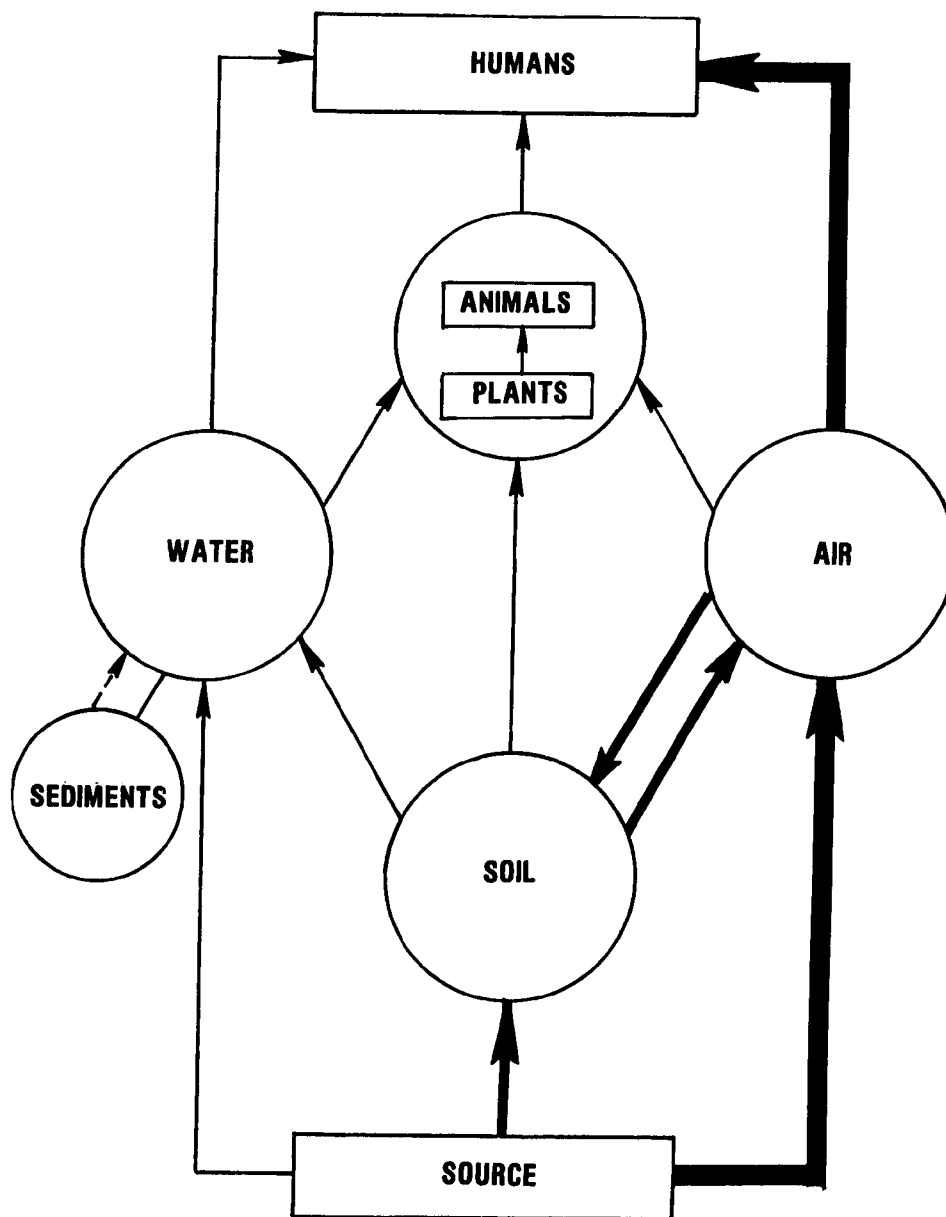
Contents

	<u>Page</u>
1. Objective	1
2. Environmental Transport	2
2.1 Aerosol Transport	2
2.2 Soil Transport	8
2.3 Aqueous Transport	10
3. Exposure Pathways	11
3.1 Inhalation	12
3.2 Ingestion	16
4. Methods of Relating Soil Concentration to Airborne Activity	18
4.1 Resuspension Factor	18
4.2 Mass Loading	19
4.3 Resuspension Rate and Other Approaches	23
5. Derivation of the Screening Level for Soil	25
5.1 Enrichment Factor	26
5.2 Correction for Area Size	29
5.3 Calculation of the Soil Screening Level	30
REFERENCES	36

1. Introduction

The purpose of this annex is to provide a brief overview of the transport of the transuranium elements through the environment and the subsequent exposure pathways which might occur as a result of their release into the biosphere. Availability, uptake, and translocation of the transuranium elements within the ecosystem depend upon many factors. Included among these are: the mode of release (e.g., accidental fire or spillage), the physical form upon release (e.g., particulate or liquid), the chemical form (e.g., elemental, oxide, or nitrate), and the nature of the environment being contaminated (e.g., desert soil or aqueous media). Also important is people's use of the environment which can significantly affect the mobility of the transuranium elements and, subsequently, their effect upon exposed populations. Because there are so many variables, potential pathways of exposure should be evaluated on a site-by-site basis. However, some general conclusions can be made based upon present knowledge of the environmental behavior of these elements although it is limited.

For a terrestrial ecosystem, the major environmental transport pathways are illustrated in Figure A 2-1. These pathways include: 1) exchange between air and soil, water, and vegetation as a result of deposition and resuspension, 2) exchange between soil and water by erosion, leaching, absorption, and precipitation and 3) uptake from air, soil, and water by plants, animals, and man. Highlighted in Figure A 2-1 are the pathways expected to produce the principal exposures to people, which will be discussed in greater detail in the following sections.



**PRINCIPAL PATHWAYS OF THE TRANSURANIUM ELEMENTS
THROUGH THE ENVIRONMENT TO MAN
FIGURE A2-1**

2. Environmental Transport

2.1 Aerosol Transport

Airborne releases of the transuranium elements result from both normal and accidental occurrences. Normal operational releases are small and are expected to decrease in the future due to improvements in containment. At present, doses from normal releases are below the limits recommended in this guidance.

Accidental releases -- such as those resulting from transportation accidents and fires -- can lead to localized contamination, and would probably necessitate some form of protective and remedial action. Generally accidental releases of the transuranium elements will be as the element, the oxide, or in liquid form. When the release is in the form of the element, it will convert rapidly to the oxide form, which is relatively insoluble and stable thermodynamically. Airborne releases of the transuranium elements will generally be as an oxide containing a substantial percentage of particles with diameters less than 10 μm . This is the size range roughly corresponding to the respirable range of particle sizes. Because of the small settling velocities associated with such particles they can be transported long distances by air currents before depositing on the ground as a result of wet and dry deposition. These particles then will become incorporated into soil and aquatic systems. When deposited on soil, the aerosol particles can attach themselves to the larger, less mobile soil particles. For example, Mork (1) found that plutonium at test sites was usually

bound to soil particles with a diameter greater than 44 μm . Likewise, Tamura (2) has analyzed the plutonium bound to soil particles at the Nevada Test Site and showed that the plutonium bound to coarse particles (5-20 μm) was present as PuO_2 , while the plutonium bound to fine particles (2-5 μm) was present as hydrated PuO_2 .

Subsequent transport will be as a result of wind and mechanical forces which transfer their energy to the surface particles causing them to roll, slide or even become airborne. The smaller the particle diameter the greater will be the tendency for the particle to stay airborne and the greater will be the distance that it will travel before returning to the surface. Some of the many factors which can influence the redistribution of surface particles by wind are listed in Table A 2-1. The multiplicity of factors and their complex interrelationship makes the prediction of soil resuspension and transport a very complex problem. Accordingly, the resuspension of soil particles has been the focus of much research over the past several years.

One of the more commonly used indexes of resuspension has been the concept of a resuspension factor which is defined as the ratio of the air concentration to soil concentration (units; meters^{-1}). A wide range of values has been reported for resuspension factors which covers a variety of surfaces and modes of disturbance. Table A 2-2 is a summary of some values reported (3) for newly deposited PuO_2 released during weapons testing. Such a wide range of values makes the prediction of the resuspension factor for a particular set of

Table A 2-1

Factors Influencing Wind Suspension

<u>AIR</u>	<u>GROUND</u>	<u>SOIL</u>
Velocity	Roughness	Structure affected by:
Turbulence	Cover	Organic Matter
Density affected by:	Obstructions	Lime Content
Temperature	Temperature	Texture
Pressure	Topographic Features	Specific Gravity
Viscosity		Moisture

SURFACE PROPERTIES

Large-scale surface roughness
 Mechanical turbulence
 Overall sheltering
Small-scale surface roughness
 Sheltering of individual particles
Area of erodible surface
Vegetative cover
 Live vegetation
 Plant residue
Cohesiveness of individual particles
 Moisture of surface
 Binding acting of organic materials

PARTICLE PROPERTIES

Particle size frequency distribution
 Ratio of erodible to nonerodible fractions
Particle density
Particle shape

METEROLOGY FACTORS

Wind velocity distribution in the surface layer
 Mean wind speed
 Wind direction
 Frequency, period, and intensity of gusts
 Vertical turbulent exchange

Moisture content on ground surface
 Precipitation
 Dew and frost
 Drying action of the air

Table A 2-2

Short Summary of Experimental Results on Resuspension
of activity in the Air [After Stewart (1967)]

Measurement Conditions	Resuspension Factor, R_f (m^{-1})	
	Range	Mean
Plutonium sampled at 1 ft above ground (1)		
Vehicle traffic	3×10^{-4} to 7×10^{-4}	
Pedestrian traffic	1.5×10^{-6} to 3×10^{-4}	
Particle size: Mainly 20-60 μm , with 1% in hazardous range ($\sim 3 \mu m$ for PuO_2)		
Uranium sampled downwind from a crater (1)		
At 1 ft. above ground (dust stirred up)		1×10^{-3}
At 1 ft. above ground		3×10^{-4}
At 2 ft. above ground		1×10^{-5}
Brick/plaster dust sample contaminated with I-131 (2)		
Enclosed space	2×10^{-4} to 4×10^{-5}	
Open space		2×10^{-6}
Sample in cab of Landrover, after a test (1)		
Round 1 (H + 18 hr)		2.5×10^{-5}
Round 2 (H + 5 hr)		6.4×10^{-5}
Airborne material without artificial disturbance of ground, consisting of limestone rock and sand with coarse grass and small bushes (3)	1×10^{-6} to 8×10^{-5} (12 results)	1×10^{-5}
Random samples following a tower shot, without artificial disturbance, near crater (3)	1×10^{-8} to 1×10^{-6} (9 results)	2×10^{-7}
On two roads formed by soil grading -no artificial disturbance (3)	1.5×10^{-6} to 1×10^{-8} (14 results)	2.5×10^{-7}
At back of a moving Landrover (3):		
D-Day + 4 (21 results)	8×10^{-7} to 3×10^{-5}	1.4×10^{-5}
D-Day + 7 (21 results)	6×10^{-7} to 4×10^{-6}	1.5×10^{-6}
D-Day + 7 over tailboard	1.6 and 3.1×10^{-5}	2.5×10^{-5}
(1) From nuclear weapon and other tests at Maralinga		
(2) From Civil Defense trial at Falfield, Gloucester		
(3) From Hurricane Trial		

conditions a difficult task. In general, however, values for newly deposited material seem to fall in the range $10^{-5}(\text{m}^{-1})$ to $10^{-7}(\text{m}^{-1})$ under conditions of low mechanical disturbance. In areas where the surface is rocky or paved, the resuspension factor may range up to $10^{-3}(\text{m}^{-1})$ due to these smoother, harder surfaces and because little mixing with noncontaminated surfaces occurs (4). Mechanical disturbances, such as vehicular traffic, will also increase the resuspension factor by as much as a factor of 10 to 100 (5).

For planning purposes, Stewart (3) and others (6) have recommended a resuspension factor of $10^{-6}(\text{m}^{-1})$ for freshly deposited material under quiescent conditions but recommended increasing this value to $10^{-5}(\text{m}^{-1})$ if there is moderate vehicular or other disturbing activity. As the freshly deposited material becomes aged, fixed to the soil or mixed with the soil, the characteristics of the contaminant approach the resuspension characteristics of the soil itself. On the basis of empirical information, a model has been proposed (7) in which the resuspension factor decreases from $10^{-5}(\text{m}^{-1})$ to $10^{-9}(\text{m}^{-1})$ within two years. Bennett has reportedly (8) estimated that in a humid eastern climate the resuspension factor reaches $10^{-6}(\text{m}^{-1})$ to $10^{-7}(\text{m}^{-1})$ after the first good rain or wet down and then rapidly decreases to $10^{-9}(\text{m}^{-1})$. If the trans-uranium material is released as a solution rather than as the oxide, its resuspension will probably be in the low range (see Section 2.2). Experiments (3) with yttrium chloride solution sorbed onto soil have indicated a resuspension factor of $10^{-9}(\text{m}^{-1})$, while measurements at

Mound Laboratory (4) of Pu-238 released from a waste transfer line produced resuspension factors in the range of $10^{-8}(\text{m}^{-1})$ to $10^{-9}(\text{m}^{-1})$.

Because of the propensity for greater mobility on the part of freshly deposited material, stabilization of newly contaminated land should be undertaken as soon as possible after the initial accident in order to reduce the resuspension and inhalation exposure. In addition to the benefit of reducing the hazard from inhalation, prompt stabilization of the contamination should result in lower cleanup costs. This lower cost arises from the fact that dispersion of the material is being minimized through stabilization and, therefore, less land area will be impacted and require cleanup.

2.2 Soil Transport

Soil contamination by plutonium has been the most prevalent situation encountered and, therefore, is the most widely studied. Plutonium dispersed onto soil has demonstrated a tendency to bond chemically and/or physically with the soil rather than exist as a separate entity (4,9,10,11,12). Plutonium oxide is relatively inert and initially attaches itself to the soil matrix as a result of adhesive forces established between the plutonium particle and the soil substrate. Over a period of time, weathering processes such as freezing, thawing, and precipitation, will begin to "solubilize" the oxide.

Although generally considered to be insoluble, plutonium oxide can undergo dissolution in a neutral aqueous media. The plutonium oxide particle dissolves producing plutonium ions until the formation of a hydrated coating inhibits further dissolution. The rate and degree of

dissolution depends on many factors including pH, temperature, the presence of oxidizing, reducing, and complexing agents, as well as the specific activity of the radionuclide. The dissolution rate of $^{238}\text{PuO}_2$, for example, has under certain circumstances been found (13) to be 100 times greater than that of $^{239}\text{PuO}_2$. Plutonium ions formed during the dissolution can undergo ion exchange reactions with the oxygenated ligands commonly found in soil (e.g., silicates) and become sorbed onto the soil, or react with other agents present in the aqueous phase and form soluble complexes. Chemicals that complex the plutonium compete with the silicate particles for the plutonium and tend to reduce the extent of plutonium sorption on soil.

When plutonium is released to soil as a solution (e.g., as a nitrate) it will already be in ionic form and, in such situations, has been shown (4) to react rapidly with soil. Plutonium ions are capable of displacing most cations (e.g., calcium, magnesium, sodium, etc.) generally found in soils and of forming strong chemical bands. Several studies (2,14,15) have shown that, after sorption of plutonium has occurred, it will not be readily displaced from the soil by natural processes.

Once in the soil, the transuranium elements can be depleted through the migration of particles down through the surface or through the resuspension of a fraction of the material back into the air stream. Of these two mechanisms, the resuspension of soil particles, with which these nuclides have associated in one form or another, will be the principal mode of further environmental transport. The resuspension of

soil particles occurs as a result of wind action and more intermittently as a result of mechanical forces; such as, plowing and vehicular disturbances.

The size of the particle will determine its distance and mode of transport. Particles with diameters greater than 1000 μm generally slide or roll along the surface (creep), while particles with diameters in the range of 50 μm to 1000 μm move in short hops along the surface, usually at a small distance from the surface (saltation). The suspension of particles is generally restricted to those below 50 μm , which will be carried along with the air stream.

2.3 Aqueous Transport

Studies have been conducted on various water bodies, streams, rivers, lakes, estuaries and oceans to determine the final disposition of plutonium in these environs. The following behaviors have been noted:

- (1) More than 90% of the plutonium becomes bound to suspended sediments and carried to the sediment bed.
- (2) Situations where reducing and complexing agents are both present can lead to resolubilization of the plutonium in the sediment bed.
- (3) Seaweeds generally have the ability to concentrate plutonium with concentration factors of ~ 1000 .
- (4) Benthic biota can alter the plutonium concentration profiles in the sediment beds.

Specifically, plutonium oxide exposed to an aqueous medium undergoes slow dissolution, producing various complex ions of plutonium as well as polymers in colloidal form and the hydrous oxide as a precipitate (13). In 1972 Langham (16) studied the fate of PuO_2

following the Thule incident and found that the majority of the plutonium agglomerated into inactive debris with only about 1% suspended as fine particulates in the water. Further studies (17) after the Thule incident showed 95% of the plutonium to be associated with the bottom sediments to a depth of at least 10 cm. In addition, a study (18) of nuclear waste discharged into the Irish Sea from Windscale has found most of the Pu-239 and Am-241 to be associated with the sediments close to the discharge area. Similar findings were observed (4) around Mound Laboratory where plutonium was accidentally discharged into a freshwater canal. Again the plutonium was found to be largely associated with the bottom sediments. Therefore, although the movement of the transuranium elements through aqueous systems is not yet well defined, the present information available would indicate a limited mobility for environmental transport via such systems.

3. Exposure Pathways

The principal hazard that arises as a consequence of soil being contaminated with the transuranium elements is exposure to radiation through the inhalation and ingestion pathways.

For a detailed discussion of the entry of plutonium and other actinides into animals and man and the resultant biological behavior, the reader is referred to ICRP Publication 19 (19) and Annex III of this document. The following section will be limited to a description of the environmental factors affecting the inhalation and ingestion pathways.

3.1 Inhalation

Inhalation exposures arise from direct injection of transuranium radionuclides into the atmosphere (e.g., normal emissions and accidental fires) and also from the resuspension of previously deposited material. For the latter pathway, only a very small fraction of the material on the surface actually becomes airborne and available to man. In general, the respirable size is considered to be that range of particles with aerodynamic diameters less than 10 μm .

An assessment can be made, using dosimetry models, of the potential health hazard resulting from the inhalation of airborne particles. Such a model requires knowledge of the total airborne activity and of the activity median aerodynamic diameter associated with it. (Aerodynamic diameter is the diameter of a unit density sphere having the same settling velocity as the particle in question of whatever shape and density.) The assumption made by most dosimetry models is that the aerosol distribution is log normal and can, therefore, be described through the use of two parameters - the activity median aerodynamic diameter (AMAD) and the geometric standard deviation of the distribution, σ_g . The PAID code used by EPA (see Annex III) assumes σ_g to be 1.5 and, therefore, only the AMAD of the distribution need be determined.

In determining the AMAD of the distribution, care should be exercised to assure that the entire distribution is being measured. Aerosol sampling is complicated by the fact that every sampler has its

own characteristic upper size cut-off, which depends on its entry shape, dimensions, and flow rate. When the aerosol being sampled contains large particles with activity associated with them, the gross air activity being measured may be underestimated. Likewise, sampling techniques may be biased against the smaller particle sizes. If particles from the resuspending soil have a disproportionate amount of activity associated with them, the inhalation hazard could be underestimated. Therefore, sampling should be conducted so that (1) the total airborne distribution is being measured, and (2) the AMAD determined actually describes the corresponding distribution of airborne activity.

Healy (20) and others (21) have emphasized the necessity of considering the resuspension of soil by mechanisms other than normal wind activity. The possibility exists that other mechanisms could, under certain circumstances, produce exposures exceeding those normally received via the resuspension pathway. Although this possibility has been recognized, relatively little experimental data is currently available to determine quantitatively the importance of the many possible secondary resuspension mechanisms. Two commonly encountered disturbances (agricultural operations and vehicular disturbance) have recently been investigated, however, and some conclusions can be drawn from these studies.

For the agricultural situation, the vicinity of a field contaminated over a period of twenty years was monitored. Increase in airborne activity was measured during such activities as plowing,

disking, and planting (22). During these operations, the air activity was found to increase by a factor of approximately 30 at the location of the tractor operator and by a factor of 6 at a distance 30 meters away from the edge of the field. Assuming that these activities take place 30 days of the year for 8 hours each day (i.e., $\frac{1}{36}$ of the year), it can be calculated that the average yearly air activity will increase by 80 percent for the tractor operator and 10 percent for an individual in the vicinity of the field. For the individual at the edge of the field, this is a conservative calculation, since it is doubtful that any one individual would be standing at that location for the entire period of time. Also, this level of increased air activity should occur for only the first year. Subsequent agricultural activities should generate lower air concentrations, because the activity originally on the surface will be diluted through mixing with soil previously below it.

The conclusion that can be drawn from such an analysis is that these agricultural operations would pose an increased inhalation hazard to the vehicle operator during the first cultivation cycle, and some protective action might be in order during that time. Subsequent cultivation, however, should not lead to significant increases in the inhalation hazard. For surrounding areas, no significant inhalation hazard would be predicted during any of these operations.

Regarding vehicular disturbances, Sehmel (5) has examined the importance of auto and truck traffic in increasing resuspension. It was concluded that such disturbances, in the case of an asphalt surface

with newly deposited material, will lead to increased resuspension, with a fraction resuspended of the order of 10^{-5} to 10^{-2} per vehicle passage. The higher rates occurred at speeds typical of freeway driving; after the passage of about 100 cars only a small fraction of the original contamination would remain on the road surface. The material resuspended from the road surface deposited on the ground at various distances from the road and was again available for resuspension, but at a much lower rate.

The potential for increased exposures from such situations will depend upon many factors in addition to the quantity of contaminating material, including the time of exposure, the frequency of vehicular passage and the speeds, and the distance from the road to the receptor. Based upon Sehmel's experiment, it can be expected that the integrated inhalation exposure due to the vehicular disturbance will be smaller than the chronic exposure received from just living within the generally contaminated area. The material deposited on the road surface will be depleted quickly and, once it is removed from there, its resuspension will be orders of magnitude lower. Sehmel's results indicate that the material transferred to the road parking strip resuspends at a rate only one tenth of that on the road itself. In addition, the total quantity of material resuspending at the higher rate would be small relative to the surrounding area; once redistributed over the larger area, it should show little increase in the average air concentration.

3.2 Ingestion

Under normal circumstances, exposures via ingestion will arise from the consumption of crops and animals grown on land contaminated by the transuranium elements. Studies to date have assessed the ingestion pathway from two directions. Some studies have looked at the uptake factors for various plant species grown in contaminated soils, while others have measured the residual amounts of fallout plutonium in processed foods.

Two recent publications (23,24) have conveniently tabulated the results of the many uptake studies performed on plutonium and other transuranium elements. These studies have shown plant and animal uptakes to be very small, with the concentration in plants (fresh weight basis) being generally less than 10^{-4} of that in dried soil and the concentration in animal tissue (fresh weight) being about 10^{-5} of that in the plants they eat. Preliminary studies (23) of transuranium elements other than plutonium produced uptake factors somewhat higher than comparable studies with plutonium. Some initial studies (25,26) seem to indicate an increase in uptake with time, possibly as a result of bacterial action or increased solubilization. The use of chelating agents as a part of agricultural practices may also increase the uptake of the transuranium elements with time (27).

Measurements of plutonium in "market basket" food samples, in which proportions of processed foods are chosen to represent the annual total diet, can be used (28) as indicators of the quantity of plutonium ingested. Such findings apply to a given soil contamination level when

all consumed food is grown on contaminated lands. The methodology gives an overestimate of activity ingested, because foods from other areas not as highly contaminated will make up part of the diet. These studies have observed uptake factors for plutonium in the range of 10^{-4} plus or minus an order of magnitude. Based upon such uptake factors and food consumption estimates, the annual estimated intake during 1972 of fall-out plutonium was 1.6 pCi while the intake for 1965 was estimated to be 2.6 pCi. There is no reason to believe that the uptake factors for crops grown on land with concentrations higher than fallout levels should vary significantly from those obtained through this "market basket" sampling technique. Some evidence indicates that americium is concentrated in certain species of plants relative to plutonium. However, preliminary analyses (29) of the "market basket" samples indicate that the Am 241/Pu 239 ratios in diets are not greatly different from current Am 241/Pu 239 ratios in soil.

The ingestion of plutonium through drinking water is another possible pathway to humans. The concentration of fallout plutonium in finished drinking water has been found (28) to be low (3 fCi/l). However, in areas of elevated levels, plutonium could migrate over-time into cisterns and wells, thus increasing the activity in drinking water. Likewise, it has been suggested (20) that a significant ingestion pathway could be the accidental ingestion of contaminated soil by adults or the deliberate ingestion of soil by children. However, for this pathway to be as significant as the inhalation pathway, extreme assumptions of soil consumption rates would be required because of the

low uptake factors and short residence time of plutonium in the gastrointestinal tract.

