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Introduction to Section II and Overview of Dose Reconstruction: Lessons Learned from Studies in the U.S.

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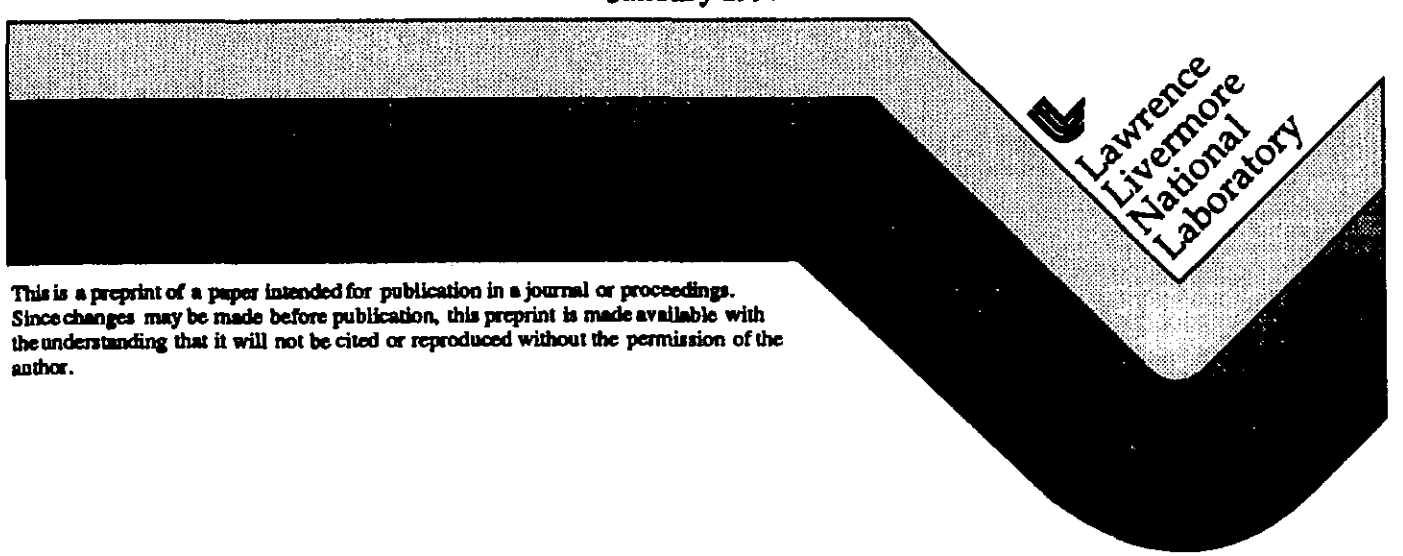

Introduction to Section II and Overview of Dose Reconstruction: Lessons Learned from Studies in the U.S.

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INTRODUCTION

Table 1 outlines our schedule for this part of the session. I am going to give an overview of dose reconstruction with some emphasis on the