4. Methods of Relating Soil Concentration to Airborne Activity

The relationship between soil and air concentrations is affected by many complex factors (see Table A 2-1). Attempts to derive values for them have resulted in many different approaches; each uses different concepts and methods of measurement, selecting some physical factors as important and tending to neglect others or include them as constants. The purpose of this section is to describe briefly some of the more commonly used approaches to relate soil contamination levels to airborne radioactivity.

4.1 Resuspension Factor

One of the earliest and still most widely used methods of predicting the relationship between soil and air contamination is the resuspension factor. It is defined as the ratio of the plutonium concentration in air, measured at some distance above the ground, to that of plutonium in the soil:

$$K(m^{-1}) = \frac{\text{concentration in air (activity/m}^3\text{)}}{\text{concentration in soil (activity/m}^2\text{)}} \quad \text{Eq. 1.}$$

The resuspension factor, however, does have limitations in its application. In the first place, it assumes that the air concentration above a contaminated surface is directly proportional to the surface contamination level, rather than on the extent of ground contamination upwind of the sampling site which is indeed the case. In the second place, the resuspension factor is an empirically determined

value which can be applied only to prevailing conditions at a given site and at a given time. Most resuspension experiments have been conducted for a relatively short duration of time and do not necessarily represent the long-term average situation for a particular area. Finally, applying a resuspension factor derived at one particular site to predict airborne contamination levels at another site would be a questionable extrapolation. However, for areas where the resuspension factor has been measured over a period of time, sufficiently long to average out the variability of the local meteorology, then this approach can be useful in assessing the potential hazard from existing soil contamination.

4.2 Mass Loading

One attempt to increase the capability of predicting soil resuspension has been the mass loading approach. This technique assumes the mass loading of the air with particulates to be an index of resuspension and derives the airborne concentration of a specific radionuclide by a comparison with its concentration on the adjacent surface. Specifically,

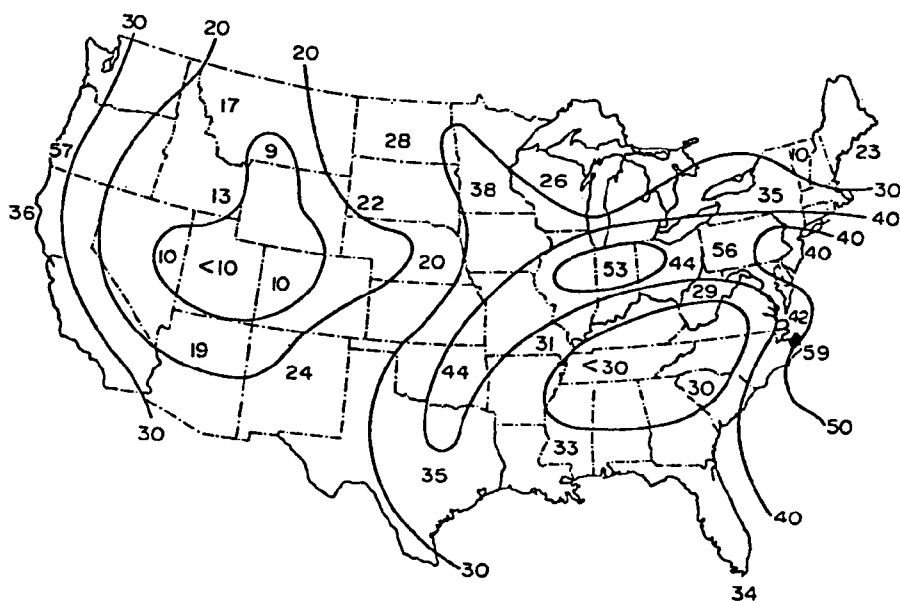
$$\begin{aligned} \text{Air Concentration (fCi/m}^3\text{)} &= \text{Soil Concentration (}\mu\text{Ci/m}^2\text{)} \times \\ \text{Mass Loading (}\mu\text{g/m}^3\text{)} &\times \text{C.F.*} \end{aligned} \quad \text{Eq. 2.}$$

Airborne particulate mass loading is one of the criteria for clean air standards and measurements are widely available for urban and

* where C.F. is the units conversion factor based upon the depth of sampling and the soil density.

nonurban locations through the National Air Surveillance Network (NASN). The data recorded at nonurban stations are a better indicator of the levels of resuspended material than are urban measurements. In general, annual mean mass concentrations of airborne particulate material at the nonurban stations range from 5-50 micrograms per cubic meter (see Fig. A 2-2); the mean arithmetic average for 1966 of all 30 nonurban NASN stations was $38 \mu\text{g}/\text{m}^3$ (30).

Ansbaugh (30,31) employed this model to predict air concentrations at a number of sites. Predicted values did not exceed measured values by more than a factor of roughly five (Table A 2-3). This fallacy of the model is in assuming that the resuspendible fraction of the soil would carry with it an equal fraction of the activity, which implies essentially that: 1) activity is distributed homogeneously in the top soil and, 2) activity exists independently of particle size. For instance, if the specific ground activity is associated mostly with particles of size greater than $50 \mu\text{m}$, a very small air concentration would result, although the model would predict the same air concentration for this case as it would for all the activity being distributed among particles of resuspendible size. In either case the model would fail. Data obtained (2,4,15) at sites of present contamination have shown a non-uniform distribution of activity with particle size, probably caused by such factors as: 1) the chemical form of the plutonium when released 2) the ion exchange capacity of the soil and 3) the surface area of the soil particles. It would seem reasonable, however, that the error associated with using the mass loading



ANNUAL MEAN MASS CONCENTRATIONS ($\mu\text{g}/\text{m}^3$) OF AIRBORNE PARTICLES FROM NON-URBAN STATIONS OF THE U.S. NATIONAL AIR SAMPLING NETWORK. 1964 - 1965

FIGURE A2-2

Table A 2-3

OBSERVED AIR CONCENTRATIONS COMPARED WITH CONCENTRATIONS PREDICTED
BY MASS LOADING MODEL [ADAPTED FROM ANSPAUGH ET AL. (1974) AND
ANSPAUGH ET AL. (1974)]

Location, etc.	Radionuclide	Air Concentration	
		Predicted ^a	Measured ^b
<u>GMX site, USAEC Nevada</u>			
<u>Test Site</u>			
NE, 1971-1972	²³⁹ Pu	7200 aCi/m ³	6600 aCi/m ³
GZ, 1972, 2 weeks	²³⁹ Pu	120 fCi/m ³	23 fCi/m ³
<u>Lawrence Livermore</u>			
<u>Laboratory</u>			
1971	²³⁸ U	150 pg/m ³	52 pg/m ³
1972	²³⁸ U	150 pg/m ³	100 pg/m ³
1973	²³⁸ U	150 pg/m ³	86 pg/m ³
1973	⁴⁰ K	1000 aCi/m ³	980 aCi/m ³
<u>Argonne National</u>			
<u>Laboratory</u>			
1972	²³² Th	320 pg/m ³	240 pg/m ³
1972	nat _U	215 pg/m ³	170 pg/m ³
<u>Sutton, England</u>			
1967-1968	nat _U	110 pg/m ³	62 pg/m ³

^a Predicted value is equal to the soil concentration (activity/g) x
10⁻⁴ g/m³.

^b Most values are annual averages.

approach would be least for soils in which the contaminant has been present for some time and in making predictions of average annual air concentrations.

4.3 Resuspension Rate and Other Approaches

Other approaches of a more sophisticated nature have been developed to describe the resuspension of particles from a soil surface. These approaches have attempted to include in their formulations as parameters some of the physical forces which control the resuspension phenomenon. One such technique proposed by Healy and Fuquay (32) is the resuspension rate approach. This model combines atmospheric transport and diffusion along with particle resuspension to calculate airborne concentration. This is achieved by assuming that the rate of pickup of particles from a surface is directly proportional to the ratio of wind forces to gravity forces on individual particles. Taking the wind force on a particle as proportional to the square of the wind velocity and to the particle area exposed to wind, the model develops a formulation for the resuspension rate, i.e., the rate at which particles will be resuspended by wind from a soil surface. Once the resuspension rate has been determined it can be used as the source term in a standard atmospheric diffusion equation to predict the resultant air concentration at some distance from the contaminated site. Recently, Healy (20) has refined the model formulations to be capable of handling various geometric configurations of the contaminated area and the variability of surface concentration within the contaminated area. The advantages of a model

of this type are that it recognizes some of the physical conditions and processes which affect resuspension as well as providing a method to calculate air concentration at various distances away from the contaminated area.

One assumption by Healy in the original formulation of the resuspension rate model (20) was that the pickup rate for particles is a function of the square of the wind velocity. Presently, studies are being conducted by Sehmel to establish the relationship between resuspension rate and wind velocity. One such study conducted by Sehmel (33) at Rocky Flats, for short time periods and for one sampling station, found the air concentration to be a function of the square of the wind velocity implying that the pickup rate was a function of the cubic power of the wind speed. However, other experiments by Sehmel (34) have shown resuspension rates to increase with windspeed to the 6.5 power. Studies are continuing to better elucidate this functional relationship.

Other approaches (35,36) are also under development which attempt to relate particle resuspension to such factors as the soil erodibility index, surface roughness factor, and quantity of vegetative cover-to mention just a few. These models generally require the determination of several empirical constants in their formulation. Although these constants may be applicable for the conditions under which they have been measured, the general applicability of these formulations in predicting air concentration has not been demonstrated at this time.

5. Derivation of the Screening Level for Soil

A screening level for soil has been derived to minimize the area around a contaminated site which must be monitored as well as the number of soil samples which must be collected and analyzed. When the trans-uranium activity in soil is at or below this concentration, it is highly unlikely that the exposure levels recommended in this Guide will be exceeded. The screening level is not to be interpreted as a soil cleanup standard to which all sites of transuranium contamination must be decontaminated; instead, when correctly applied, it will identify land areas where no additional monitoring is required. Because of the conservative assumptions that have been incorporated into the calculation of the screening level, it is anticipated that present and future contaminated sites will not require cleanup to the screening level.

The screening level was derived after careful consideration of all currently contaminated sites, placing particular emphasis on areas for which enough site-specific data is available covering factors as particle size and soil activity distributions. After examining these data, a hypothetical site was defined with a combination of parameters chosen to be conservative, i.e., to produce an acceptable level of transuranium activity more restrictive than that which would be derived for any of the existing sites. This conservative approach has been taken due to the uncertainties inherent in any calculational model, and because of the limited experience with contamination by the trans-uranium elements. Sites of future contamination are also likely to have site characteristics which would permit levels of contamination higher than the screening level.

Of the various models that have been suggested for relating soil contamination levels to airborne concentrations, the Agency has opted to use the mass loading approach in deriving a soil screening level. This approach has been shown (30,31) to provide some capability in predicting air concentrations on an annual basis at existing contaminated sites and, since the objective of this Guidance is to relate soil contamination levels to annual dose levels, annual average air concentrations are required. Additionally, since the screening level is a generic value with application at all sites, the Agency chose not to use one of the more sophisticated resuspension models requiring detailed site-specific parameters such as wind speed, atmospheric stability class, soil erodibility index, etc. In applying the mass loading model to calculating a soil screening level, some modifications have been made, however, in an effort to overcome some of the shortcomings which are fundamental to the approach and which were discussed earlier (Sect. 4.2).

5.1 Enrichment Factor

In an effort to take into consideration the non-uniform distribution of activity with soil particle size as well as the non-uniform resuspension of particle sizes, the Agency has derived an "enrichment factor" which is included in the mass loading calculation. Potential exposure due to contaminated soil depends largely on the amount of activity associated with particles in the respirable size range (generally $< 10 \mu\text{m}$). It has been suggested by Johnson (21) that sampling of only those particles in a soil sample

which are within the inhalable size range would give the best measure of risk to the public health. However, the weight fraction of particles in the less than 10 μm range is small in most soils, and sampling, separation, and analysis techniques are correspondingly more difficult and inaccurate. There is also considerable evidence that some of the larger particles really consist of aggregates and are relatively easily broken down into smaller ones, so that an instantaneous measurement of a single size range may not give a good picture of long-term trends. Another important objection to limited sampling is that larger particle sizes may make a substantial contribution to other possible pathways (e.g., ingestion), and hence should be measured.

To assess the potential hazard of the inhalable fraction of soils, while retaining the advantages and convenience of analyzing the entire soil sample, the Agency has modified the mass loading approach by use of an "enrichment factor". The proposed method weights the fraction of the activity contained within the respirable range in terms of its deviation from the activity to mass ratio for the entire sample and at the same time addresses the problem of the nonuniform resuspension of particle sizes mentioned in the previous section.

The inhalable fraction of the soil is weighted by considering the relative distribution of activity and soil mass as a function of particle size for representative samples of soil. To accomplish this, the sample of contaminated soil is segregated into size increments and the activity and mass contained within each size increment is determined. The factor g_i is then defined as the ratio of the fraction

of the total activity contained within increment i to the fraction of the total mass contained within that increment. A value greater than 1 for g_i implies an enrichment of activity in relation to mass within that incremental fraction, while a value less than 1 indicates a dilution of the activity with respect to mass. For g_i equal to 1, the fraction of the activity and the mass contained with increment i are the same.

The nonuniform resuspension of particle sizes must also be considered. This is achieved in the modification of the mass loading calculation by measuring the mass loading as a function of particle size. The fraction of the airborne mass contained within each size increment, i , is then calculated and designated as f_i . The factors of f_i and g_i can then be incorporated into the mass loading formulation as follows:

$$\text{Air Concentration}_i = \text{Air Mass Loading} \times f_i \times \text{Soil Concentration} \times g_i \quad \text{Eq.3.}$$

Summation over all the size increments results in the total air concentration:

$$\text{Air Concentration} = \text{Air Mass Loading} \times \text{Soil Concentration} \times \sum_i f_i g_i \quad \text{Eq.4.}$$

The term $\sum_i f_i g_i$ weights the contribution of the plutonium from each soil size fraction to the total resuspended material, thereby taking into account both the nonuniform resuspension of particle sizes as well as the nonhomogeneous distribution of activity.

For purposes of this guidance, $\sum_i f_i g_i$ will be referred to as the "enrichment factor" where the f factor accounts for the distribution of airborne mass as a function of particle size and the factor g accounts for the variability of both soil activity and soil mass as a function of particle size.

5.2 Correction for Area Size

Use of the mass loading approach implies that the air concentration is at equilibrium with the ground surface, i.e., a steady state situation exists in which the amount of material coming up from the surface is balanced by the amount of material depositing back onto the surface. In the strictest sense this limit can only be achieved for source areas approaching infinite dimensions. For source areas of finite dimensions a fraction of the airborne mass loading can be arising from an uncontaminated area upwind which, although contributing dust to the atmosphere, contributes no radioactivity. The smaller the size of the contaminated area the less it will contribute to the mass loading level and the greater the uncertainties involved in applying the mass loading model.

Healy (37) has recently attempted to quantify the relationship between the size of the contaminated area and the air concentration that would result from it. His calculations show that, for a contaminated area which is 50 meters in horizontal depth, the air concentration would be approximately a factor of one hundred smaller than from an area 5000 meters in depth (based upon certain assumptions regarding prevailing meteorology). Obviously, a correction for area size becomes necessary when applying the mass loading approach to small

areas of contamination.

In deriving the screening level for soil, the Agency has assumed the area contaminated to be sufficiently large that a correction for area size is not necessary. It is recognized that this is a conservative assumption and that areas of actual contamination will require a correction for area size; however, since one cannot predict a priori the extent of a contamination incident nor the prevalent meteorology, the conservative case has been assumed.

5.3 Calculation of the Screening Level

The following assumptions were made in deriving the screening level: 1) the mass loading for the hypothetical site was taken to be $100 \mu\text{g}/\text{m}^3$ and to have a particle size distribution similar to that reported by Chepil (38) for resuspended dust, 2) the soil is enriched with activity in the respirable size range relative to the soil as a whole, 3) the contamination is widely dispersed and a correction for area size is not appropriate and 4) there are no restrictions as to land use.

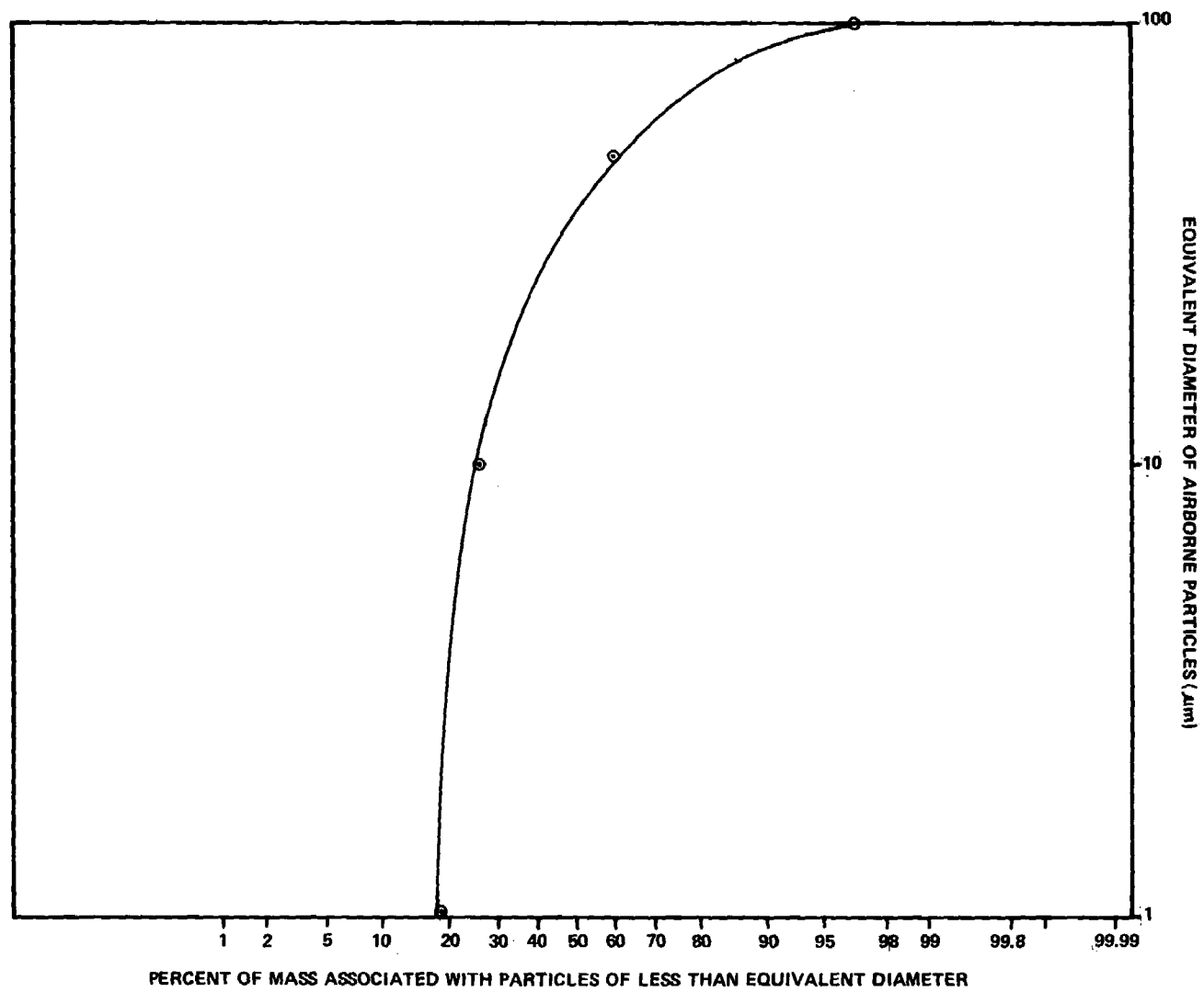
An annual average mass loading of $100 \mu\text{g}/\text{m}^3$ is higher than the annual average for any non-urban site reported by the NASN (Fig. A2-2) and is indicative of higher resuspension for the hypothetical site. Anspaugh (30) has examined the data from the NASN stations and has concluded that $100 \mu\text{g}/\text{m}^3$ is a sufficiently conservative value to use in mass loading calculations. This level of mass loading is also consistent with the value of $120 \mu\text{g}/\text{m}^3$ arrived at by Healy (20) in proposing an interim standard for plutonium in soil. In addition,

100 $\mu\text{g}/\text{m}^3$ was considered to be a sufficiently conservative mass loading value and was applied in assessing the potential inhalation hazard from plutonium contamination around Mound Laboratory (4).

The particle size distribution of the resuspended soil, for use in calculating the screening level, is from Chepil's (38) data obtained from fields undergoing wind erosion in Colorado and Kansas. The results of his findings have been plotted by Slinn (35) and adapted as Figure A 2-3. Comparision of Chepil's data with other studies substantiates the applicability of his results to other areas. For example, Chepil found 30% of the airborne mass to be below 10 μm versus a study by Sehmel (39) around the Hanford site where 28% of the mass was below 10 μm (under mass loading conditions of approximately 30 $\mu\text{g}/\text{m}^3$) and a study by Willeke (40) in the area around Denver where 33% of the measured airborne mass was below 10 μm (mass loading < 100 $\mu\text{g}/\text{m}^3$).

Currently, soil particle size and activity distribution data are available for five sites with plutonium contamination: Mound Laboratory, Oak Ridge National Laboratory, and the Nevada Test Site (analyses by Tamura [15]), and the Trinity Test Site and the Rocky Flats Plant (analysis by the USEPA). Of these sites, the greatest enrichment of activity within the fine particle size range is found in samples from the Rocky Flats area. For this reason, the Rocky Flats soil distribution (see Table A 2-3) was used in calculating the screening level.

Since the size of the contaminated area varies greatly from site to site, and because of the inability to predict the extent of future



PARTICLE SIZE DISTRIBUTION OF RESUSPENDED SOIL

FIGURE A2-3

contaminated areas, no reduction for area size was incorporated into the calculation of the screening level. Likewise, the conservative assumptions of free access to the contaminated site with no limitations on land usage were made in determining the screening level.

Finally, the air concentration which corresponds to the pulmonary dose rate limit of 1 mrad/yr is equal to 2.6×10^{-15} Ci/m³. This concentration was obtained from Table A 3-4 of Annex III by assuming an activity median aerodynamic diameter of 1 μ m.

The above assumptions have been incorporated into the modified mass loading formula (Eq. 4) and the screening level calculated as follows:

$$\text{Screening Level } (\mu\text{Ci}/\text{m}^2) = \frac{\text{Air Concentration } (\text{fCi}/\text{m}^3)}{\text{Mass Loading } (\mu\text{g}/\text{m}^3) \times \sum_i f_i g_i \times \text{C.F.*}} \quad \text{Eq. 5}$$

$$\text{Screening Level } (\mu\text{Ci}/\text{m}^2) = \frac{2.6 \text{ fCi}/\text{m}^3}{100 \mu\text{g}/\text{m}^3 \times 1.5 \times 6.6 \times 10^{-2}} \quad \text{Eq. 6}$$

$$\text{Screening Level} = .26 \mu\text{Ci}/\text{m}^2 \quad \text{Eq. 7}$$

As discussed earlier, it is highly unlikely that any present or future site would have a combination of site-specific factors which would produce an acceptable soil concentration more restrictive than

* C.F. is the units correction factor and is equal to 6.6×10^{-2} when a soil density of 1.5 g/cm³ is assumed for a 1 cm dry soil sample.

Table A 2-3

<u>Sample</u>	<u>Size Increment (μm)</u>	<u>Wgt. Fract.</u>	<u>Act. Fract.</u>	<u>g₁</u>	<u>f₁</u>	<u>Σ f₁ g₁</u>
RF 1A	2000-105	.62	.07	.12	-	
	105-10	.18	.40	2.21	.7	
	<10	.20	.53	2.65	.3	2.34
RF 1B	2000-105	.63	.39	.63	-	
	105-10	.17	.06	.34	.7	
	<10	.20	.55	2.74	.3	1.06
RF 1C	2000-105	.64	.43	.68	-	
	105-10	.16	.07	.46	.7	
	<10	.20	.49	2.47	.3	1.06
RF 2A	2000-105	.46	.13	.28	-	
	105-10	.34	.37	1.10	.7	
	<10	.20	.50	2.48	.3	
						av. <u>1.51</u> 1.49

*Sampling and analysis by USEPA, Office of Radiation Programs, Las Vegas Facility.

this screening level. To illustrate this, the resuspension factor for this hypothetical site can be calculated:

$$R. F. (m^{-1}) = \frac{2.6 \times 10^{-15} \text{ Ci/m}^3}{2.6 \times 10^{-7} \text{ Ci/m}^2} \quad \text{Eq. 8}$$

$$R. F. = 1.0 \times 10^{-8} \text{ m}^{-1} \quad \text{Eq. 9}$$

This value for the resuspension factor is a factor of 5-10 higher than values reported for any of the existing sites. Sites of future contamination, after stabilization, should have resuspension factors no higher than the present unstabilized sites and, therefore, the screening level value has applicability to sites of future contamination as well.

References

1. H. M. Mork, Redistribution of Plutonium in the Environs of the Nevada Test Site, UCLA-12-590, University of California Press, Los Angeles (1970).
2. T. Tamura, "Distribution and Characterization of Plutonium in Soils from Nevada Test Site," in The Dynamics of Plutonium in Desert Environments, NVO-142, USAEC, Las Vegas, Nevada (1974).
3. K. Stewart, "The Resuspension of Particulate Material from Surfaces," in Surface Contamination, B. R. Fish (ed.), Pergamon Press, New York, N. Y. (1964), pp. 63-64.
4. D. R. Rogers, Mound Laboratory Environmental Plutonium Study 1974, MLM-2249, Mound Laboratory (1975).
5. G. A. Sehmel, "Particle Resuspension from an Asphalt Road Caused by Car and Truck Traffic," Atm. Env., 7, p. 291 (1975).
6. W. H. Langham, Biological Considerations of Nonnuclear Incidents Involving Nuclear Warheads, UCRL-50639, Lawrence Livermore Laboratory (1969).
7. Environmental Statement for LMFBR, WASH-1535, Appendix 11.G, USAEC, Washington, D. C. (1974).
8. B. L. Cohen, The Hazards in Plutonium Dispersal, GEZ-6521, General Electric (1975).
9. J. A. Hayden, "Characterization of Environmental Plutonium by Nuclear Track Techniques," in Atmospheric-Surface Exchange of Particulate and Gaseous Pollutants, CONF-740921, ERDA, Washington, D. C. (1974).
10. M. W. Nathans, R. Reinhart, and W. D. Holland, "Methods of Analysis Useful in the Study of Alpha-Emitting and Fissionable Material Containing Particles," in Atmospheric-Surface Exchange of Particulate and Gaseous Pollutants, CONF 740921, ERDA, Washington, D. C. (1974).
11. G. A. Sehmel, "A Possible Explanation of Apparent Anomalous Airborne Concentration Profiles of Plutonium at Rocky Flats," Pacific Northwest Laboratory Annual Report for 1974, to the USAEC Division of Biomedical and Environmental Research, BNWL-1950, Pt. 3, Atmos. Sci., p. 221 (1975).
12. M. W. Nathans, The Size Distribution and Plutonium Concentration of Particles from the Rocky Flats Area, TLW-6111, LFE Corporation (1972).

13. J. H. Patterson, G. B. Nelson, G. M. Matlock, The Dissolution of ^{239}Pu in Environmental and Biological Systems, LA 5624, Los Alamos Laboratory (1974).
14. M. Sakanoue and T. Tsuji, "Plutonium Content of Soil at Nagasaki," Nature, 234, p. 92 (1971).
15. T. Tamura, "Physical and Chemical Characteristics of Plutonium in Existing Contaminated Soils and Sediments," in Proceedings of the International Symposium on Transuranium Nuclides in the Environment (Nov. 1975), IAEA, Vienna.
16. W. H. Langham, "Biological Implications of the Transuranium Elements for Man," Health Physics, 22, p. 943 (1972).
17. A. Aarkrog, "Radioecological Investigation of Plutonium in an Arctic Marine Environment," Health Physics, 20, p. 30 (1971).
18. A. Preston and N. Mitchell, "Evaluation of Public Radiation Exposure from the Controlled Marine Disposal of Radioactive Waste," in Radioactive Contamination of the Marine Environment, IAEA STI/PUB/313, International Atomic Energy Agency, Vienna, p. 575 (1973).
19. ICRP Publication 19, 1972. The Metabolism of Compounds of Plutonium and Other Actinides, Pergaman Press.
20. J. W. Healy, A Proposed Interim Standard for Plutonium in Soil, LA 5483-MS, Los Alamos Scientific Laboratory (1974).
21. C. J. Johnson, R. R. Tidball, and R. C. Severson, "Plutonium Hazard in Respirable Dust on the Surface of Soil," Science, 193, p. 488 (1976).
22. R. C. Milham, J. F. Schubert, J. R. Watts, A. L. Boni, and J. C. Corey, "Measured Plutonium Resuspension and Resulting Dose from Agricultural Operations on an Old Field at the Savannah River Plant in the Southeastern U. S.," in Proceedings of the International Symposium on Transuranium Nuclides in the Environment, (Nov. 1975), IAEA, Vienna.
23. R. L. Thomas and J. W. Healy, An Appraisal of Available Information on Uptake by Plants of Transplutonium Elements and Neptunium, LA 6460-MS, Los Alamos Scientific Laboratory (1976).
24. R. A. Bulman, Concentration of Actinides in the Food Chain, NRPB-R44, National Radiological Protection Board, Harwell, England (1976).
25. E. M. Romney, H. M. Mark, and K. H. Larson, "Persistence of Plutonium in Soil, Plants, and Small Mammals," Health Physics, 19, p. 487 (1970).

26. P. Neubold, Absorption of Plutonium-239 by Plants, ARCRL-8, St. Brit. Agr. Res. Council (1962).
27. W. V. Lipton and A. S. Goldin, "Some Factors Influencing the Uptake of Plutonium-239 by Pea Plants," Health Physics, 31, p.425 (1976).
28. B. G. Bennett, "Environmental Pathways of Transuranic Elements," in Proceedings of Public Hearings: Plutonium and the Other Transuranium Elements, Vol. 1, ORP-CSD-75-1, USEPA, Washington, D.C., (1974).
29. B. G. Bennett, private communication.
30. L. R. Anspaugh, "The Use of NTS DATA and Experience to Predict Air Concentration of Plutonium Due to Resuspension on the Enewetak Atoll," in The Dynamics of Plutonium in Desert Environment, NVO-142, USAEC, Las Vegas, Nevada (1974).
31. L. R. Anspaugh, J. H. Slinn, D. W. Wilson, "Evaluation of the Resuspension Pathway Toward Protection Guidelines for Soil Contamination with Radioactivity," in Proceedings of the International Symposium on Transuranium Nuclides in the Environment (Nov. 1975), IAEA, Vienna.
32. J. W. Healy and J. J. Fuquay, "Wind Pickup of Radioactive Particles from the Ground," Progress in Nuclear Energy Series XII, Health Physics, V.1, Pergamon Press, Oxford, (1959).
33. G. A. Sehmel and M. M. Orgill, "Resuspension by Wind at Rocky Flats," Annual Report for 1972, BNWL-1751, pt. 1, Battelle Pacific Northwest Laboratory, Richland (1973).
34. G. A. Sehmel and F. D. Lloyd, "Particle Resuspension Rates" in Atmospheric-Surface Exchange of Particulate and Gaseous Pollutants, CONF 740921, ERDA, Washington, D.C., (1974).
35. W. G. N. Slinn, "Dry Deposition and Resuspension of Aerosol Particles - A New Look at Some Old Problems," *ibid.*
36. J. H. Shinn, N. C. Kennedy, J. S. Koval, B. R. Clegg, and W. M. Porch, "Observations of Dust Flux in the Surface Boundary Layer for Steady and Non-Steady Cases," *ibid.*
37. J. W. Healy, An Examination of the Pathways from Soil to Man for Plutonium, LA-6741-MS, Los Alamos Scientific Laboratory (1977).
38. W. S. Chepil, "Sedimentary Characteristic of Dust Storms: III Composition of Suspended Dust," Am. J. Sci., 225, p. 206 (1957).

39. G. A. Sehmel, Radioactive Particle Resuspension Research Experiments on the Hanford Reservation, BNWL-2081, Battelle Pacific Northwest Laboratory, Richland (1977).
40. K. Willeke, K. Whitby, W. Clark, and V. Marple, "Size Distribution of Denver Aerosols-A Comparison of Two Sites," Atm. Env., 8, p. 609 (1974).

Annex III

THE DOSE AND RISK TO HEALTH DUE TO THE
INHALATION AND INGESTION OF TRANSURANIUM NUCLIDES

U. S. Environmental Protection Agency
Office of Radiation Programs
Washington, D.C. 20460

CONTENTS

- 3.1 Introduction
- 3.2 Risks
- 3.3 Exposure Pathways
- 3.4 Dosimetry of Inhaled and Ingested Plutonium, Americium, and Curium
 - 3.4.1 The Dose to Lung Tissues
 - 3.4.2 The Dose to Bone, Liver, and the Total Body
 - 3.4.3 The Dose to Gonadal Tissue
 - 3.4.4 Dose Models for the Ingestion Pathway
- 3.5 The Risk of Lung Cancer from Inhaled Transuranics
- 3.6 The Risk of Bone Cancer
 - 3.6.1 Inhalation Risk Estimates
 - 3.6.2 Ingestion Risk Estimates
- 3.7 The Risk of Inducing Cancer of the Liver
 - 3.7.1 Inhalation Risk Estimates
 - 3.7.2 Ingestion Risk Estimates
- 3.8 The Risk of Genetic Damage
 - 3.8.1 Genetic Risk Estimates
- 3.9 Other Risks Due to the Inhalation and Ingestion of Plutonium
 - 3.9.1 Leukemia due to Bone Marrow Irradiation
- 3.10 Summary of Health Risks
 - 3.10.1 Inhalation Pathway
 - 3.10.2 Ingestion Pathway

References

Tables

Figures

Annex 3

The Dose and Risk to Health Due to the Inhalation and Ingestion of Transuranium Nuclides

May 23, 1977

3.1. INTRODUCTION

The purpose of this chapter is to outline the methods recommended by the Agency to estimate the dose and potential health effects from some of the transuranium elements in the environment. An attempt has been made to balance the discussion between scientific details and understandability by laymen. Where it is impossible to satisfy both needs, the documentation cited should supply more details for the interested reader.

Information on the biological properties of transuranium elements is reviewed in "Selected Topics: Plutonium in the Environment" which includes an extensive bibliography (1). The amount known about the metabolisms of each of the transuranic elements varies depending on their commercial importance as well as other factors. Considerable information is available on the distribution of plutonium in most human tissues, the gonads being a notable exception. The biological data base for the dosimetry of americium and curium is not as extensive, which limits the accuracy of estimates of the dose due to these radionuclides.

3.2. Risks

Although much information is available on the somatic effects of plutonium inhalation and ingestion by laboratory animals, no relevant epidemiological information is available on the effects of plutonium

or other transuranium elements on humans via these exposure pathways (1). When human data is available, the Environmental Protection Agency prefers to base estimates of health risk due to radiation on the results of human epidemiological studies rather than animal experiments. To derive estimates of the cancer risk due to plutonium and other transuranium elements, the Agency relies on human experience with other alpha particle emitters. (In a few cases transuranics emit radiation other than alpha particles but such emissions make relatively unimportant contributions to the dose.) Estimates of genetic risk are not based directly on human studies. Assumed doubling doses for genetic injury are based almost exclusively on irradiated animal populations.

Data on human experience following alpha particle irradiation is largely confined to occupational and medical exposures. The Agency's chief reference documents for the effects of ionizing radiation on health is the 1972 BEIR Report prepared by the National Academy of Sciences (2), and the NAS Report, "Health Effects of Alpha-Emitting Particles in the Respiratory Tract" (3), the latter document being used to estimate lung cancer risk. Following the procedure used by the BEIR Committee in arriving at its "best estimate" of radiation risk, it is the practice of the Agency to average results obtained by the two types of risk models utilized in the NAS-BEIR Report (2), absolute¹

¹Probability of cancer per organ rad.

and relative risk¹, respectively. These two models can yield results differing by as much as a factor of about seven depending on the particular cancer being considered, duration of risk following exposure, etc. (2). Therefore, in addition to other uncertainties in the risk estimates there is a residual uncertainty of a factor of three or more in the average of the risk estimates listed below, depending on which of these models is appropriate for a particular organ system.

The Agency's risk estimates are based on an assumed linear relationship between dose and the probability that a cancer is induced. No threshold dose for effects is hypothesized and no allowance is made for enhanced or reduced effects due to the relatively low dose and dose rates realized from alpha particle exposures. It should be noted that the Agency does not consider the risks due to ionizing radiation hypothetical and that for highly ionizing radiation such as alpha particles from plutonium, the linear non-threshold hypothesis is unlikely to overestimate the actual risks.

In the case of estimating genetic risks, the overall uncertainty is larger than for estimating the risk of cancer induction. The NAS-BEIR Committee report (2) indicates that this uncertainty has two components: an estimated uncertainty of a factor of ten in the dose required to double the human mutation rate and for common diseases

¹Percent increase of cancer per organ rad.

thought to have a mutational component, an additional uncertainty of a factor of ten. Obviously, only a broadly defined range of genetic risk due to transuranium radionuclides can be estimated at the present time.

Risk estimates for ionizing radiation are often expressed in terms of the rem, a unit of dose equivalence used in radiation protection practice to provide at least some degree of equality between the biological damage produced by different kinds of radiation. Since this guidance is limited to alpha particle emitters, both dose and risk estimates are expressed in a more fundamental physical unit, the rad, the specific energy imparted. The advantages of this approach are that it is more straightforward and that the guidance will not need periodic revision to account for differences between various kinds of radiation that may be proposed. Future information on health effects will, however, be factored into these guides as the need arises.

An air concentration yielding 1 mrad per annum to adults will result in children receiving a somewhat larger dose rate. The dose rate to children cannot be calculated as accurately as for adults because the necessary lung model parameters are not known. As a first approximation to the dose rate as a function of age, the reduced breathing rate (minute volume) and smaller organ mass of children have been used to estimate their increased annual dose. This provides a conservative estimate of childhood dose since deposition and retention in the lung should be less for children due to their smaller lung

area. A more detailed treatment of the dose to children is not likely to result in appreciable difference in the risk since most of a person's body burden is accumulated during adult life, not childhood.

Specific information on the carcinogenic effects of alpha particle or other highly ionizing radiations on children's lungs is not available. No lung cancer deaths have been reported as yet in children irradiated at Hiroshima. Nevertheless, as mentioned in Sections 3.5 and 3.6, the risk estimate of excess lung and liver cancer mortality allow for observed differences in the radiosensitivity of children and adults, based on data for other radiogenic cancers (2). In the case of lung cancer, these differences have a large effect on the estimated risks. Estimates based on the BEIR absolute risk model include an assumption that children are less sensitive to radiation than adults. Estimates based on the relative risk model assume children are ten times more sensitive.

Unfortunately, there is paucity of information which can lead to a choice between these two models on scientific grounds. It should be noted that the Agency's use of a logarithmic averaging of the results obtained by the absolute and relative risk models results in a bias in favor of the former. However other assumptions in the risk model are somewhat conservative so that on balance it is unlikely that the true risks are not underestimated.

3.3. EXPOSURE PATHWAYS

In general, the most important pathway for human exposure from plutonium oxide and other transuranium radionuclides in the environment is expected to be inhalation. This route provides a direct pathway for alpha particles to enter a sensitive organ, the pulmonary lung. Subsequently, a fraction of the inhaled material is redistributed via the blood to such important organs as the bone, liver and gonadal tissues. This is in contrast to the ingestion pathway, where the gut walls act as a barrier to plutonium absorption by blood. The dose to the gut wall itself is not a major cause of concern because, unlike other radiations, plutonium alpha particles have a short finite range in tissue, 41 microns (u), i.e., less than two-thousandths of an inch. Radiosensitive dividing cells in the gut wall are over 100 u distant from the gut contents and are effectively isolated from the alpha radiation.

The dose to various tissues from inhaled plutonium is highly time dependent. Insoluble materials deposited in the pulmonary lung are removed fairly rapidly; half is assumed to be cleared within 500 days. Clearance from other organs is much slower; the estimated biological half-life¹ in the liver is assumed to be 40 years, and in the case of bone, 100 years (4). The dose delivered to an organ is directly related to the residence time of the radioactive material. Following

¹The time required to eliminate one-half of the initial organ burden.

a single acute exposure to airborne plutonium, the lung (pulmonary) dose rate decreases due to the clearance of particles from the lung so that almost all of the dose is received in a few years. In contrast, the dose rates to the liver and bone are relatively constant over this time span.

In the case of chronic environmental contamination leading to a constant annual intake, the temporal pattern of the dose rate to various organs due to inhalation is different. For pulmonary tissues, a constant (equilibrium) dose rate is realized within a relatively few years of the start of exposure. The dose rate to liver increases more slowly and does not equal that to lung tissue until after about 70 years of exposure. The dose rate to bone never approaches that in the lung and liver (Figure A3-1). Therefore, the total risk from chronic inhalation will vary with the duration of exposure. In setting guides for the dose due to inhalation, the Agency has selected the annual dose rate to lung (pulmonary tissue) as the appropriate limit. This choice is warranted because administrative controls for the inhalation pathway can be more easily instituted on the basis of an annual dose limit to lung rather than a lifetime dose limit for lung and other tissues. This does not mean the risks from exposure to organs other than lung have been overlooked. The estimates of the total risk from inhaled radionuclides of transuranium elements made below account for the dose to all important organ systems and are, necessarily, a function of the duration of exposure so as to reflect the varying dose rate for liver and bone shown in Figure A3-1. For highly insoluble

transuranics, the additional risks due to liver and bone irradiation due to inhalation are believed to be small compared to the risk of lung cancer, as outlined below.

Use of lands contaminated with low concentrations of plutonium or other transuranium radionuclides are expected to be strictly controlled, unless the resulting doses are below the level specified in this guidance. Uncontrolled land utilization implies that such ordinary uses of land as farming, residency, industrial use, etc. are not precluded. All of these activities and particularly residential use could lead to exposures extending over several decades and in some cases lifetime exposure. In the section below, the lung cancer risk from plutonium inhalation is calculated on the basis of an annual limiting dose rate of one mrad per annum occurring throughout a persons life. In many cases of environmental contamination, the concentration of plutonium in air would be expected to decrease with time so that the life time dose, and risk, would be smaller.

3.4. DOSIMETRY OF INHALED AND INGESTED PLUTONIUM, AMERICIUM, AND CURIUM

3.4.1 The Dose to Lung Tissues

Dosimetric models for projecting the average distribution of ionizing radiation within body organs due to the inhalation of radioactive aerosols are still somewhat crude (1). The most promising general model is that developed and published in ICRP Report #19, "The Metabolism of Compounds of Plutonium and Other Actinides" (4), as an amended version of a model developed earlier for the ICRP (5). In

general, ICRP report #19 is the basis for the estimates of dose made in this section. Where it has been supplemented by more recent data, specific reference is made. Since the inhalation model is documented in ICRP #19 (4) and in reference 5, only its basic outline is described here.

Inhaled aerosols are considered on the basis of particle size and other aerodynamic parameters. The fraction of inhaled materials initially retained or exhaled, and the deposition in various portions of the respiratory tract, is a given function of a particle's activity median aerodynamic diameter (AMAD). The physiological parameters used in this study were taken from reference 5. A diagram of the ICRP Task Group Lung Model is shown in Figure A3-2. The rate at which deposited material is removed from the lungs is considered to be a function only of the chemical state of the inhaled material, and not of size or radioactive content. Environmental sources of plutonium and other transuranium elements are likely to be in the oxide or hydroxide form. Actinides in either form are currently classified as Class Y (insoluble) materials by the ICRP (4). Such materials take years to be cleared from the lung; their estimated biological half life in pulmonary tissue being 500 days (4). The dose estimates made below apply only to Class Y compounds. Dose estimates for the inhalation of more soluble, Class W, materials are given in reference 6. The breathing rate, retention half-time and fraction of material handled by the various elimination pathways for relatively insoluble material deposited in the lung that are assumed in this model are shown in

Table A3-1, from ICRP #19 (4). A critique of the ICRP model is included in (1).

In assessing the dose and risk due to the inhalation of transuranium elements only retention in the pulmonary region is of primary importance. Residence time of inhaled materials in the nasopharyngeal and tracheobronchial regions is short compared to that in pulmonary tissues.

Radioactivity is assumed to leave the pulmonary tissues by three routes: elevation up the tracheobronchial tree via the mucus elevator into the gut by way of the esophagus and stomach, transport of particulate materials into the lymphatic system and lymph nodes and most important by dissolution into the blood stream. Most of the activity in blood is redeposited into the liver and bone.

The dose to different organs from inhaled aerosols of various sizes has been considered by a number of workers who have written computer codes to quantify the ICRP model (7,9). Since the ICRP model is not described in the form of unambiguous equations, there are minor differences between the results obtained with various codes. The EPA code PAID (9) has been used to calculate the annual dose rates due to isotopes of plutonium, americium, and curium for a number of particle sizes. Typical results for five micron, one micron, and five one-hundredths micron, AMAD plutonium-239 aerosols (1 fCi/m³) are shown in Table A3-2 for the various compartments in the lung for the case of lifetime exposure (70 years). It is seen from the Table that most of the radiation insult is delivered to pulmonary tissue. Given

equal concentrations in air, the dose to the pulmonary lung is not very sensitive to particle size. For example, the size range from 0.05 to 5.0 micron (AMAD), the pulmonary dose decreases by a factor of about five, as shown in Figure A3-3.

Doses calculated by the PAID code are obtained by averaging the energy deposited by alpha particles throughout the entire organ mass (570 grams in the case of pulmonary tissues) (10). While some have argued (11) that the energy imparted from deposited aerosols should not be averaged in such a manner, current scientific opinion holds that it is a sufficiently conservative way to estimate the radiation damage even though some cells receive more or less dose than the average for all cells (3).

The ICRP model assumes that 10% of the radioactive material transferred to the lymph nodes from pulmonary lung is retained permanently, and 90% is retained for a half time of 1000 days before being released as soluble material into the bloodstream. As a result, the dose rate to these nodes is high compared to that received by pulmonary tissue, as shown in Table A3-3. This is not believed to be an important consideration in estimating risk, since the frequency of radiation induced cancers in respiratory lymph nodes appears to be very small if not zero. From animal studies, it is certain that this risk is small compared to the frequency of radiogenic cancers at other sites (12,13).

3.4.2. The Dose to Bone, Liver and the Total Body

A portion of the aerosols initially deposited in lung tissue is soluble, eventually enters the bloodstream and is redeposited into other body organs. The current ICRP model assumes that 45% of such material is redeposited in bone, 45% in liver, and 10% in soft tissue and excreta (4). This 10% in soft tissue and excreta is roughly divided into 7% in soft tissue and 3% in excreta based on data in reference 14. Release from these organs is slow; 40 years is the assumed half-life in liver and 100 years the assumed half-life in bone (4) and perhaps longer (no observed release) in gonadal tissue. For this assessment it is assumed that a 100 year half-life is appropriate for both bone and gonadal tissue. Percent deposition of other transuranium elements is similar to that for plutonium and ICRP Report #19 has recommended that the percentages shown above be used for all transuranics, a practice followed here. Animal data which differentiates between deposition by the various transuranium elements is discussed in (1).

Table A3-3 shows the dose rates to various body organs from continuously inhaled plutonium-239 (oxide), americium-241, curium-244 and plutonium-241 as a function of years of exposure based on the assumption that all are class y particulates. Dose rates have been calculated by averaging the energy absorbed over the total organ: 1800 grams for liver; 5000 grams for bone; 15 grams for the respiratory lymph nodes (10). Figure A3-2 shows the pathways for these translocated inhaled materials. Almost all of the dose to liver and

bone is from material in the lungs and lymph nodes that has been dissolved and transferred via the blood. Transfer to blood of swallowed materials is much less important. For example, depending on particle size, only 0.06% to 0.35% of the annual dose to bone shown in Table A3-3 is due to inhaled plutonium oxide crossing the gut wall.

Lifetime dose rates for plutonium-238, plutonium-239, plutonium-240, and the two member radionuclide chains, Pu-241/Am-241, Am-241/Np-237, and Cm-244/Pu-240 have been calculated to aid implementation of this guidance (6, 9). Table A3-4 lists the concentration in air of transuranium element aerosols that should deliver an annual alpha dose rate of one mrad per year to adult pulmonary tissues. Particle sizes in Table A3-4 range from 0.05 μ to 5 μ (AMAD). Over this range of particle sizes the limiting concentrations varies by a factor of about five.

3.4.3. The Dose to Gonadal Tissue

The degree to which transuranium elements are translocated from human blood to gonadal tissues is not well known due to the analytical difficulty of making reasonably precise measurements at the low activity levels usually involved, and the high variability between various individuals. Besides limited information from studies of laboratory animals, there are three sources of post-mortem human data; the general population exposed to fallout plutonium, industrially exposed radiation workers, and a few clinical studies with hospital patients. Richmond and Thomas reported that for the five animal species considered in their 1974 review, an average of 0.03% of the

plutonium in blood was transferred to gonadal tissue (15). The data on which this average is based varied by a factor of about ten. A review of clinical data, based on only four persons, also leads to an estimate of about .03% for transfer from blood to gonadal tissue (14).

The Medical Research Council (MRC) also reviewed this problem in their 1975 analysis of plutonium toxicity and concluded that 0.05% of the plutonium in blood would be transferred to gonadal tissue (16). Since the mass of the ovary is 11 grams, the MRC estimate on transfer from blood is equivalent to 0.005% per gram of ovary. The mass of testes is greater than that of the ovary by a factor of about 3. The MRC assumed equal quantities of plutonium in each, so that the concentration (percent per gram) in the testes would be about one-third of that in ovarian tissue i.e., 0.002% per gram. Richmond and Thomas estimated that the amount of plutonium in the smaller female gonad was a factor of five to ten less than males, a somewhat less conservative assumption. Recent data reported for plutonium in beagle gonads indicates that the concentration per gram is about 0.0055% in ovaries and 0.0012% in testes which supports the MRC viewpoint (17).

For calculational purposes it is convenient to relate concentration per gram of transuranics in gonadal tissue to that in bone. According to the ICRP model, 0.09% of the plutonium in blood is concentrated in one gram of bone (4). Based on the MRC study and the results cited above this is about twice the concentration (% per gram) of plutonium in the female gonads; and for testes about five times greater.

Autoradiographic studies in mouse testes indicate that because of the deposition of plutonium near the spermatogonial stem cells, the effective dose to these cells is about 2 to 3 times greater than that to the testes as a whole. (No comparable study of the pattern of deposition for the ovary has been reported). To account for this factor, it is assumed that the effective genetic dose due to transuranium elements in male gonads will be the same as in the female.

The turnover rate of transuranium elements in gonadal tissues is known to be small compared to that of other soft tissues (4, 18, 19). For these calculations the biological half-life in gonadal tissue is assumed to be the same as in bone, 100 years. As in bone, the buildup of transuranium elements in gonadal tissue will occur rather slowly in the case of chronic ingestion or inhalation. The significant gonadal dose in terms of genetic risk is that received during the first 30 years of life (1). From Figure A3-1 it is seen that the dose rate to gonadal tissue over a 30-year period is considerably less than that received by pulmonary tissue. For an equilibrium pulmonary dose rate of 1 mrad per year, the dose to gonadal tissue in the first 30 years of life is calculated to be 1.4. mrad (15). The dose to gonads and other organs due to ingestion is considered below.

3.4.4. Dose Models for the Ingestion Pathway

The magnitude of calculated doses due to the ingestion of transuranic materials is directly proportional to the fraction of the ingested material that is assumed to cross the gut wall and enter the blood stream. This fraction is not well known and it is reasonable to assume that it varies considerably depending on the solubility of the ingested material. Animal experiments to measure gut transfer that have used highly insoluble laboratory prepared materials in the oxide form yield transfers in the parts per million range (4). However, plutonium oxide found in the environment has been shown to be much more soluble than the refractory oxides utilized in animal experiments (20). This is in agreement with recent experiments showing plutonium oxides formed at low temperatures are more soluble than those formed at high temperatures (21). Therefore it is reasonable to assume that plutonium oxidized in the environment will not be as insoluble as the materials which have been used to determine plutonium gut transfer in animals. Moreover, because the quantitative applicability of animal data to man is unknown, conservative estimates that will not underestimate the dose to humans are required. Information on the transfer of transuranium elements across the gut wall, as reported in references 4 and 22 as well as in the reports of more recent studies (23, 24, 25, 26) were reviewed and the transfer coefficients shown in Table A3-5 adopted for use in the dose calculations for this Guidance. The fractional transfers listed are much higher than currently assumed

by the ICRP (4) and are thought to be conservative estimates applicable to public health problems.

The degree of conservatism in these transfer fractions varies depending on how much is known about a particular material, more conservatism being applied to materials for which information is less available. The lowest transfer, 10^{-4} , is assumed to occur for low specific activity plutonium oxides, which have been utilized in many experimental situations with several species. Oxides of some of the other transuranium elements may behave similarly but have been studied less extensively. Transuranium elements in non oxide form, and the oxide of high specific activity transuranium elements such as Pu-238, are somewhat more soluble than plutonium-239 oxide. Present evidence indicates the transfer to blood may increase by an order of magnitude or even more if the element is incorporated into organic materials (1). It should be noted that the fractions listed in Table A3-5 are applicable to adults and children over one year of age. The special case of infants is discussed in Section 3.6.

The estimates shown in Table A3-5 have been used in the dose calculations described below. Most of the plutonium-239 and plutonium-240 in the environment is assumed to be in the oxide form. Dose calculations for these radionuclides assume one pCi in 10^4 pCi ingested is transferred to blood; for all other transuranium elements; ten pCi per 10^4 pCi ingested. No allowance was made for transuranium elements "biologically" incorporated into ingested materials. This is assumed to happen when transuranium elements are incorporated into

plant and animal tissues at the molecular level in contrast to the surface contamination of foodstuffs. Where the fraction of such material in the diet can be estimated, the dose estimates made below should be increased in proportion to the increased amount transferred to blood. For example, if 10% is incorporated into biological material, the dose (and risk) would be increased by 40%.

Subsequent to their transfer across the gut wall, ingested radionuclides enter the blood stream and are deposited primarily in liver and bone and to a lesser extent gonadal tissue, as outlined above in the description of the lung model. As stated in Section 3.2, the dose to the intestinal walls is not considered in this analysis because of the low likelihood of alpha particle penetration to sensitive cells in the intestines. An ICRP Task Group has suggested, as surrogate for information on the dose to dividing cells, that calculations of maximum permissible concentration of transuranium elements in air and water be based on the assumption that 1% of the alpha energy is absorbed (27,28). However, this is highly unrealistic for the purpose of predicting health effects and is not used here.

The transfer factors from gut to blood shown in Table A3-5 have been utilized to calculate annual dose rates as functions of the duration of ingestion. Dose rates to bone, liver, and the total body of reference man (10) due to the chronic ingestion of 1000 pCi/annum of plutonium-239 oxide, americium-241, and curium-244 are shown in Table A3-6. Pu-241, half life 14.8 years, and Cm-244, half life 17.9 years, are the most rapidly decaying transuranium elements capable of

causing chronic exposures in a contaminated environment. Because of their short half lives, the occurrence of lifetime ingestion of these radionuclides is remote. This is even more true of curium-242, half life 0.45 years, where only acute intake is a plausible mode of exposure.

The dose rate to bone in the 70th year due to a constant rate of ingestion of 10 nCi per year are listed in Table A3-7 for a number of transuranium elements. To determine if the bone dose limit is being exceeded, the total dose rate due to the ingestion of a combination of transuranium elements can be calculated from these data.

Table A3-8 lists the cumulative 30-year dose to gonadal tissue due to the chronic ingestion of several transuranium elements at an annual intake of 1000 pCi per year. The gonadal dose has been calculated as described above in Section 3.4.3.

The organ dose rates listed in Table A3-6 were calculated on the basis of organ masses appropriate for reference man, not children. However, there is enough proportionality between food intake and organ mass as a function of age so that the results are applicable to a lifetime exposure situation. Because there is some evidence from animal studies that the newborn have a particularly high transfer of transuranium elements across the gut wall to blood, the transfer fractions listed in Table A3-5 may not be applicable to infants, (less than one year old). The duration of this increased transfer is unknown for humans, perhaps a few weeks or considerably longer. To test for what effect this may have on lifetime doses, it has been

assumed below that the GI tract to blood transfer factor during the first year of life is one hundred times greater than the value shown in Table A3-5, i.e., 10^{-2} in the case of plutonium oxide (26). Any increased transfer due to the incorporation of the plutonium into food is included in this factor of 100.

Ingestion of contaminated food by infants would cause a large initial dose rate to body organs. After the first year, the dose rate decreases due to organ growth, and possibly a more rapid turnover rate of minerals in infant bone compared to adults. However, because turnover rates for children are poorly known, this factor has not been included in calculating the skeletal dose. As an example of enhanced transfer of radioactivity across the infant gut, Table A3-9 compares the skeletal dose rates when transfer is increased by a factor of 100 in the first year of life with the dose rate pattern to reference man. Increased dose rate during the first year of life has less effect on lifetime dose rates than might be expected. Risk analyses of the type outlined in Section 3.6 indicate that the dose pattern shown for enhanced transfer in Table 3.9 would result in 50% more bone cancers than are estimated below, for reference man.

Because infant feeding in the U.S. relies heavily on a variety of non-local foods, it is unrealistic to assume that 100% of the first year's intake would be produced only in contaminated areas. A possible exception, milk, would be less contaminated than other locally produced food stuffs because the transfer of most transuranium elements into milk (animal) following ingestion of the radioisotope is

only about 0.0001 percent per liter (29, 30). Transfer of americium and curium to milk is about 100 times greater than other transuranium elements (31) but is still small.

3.5. THE RISK OF LUNG CANCER FROM INHALED TRANSURANICS

In most cases, the lung is the organ of primary concern when assessing the risks from plutonium and other transuranium elements in soil. Animal studies, particularly those with dogs (15), which have a relatively long life span, indicate that lung cancer can result from inhaled plutonium aerosols as do extensive experiments with rats (32). Even so, the assessment of the risk to humans cannot be directly inferred from animal evidence. Almost all lung cancers in dogs exposed to plutonium occur in a different location than radiogenic cancers in humans following exposure to radon daughters. The animal cancers are in the peripheral parts of the lung and often of different cell types than human lung cancers. They are histologically classified as bronchiolar-alveolar carcinomas (a type of adenocarcinoma) (3). In humans, the inhalation of alpha particle emitters (but not transuranium elements) has usually resulted in bronchial cancers (hilar bronchogenic carcinomas). These are primarily epidermoid and anaplastic carcinomas, but include some adenocarcinomas. Cancers histologically similar to those in animals (peripheral adenocarcinomas) are found much less frequently (3). This difference may be due to differing exposure conditions but must, in part, be due to differences in tissue sensitivity between species (3).

Some insight into this problem is obtained by considering the type of cancers resulting from highly ionizing radiation that have occurred in survivors of the Hiroshima bombing. Examination of these data show that, even though the dose to the pulmonary lung must have been higher than that delivered to the bronchi (33), the significant increases in observed cancer occurred in the bronchi rather than in the region of higher dose (34).

An NAS committee recently concluded that "the risk from alpha irradiation of the deep lung tissues would not be underestimated by applying risk factors from human experience with cancer induced by irradiation of the bronchial tree" (3). Therefore, the risk estimates (Tables A3-10,11) are based on the highest dose rate received by any lung tissues (pulmonary lung) and risk estimates appropriate for the most sensitive tissues within the lung, the site of bronchial cancers. It should be noted that the dose to bronchial tissues following plutonium inhalation is small compared to that received in the pulmonary lung, Tables A3-2.

The number of lung cancers from alpha irradiation at a given dose appears to be increasing as the years at risk in relevant epidemiological studies are extended. The 1976 NAS report states that the absolute risk (the number of cases that will result from exposure of a given population) estimate for bronchial cancer in uranium miners is twenty cases per million organ rad per year at risk, not ten as in the 1972 BEIR Report. The relative risk (the ratio of the risk in those exposed to the risk to those not exposed) estimate in the 1972

BEIR Report is also likely to be low. Assuming that the relative risk for U.S. miners has increased in a manner similar to their absolute risk, it would be comparable to the 1972 BEIR estimate for Canadian miners who have been similarly exposed, i.e. 6% increase in annual incidence per rad. Because an updated analysis of the U.S. uranium miner cancer experience is not available, this estimate has been utilized in the life table analysis described below.

Tumors may occur at any time after a latent period during which the affected cell or cells progress from the state of initial injury to a tumor which can be identified clinically. The duration of this period is, of course, variable depending on the kind of cancer and host response. For many cancers it is estimated as 10 to 20 years. The 1972 BEIR Report assumed a 15 year latent period for all solid tumors induced in both children and adults.

Following the latent period, tumors occur with an increased frequency in the groups irradiated. The temporal pattern of cancer induction is poorly known for human populations exposed to radiation, mainly because exposed groups have not, as yet, been followed for sufficiently long enough times. The 1972 BEIR Committee made two approximations of the temporal pattern for tumor occurrence. One assumes a constant risk per year for the balance of a person's lifetime following a 15-year latent period; the other, a constant risk for a 30-year plateau following the latent period. This may not be long enough, since 30 years is the duration of most of the epidemiological studies used to establish the risk estimate.

Estimates of the risk of cancer following irradiation in the 1972 BEIR Report are a function of two parameters: the type of risk model, absolute or relative, and the duration of the plateau period. In the case of absolute risk the estimates are relatively independent of the plateau period, for either children or adults. However, results obtained with a relative risk model are more sensitive to the plateau period selected, particularly when children are included in the population at risk. If a 30-year plateau, relative risk model is assumed, the calculated risk of radiation carcinogenesis is small from early exposure when a child's sensitivity to radiation injury is high. Only in the later years of life is the natural incidence of cancer an important cause of death, and the plateau period does not extend into these years.

The life table analysis used to estimate the risk of premature death following irradiation is described in reference 35 and in Annex 4. Table A3-10 compares the risk models and Plateau periods discussed above, for the case of lung cancer due to lifetime inhalation causing a dose rate (to adults of 1 mrad per year. The absolute risk from exposures before ten years of age was assumed to be one-fifth that of adults; the relative risk ten times the adult value, as shown in Table A3-2 of reference 2. In Table A3-10, the risks to the exposed population are accounted for by three measures: (1) number of early deaths per 100,000 exposed, (2) the total life-shortening for the group as a whole and finally, (3) the average number of years of life lost by an afflicted individual. The much larger number of lung

cancers expected to occur in a cohort of 100,000 persons not exposed to this additional insult is estimated in Annex 4 (Table A4-1).

For the purpose of this report, an estimate of the lung cancer risk is obtained by averaging the geometric means of the absolute and relative risk for 30-year and lifetime plateaus, shown in Table A3-10. The Agency recognizes that these risk estimates must be regarded as tentative, because information on the radiocarcinogenicity of high LET particles is being reassessed by several competent scientific groups. The Agency has an ongoing contract with the NAS to have the BEIR committee re-evaluate radiation risk, including the risk of lung cancer. Pending completion of the new NAS study, the average risk of early death from a one millirad annual dose to adult pulmonary tissue has been used to assess the lung cancer risk from inhaled transuranics. At this level, lifetime exposure experienced by 100,000 persons could induce about 8 premature deaths.

3.6 The Risk of Bone Cancer

Unlike radium-226, which is distributed throughout the bone volume following long term ingestion, plutonium is preferentially deposited and retained on endostial bone surfaces, principally in the organic matrix. In some cases as much as 30-50% of the endostial plutonium has been shown to be retained on osteogenic cells (36). Americium and curium are also retained on bone surfaces. Alpha particle emitters which are retained on bone surfaces have been shown to be more tumorigenic than radium-226 and other bone volume seekers. Surface seekers deliver a higher dose to osteogenic cells adjacent to

bone surfaces, and such injury is thought to be the cause of radiogenic bone sarcomas.

There is no clinical evidence of bone cancer being caused by plutonium. The most relevant human data is for medical patients treated with radium-224, which, because of its short half-life (3.64 days), is retained mainly on bone surfaces. A large number of patients (approximately 900) who were treated with radium-224 for tuberculosis and ankylosing spondylitis have been followed for bone cancer incidence by Spiess and Mays (37). The dose to these patients has been calculated by the authors in terms of the average skeletal dose, defined as total alpha energy emitted divided by bone mass, even though the dose distribution is very nonuniform. On this basis Mays has estimated that for chronic irradiation due to Pu-239, 200 bone cancers will be produced per 10^6 rad to a 7 kg skeletal mass (38). In terms of the dose to mineral bone, mass 5 kg, utilized in this analysis, these results yield 140 bone cancers per rad to osseous tissue. As noted below this estimate is likely to be too low.

Because of uncertainties in the redistribution of Ra-224 following its initial deposition on bone surfaces, Mays' estimate of the average skeletal dose delivered in the Ra-224 cases may be too high, leading to an underestimate of the risk per rad for radium-224. While Mays assumes that half of the skeletal radium-224 decays on bone surfaces and half in the bone volume, Marshall has stated that only 1.5% of the skeletal radium-224 decays within the bone volume away from bone surfaces. This would increase the risk per rad by 174%.

Furthermore, Marshall's model predicts that, for radium-224 on bone surfaces, the dose rate to osteogenic cells near the bone surface is 8.9 times the average skeletal dose rate; for plutonium-239, 12.8 times (39). In terms of average dose to osseous tissue as calculated for these risk estimates, plutonium-239 would therefore deliver 1.44 times as much alpha particle dose to osteogenic cells as radium-224. This estimate is likely to be too high, since it assumes all the plutonium is retained on bone surfaces and none is buried in the remodelling or bone growth process.

The residence time of plutonium on bone surfaces depends on age. In rapidly growing animals, it is relatively short (40), while in adults, as Jee, et al. have pointed out, "Not only is there a prolonged ^{239}Pu bone surface residence time in adult bone, but they accumulate more ^{239}Pu with time (40)." Since almost all of the body burden is assumed during adult life, the exposure regime due to chronic plutonium inhalation and ingestion may favor a surface dose distribution more analogous to Marshall's model than Mays'. After giving due consideration to the smaller number of bone precursor cells in adults, Jee, et al. have characterized the plutonium-239 injury to bone as low in rapidly growing beagles, moderate for young adults, and high for adults (40). A similar characterization for degrees of insult would appear to hold for humans, particularly when subject to chronic exposure.

Based on these considerations, below is an upper bound estimate of the risk from plutonium and other bone surface seekers in terms of average skeletal dose:

$$\frac{140}{10^6 \text{ rad}} \times 1.74 \times 1.44 = \frac{350 \text{ bone cancers}}{10^6 \text{ rad}}$$

Even though the redistribution of plutonium on adult bone surfaces is expected to be very uneven on both macroscopic and microscopic scales, it seems likely that the amount of retained plutonium irradiating bone surfaces will be between 50% and 90% of total bone burden. It should be noted that the extent of plutonium redistribution affects only the factor of 1.44 in the relationship shown above.

It is assumed that an average of EPA's upper and lower bound figures, 250 bone carcinomas per 10^6 average skeletal rad, is a reasonable estimate of the actual bone cancer risk. The same risk is applied to americium and curium, whose metabolism in bone tissue is expected to be similar if not identical to plutonium's.

The latent period observed for the radium-224 patients was five years, with a plateau period of ten years. Therefore, the average annual risk would be 25 sarcomas per 10^6 skeletal rad per year at risk. The same risk is assumed for both juveniles and adults, since Mays' analysis of the radium-224 results indicates little difference between these two groups.

This risk estimate for bone cancer has been used to determine the risk to a cohort of 100,000 persons receiving a lifetime exposure, as

outlined in Annex 4. A cohort analysis is essential for this risk estimate since the correlation between accumulated dose and the duration of exposure is an important factor. To provide input data for this risk analysis, annual dose rates to bone have been calculated using the PAID code, as a function of the duration of exposure from inhaled and ingested transuranium elements (see Tables A3-3 and A3-6).

3.6.1 Inhalation Risk Estimates

The risk of bone cancer due to the lifetime inhalation of plutonium causing a one mrad annual dose to pulmonary lung tissue is shown in Table A3-11. The estimated excess risk is relatively small compared to that for lung cancer cf. Table A3-10. Estimates of relative risk has not been considered for the case of bone cancer. In view of the short duration of the plateau period used in the current model, ten years, and the small risk of naturally occurring bone cancer, the Agency believes that the absolute risk provides a suitable estimate to use for public health protection.

3.6.2 Ingestion Risk Estimates

The risk of bone cancer mortality occurring in a cohort due to ingestion has been calculated by means of a life table analysis. It has been assumed that the cohort is irradiated for a lifetime due to a constant intake of plutonium-239 oxide, so that the bone dose in the 70th year is 3.0 mrad. The estimated risk of bone cancer is shown in Table A3-12. Note that while the risk due to the ingestion of Am-241 or Pu-239 in a non-oxide form would be the same, the annual intake

needed to produce the limiting dose rate to bone would be ten times smaller.

Included in Table A3-12 is the bone cancer risk due to the chronic ingestion of Am-241, Cm-244, and Pu-241. The latter is a short half life (14.8 years) beta emitter having an alpha emitting daughter Am-241. The temporal pattern of the alpha dose to bone for Pu-241 and Cm-244 differs from that due to Pu-239 and Am-241. Because of the delay caused by the short half life parent, the alpha dose from the Am-241 daughter is delivered relatively late in life and the cancer risk is somewhat smaller. In contrast, Cm-244 (half life 17.9 years) approaches the equilibrium dose rate more rapidly than Pu-239 with a larger dose delivered early in life and thus a greater cancer risk (see Table A3-12).

3.7 THE RISK OF INDUCING CANCER OF THE LIVER

The magnitude of the potential risk of inducing liver cancer by means of plutonium and other transuranium elements has been recognized only recently. Existing governmental regulations are based on the 1959 NCRP-ICRP assumption that the critical organ for plutonium deposited from blood is bone (28). More recently, it has been recognized that deposition in liver is as likely as in bone. ICRP 19 assumes 45% of the plutonium and other transuranium elements dissolved in blood is deposited in the liver and an equal amount in bone (4). Based on the results of animal studies, other authorities have estimated a somewhat lower deposition in liver for plutonium and somewhat higher for americium and curium (41). The expected variation

between the transuranium elements is not large enough to change, by an appreciable extent, the overall risk estimate made below. Only the type of cancer might differ.

The risk to humans from alpha emitters deposited in the liver can be assessed on the basis of rather limited information obtained from epidemiological studies of medical patients. Earlier in this century, a low specific activity alpha particle emitting contrast medium called Thorotrast, was utilized in some diagnostic x-ray procedures. In subsequent years patients who were treated with it, mainly European, have been followed clinically and shown to have a higher than expected incidence of liver cancer. These data are pertinent although they do have limitations. Because the amount of material injected into the blood in these studies was quite large, its deposition in the liver was uneven (42, 43). Liver cancer incidence in this group would not necessarily be higher than might be expected for a more uniform deposition. On the contrary, there is a general consensus that highly localized concentrations of alpha particle emitters are likely to be less carcinogenic than a more uniform distribution (3). Another possible limitation of these data is that the relatively large quantity of Thorotrast deposited in the liver could lead to a foreign body response, which might in turn result in cancer. While the quantitative applicability of the human experience with Thorotrast to the prediction of plutonium risks to liver is tentative, there is an abundance of experimental animal data showing that liver cancers can be induced by plutonium. Liver cancers are seen less frequently than

bone cancers in most experiments with animals, but since liver cancers have a longer latent period than bone cancers, they may be more important in a longer-lived species such as man. The primary source of data on Thorotrast patients is Faber's review (44) also cited by the MRC as a basis for their assessment of plutonium toxicity (16). Faber's estimates are: absolute risk, 4.2×10^{-6} liver cancer for each organ rad per year at risk; relative risk, an 11% increase per rad.

3.7.1 Inhalation Risk Estimates

In Table A3-13, the risk of liver cancer has been evaluated for the airborne concentration of a plutonium-239 oxide aerosol (1 μ AMAD) that will cause a pulmonary dose rate to adults of 1 mrad per year. Unlike the case of ingested material discussed below, the risk of liver cancer due to various inhaled long half-life transuranics is nearly the same (for a given lung dose) as for plutonium oxide. This is because gut transfer has little effect on the dose to liver due to inhaled materials. For Cm-244 and other comparatively short half life transuranium elements, the dose to liver relative to lung is much smaller, cf, Table A3-3 and A3-4 so that the risk of inducing liver cancer is less for such radionuclides than for plutonium. The average dose rate to liver relative to lung is nearly independent of particle size (15). In computing Table A3-13, the risk to children less than 10 years of age was assumed to be different from adults. Since no clinical data is available on the effect of Thorotrast on the young, their risk was estimated from Table 3-2 in reference 2, as described above for the case of lung cancer.

The average of the estimated excess deaths shown in Table A3-13, is 0.34 for a lifetime exposure to 100,000 persons. This is a much smaller number of excess cancers than the estimated mortality due to lung cancer from the same aerosol concentration, cf. Table A3-10.

3.7.2 Ingestion Risk Estimates

In contrast to inhalation, the ingestion dose to liver and the resultant risk is a direct function of the amount of radioactivity transferred to blood via the gastrointestinal tract. Table A3-14 lists the risk of liver cancer from a lifetime ingestion pattern that results in a 3 mrad dose to bone after 70 years. The results shown are for plutonium-239. For transuranium elements having a physical half life comparable to the elements' residence time in liver the risk will be somewhat greater since the dose approaches the equilibrium value earlier in life. Averaging the estimates of early death shown in Table A3-14 yields an estimated risk of two deaths per 100,000 exposed persons, somewhat less than for cancer of the bone, cf Table A3-12. For Am-241, Pu-241 and Cm-244 this average lifetime risk is 2.1, 1.2 and 3.2 per 100,000 exposed, respectively.

3.8 THE RISK OF GENETIC DAMAGE

Risks due to transuranium elements are not only to persons directly exposed to the radiation but also to their progeny. Alpha particles can damage the male progenitor cells producing sperm and the egg cell (oocyte) in the female. The expression of this damage is either genetic impairment of the live-born offspring or fetal death.

Only the former has, as yet, been quantified in current risk estimates of health effects due to radiation.

NAS-BEIR estimates of genetic risk are based on chronic x-irradiation to males, not the dose to both sexes due to alpha particles. For the reasons given below genetic damage from alpha particles is expected to be about a factor of 100 greater than that assumed in the BEIR report (2). Based on current recommendations of the ICRP, alpha particles are 20 times more damaging than x-irradiation for a number of biological end points, including genetic effects. This is not necessarily overly conservative since an increase of about 20 compared to x-irradiation has been reported for genetic damage from highly ionizing neutron irradiations (46).

Alpha particle damage is believed to be independent of dose rate. However BEIR estimates of genetic risk are based on low dose rate x-ray exposures which had the effect of lowering genetic damage to males by a factor of 3.4 and eliminating this damage to females. The British Medical Radiation Council also assumed no damage to the oocyte of females (16), an assumption based on Searle's evaluation (47) of mouse experiments with fission neutrons, performed by Batchelor, et. al. (46). However, more recent analyses of the efficacy of chronic irradiation in producing genetic damage indicate that it may not be wise to disregard potential damage to the oocytes of the female (48). Therefore, at this time it appears prudent to increase the BEIR risk estimate for genetic damage due to chronic x-irradiation by a factor

of 100 (5 x 20) when assessing the risk of genetic damage due to alpha emitting transuranics in the gonads.

The range of the BEIR estimates of genetic risk is quite large Table 4, on page 57 of (2). They reflect the uncertainty in the extent of the genetic component in diseases classified as having a "complex etiology", i.e., those with a mutational component among many other causes, such as heart disease. One recent analysis indicates that this type of genetic risk may be so small that the actual risk is near the lower end of the range estimated by the BEIR Committee in 1972 (49). On the other hand, there are other analyses which indicate that the 1972 BEIR report may have underestimated the amount of multifactorial diseases having a genetic component (50).

In 1976 EPA contracted with the National Academy of Sciences to provide it with guidance on genetic risk, as part of the BEIR Committee's reevaluation of radiation risk. The degree to which the estimate made above may be high or low may be resolved when the BEIR Committee's ongoing review of this problem is completed.

It is estimated here that a 30-year dose of one millirad due to alpha-emitting transuranium elements in gonadal tissue may cause between 0.1 and 2 genetic effects per 100,000 live births in the first generation. If this gonadal dose were to continue indefinitely so that a new equilibrium of genetic damage was established in the population, the risk might increase to 0.6 to 15 per 100,000 live births. Currently, the observed genetic effects in the U.S. are about 6000 per 100,000 live births.

3.8.1 Genetic Risk Estimates

Risk estimates for genetic damage are based on the gonadal dose received in a reproductive generation, i.e., the first 30 years of life. Table A3-15 lists the 30-year gonadal doses due to chronic ingestion of transuranium elements that would also cause a 3 millirad per year dose rate to bone after 70 years. Except for cm-244, the guidance's limitation on the annual dose rate to bone limits the relevant gonadal dose to about 10 millirad. The estimated genetic risks due to this 30 year dose is shown in Table A3-16. Chronic ingestion of Cm-244, would cause genetic risks about two times larger which would cause genetic risks about two times larger than those shown in Table A3-16. However, because of Cm-244's relatively short half life circumstances that would lead to a constant rate of ingestion for 30-years are difficult to imagine. For the limiting dose rates permitted by this guidance, genetic effects due to inhalation are substantially less important than those due to ingestion. Where the gonadal dose is from chronically inhaled transuranics causing a one millirad dose to lung, the gonadal dose is 1.4 mrad in 30 years (Section 3.4.3). and the estimated genetic effects are a factor of seven smaller than the estimates shown in Table A3-16.

3.9 OTHER RISKS DUE TO THE INHALATION AND INGESTION OF PLUTONIUM

3.9.1 Leukemia due to Bone Marrow Irradiation

Recently, several authors have pointed out that leukemia is a potential risk from plutonium incorporated into bone tissues (51). Alpha particles originating in trabeculae may irradiate a significant fraction of the bone marrow, and the plutonium in marrow itself will act as a source. Based on autoradiographic studies of bone and bone marrow, Spiers and Vaughan have estimated that the dose to trabecular marrow is 88 percent of the average skeletal dose due to plutonium-239 (52).

Using this factor and the skeletal dose rate from Pu-239, shown in Figure A3-1, the risk of leukemia has been calculated in a cohort of 100,000 persons exposed to plutonium-239 aerosol having a 1 μ AMAD and concentration of 2.6 f Ci/m³. This concentration results in the limiting dose rate to pulmonary lung of one mrad per annum. Excess leukemia deaths were estimated utilizing the risk coefficients given in Table 3-2 of the 1972 BEIR Report (2) and a quality factor of 20 for alpha particle irradiation (45). Results are listed in Table A3-17, which shows that the incremental risk due to leukemia is rather small compared to the risk for other cancers associated with a dose of 1 mrad per annum to pulmonary tissue, cf. Table A3-10.

In the case of ingested plutonium-239 the estimated risk of leukemia induction is somewhat greater. For a 3 mrad limiting dose to have in the 70th year, the leukemia risk ranges from 0.4 to 1.6 cases per 100,000 exposed for absolute and relative risk models respectively

and is therefore smaller but comparable to the estimated risk of liver cancer due to the ingestion of transuranic elements.

Over a lifetime the average dose to the whole body (soft tissues) due to the 7% of actinides distributed in the body tissues after inhalation is about a factor of 100 less than the dose received by skeletal tissues. Leukemia mortality due to excess radiation is about one-fifth of all radiation induced cancers (2). Therefore, health effects due to the plutonium burden in the whole body are expected to be about 5% of the early deaths due to leukemia indicated in Table A3-17. Inhalation of other transuranium elements at these limits set by those guides would cause a similar risk relative to leukemia.

3.10 SUMMARY OF HEALTH RISKS

3.10.1 Inhalation Pathway

Table A3-18 below summarizes the somatic and genetic risk due to the inhalation of transuranium element aerosols causing an annual dose to the pulmonary region of 1 mrad per year. The estimated cancer risk to a cohort of 100,000 is nine premature deaths, with an estimated range of between 3 and 30 early deaths. Since the average lifespan of this cohort would be about 71 years, the annual risk of cancer from lifetime exposure is about 1×10^{-6} per year. The estimated genetic risk to the first generation is considerably smaller numerically than the risk of cancer due to the inhalation of transuranium elements.

3.10.2 Ingestion Pathway

The total risk due to the ingestion of transuranium elements may include an appreciable genetic component as well as the risk of cancer. Therefore, in this guidance the cancer risk due to ingestion is less than that due to inhalation. Exact correspondence between the risks from inhalation and ingestion is not possible, since it depends on value judgements concerning the acceptability of risk to future generations as compared to the present. Moreover, the uncertainty of the genetic risk estimates makes such a comparison uncertain.

The guides are expressed in terms of the dose rate to bone after 70 years of chronic ingestion. Including the risk of leukemia induction, this would result in an estimated incidence of about five cases of fatal cancer in a cohort of 100,000 persons. The range of estimated genetic effects for the same pattern of exposure are shown in Table A3-16. The estimated genetic risk to first generation progeny may be numerically comparable to the cancer risk to the parents, while the genetic risk to succeeding generations may exceed the risk of cancer.

REFERENCES

1. Selected Topics: Plutonium in the Environment, ORP Technical Report (in preparation, 1977).
2. BEIR Report, 1972. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation, Report of the Advisory Committee on the Biological Effects of Ionizing Radiations, National Academy of Sciences, Washington, D.C.
3. Health Effects of Alpha-Emitting Particles in the Respiratory Tract, Report of Ad Hoc Committee on "hot particles" of the Advisory Committee on the Biological Effects of Ionizing Radiations, National Academy of Sciences, EPA 520/4-76-013, Office of Radiation Programs, Environmental Protection Agency, Washington, D.C. 1976.
4. ICRP Publication 19, 1972. The Metabolism of Compounds of Plutonium and Other Actinides, Pergamon Press, New York.
5. TGLD (Task Group on Lung Dynamics) 1966. Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract, Health Physics 12:173-208.
6. Hodge, F. A. and Ellett, W. H., 1977. Dose and Dose Rates Due to the Chronic Inhalation and Ingestion of Transuranic Radionuclides, EPA/ORP (to be published).
7. Kotrappa, P., 1969. Calculation of the Burden and Dose to the Respiratory Tract from Continuous Inhalation of a Radioactive Aerosol, Health Physics, 17:429-432
8. Houston, J. R., Strenge, D. L. and Watson, E. C., 1974. DACRIN - A Computer Program for Calculating Organ Dose from Acute or Chronic Radionuclide Inhalation, BNWL-B-389, Battelle Pacific Northwest Laboratories, Richland, Washington.
9. Sullivan, R., 1977. Plutonium Air Inhalation Dose (PAID), ORP/CSD Technical Note, 77-4 (in press).
10. ICRP Publication 23, 1975. Report of the Task Group on Reference Man, Pergamon Press, New York.
11. Tamplin, A. R. and Cochran, T. B., 1974. Radiation Standards for Hot Particles: A Report on the Inadequacy of Existing Radiation Protection Standards Related to Internal Exposure of Man to

Insoluble Particles of Plutonium and Other Alpha-Emitting Hot Particles, Natural Resources Defense Council, Washington, D.C.

12. ICRP Publication 14, 1969. Radiosensitivity and Spatial Distribution of Dose, Pergamon Press, New York.
13. Bair, W. J., 1974. Toxicology of Plutonium, Adv. Rad. Biol., 4:255-313
14. Durbin, P.W. Plutonium in Man: A New Look at Old Data in Radiobiology of Plutonium, Ed. by B.J. Stoer and W.S. Jee, J.W. Press, University of Utah, Salt Lake City, 1972.
15. Richmond, C. R. and Thomas, R. L., 1975. Plutonium and Other Actinide Elements in Gonadal Tissue of Man and Animals, Health Physics, 29:241-250.
16. The Toxicity of Plutonium, 1975. Medical Research Council, Her Majesty's Stationary Office, London.
17. Stevens, W., Atherton, D.R., Bruenger, F. W., Buster, D. and Bates, D., 1977. Deposition of ^{239}Pu , ^{226}Ra , ^{233}U and ^{241}Am in the Gonads, pp 223-227 in Research in Radiobiology, COO-119-252, Annual Report of the Radiobiology Laboratory, University of Utah, College of Medicine, Salt Lake City, Utah.
18. Taylor, D.M., 1976. The Uptake, Retention and Distribution of Plutonium-239 in Rat Gonads, Health Physics, 32:29-31.
19. Fish, B. R., Keilholz, G. W., Snyder, W.S. and Swisher, S.D., 1972. Calculation of Doses Due to Accidentally Released Plutonium from an LMFB, ORNL-NSIC-74, Oak Ridge National Laboratory, Oak Ridge.
20. Raabe, O.G. Kanapilly, G.M., and Boyd, H.A., 1973. Studies of in vitro Solubility of Respirable Particles of ^{238}Pu and ^{239}Pu , pp 24-30 in Fission Product Inhalation Program Annual Report, 1972-1973, LF-46, Lovelace Foundation for Medical Education and Research, Albuquerque.
21. Muggenburg, B.A., Mewhinney, J.A., Miglio, J.J. Slauson, D.O. and McClellan, R.O., 1974. Bronchopulmonary Lavage and DTPA Treatment for the Removal of Inhaled ^{239}Pu of Vaired Solubility in Beagle Dogs, II, pp 269-273 in Inhalation Toxicology Research Institute Annual Report 1973-1974, LF-49, Lovelace Foundation for Medical Education and Research, Albuquerque.

22. Durbin, P.W., 1974. Behavior of Plutonium in Animals and Man, pp 30-56 in Plutonium Information Meeting, CONF-740115, US Atomic Energy Commission, Oak Ridge.
23. Sullivan, M.F. and Crosby, A.L., 1976. Absorption of Transuranic Elements from Rat. Gut, pp91-93 in Pacific Northwest Laboratory Annual Report for 1975, Part I Biomedical Sciences, BNWL-2000, PT 1, Battelle Pacific Northwest Laboratories, Richland.
24. Sullivan, M.F. and Crosby, A.L., 1975. Absorption of Uranium-223, Neptunium-237, Plutonium-238, Americium-241, Curium-244, and Einsteinium-253 from the Gastro-intestinal Tract of Newborn and Adult Rats, pp 105-108 in Battelle Pacific Northwest Laboratories Annual Report for 1974, BNWL-1950, Battelle Pacific Northwest Laboratories, Richland.
25. Bair, N.J., 1975. The Biological Effects of Transuranium Elements in Experimental Animals, pp 464-536 in Proceedings of Public Hearings: Plutonium and Other Transuranium Elements, ORP/CSD-75-1, Volume 1, U.S. Environmental Protection Agency, Washington, D.C.
26. Bair, W. J. and Thompson, R. C., 1977. Battelle Northwest Laboratory, letter communication.
27. Dolphin, G. W. and Eve, I. S., 1966. Dosimetry of the Gastrointestinal Tract, Health Physics, 12:163-172.
28. ICRP Publication 2, 1959. Report of Committee II on Permissible Dose for Internal Radiation, Pergamon Press, New York.
29. Sansom, B.F., 1964. The Transfer of Plutonium-239 from the Diet of a Cow to its Milk, Brit. net. J. 120: 158-161.
30. Stanley, R.E., Bretthauer, E.W. and Sutton, W.S., 1974. Absorption, Distribution and Excretion of Plutonium by Dairy Cattle, pp 163-185 in Dynamics of Plutonium in Desert Environments, NVO-142, U.S. Atomic Energy Commission, Nevada Operations Office, Las Vegas.
31. McClellan, R.O., Casey, H.W., and Bustad, L.K., 1962. Transfer of Some Transuranic Elements to Milk, Health Physics, 8:689-694.
32. Bair, W. J. and Thomas, J. M., 1976. Prediction of the Health Effects of Inhaled Transuranium Elements from Experimental Animal Data, pp 569-585 in Transuranium Nuclides in the Environment, International Atomic Energy Agency, Vienna.

33. Ken, G. D. and John, T. D. A Reanalysis of Leukemia Data on Atomic Bomb Survivors Based on Estimates of Absorbed Dose to Bone Marrow, ORNL paper IL-1, Twenty-first Annual Health Physics Meeting, June 23-July 2, 1976.
34. Ishimaru, T., Cihak, R. W., Land, C. E., Steer, A. and Yamada, A., 1972. Lung Cancer at Autopsy in Atomic Bomb Survivors and Controls, Hiroshima nad Nagasaki, 1961-1970, ABCC Technical Report 33-72, Atomic Bomb Casualty Commission, Hiroshima.
35. Bunker, B., Cook, J. and Barrick, K., 1977. Life Table Methodology for Evaluating Radiation Risk. ORP/CSD Report - in preparation.
36. Vaughan, J. M., 1973. The Effects of Irradiation on The Skeleton, Clarendon Press, Oxford.
37. Spiess, H. and Mays, C. W., 1973. Protraction Effect on Bone Sarcoma Induction of ^{224}Ra in Children and Adults. pp 437-450 in Radionuclide Carcinogenesis, CONF-720505, C. L. Saunders, R. H. Busch, J. E. Ballou and D. D. Mahlum, editors, AEC Symposium Series 29, U.S. Atomic Energy Commission, Oak Ridge.
38. Mays, C. W., et al., 1976. Estimated Risk to Human Bone from ^{239}Pu , pp 343-362 in The Health Effects of Plutonium and Radium, W. S. S. Jee, editor, The J. W. Press, Salt Lake City.
39. Rowland, R. E. and Durbin, P. W., 1976. Survival, Causes of Death and Estimated Tissue Doses in a Group of Human Beings Injected with Plutonium, pp 329-341 in The Health Effects of Plutonium and Radium, W. S. S. Jee, editor, The J. W. Press, Salt Lake City.
40. Jee, W. S. S., et al., 1976. The Current Status of Utah Long-Term ^{239}Pu Studies, in Biological and Environmental Effects of Low-Level Radiation, Vol II, International Atomic Energy Agency, Vienna.
41. Durbin, P. W., 1975. Plutonium in Mammals: Influence of Plutonium Chemistry, Route of Administration, and Physiological Status of the Animal on Initial Distribution and Long-Term Metabolism, Health Physics 29:495-510.
42. Tessmer, C. F. and Chang, J. P., 1967. Thorotrast Localization by Light and Electron Microscopy. Ann. N. Y. Aca. Sci., 145(3):545-575.
43. Riedel, W., Miller, B. and Kaul, A., 1973. Non-Radiation Effects of Thorotrast and Other Colloidal Substances pp 281-293 in

Proceedings of the Third International Meeting on the Toxicity of Thorotrast, Risø Report No. 294, Danish Atomic Energy Commission, Copenhagen.

44. Faber, M., 1973. Dose Effect Relationships in Hepatic Carcinogenesis, pp 308-316 in Proceedings of the Third International Meeting on the Toxicity of Thorotrast, Risø Report No. 294, Danish Atomic Energy Commission, Copenhagen.
45. ICRP Publication 26, 1977. Radiation Protection. Pergamon Press, N.Y.
46. Searle, A. G., 1974. Mutation Induction in Mice, Adv. Radiat. Biol. 4:131-207.
47. Batchelor, A. L.; Phillips, R. J. S. and Searle, A. G., 1969. The Ineffectiveness of chronic irradiation with neutrons and gamma rays in inducing mutations in female mice, Brit. J. Radiol. 42:448-451
48. Abrahamson, S. and Wolff, S., 1976. Re-analysis of Radiation-Induced Specific Locus Mutations in the Mouse, Nature, 264:715-719.
49. Newcombe, H. B., 1975. Mutation and the Amount of Human Ill Health, pp 937-946 in Radiation Research: Biomedical, Chemical, and Physical Perspectives, O. F. Nygaard, H. I. Adler and W. K. Sinclair, editors, Academic Press, Inc., New York.
50. Trimble, B. K. Induced Mutation and Human Disease presented at the Radiation Research Society Meeting, San Francisco, 1976.
51. Vaughan, J., 1976. Plutonium a Possible Leukemia Risk, pp 691-705 in The Health Effects of Plutonium and Radium, W. S. S. Jee, editor, The J. W. Press, Salt Lake City, Utah
52. Spiers, F. W. and Vaughan, J., 1976. Hazards of Plutonium with Special Reference to the Skeleton, Nature 259:531-534.

Table A3-1

Retention Halflives, Clearance Patterns and
Breathing Rate for Class Y Aerosols
Inhaled by Referenced Man (4,10)

Lung Compartment	Compartmental** Half life (days)	Transfer Fraction	Target
Nasopharyngeal	(a) 0.01	0.01	to blood
	(b) 0.4	0.99	to GI tract
Tracheobronchial T-B	(c) 0.01	0.01	to blood
	(d) 0.2	0.99	to GI tract
	(e) 500	0.05	to blood
Pulmonary	(f) 1	0.4	to GI tract via T-B
	(g) 500	0.4	to GI tract via T-B
	(h) 500	0.15	to lymph nodes
T-B lymph nodes	(i) 1000	0.9	to blood
	(j)	0.1	retained

Breathing rate 2.3×10^4 liters per day (16 hours lightwork;
8 hours resting).

**See Figure A3-2 for designation of compartmental pathways (a),
(b), etc.

Table A3-2

Annual Dose Rate to Various Lung Compartments from
 Chronic Exposure to Plutonium-239 Aerosols
 Concentration: 1.0 fCi/m³ Particle AMAD: 0.05, 1.0 and 5.0 Microns

Duration of Exposure Years	Pulmonary mrad/yr. x 10 ⁻¹			Tracheobronchial mrad/yr. x 10 ⁻²			Nasopharyngeal mrad/yr. x 10 ⁻⁶		
	0.05u	1.0u	5.0u	0.05u	1.0u	5.0u	0.05u	1.0u	5.0u
1	3.9	1.5	.7	2.7	1.1	6.1	.04	11.	30.
5	9.1	3.5	1.7	3.7	1.5	7.9	.04	11.	30.
10	9.8	3.8	1.8	3.8	1.6	8.1	.04	11.	30.
70	9.9	3.8	1.8	3.8	1.6	8.1	.04	11.	30.

Table A3-3

Annual Dose Rates to Various Organs from Chronic
Exposure to Plutonium-239 and Americium-241
Aerosols AMAD=1 μ ; Concentration 1 fCi/m³; Class Y Clearance

Duration of Exposure (years)	Pu ²³⁹ (mrad/year)			Am ²⁴¹ (millirad/year)		
	Liver	Bone	T-B Lymph Nodes	Liver	Bone	T-B Lymph Nodes
1	.001	.0005	.40	.0015	.0005	.39
5	.018	.0065	4.0	.019	.007	4.2
10	.052	.019	7.0	.055	.021	7.4
15	.089	.034	8.7	.095	.036	9.1
20	.13	.049	9.8	.13	.052	10
30	.19	.078	12	.20	.082	12
40	.24	.11	14	.26	.11	14
50	.29	.13	15	.30	.14	16
70	.36	.17	19	.37	.18	20

Duration of Exposure (years)	Pu ²⁴¹ /Am ²⁴¹ * (microrad/year)			Cm ²⁴⁴ /Pu ²⁴⁰ (millirad/year)		
	Liver	Bone	T-B Lymph Nodes	Liver	Bone	T-B Lymph Nodes
1	.0014	.0005	.396	.0016	.0006	.040
5	.087	.032	17.1	.018	.0016	4.0
10	.46	.17	45	.047	.017	7.5
15	1.1	.41	64	.073	.028	7.8
20	1.8	.71	75	.094	.037	8.4
30	3.4	1.4	92	.12	.049	9.3
40	5.0	2.2	110	.14	.057	9.8
50	6.4	3.0	120	.15	.063	10
70	8.7	4.4	150	.16	.068	11

* α dose only - 70th year beta dose rates: liver, 0.11 urad; bone, 0.049 urad.

Table A3-4

Aerosol Concentrations in fCi/m³ Producing a
1.0 mrad Annual Dose Rate to the Pulmonary Region
of Reference Man; Class Y Clearance

Aerosol	²³⁸ Pu (α) 94	²³⁹ Pu (α) 94	²⁴⁰ Pu (α) 94	²⁴¹ Pu (β) * 94
AMAD (u)	²³⁴ U (α) 92	²³⁵ U (α) 92	²³⁶ U (α) 92	²⁴¹ Am (α) 95
0.05	1.0	1.0	1.0	330
0.10	1.1	1.2	1.2	390
0.30	1.6	1.7	1.7	540
0.50	1.8	1.9	1.9	630
1.0	2.5	2.6	2.6	850
2.0	3.4	3.5	3.5	1,100
3.0	4.1	4.3	4.3	1,400
5.0	5.2	5.4	5.4	1,800
Aerosol	²⁴¹ Am (α) 95	²⁴⁴ Cm (α) 96		
AMAD (u)	²³⁷ Np (α) 93	²⁴⁰ Pu (α) 94		
0.05	1.0	1.0		
0.10	1.1	1.1		
0.30	1.6	1.6		
0.50	1.8	1.8		
1.0	2.4	2.5		
2.0	3.3	3.4		
3.0	4.0	4.1		
5.0	5.1	5.2		

* β dose rate < 40% of α dose - only α dose is considered in setting limit.

Table A3-5

Fraction of Ingestion Material Transferred to
Blood from the Gastrointestinal Tract

Radionuclide	Transfer Fraction		Biologically Incorporated
	Non-oxide	Oxide	
Plutonium-238	10^{-3}	10^{-3}	5×10^{-3}
Plutonium-239	10^{-3}	10^{-4}	5×10^{-3}
Plutonium-240	10^{-3}	10^{-4}	5×10^{-3}
Plutonium-241	10^{-3}	10^{-3}	5×10^{-3}
Americium-241	10^{-3}	10^{-3}	5×10^{-3}
Curium-244	10^{-3}	10^{-3}	5×10^{-3}

* Persons over one year of age (see text).

Table A3-6

Annual Dose Rate Due to Chronic Ingestion of
Plutonium-239 Oxide, Americium-241, Plutonium-241 and Curium-244
Annual Intake 1000 pCi/Year*

Duration of Ingestion Years	Plutonium-239 Oxide (urad/year)			Americium-241 (urad/year)		
	Bone	Liver	Whole Body	Bone	Liver	Whole Body
1	0.9	2.4	1.1×10^{-2}	9.2	2.5×10^1	1.1×10^{-1}
5	4.3	1.2×10^1	6.3×10^{-2}	4.5×10^1	1.2×10^2	5.6×10^{-1}
10	8.4	2.2×10^1	1.0×10^{-1}	8.8×10^1	2.3×10^2	1.1
15	1.2×10^1	3.2×10^1	1.5×10^{-1}	1.3×10^2	3.4×10^2	1.6
20	1.6×10^1	4.1×10^1	2.0×10^{-1}	1.7×10^2	4.3×10^2	2.1
30	2.4×10^1	5.6×10^1	3.0×10^{-1}	2.4×10^2	5.9×10^2	3.0
40	3.0×10^1	6.9×10^1	3.7×10^{-1}	3.1×10^2	7.2×10^2	4.0
50	3.7×10^1	8.1×10^1	4.4×10^{-1}	3.8×10^2	8.3×10^2	4.7
70	4.8×10^1	9.8×10^1	6.1×10^{-1}	4.9×10^2	9.9×10^2	6.1
Duration of Ingestion Years	Pu ²⁴¹ /Am ²⁴¹ (urad/year) **			Cm ²⁴⁴ /Pu ²⁴⁰ (urad/year)		
	Bone	Liver	Whole Body	Bone	Liver	Whole Body
1	.007	.020	9.0×10^{-5}	9.5	2.6×10^1	1.2×10^{-1}
5	.17	.45	2.1×10^{-3}	4.4×10^1	1.2×10^2	5.4×10^{-1}
10	.60	1.6	7.5×10^{-3}	7.8×10^1	2.1×10^2	9.8×10^{-1}
15	1.2	3.1	1.5×10^{-2}	1.1×10^2	2.8×10^2	1.3
20	2.0	4.9	2.6×10^{-2}	1.3×10^2	3.3×10^2	6.8×10^{-1}
30	3.8	8.7	4.7×10^{-2}	1.6×10^2	3.9×10^2	2.0
40	5.7	12	7.0×10^{-2}	1.8×10^2	4.3×10^2	2.2
50	7.6	16	9.6×10^{-2}	1.9×10^2	4.6×10^2	2.3
70	11	21	1.4×10^{-1}	2.1×10^2	4.8×10^2	2.6

*Reference Man (10).

**Alpha dose rate.

Table A3-7

Average Skeletal Dose Rate in the 70th Year Due to
the Lifetime Ingestion by Reference Man of 10 nCi/yr.

Radionuclide Chain	Millirad Per Year	
	Oxide	Other Inorganics*
$^{238}\text{Pu}(\alpha) / ^{238}\text{U}(\alpha)$	4.0	4.0
$^{239}\text{Pu}(\alpha) / ^{235}\text{U}(\alpha)$	0.48	4.8
$^{240}\text{Pu}(\alpha) / ^{234}\text{U}(\alpha)$	0.48	4.8
$^{241}\text{Pu}(\beta) / ^{241}\text{Am}(\alpha)$	0.11**	0.11**
$^{241}\text{Am}(\alpha) / ^{237}\text{Np}(\alpha)$	4.9	4.9
$^{244}\text{Cm}(\alpha) / ^{240}\text{Pu}(\alpha)$	2.1	2.1

*Biologically incorporated transuranic multiply by 5.0.

**Only the alpha dose listed in applicable to this guide.
The beta dose rate is 1.5 urad/yr. in the 70th year.

Table A3-8

Cumulative 30-year Alpha Particle Dose to
Gonadal Tissue
Annual Intake 1000 pCi/y

Radionuclide	30-year Dose millirad
Pu-238	1.80
Pu-239 (oxide)	0.18
Pu-240 (oxide)	0.18
*Pu-241	0.02
Am-241	1.9
Cm-244	1.5

* α dose only - the β dose is 1.2×10^{-6} rads.

Table A3-9

**Average Skeletal Dose Rates Due to Chronic Ingestion of
Plutonium-239 Oxide with and Without Increased Infant Uptake
Annual Intake 1000 pCi/yr.**

Age	Average Skeletal Dose Rate (uRad/yr.)	
	Without Enhanced Infant Absorption*	With Enhanced Infant Absorption**
1	0.86	86.4
5	4.26	37.7
10	8.37	32.6
15	12.4	21.6
20	16.2	23.7
30	23.5	30.5
40	30.3	36.9
50	36.6	42.7
70	48.0	53.3

*GI tract to blood transfer 10^{-4} all ages.

**GI tract to blood transfer 10^{-2} first year of life.

Table A3-10

Measures of the Lifetime Health Risk Due to Lung
Cancer Mortality Pulmonary Dose Rate to Adults
1mrad/yr. Cohort Size 100,000 Persons - Latent Period = 15 Years.

Measure of Risk	Relative Risk Estimate 30-Year Plateau	Lifetime Plateau	Absolute Risk Estimate 30-year Plateau	Lifetime Plateau
Premature Deaths	7.02	25.0	2.15	2.80
Aggregate Years of Life Lost to Cohort	129	368	47	54
Average Years of Life Lost to Premature Deaths	18.4	14.7	22.0	19.4

Table A3-11

Estimated Risk of Bone Cancer Mortality Due to the Inhalation
of Plutonium-239 Oxide (AMAD=1.0 u)
Pulmonary Dose Rate = 1 mrad/yr.
Average Skeletal Dose Rate in the 70th Year .46 mrad
Cohort Size = 100,000 Persons

Measure of risk	Absolute Risk Estimate 10 Year Risk Plateau
Premature Deaths	0.37
Aggregate Years of Life Lost to Cohort	8.0
Average Years of Life Lost to Premature Deaths	23.3

Table A3-12

Estimated Risk of Bone Cancer Due to a Lifetime Ingestion
Pattern that Results in a 3.0 mrad Alpha Particle Dose Rate Per Year
to Bone in the 70th Year

Radionuclide	Absolute Risk
	10-Year Risk Plateau
Premature Deaths per 100,000 Exposed	
Pu-239	2.4
Am-241	2.5
Pu-241	1.9
Cm-244	3.2

Average years of life lost per premature death — 24 years.

Table A3-13

Measures of the Lifetime Health Risk of Liver Cancer Mortality
 Due to Inhalation of Plutonium-239 Oxide (AMAD=1u)
 Pulmonary Dose Rate 1 mrad/yr.
 Liver Dose Rate on the 70th Year 0.95 mrad/yr.
 Latent Period = 15 Years; Cohort 100,000 Persons

Measure of Risk	Relative Risk Estimate 30-Year Plateau	Risk Estimate Lifetime Plateau	Absolute Risk Estimate 30-year Plateau	Risk Estimate Lifetime Plateau
Premature Deaths	.261	.635	.236	.296
Aggregate Years of Life Lost to Cohort	4.0	8.4	4.7	5.3
Average Years of Life Lost to Premature Deaths	15.3	13.2	19.7	17.9

Table A3-14

Estimated Risk of Liver Cancer Due to A Lifetime Ingestion Pattern
That Results in a 3.0 mrad Alpha Particle Dose Rate Per Year
to Bone in the 70th Year for Long Half Life Transuranic Elements*
Deaths per 100,000 exposed

Risk Model	30-year Plateau	Lifetime Plateau
Relative Risk	1.6	2.8
Absolute Risk	1.5	1.8

*T $1/2$ 100 years.

Table A3-15

**Thirty-year Gonadal Dose Due to an Ingestion Pattern
Causing a 3 Millirad/yr. Alpha Particle Dose Rate to Bone in the 70th Year
(Chronic Lifetime Ingestion at a Constant Rate)**

Transuranium Element	Gonadal Dose (millirad)
Pu-238	13
Pu-239	11
Pu-240	11
Pu-241	6
Am-241	12
Cm-244	21

Table A3-16

**Estimated Genetic Risk Due to an Alpha Particle Dose
of 10 Millirads to the Gonads in the First 30 Years of Life**

Type of Genetic Disease	Number of Effects Per 100,000 Live Births	
	1st Generation	All Generations
Dominant	1 - 10	5 - 50
Multifactorial	0.1 - 10	1 - 100
Total	1 20	6 150

Table A3-17

**Measures of the Lifetime Health Risk of Leukemia Due to
Inhalation of Plutonium-239 Oxide (AMAD = 1u)
Pulmonary Dose Rate 1 mrad/yr.
Cohort Size 100,000 Persons**

Measure of Risk	Relative Risk	Absolute Risk
Early deaths	0.24	0.06
Decrease in population life expectancy (years)	2.8	1.4
Average years of life lost per early death	11.8	23.1

Table A3-18

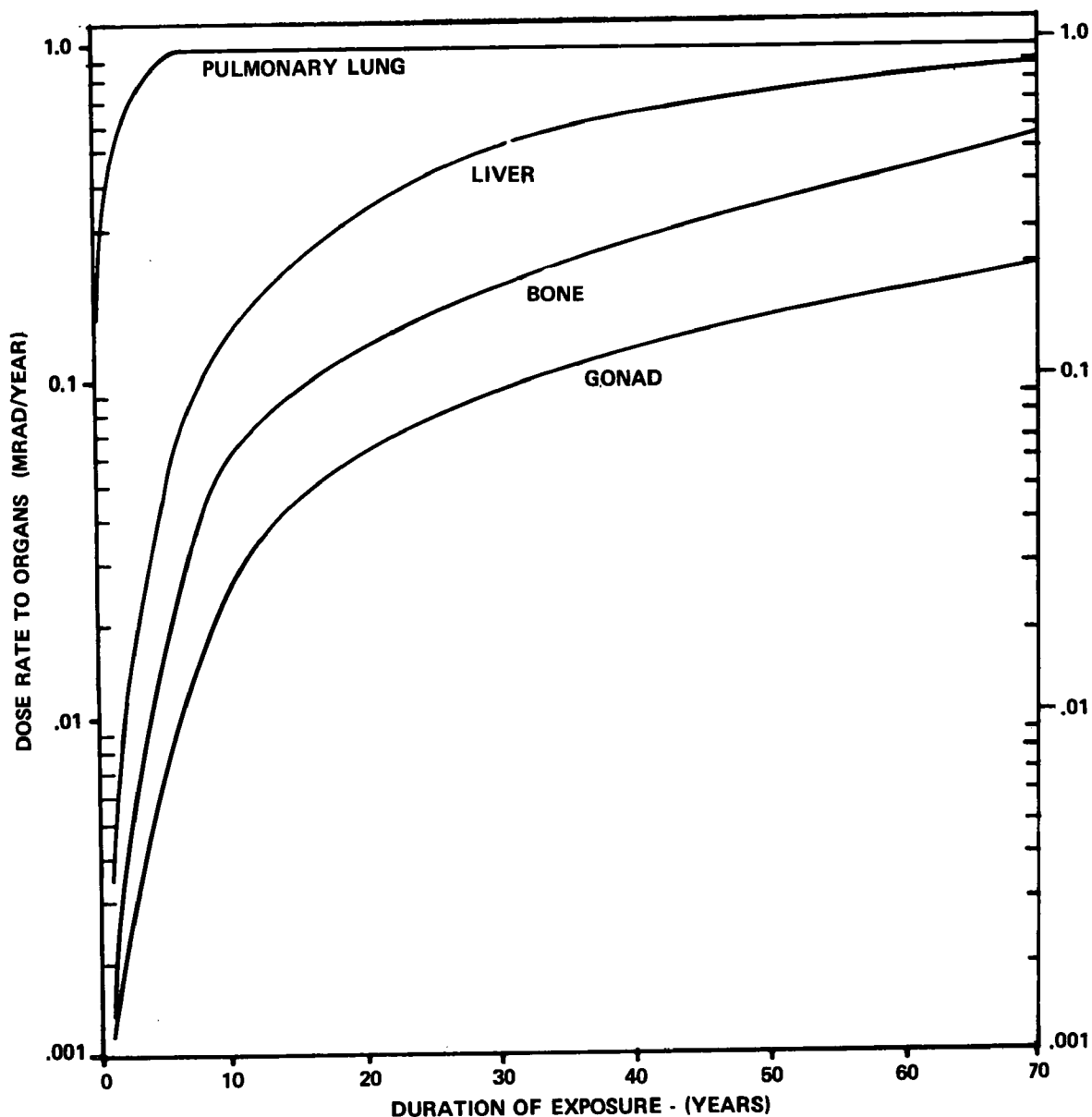
Estimated Risk Due to the Inhalation of Transuranic
Aerosols for a 1 mrad/yr. Lifetime Exposure

Estimated Somatic Risk in a
Cohort of 100,000 Persons

<u>Cause of early death</u>	<u>Premature deaths</u>	<u>Range</u>
Lung Cancer	8	2-25
Bone Cancer	0.3	0.2-.7
Liver Cancer	0.3	0.2-0.6
Leukemia & other causes	0.12	0.06-0.25
Estimated total premature deaths	9	3-20

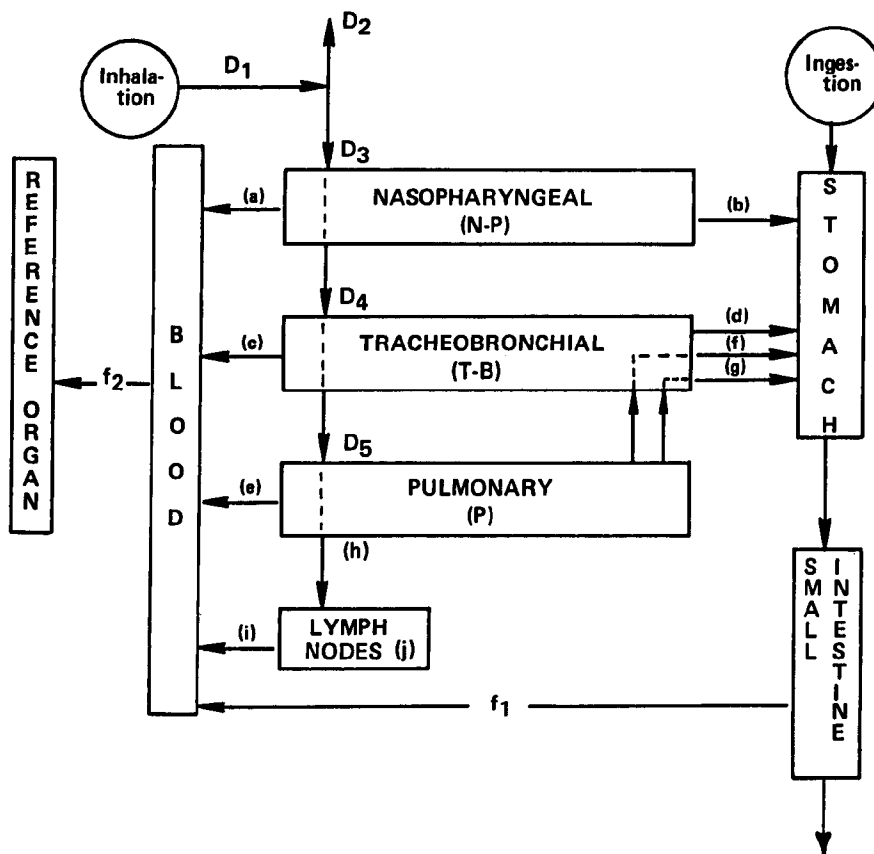
Estimated Genetic Risk
per 100,000 Live Births

First generation	---	0.1-3
All generations	---	1-20



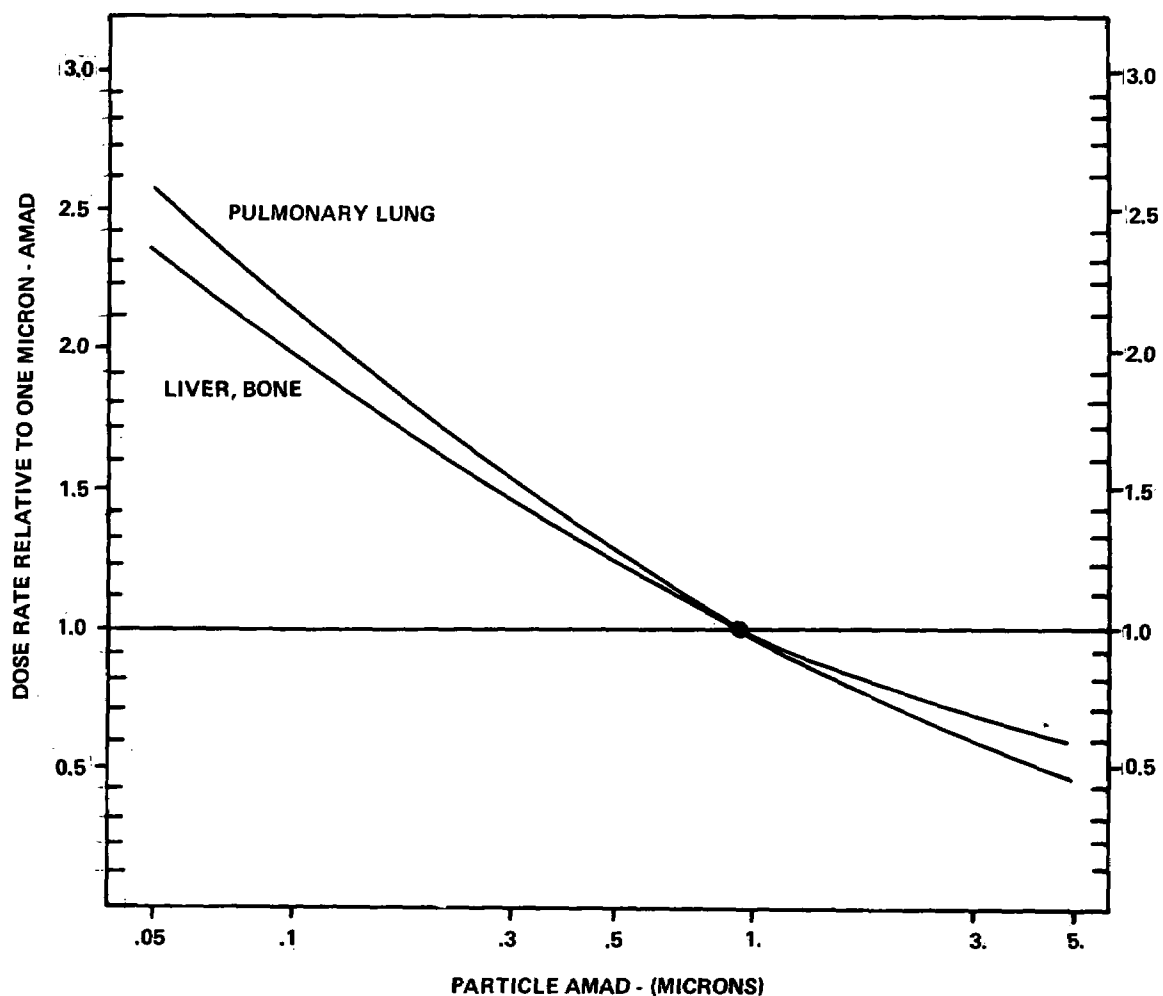
DOSE RATE TO ORGANS AS A FUNCTION OF TIME DUE TO CHRONIC INHALATION OF ONE MICRON (AMAD) PLUTONIUM PARTICLES AT A CONCENTRATION OF 2.6 fCi/m³. EQUILIBRIUM DOSE RATE TO PULMONARY LUNG 1 MRAD PER YEAR ADULT REFERENCE MAN — BREATHING RATE 2.3 x 10⁴ LITERS PER DAY.

FIGURE A3-1



D^1 IS THE TOTAL AEROSOL INHALED; D^2 IS THE AEROSOL IN THE EXHALED AIR; D^3 , D^4 , AND D^5 ARE THE AMOUNTS DEPOSITED IN THE NASOPHARYNGEAL, TRACHEOBRONCHIAL, AND PULMONARY LUNG RESPECTIVELY. THE LETTERS (a) THROUGH (j) INDICATE THE PROCESSES WHICH TRANSLOCATE MATERIAL FROM ONE COMPARTMENT TO ANOTHER. VALUES FOR THESE PARAMETERS ARE LISTED IN TABLE A3-1.

FIGURE A3-2



RELATIVE DOSE RATES DUE TO CHRONIC INHALATION AS A FUNCTION OF PARTICLE SIZE (AMAD). FOR THE PULMONARY LUNG, THE DOSE RATE AT EQUILIBRIUM; FOR LIVER AND BONE, IN THE 70th YEAR. THE CASE SHOWN IS Pu-239. THE CURVES ARE VIRTUALLY IDENTICAL FOR ALL THE TRANSURANIUM ELEMENTS CONSIDERED IN THIS STUDY.

FIGURE A3-3

Annex IV
RISK PERSPECTIVES

U. S. Environmental Protection Agency
Office of Radiation Programs
Washington, D.C. 20460

Annex IV

Table of Contents

	<u>Page</u>
1. Introduction	1
2. Life Table Methodology	1
3. Risk From Background Radiation	4
4. Other Risks Experienced By the General Population....	7

Annex IV

Risk Perspective

1. Introduction

The purpose of this annex is to provide a perspective for evaluating the somatic component of the risk from transuranium elements. Life table measures of risk of death are developed for some of the diseases and accidents presently experienced by the U.S. population. They are comparable to the life table risk measures provided in Annex III.

Before proceeding with the discussion of what has been done to provide a perspective for evaluating this risk, the general characteristics of life table models will be described.

2. Life Table Methodology

Life tables are a method for following hypothetical cohorts of individuals through their life spans, from birth to death. The cohort is assumed to be subject throughout its existence to the age specific mortality rates observed for an actual population.

The National Center for Health Statistics (NCHS) publishes life table models for the U.S. population, which incorporate age specific mortality rates for the U.S. population (1,2). They are used to estimate the life expectancy of the U.S. population.

EPA has developed life table models (based on the NCHS models) for measuring the impact of changes in the rate of mortality. Changes

are measured from the mortality rates incorporated into the NCHS tables. These changes may represent increases or decreases in the mortality rate. Increases result when people are exposed to additional risks not a component of the U.S. population average. Exposure to the ionizing radiation emitted by transuranium elements is an example of an increased mortality risk.

Decreases in mortality would result from the elimination of one or more of the causes of death presently experienced. An example (which is used in this annex) is the hypothetical elimination of lung cancer as a cause of death in the U.S. population. When these calculated results from the EPA model are compared to the results of the NCHS model, the impact of lung cancer mortality on the U.S. population can be estimated.

Because of methodological differences, the discussion of the EPA life tables will consider the case of increased and decreased mortality separately.

The life table concept of life expectancy has attractive features for measuring risk, for it is based on the years of life lived by the members of the cohort. EPA's models rely on standard life table methodology to determine the sum of the years of life lived. The difference in years of life lived in the EPA and NCHS models is the net change in total years of life lived by the two cohorts.

For the case of increases in the rate of mortality caused by exposure to transuranium elements, the net change is downward (the years

of life lived by the cohort in the EPA model is less than that for the NCHS model). The EPA model has been designed so that the numbers of deaths attributable to the increased rate of mortality can be calculated. These are premature or early deaths because the individuals die at an earlier age than they would have, had there been no increase in the mortality rate (i.e. had they not been exposed to transuranium elements). The net decrease in years of life lived divided by the number of premature deaths is the measure of the average years of life lost to those dying prematurely as a result of the exposure.

Lung cancer will be used as an example for discussing decreased mortality rates resulting from the elimination of a cause of death. The elimination of other causes of death uses the same methodology. The years of life lived increases when the mortality effects of lung cancer are eliminated. The EPA model determines the numbers of lung cancer deaths averted by eliminating lung cancer as a mortality risk. The net increase in years of life lived divided by the number of lung cancer deaths is the average years of life gained to those whose deaths by lung cancer have been averted. This effect can also be interpreted as a change in the opposite direction, from a situation where there is no lung cancer mortality to one where lung cancer mortality is the same as presently experienced in the U.S. With this change in interpretation, the net change in years of life lived is considered a decrease, for it measures the years of life lost to the cohort because of lung cancer mortality. This value divided by the number of lung cancer deaths gives the average years of life lost to those dying of lung cancer. This change in

interpretation facilitates the comparison of the risks from transuranium elements with the risk of lung cancer. This is the interpretation to be used in this Annex.

A discussion of life table methodology and the technique for removing specified causes of death from the life table are discussed in Chiang (3). Life tables similar to the EPA models, with specified causes of death removed, are published by NCHS (4). The basic methodology used here for developing life table estimates is similar to that discussed in the technical report, "Life Table Methodology for Evaluating Radiation Risk" (5).

The remainder of this annex is devoted to the estimation of the risk of death from a variety of diseases and accidents. Results are shown in Tables A 4-1 and A 4-2. These can be compared to the risk from transuranium elements calculated in Annex III.

3. Risk From Background Radiation

The average annual background radiation equivalent dose received by the U.S. population is approximately 100 mrem over the whole body. It is assumed that on a per rem basis background radiation will have the same impact on cancer incidence as other forms of radiation. Two situations can be evaluated: one where all risks of death are the same as presently experienced and the other (a hypothetical situation) where the risk of mortality from 100 mrem whole body dose is removed. The results are shown in Table A 4-1.

All risk estimates for whole body dose are taken from the BEIR report, Table 3-2, p. 171. Results of four models are shown, based on

relative and absolute risk and on the 30 year and life plateaus for cancer other than leukemia as defined in the BEIR report (6).

Four measures of risk are shown for each life table model shown in Table 1. The first measure is the numbers of deaths attributed to background radiation. Numbers of deaths furnishes no perspective on the prematurity of the deaths, since it provides no measure of the years of lifetime lost because of these deaths. The other values in Table A 4-1 are measures of the lifetime lost.

The basic measure of prematurity of death is the aggregate years of life lost. Values for each model are shown in Table A 4-1. This aggregate can be used to measure the reduction in average length of life for the cohort. This measure expresses the viewpoint at the beginning of life when all members of the cohort face equal risk of death from background radiation. The average reduction in years of life lived by the cohort (reduction in life expectancy at birth) is calculated by dividing aggregate years of life lost by 100,000 (the cohort size at birth). This value is shown in the table. The aggregate years of life lost can also be used to measure the average loss of life to those dying of background radiation induced cancer. The members of the cohort can be considered in two groups, those that die of background radiation induced cancer and those that do not. The second group has the same life expectancy as it would if there were no background radiation. Those that do die from background radiation have a reduction in life expectancy equal to the average years of life lost to premature deaths.

The four models (for absolute and relative risk and for 30 year and life plateaus) provide estimates of cancer deaths ranging from 69 to 490 over the life of the cohort. Aggregate loss of life ranges from 1700 to 6600 years. Reduction in life expectancy at birth ranges from .0017 to .0066 years. The individuals suffering the deaths induced from the background radiation suffer an average shortening of their lives ranging from 14 to 25 years depending on the model.

These results can be compared with the measures of risk presented in Tables A 3-10 through A 3-14 and A 3-17 and 18 in Annex III. Table A 3-10 displays the estimated measures of lifetime risk of lung cancer induced by inhaled transuranium elements. Estimated numbers of early deaths range from 2.15 to 25.0 with an aggregate years of life lost ranging from 47 to 368 years. The average years of life lost to those suffering early deaths ranges from 14.7 to 22.0 years. Exposure to 1 mrad per year is therefore estimated to have a much smaller lifetime impact than does background radiation.

Measures of lifetime risk of liver cancer mortality from inhaled plutonium are shown in Table A 3-13. This impact is considerably below that of lung cancer induced by inhaled transuranium elements, and is therefore lower in comparison to background radiation. Tables A 3-10,12,14 and 17 of Annex III display measures of risk to other organs from inhaled and ingested transuranics. Results show that lung cancer induced from inhaled plutonium is the major contributor to the risk from exposure to transuranium elements. The cumulative risk from all organs is not large in comparison to that from background

radiation, as can be seen from the summary of premature deaths from inhaled transuranic aerosols for a 1 mrad per year lifetime exposure (Table A 3-18).

These life table models show that lost years of life have an almost insignificant impact on life expectancy at birth, but that those that die prematurely from the exposure suffer a substantial shortening of their lives. The life shortening is approximately the same for both forms of risk: exposure to transuranium elements and background radiation.

4. Other Risks Experienced By The General Population

Table A 4-2 illustrates a few of the risks of death the U.S. population presently experiences from various causes. All estimates are based on the life table model. Mortality rates are derived from 1969 mortality statistics for the U.S (2). The measures of risk are the same as those used in Table A 4-1 and are interpreted in an analogous manner. The first risk shown is that from malignant neoplasms. Risk is measured by removing malignant neoplasms as a cause of death and comparing the situation that would then prevail against that for the case where it exists at the presently experienced level.

The Table shows that a cohort of 100,000 individuals suffers 2900 spontaneously occurring lung cancer deaths. These deaths take an aggregate of 45,000 years of life from the cohort. Therefore, life expectancy at birth is reduced .45 years due to lung cancer. The average loss of life to those individuals that die of lung cancer is 15 years. These results can be compared to the results in Table A 3-10. Although the impact of lung cancer is much greater than that for inhaled

plutonium or for background radiation, the years of life shortening to those that die of lung cancer is approximately the same.

Other models presented in Table A 4-2 measure the impact of a variety of non-cancerous causes of death.' The numbers of deaths range from a high of 1000 to a low of 2, and the aggregate years of life lost to the cohort ranges from 12000 to 80. These impacts are considerably smaller than those for malignant neoplasms or for lung cancer.

Some well known diseases are represented in the table. All incorporate some risk of death. Some, such as chicken pox, are not normally believed to cause death. As can be seen chicken pox does involve a small risk of death. The average of 56 years of life lost to those dying of chicken pox reflects that its impact is primarily on the young.

The table also has measures of the impact of various accidental causes of death. The accidental risks shown range from 1000 deaths due to falls to 8 for deaths from accidents caused by electric current from home wiring and appliances. The average years of life lost to those that die of these accidents reveals that different forms of accidental death impact at different ages.

Table IV-1
Measure of Lifetime Risk of Mortality from Background Radiation*
(Cohort size = 100,000)

<u>Measure of Risk</u>	<u>Risk Models</u>			
	<u>Relative Risk Estimate</u>		<u>Absolute Risk Estimate</u>	
	<u>30 Year Plateau</u>	<u>Life Plateau</u>	<u>30 Year Plateau</u>	<u>Life Plateau</u>
Premature Deaths	150	490	69	84
Aggregate Years of Life Lost to Cohort	2700	6600	1700	1900
Reduction in Life Expectancy at Birth (in years)	0.027	0.066	0.017	0.019
Average Years of Life Lost to the Premature Deaths	17	14	25	22

*All mortality effects shown are calculated as changes from the U.S. Life Tables for 1970 to life tables with the risk of mortality from all forms of cancer reduced to account for the removal of background radiation. These effects also can be interpreted as changes in the opposite direction; from life tables with the cause of death removed to the 1970 life table. Therefore, the premature deaths and years of life lost are those that would be experienced in changing from an environment where there is no background radiation to one where background radiation is present. Background radiation is assumed to be equal 100 mrem per year. Risk estimates are based on the NAS-BEIR Report. All values rounded to no more than two significant figures.

Table IV-2
Measure of Lifetime Risk of Mortality from a Variety of Causes¹
(Cohort size = 100,000)

Cause of Death	Premature Deaths	Aggregate Years of Life Lost to Cohort	Reduction in Life Expectancy at Birth (in Years)	Average Years of Life Lost to Premature Deaths
Malignant Neoplasms (140-209) ²	16,000	250,000	2.5	15
Malignant Neoplasms of Trachea Bronchus and Lung (162)	2,900	45,000	0.45	15
Accidental Falls (E880-E887)	1,000	12,000	0.12	11
Accidents Caused by Fires and Flames (E890-E899)	300	7,600	0.076	26
Tuberculosis, All Forms (010-019)	270	4,300	0.043	16
Accidental Drowning and Submersion (E910)	190	8,700	0.087	45
Asthma (493)	100	2,100	0.021	20
Accidental Poisoning by Drugs and Medicaments (E850-E859)	69	2,500	0.025	37
Appendicitis (540-543)	67	1,200	0.012	17
Accidents Caused by Cataclysm (E908) ³	17	490	0.005	30
Accidents Caused by Bites and Stings of Venomous Animals and Insects, and Other Accidents Caused by Animals (E905, E906) ⁴	8	220	0.002	27

Table IV-2
continued

<u>Cause of Death</u>	<u>Premature Deaths</u>	<u>Aggregate Years of Life Lost to Cohort</u>	<u>Reduction in Life Expectancy at Birth (in Years)</u>	<u>Average Years of Life Lost to Premature Deaths</u>
Accidents Caused by Electric Current in Home Wiring and Appliances (E925.0) ⁵	8	290	0.003	37
Tetanus (037)	4	80	0.001	20
Chicken pox (052)	2	130	0.001	56

¹All mortality effects shown are calculated as changes from the U.S. Life Tables for 1970 to life tables with the cause of death under investigation removed. These effects also can be interpreted as changes in the opposite direction, from life tables with the cause of death removed to the 1970 Life Table. Therefore the premature deaths and years of life lost are those that would be experienced in changing from an environment where the indicated cause of death is not present to one where it is present. All values rounded to no more than two significant figures.

²ICDA Codes, 8th Revision.

³Cataclysm is defined to include cloudburst, cyclone, earthquake, flood, hurricane, tidal waves, tornado, torrential rain and volcanic eruption.

⁴Accidents by bite and sting of venomous animals and insects includes bites by centipede, venomous sea animals, snakes and spiders; stings of bees, insects, scorpions and wasps; and other venomous bites and stings. Other accidents caused by animals includes bites by any animal and non-venomous insect; fallen on by horse or other animal; gored; kicked or stepped on by animal; ant bites; and run over by horse or other animal. It excludes transport accidents involving ridden animals; and tripping, falling over an animal. Rabies is also excluded.

⁵Accident caused by electric current from home wiring and appliance includes burn by electric current, electric shock or electrocution from exposed wires, faulty appliance, high voltage cable, live rail and open socket. It excludes burn by heat from electrical appliance and lightning.

References

1. U.S. Department of Health, Education, and Welfare, Public Health Service. U.S. Decennial Life Tables for 1969-71, Volume 1, Number 1, May 1975.
2. U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics. Excerpt from Vital Statistics of the United States 1969, Volume II-Mortality.
3. Chiang, Chin Long. Introduction to Stochastic Processes in Biostatistics. John Wiley & Sons, Inc., New York, London, Sydney. (A Wiley Publication in Applied Statistics).
4. U.S. Department of Health, Education, and Welfare, Public Health Service, U.S. Decennial Life Tables for 1969-71, Volume 1, Number 5, May 1975.
5. Bunker, Byron M., Barrick, Mary Kay, and Cook, John, "Life Table Methodology for Evaluating Radiation Risk". Office of Radiation Programs, U.S. Environmental Protection Agency, Technical Note (in preparation).
6. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. National Academy of Sciences, National Research Council Washington, D. C., November 1972.

ANNEX V

Guidance Implementation

**U. S. Environmental Protection Agency
Office of Radiation Programs
Washington, D. C. 20460**

Contents

	<u>Page</u>
1.0 Introduction	1
2.0 Implementation of Guidance by Estimating Dose Rates to Lung and Bone	4
2.1 Dose Rate to the Lung	4
2.2 Dose Rate to the Bone	6
3.0 Implementation of Guidance by Use of a Soil "Screening Level"	8
4.0 Implementation of Guidance by Means of Soil Data Using Site-Specific Parameters	9
5.0 Sampling and Analysis Methods	11
5.1 Statistical Criteria	12
5.1.1 Soil Sampling	12
5.1.2 Area Acceptance Criteria	14
6.0 Remedial Actions	15
6.1 Costs of Remedial Actions	17

1.0 Introduction

The Environmental Protection Agency is issuing guidance directed to all Federal Agencies in response to the problem of environmental contamination by the transuranium elements. The guidance recommends annual dose rate limits to members of the public in the critical segment of the exposed population for pulmonary lung and for bone. Implementation procedures that may be used to determine compliance with this guidance are discussed in this annex.

Implementation of these recommendations should include an evaluation of the site, a projection of the radiation dose rate to determine whether or not guidance values are being exceeded, and remedial actions if there is indication that guidance values are, or may in the future be exceeded. A reasonable evaluation of a contaminated site should include a description of the site and environmental measurements of contamination levels in environmental media at a level of detail sufficient to convey adequate information to the general public. All dosimetry and environmental pathway models used in estimation of radiation doses to persons should be described in sufficient detail to permit evaluation of the procedures used. If projected dose rates are greater than the guides, protective or remedial actions should be performed to the extent necessary, so that guidance values are not exceeded and will not be exceeded in the foreseeable future. The implementation of these recommendations is the responsibility of those Agencies having regulatory and administrative responsibilities for the site in question and/or the materials in use at that site.

The Agency believes that these recommendations can be implemented by using one of three general procedures without requiring unreasonable, unnecessary, and expensive regulatory actions. These procedures, which are described in more detail in the following sections of this annex, may be used for the entire site or for portions of the site as appropriate:

a. dose rates can be calculated, using the appropriate dosimetry models, from measurements of the concentration of the transuranium elements in air, food, and water at the point of inhalation and/or ingestion by people. This is the most direct and preferred method.

b. soil concentration levels of the transuranium elements can be compared to a "screening level" for soil; defined as that level below which the concentration of the transuranium elements are not likely to lead to dose rates in excess of guidance recommendations.

c. dose rates can be calculated from the soil contamination levels of the transuranium elements using site-specific parameters for transport models and the appropriate dosimetry models.

An implementation program for obtaining needed information at minimum cost may best be designed according to statistical criteria for sampling, measurements, and analysis. By selecting such criteria appropriate to the site, adequate data can be obtained for making a decision on any required action without unnecessary cost due to overly restrictive conservatism or inefficient design.

In the context of this guidance, the objective of environmental sampling and analysis is to derive information for the purpose of

estimating dose rates to pulmonary lung and to bone of exposed individuals. These dose estimates are derived on the basis of models which depict the various pathways by which transuranium elements in the environment may interact with man and produce exposure to radiation. These models include parameters which describe the characteristics of transuranium elements in the environment, the manner in which they may be transported through the air or through food pathways, modes of interaction with man (including inhalation or ingestion) and, finally, factors related to the radiation energy deposition in organs and tissues. In general, the best dose estimates are derived from data acquired from measurements in the dose pathway as close as possible to the point where transuranium elements interact with man, although it is sometimes necessary to make measurements of the radionuclide concentrations at points in the environment further from the receptor, followed by the use of pathway models to estimate doses to individuals or population groups.

A soil sampling program may be utilized to determine compliance with the recommendations. Most sites where contamination of the soil presently exists have been surveyed to determine levels of transuranium elements. The data should be used to indicate areas which are clearly much greater or much smaller than the limiting soil concentration derived from the guidance recommendations. It is only for those land areas in between, which may or may not exceed the guidance recommendations, that it will be necessary to conduct a sampling program.

The guidance is intended to be applicable to all sites and all types of land utilization. Evaluation must be made on the basis of

present and projected conditions and include recognition that disturbance of the soil surface may change both the pathway to humans and the magnitude of the accumulated dose. For the inhalation pathway, most of the potential hazard is derived from contamination at, or near the surface. Most man-made disturbance will reduce the concentration in the top layer either by dilution or removal, but may increase the resuspension rate. The overall effect of such activities must be considered in the implementation plans.

2.0 Implementation of Guidance by Estimating Dose Rates to Lung and Bone

Federal Agencies may show compliance for a specific site, or for sub-areas of a specific site, by certifying that guidance values for dose rates to the lung and bone of members of the critical segment of the exposed population are not being exceeded. The most direct method is to measure transuranium element concentrations in environmental media such as air, food, and water at the point of interaction with man and to then calculate the potential radiation dose rates using the appropriate dose conversion factors and dose model parameters. When this procedure is used, adequate documentation should be provided to demonstrate how dose rates are calculated. The Agency favors the use of realistic environmental measurements and realistic model input parameters; conservative parameters should only be used to the extent necessary to compensate for uncertainties.

2.1 Dose Rate to the Lung

Lung dose rates are calculated using appropriate dosimetry models, which require knowledge of the annual average transuranium element

concentration in air, aerosol particle size distributions, and solubility class of the specific radionuclides present. Procedures for the sampling and analysis of near-ground level air for the transuranium elements have been published and have been used for many years. Procedures of sufficient sensitivity, accuracy, and precision are available for implementation of this guidance.

Judgment should be exercised in the design of an air sampling program to ensure that air concentration levels are representative of actual exposure conditions. Environmental measurements of airborne particulates which bias the dose estimates by the collection of only certain particle size ranges should be avoided or a suitable correction should be made. It is preferable that the particle size distribution be experimentally measured for a specific site. Reasonable values can be assumed based on analogies with similar sites when projected lung dose rates are small compared to the guidance level. The solubility class of an aerosol can usually be determined from the history of the contaminating event and the subsequent environmental weathering mechanisms. Dose conversion factors for lung dose rates that the Agency believes to be reasonable for the purpose of implementation of the guidance are presented in Annex III.

In certain cases, the determination of site-specific parameters for use in the dosimetry models may be difficult or impossible with the equipment available or within the time constraints allowed. Under these circumstances, a derived air concentration limit, which will have a

very large probability that the guidance recommendations will not be exceeded, may be substituted for the site-specific value. The Agency suggests that such a derived air concentration limit be based on an activity median aerodynamic particle diameter (AMAD) not to exceed 0.1 μm , which is substantially smaller than observed values at all known contamination sites. The calculated limiting concentration for this procedure would be about 1 fCi/m³ of alpha emitting transuranium nuclides, for air samples averaged over a period of one year or more. Air concentrations above this value do not necessarily mean that the guidance recommendations may be exceeded, but rather dictate that a more thorough evaluation of existing conditions be made.

Elevated levels of transuranium elements in air indicate that these elements may be found in nearby soils. When these levels approach that of the guidance recommendation, implementation should include a characterization of the environmental source term, to provide a means of judgment with respect to the potential for future exposure levels and the practicality of remedial measures.

2.2 Dose Rate to the Bone

Bone dose rates are calculated with appropriate dosimetry models using a knowledge of the average amounts of transuranium elements that are ingested in a year, the chemical state of the transuranium elements at the time of ingestion, and the proper dose conversion factor. Inhalation of transuranium elements, especially in soluble forms, can also lead to radiation doses to bone and should be considered where appropriate.

Sampling and measurements of transuranium elements in food and water at the point of human consumption is the most direct and preferred procedure for determining the annual average ingested amount of these elements. Alternatively, the amounts of ingested radionuclides may be estimated using environmental pathway models. The chemical state at the time of ingestion is inferred from the medium into which the transuranium elements are incorporated at the time of ingestion. In particular, transuranium elements which are incorporated into biological tissue when ingested should be considered as "organically complexed" and require a special dose conversion factor. Dose conversion factors that the Agency believes appropriate for the implementation of this guidance with respect to bone dose rates are given in Annex III.

The Agency believes that suitable sampling and analytical procedures are available for the analysis of the transuranium elements in food and water and that they have the necessary sensitivity, accuracy, and precision for purposes of implementing of this guidance. Also, as with the inhalation pathway, elevated levels of plutonium and the transuranium elements in food or water indicate that these elements may be found in nearby soil or in sediments. Under such conditions, implementation of the guidance should include a characterization of the environmental source term, to provide a means of judgment with respect to the potential for future exposure levels and the practicality of remedial measures.

3.0 Implementation of Guidance by Use of a Soil "Screening Level"

Federal Agencies may show compliance for the total area of a site, or for subareas of a site, by certifying that such areas have transuranium element soil concentration levels less than a screening level value of $0.2 \mu\text{Ci}/\text{m}^2$. The "screening level" is a total transuranium element soil concentration level in the top 1 cm of soil such that, in the Agency's opinion, dose rates will not exceed guidance recommendations under the vast majority of land use conditions. Its usefulness is limited to the area in close proximity to the measurement, in that a dynamic equilibrium between the surface and adjacent air column is localized and only indirectly contributes to the adjacent areas.

Because of present uncertainties in the amount of plant uptake for the more soluble transuranium nuclides, such as americium and curium, and the resultant possibility of larger doses via the ingestion pathway than calculated, the screening level concept may not be applicable when the soils of a contaminated area contain these nuclides in amounts greater than 20% of the total activity. Lands with concentration levels less than the screening level are judged to be suitable for all normal activities including residential and agricultural uses. The use of this screening level is intended to reduce the land areas requiring extensive evaluation and to minimize the number of measurements needed.

If land areas have transuranium element levels greater than the screening level, it should not be presumed that guidance values are necessarily exceeded, because conservative assumptions were used in the

derivation of the screening level. Additional site specific evaluations of potential dose rates to lung and bone (Section 4) should be made before remedial actions are initiated.

Inherent in the application of the screening level is the assumption that soil contamination by the transuranium elements will cause radiation exposure through pathways such as the inhalation of resuspended soil, the ingestion of foodstuffs grown on the soil, the ingestion of soil by children, and the ingestion of drinking water contaminated by soil runoff. In all cases the cumulative doses to the critical segment of the population must be considered, with the admonition that the accumulated doses from all pathways should not exceed those recommended in this guidance.

4.0 Implementation of Guidance by Means of Soil Data Using Site-Specific Parameters

Federal Agencies may show compliance with this guidance for a specific site, or for subareas of a specific site, by means of soil measurements and by using pathway and dosimetry models with parameters determined for that specific site to certify that the resulting dose rates do not exceed guidance values. This approach differs from the use of a soil screening level because parameters such as the resuspension factor are determined for a specific site. It is expected that use of site-specific parameters will show that soil contamination levels higher than the suggested screening level may correspond to organ doses well below guidance level. Implementation by site-specific parameters is appropriate where land areas have transuranium element levels greater than the screening level and further evaluation is necessary to

determine whether or not guidance dose limits are being exceeded.

The air concentration at the site of the receptor can be generally correlated with the adjacent soil concentration by use of a resuspension factor, and used to estimate the inhalation dose rate.

The site-specific resuspension factor may be either measured directly or calculated from other data. Direct experimental determinations are often difficult to make and not very reproducible. Therefore, calculational techniques are sometimes preferred although their correlation with measured values is subject to considerable uncertainty. The Agency has developed a method, based on the concept of air mass loading, which may be useful for this purpose (see Annex II). An "effective" resuspension factor is derived, defined as the resuspension factor derived from the air mass loading for the given location and modified by an "enrichment factor" which takes into account the generally observed nonuniform distribution of the activity with size of particles in calculating the amount of transuranium element activity in the inhalable fraction of the resuspended material. The "enrichment factor" is a theoretically derived parameter, and its correlation to actually observed situations has not yet been established. The resuspension factor derived in this manner is applicable only to an infinite plane source, and must be further corrected for the dilution by uncontaminated materials carried into the generally small contaminated area.

The ingestion pathway must be evaluated separately, using data applicable to the specific site in terms of type of crops, plant uptake

parameters, and pathway to a critical segment of the population. The more unusual transfer mechanisms to people, such as the ingestion of dirt by children and the contamination of drinking water wells, may also need to be examined if shown to be of importance.

5.0 Sampling and Analysis Methods

Choice of Methods

The choice of suitable methods for sampling and analysis is the responsibility of the Agency implementing the guidance. The implementing Agency should demonstrate that the methods that are used have the necessary sensitivity, accuracy and precision for purposes of implementing this guidance. A description of the tools and techniques used to collect the samples, the procedures for preparing the samples for analysis, and the method used for radiochemical analysis should be included.

Sample Collection

The Agency recommends that for undisturbed sites where soil measurements are taken to evaluate the inhalation pathway, soil samples should be taken to a depth of one centimeter and transuranium element activity be measured in all soil particles less than two millimeters in size. Several individual samples may be composited for a single measurement. At some sampling points it may not be possible to collect samples to a depth of one centimeter (for example, very stony soil or a thick grassy area). In such cases, other means must be found so that representative samples are collected.

Soil Particle Size Distribution Analysis

If a model for determining a transuranium element soil concentration corresponding to the lung dose rate limit is chosen which

involves the calculation of a soil particle resuspension factor, measurement of the soil particle size distribution may need to be made. The usual method for determining the distribution is by sedimentation analysis (1). The standard sedimentation technique employs wetting agents for dispersing agglomerated soil particles. Another method uses an oscillating air column for the dry separation of particles down to 5 μ m. For purposes of this guidance, soil characteristics should be altered as little as possible in the collection and preparation of the soil sample and care should be taken to choose a method which does not cause the breaking up of soil aggregates that were present when the sample was taken.

Radiochemical Analysis

Techniques for the determination of transuranium elements in soil have been published (2). Each of the more widely used techniques has been shown to be accurate under certain conditions. The principal differences between them are the techniques used to solublize the plutonium in the sample. Three solubilization techniques are most commonly used: acid leaching, acid dissolution, and fusion. The fusion method is considered to be applicable to a wider variety of soils than the other two methods.

5.1 Statistical Criteria

5.1.1 Soil Sampling

When planning a soil survey it is advisable to divide the total area under investigation into units at the very beginning of the survey rather than to collect samples more or less haphazardly. Then samples

taken to determine the acceptability of the land by comparison of measured concentration levels to the screening level may be collected from sampling units in accordance with a sampling plan. If it is later decided that more sampling is necessary, no change in the sampling plan is necessary, and the location for additional samples will have already been determined.

The number of samples to take within a sampling unit may be estimated from the specific statistical approach used in the sampling plan. An important factor affecting the number of samples to be taken is the risk of making the wrong decision in deciding whether a sampling unit is acceptable or requires remedial action. To reduce the risk of making the wrong decision, larger numbers of samples must be taken. Judgment must be used to strike a balance between the desirability of making the right decision and the difficulties and expense involved in taking large numbers of samples. An additional factor affecting the number of samples is the variability of the transuranium element concentration within a sampling unit. If detailed information is not available on the variability, a simple approach is to take the same number of samples within each unit. These could be taken on a grid system to ensure that all subareas of the sampling unit are sampled. A disadvantage of this approach is that if the variability is substantially different in different units, then the probability of detecting concentration levels requiring remedial action will vary from unit to unit. If estimates of variability are available from past studies, these can be used to help determine the number of samples required within each

unit so that the probability of making a correct decision will be the same for all units.

5.1.2 Area Acceptance Criteria

After soil concentration levels have been determined, it must be decided if the area under consideration complies with the guidance recommendations or whether further evaluation will be needed. The statistical methodology that is used must be such that few assumptions regarding the form of the soil concentration distribution will be necessary to ensure the validity of the statistical test. The method should also ensure reasonably low bounds on the risk of making the wrong decision, and the probability of not accepting an area which meets the guidance, or accepting one which does not, should be small. Acceptance criteria which allow a maximum chance of error of 5-10% are generally considered appropriate.

Considerable variation generally occurs in environmental samples taken even in closely adjacent locations. If one or more samples from any sampling unit exceed the air or soil concentration limits corresponding to the guidance recommendations, a decision must be made on whether the sampling unit is acceptable. Such a decision is best based on statistical tests which consider both the magnitude of the deviations from the average and the number of samples which are involved. A number of statistical methods are available for performing such an evaluation, and the choice must be made on the basis of the data available and the results desired.

The number of samples collected and analyzed should be sufficient to adequately guard against the errors of falsely failing to accept a land area when the true fraction does not exceed a lower bound and of falsely accepting an area when the true fraction is equal to or greater than the upper bound. The upper bound is a measure of that portion of the land area which would be considered reasonable to exceed the limiting soil concentration. A small fraction would provide assurance that the mean for the entire area, assuming a normal or known skewed distribution, would not exceed the screening level.

6.0 Remedial Actions

Remedial actions are required at a site when the projected radiation dose exceeds the recommended guidance values. Choosing a specific action is usually a complex decision affected by the physical characteristics of the site, the variety of appropriate remedial actions possible, and on monetary and non-monetary costs. The long half-lives of the transuranium elements makes the decision more difficult because consideration must be given to long-term care. The objective at a specific site is the selection of remedial actions that best assure that guidance recommendations will not be exceeded at the least possible cost.

Most remedial actions can be categorized into one of five general classifications: removal of the contamination for off-site disposal, removal for on-site shallow burial, stabilization with no removal of contamination, dilution of the contaminant with no removal, and measures restricting the use of the site by members of the public.

Methods of removing contamination include raking and grubbing out vegetation, stripping the top layer of soil by scraping, vacuuming, or other similar techniques. The contaminated soil and other material can be transported to especially designated locations on site for storage or shallow burial, or they can be shipped to off site depositories with provision for long-term care.

Stabilization of a contaminated site includes actions such as covering the land with an impermeable cover such as oil, polymerized plastics or asphalt, or with soil and vegetation, or by applying chemical stabilizers to the land area. The stabilization leaves the contamination on site, but reduces its accessibility to wind and surface water erosion and therefore reduces the potential exposure dose.

Plowing and cultivating are the principal methods of dilution of contamination. The goal is to mix the surface contamination into the top 20 cm or more of soil, attaining a form of stabilization in place. Estimates of the amount of dilution achieved by cultivation are suggested in Table A 1-2 of Annex I.

At some sites, it may be feasible to perform no cleanup, but rely completely on restricting land use. Restrictions are applied to the contaminated area itself, and may include a buffer zone bordering the contamination site. Land-use restrictions serve two purposes; they provide a means of controlling access to areas where the radiation health risk is excessive and they help in preventing the disturbance of the land surface. Land-use restrictions may limit or prohibit access to an area, or they may be limited to prescribing the types of activities carried out within an area.

6.1 Costs of Remedial Actions

It is to be expected that a variety of remedial actions should be effective in reducing the exposure dose. Under these conditions, the least costly action or set of actions should be selected. When costs are evaluated both monetary costs and nonmonetary costs, including the environmental costs, are to be considered. Whenever possible, it is desirable that all costs be quantified monetarily. Costs that cannot be quantified in monetary terms should, whenever possible, be quantified in other ways, with narrative descriptions used when quantification is not possible. A major difficulty is likely to be that different combinations of decontamination procedures are expected to have somewhat different combinations of monetarily and nonmonetarily, quantifiable and nonquantifiable costs.

Although it probably is impossible to identify and measure all costs in the evaluation of various remedial actions, it is desirable that as many as possible of the larger costs be evaluated. One constraint on any attempt at measuring costs is the cost of acquiring the information needed. Some costs may be easy to identify but expensive to quantify. Other costs may be quantifiable in nonmonetary terms, but it may be difficult or impossible to place a monetary value on them. Models may be useful in estimating the difference in costs of some remedial procedures. It should be noted that the cost of any radiological surveillance carried out to determine if a site exceeds the guidance does not impact on the selection of the least cost remedial action. However, all radiological surveillance costs necessary for the

performance of a cleanup procedure are a cost to that particular procedure.

Any general technique of remedial action is likely to incorporate costs associated with some combination of the following actions or procedures: (1) radiological surveillance, (2) protection of workers, (3) stabilization of land surface, (4) dilution, (5) removal of contaminated material from surface, (6) packaging, (7) transportation of contaminated materials, (8) ultimate disposal at storage site, (9) restoration of site, and (10) maintenance of restricted access to site. Costs for each must be evaluated as appropriate. When services are needed indefinitely, these future costs should be expressed as present worth.

Nonmonetary costs include all costs that are difficult or impossible to quantify in monetary terms. They should be quantified in nonmonetary terms to the extent possible. Descriptive discussion is appropriate where quantification is not complete or is impossible.

Examples of such costs would include the increased health risk to workers performing the remedial actions, the risk of health effects to members of the general population that may result when land surfaces are disturbed during remedial actions, and psychological costs resulting from fears of a little understood environmental contaminant.

References

1. Day, P.R., Particle Fractionation and Particle-Size Analysis, in Methods of Soil Analysis, Part I, C. A. Black, Editor, Amer. Soc. of Agronomy, Inc., Madison, Wisconsin 1965.
2. Bernhardt, D. E., "Evaluation of Sample Collection and Analysis Techniques for Environmental Plutonium:" U.S. Environmental Protection Agency, Technical Note, ORP/LV765.
3. Burstein, H. 1971 Attribute Sampling: Tables and Explanation. McGraw-Hill, New York.
4. Burr, I. W., Statistical Quality Control Methods, Marcel Decker, Inc., New York 1976.

ANNEX VI
ENVIRONMENTAL ASSESSMENT

U. S. Environmental Protection Agency
Office of Radiation Programs
Washington, D.C. 20460

Annex VI

Table of Contents

	<u>Page</u>
I. Introduction	1
II. Generic Impact Assessment	2
III. Alternatives to Proposed Action	4
IV. Projected Impact of Guidance at Existing Sites	8
V. Costs of Remedial Actions	13

Annex VI

ENVIRONMENTAL ASSESSMENT

I. Introduction

Under the provisions of the National Environmental Policy Act of 1969, it is intended that every major Federal action be examined in terms of projected impacts and that all available alternatives be considered. The purpose of such an analysis is to compare the costs and benefits of the recommended action with other options in terms of the broad range of projected health, sociological, economic, and environmental impacts.

The implementation of the proposed action would be site-specific. The portion of the environment affected is that where present or future transuranium element concentrations may exceed guidance values. Such areas are likely to be those where the transuranium elements are prepared, fabricated, used, stored, or transported. A number of such areas currently exist within the limits of the continental United States. Most such contamination is located on lands with restricted or limited access, but a few instances exist where significant contamination has spread to privately owned property.

The proposed guidance does not include recommendations on specific methods of cleanup and restoration. Such methods are to be determined for each contaminated site by consideration of the effectiveness of the techniques, the cost-benefit evaluation, and the specific environmental impacts. Therefore, the range of total impacts must be evaluated separately and independently for each proposed major remedial action in terms of all available and applicable methods.

II. Generic Impact Assessment

The probable impacts of implementation of the proposed action will vary according to the nature and scale of the method used for affecting cleanup and restoration, and may be particularly sensitive to the location of the proposed actions. The primary impacts of most methods of effecting desired cleanup and restoration of contaminated areas will result in some temporary disruption of normal activities on and near the site, slight and temporary impairment of air and water quality, and possibly significant effects on animals, flora, and fauna. The exact nature of such environmental impact will be site specific and dependent on the nature and extent of the site, the degree of contamination, and the procedures chosen for cleanup and restoration. Examples of adverse environmental impacts are: loss of habitat of fish, birds and mammals, increased mortality among displaced animals as well as loss of trees and other vegetation. Run-offs from disturbed land can lead to pronounced short-term aquatic impacts depending upon the amount of the chemical and biotic components of the system. A detailed study of such short- and long-term ecological impacts has been commissioned by the Environmental Protection Agency and results are expected to be available by late 1977.

Of special concern in the evaluation of potential environmental impacts is the irreversible or irretrievable commitment of resources. Such commitments range from permanent removal of the land from useful productivity to loss of scenic beauty and other intangible values. Cleanup and restoration inevitably involves a commitment of money,

labor, and equipment and this must be taken into consideration in an overall assessment. Both the temporary and permanent commitment of resources can be expected to increase as the stringency of the regulation becomes more severe, but not necessarily on a linear proportional basis.

The principal expected effects of a land cleanup and restoration action are those related to the anticipated benefit on the health and safety of individuals in the general population, the short- and long-term effects on the environment, the costs related to the total action, and the sociological and political consequences of these changes. A number of alternative levels of control need to be considered to gain an overall perspective from which to evaluate the overall impact.

Under Section 102(2)D of the National Environmental Policy Act of 1969, it is required to study, develop, and describe appropriate alternatives to the proposed or recommended courses of action. The purpose is to analyze the environmental benefits, costs and risks so as not to foreclose prematurely options which might better advance environmental quality or have less detrimental effect. Examples of such alternatives are those of taking no action, of postponing action pending further study, of taking actions of a significantly different nature which could provide similar benefits with less severe environmental impacts, or the acquisition or condemnation of land and waters. The analysis of each alternative should compare the environmental benefit, costs and risks with the proposed action. In summary, alternatives to the proposed action consist of (1) no action, (2) more stringent limits (3)

less stringent limits (4) alternative ways of implementing guidance (5) action which will achieve desired results by other means.

III. Alternatives to Proposed Action

A number of realistic alternatives were considered in the development of this guidance. The lowest level of effort would be that of maintenance of the status quo, with no remedial actions. This would gain zero benefit in that committed adverse health effects would not be reduced, have zero costs, and have no detrimental effect on the environment. The highest level of effort would be a uniform cleanup to fallout level background. This would gain a benefit of reducing the future number of adverse health effects to a very low number, but have a potential for very large monetary cost and widespread disruption of the environment. Such a level would be virtually impossible to achieve on a uniform basis and difficult to enforce because of major variations in the background level. Uniform cleanup to such a limit on a national scale would be prohibitively expensive, involve significant relocation of populations and disruption of activities, and result in major ecological damage.

A reasonable range of numerical limits for environmental contamination by the transuranium elements would appear to range from a lower bound set at some reasonable multiple of the average background level to an upper bound set at the limit required by consideration of public health criteria. These limits, and the costs and benefits at these levels, are considered below in more detail.

The least restrictive available guidance for population exposures resulting from soils contaminated by the transuranium elements is that derived from the numerical guidelines of the International Commission on Radiological Protection (ICRP) and given as recommended maximum permissible concentrations in air and water for specific radionuclides. For unrestricted occupancy this is 1 pCi/m^3 for insoluble and 0.06 pCi/m^3 for soluble plutonium-239 in air. Soil concentrations derived from these recommendations are of the order of $1000 \text{ } \mu\text{Ci/m}^2$, or higher than the proposed "screening level" by a factor of 5000. Numerical values of this magnitude would appear unacceptable for protection of an individual in the general population, in that the total permissible radiation exposure of that individual would be allocated to a single source or activity. Such numerical values could well be acceptable, however, for remote or intermittently inhabited areas where the maximum cumulative individual exposures would be considerable lower. Such relatively high contamination limits may also be appropriate where the decontamination costs are prohibitive and remedial actions must be limited to on-site stabilization and restricted occupancy. The costs associated with remedial actions only to this level could generally be expected to be lower than for the more restrictive guidance proposed.

The lowest presently existing limit applicable to land use of contaminated areas is the plutonium-in-soils standard of 2 dpm/g of dry soil adopted by the State of Colorado in 1973. This value represents approximately twenty times the average plutonium fallout background for Colorado, and also represents the threshold below which no increase in

the adjacent airborne particulate concentration was reported observable. The Colorado standard is intended as a worker protection level above which some control action needs to take place before construction work can proceed in a contaminated area. Because of the increased dust loading created by such activities the actual inhalation dose to exposed individuals could exceed the proposed guidance recommendation and no direct comparison of long-term impacts is possible. Because it is intended to achieve only a limited objective for a relatively short period of time, the Colorado standard cannot be considered in the context of long-term general public health protection.

On an administrative basis, a number of alternatives were available to this Agency for promulgation of appropriate guides or standards. The President's Reorganization Plan No. 3 of 1970 transferred certain functions from the Atomic Energy Commission to the Environmental Protection Agency "...to the extent that such functions of the Commission consist of establishing generally applicable environmental standards for the protection of the general environment from radioactive material." As a result of this transfer, Section 161(b) of the Atomic Energy Act provides that the Administrator may, within the above framework, "establish by rule, regulation, or order, such standards to govern the use of special nuclear material, source material, and by-product material as (he) may deem necessary or desirable to... protect health or to minimize changes of life or property."

The same Reorganization Plan also transferred all the functions of the former Federal Radiation Council, as specified in the Atomic Energy

Act, to the Agency. Section 274(h) provides that "the Administrator shall advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with States."

The Agency has considered both of the above options and decided in this instance to promulgate Federal Radiation Guidance. The decision was based, in part, on the realization that all transuranium elements are currently under the direct or indirect control of the Federal government, that the extent of current contamination is very limited, and that considerable flexibility may be required in implementation because of substantial differences between sites.

Other applicable legislative authorities delegated to the Agency include sections of the Clean Air Act, as amended, the Safe Drinking Water Act, and the Hazardous Materials Control Act. These other authorities are generally considered to apply to effluent releases rather than to existing environmental contamination, and therefore have limited applicability to the problems addressed in this guide.

The above discussion is intended only to convey the basis for the decision making process for a generic guidance. Application to specific sites will necessarily require a more detailed consideration of alternatives for possible remedial actions and/or land use restrictions where required. It is therefore important that a detailed impact evaluation be made in every instance prior to implementation of these guides.

A further alternative considered would involve issuance of no Federal guides, with remedial actions determined solely by State and local authorities. Such a course of action could be expected to result in a diversity of standards and regulations, not all of which may be derived on a timely or rational technical basis. In general, such regulations tend to be unduly restrictive when viewed from the objective of protecting the public health and reflect much of the fear of the unknown. Because the exact scope and application of such regulations cannot be predicted, no analysis of expected impacts can reasonably be made.

IV. Projected Impact of Guidance at Existing Sites

The proposed guidance is intended to provide a minimization of health risk to that level where no individual is exposed to greater than an equilibrium lung dose rate of 1 mrad per year, with an attendant maximum risk of one additional fatality per million persons exposed to that level per year. In terms of environmental contamination levels, this can be equated to approximately one hundred times the current contamination levels derived from fallout. For a parametric analysis of the effect of changing the proposed guidance in either the positive or negative direction, a reference case of a deviation by a factor of ten can be assumed. The lower bound will then be of the order of magnitude of the Colorado standard and the upper bound at a level currently exceeded in only a few instances on unrestricted lands.

There are four Federal sites in the United States that presently have transuranium element contamination above ambient levels beyond

their boundaries. These include the Rocky Flats Plant in Jefferson County, Colorado, Mound Laboratory in Miamisburg, Ohio, Nevada Test Site in southern Nevada, and Trinity Test Site near Alamogordo, New Mexico. The majority of all contamination released is confined within areas under the direct control of the Federal government, which imposes restrictions on the access and use of these areas. Relatively small amounts of transuranium element contamination exists outside the boundaries of these sites on lands generally accessible to the public. The following discussion is intended to supply a perspective of applying the guidance recommendations to these sites in terms of a soil concentration reference level derived on the basis of generic data, which with a very high degree of probability would be expected to result in an inhalation dose to an individual not to exceed the guidance recommendations. Use of such a soil contamination level is intended solely to provide a basis for comparisons and does not imply direct correlation with the dose rate recommendations. A brief description is given for each site and the general contamination pattern is indicated. Numerical comparisons to show the estimated areas of limiting contamination to one-third and one-tenth the reference level, and of allowing ten times greater a value are given in Table VI-1. Comparisons are made in terms of areas outside the boundaries of these sites.

The Rocky Flats Plant (RFP) produces components for nuclear weapons. Barrels containing plutonium contaminated cutting oil slowly corroded and some of the contents eventually leaked into the environment and were dispersed. On the basis of soil concentration data, all

Annex VI

Table VI-1

Comparison of Costs of Remedial Actions At Various Sites
of Existing Plutonium Contamination For Several Possible
Levels of Maximum Soil Concentrations (Areas are Estimated
from Contour Maps and Costs Are Arbitrarily Assumed as \$500/acre)

	Reference Level 0.2 $\mu\text{Ci}/\text{m}^2$		10 x Ref. Level 2 $\mu\text{Ci}/\text{m}^2$		1/3 Ref. Level 0.07 $\mu\text{Ci}/\text{m}^2$		1/10 x Ref. Level 0.02 $\mu\text{Ci}/\text{m}^2$	
	Area	Cost	Area	Cost	Area	Cost	Area	Cost
Rocky Flats Plant	0	0	0	0	0.3 mi^2	100K	$\sim 1.6 \text{ m}^2$	500K
Nevada Test Site	0	0	0	0	<80 mi^2	25M	< 165 mi^2	50M
Trinity Site	0	0	0	0	<20 mi^2	<6M	$\sim 300 \text{ mi}^2$	100M
Mound Lab.	$\sim 0.01 \text{ m}^2$	*	$\sim 0.01 \text{ mi}^2$	*	$\sim 0.01 \text{ mi}^2$	*	$\sim 0.01 \text{ mi}^2$	*

* Most of the existing contamination is in sediments of canals, and does not represent a hazard to humans. Costs of eventual remedial actions are indeterminate.

off-site areas at the Rocky Flats Plant would probably be in compliance with guidance recommendations. However, more intensive evaluation may be needed to determine the actual dose rates to the general population, particularly in the most highly contaminated areas east of the plant. The area is sparsely inhabited and there are few people living in the particular area of concern. The off-site area contaminated to a level one-tenth the screening level comprises about 1.6 mi^2 with a current population of less than 600. No uncontrolled areas are contaminated to a level greater than ten times the screening level. All local water supplies are expected to yield ingestion dose rates well below the guidance recommendation.

Mound Laboratory is a major research and development site for fabrication of radioisotopic heat sources used for space and terrestrial applications. In 1969 a pipeline transporting a Pu-238 waste solution ruptured, spilling the contaminated solution. The plutonium migrated slowly into nearby waterways. The majority of the plutonium is now sorbed and fixed on the sediments of the North and South Canals. Maximum concentrations are 1 to 3 ft below the sediment surface and currently do not pose any radiation problem, since very little of the plutonium is in soluble form and the canal water is not used for drinking purposes. Banks immediately adjacent to the canal and overflow creek subject to occasional flooding have maximum plutonium concentrations exceeding the reference level. The amount of land in question is about $.01 \text{ mi}^2$ and there are no people living on this land. There are no areas with transuranium element contamination 10 times the screening

level. The amount of land contaminated to one-tenth the screening level is the same as the amount of land above the screening level, because the nature of the contaminating event limited the contamination to the waterways and adjacent banks. No immediate cleanup is indicated for this site, but continued surveillance will be required.

The Nevada Test Site (NTS) covers an area of 1400 mi^2 with an additional exclusion zone extending 16 to 48 miles. Major programs at NTS have included nuclear weapons tests, testing for peaceful uses of nuclear explosives, and nuclear reactor engine development. These activities have resulted in plutonium contamination in certain areas of the test site and exclusion areas and slight contamination (above background levels) outside the exclusion areas. There are no known uncontrolled areas which have transuranium element contamination exceeding the reference level. Land contaminated to one-tenth the reference level or less covers approximately 165 mi^2 with a resident population of less than 240 people.

The Trinity Test Site was the location of the first nuclear explosion. No other nuclear explosion tests were performed at Trinity. A site survey was performed by EPA during 1973-74 to determine residual plutonium concentration contours. The highest plutonium contamination levels in uncontrolled areas ranged from .02 to .09 $\mu\text{Ci}/\text{m}^2$. The amount of land contaminated to a level one-tenth the reference level covers less than 300 mi^2 , with fewer than 500 people living in the area in small towns, ranches, and farms. On the basis of the limited available

data, no major remedial actions would appear to be indicated for this site.

V. Costs of Remedial Actions

The Agency has evaluated the available methods and costs for cleanup and restoration of contaminated land areas. For soils with transuranium element concentrations no higher than about 10-100 times the guidance recommendations, remedial actions to bring such areas into compliance would generally involve only plowing or surface removal followed by restorative actions needed to prevent erosion and assist ecological recovery. The most common recommended methods include plowing, surface stabilization, soil cover, and soil removal. Specific steps required for each of these methods, and the associated costs, are summarized in Table VI-2. The costs of implementing the guidance can be expected to vary by location, contamination level, and other factors. Therefore, the numerical estimates shown can only be considered as a general approximation and specific values must be established for each site. Dollar values are given on a 1975 basis and must be adjusted by suitable indicators.

The range of options available for bringing an area into compliance with the guidance recommendations includes both the method(s) used for dilution or removal of surface contamination, the stabilization and restoration of the area, and the ultimate disposal of displaced soils. Certain alternatives may have to be considered, including those of changing land use or long-term restrictions. In all cases, the total costs of remedial actions should be evaluated to the maximum extent possible in the selection of alternatives.

TABLE 1 RECONTAMINATION COSTS

RECONTAMINATION TECHNIQUE	STABILIZATION ONLY				REMOVAL WITH GRIOTE DISPOSAL				REMOVAL WITH OFFSITE DISPOSAL AT A LICENSED BURIAL GROUND	
	1. FILLING	2. CHEMICAL VEGETATIVE	3A. SOIL COVER - 4"	3B. SOIL COVER - 12"	4A. SCRAPING INTO BARRIERS	4B. SCRAPING INTO BARRIERS	4C. SCRAPING INTO TRENCHES	4D. VACUATING WITH THICK DISPOSAL	5A. SCRAPING	5B. VACUATING
A. RECONTAMINATION SURVEILLANCE	RANGE \$200 - \$1000/A				RANGE \$200 - \$1000/A				RANGE \$200 - \$1000/A	
B. STABILIZATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
C. REMOVAL	NO SOIL REMOVAL COSTS				NO SOIL REMOVAL COSTS				NO SOIL REMOVAL COSTS	
D. PACKAGING	NO PACKAGING COSTS INVOLVED				NO PACKAGING COSTS INVOLVED				NO PACKAGING COSTS INVOLVED	
E. TRANSPORTATION	NO TRANSPORTATION COSTS INVOLVED				COSTS FOR FIRST 100 FT. OF GRIOTE HAUL INCLUDED IN REMOVAL COSTS. EACH ADDITIONAL 100 FT. OF HAUL -				COSTS FOR FIRST 100 FT. OF GRIOTE HAUL INCLUDED IN REMOVAL COSTS. EACH ADDITIONAL 100 FT. OF HAUL -	
ULTIMATE DISPOSAL	COSTS AND REMEDIATION MAY OR MAY NOT BE DEPENDENT UPON THE TYPE OF CONTAMINATION				COSTS AND REMEDIATION MAY OR MAY NOT BE DEPENDENT UPON THE TYPE OF CONTAMINATION				COSTS AND REMEDIATION MAY OR MAY NOT BE DEPENDENT UPON THE TYPE OF CONTAMINATION	
F. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
G. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
H. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
I. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
J. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
K. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
L. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
M. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
N. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
O. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
P. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
Q. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
R. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
S. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
T. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
U. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
V. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
W. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
X. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
Y. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	
Z. RECONTAMINATION	COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT				COSTS ARE ESTIMATED FOR A 3 MONTH PROJECT	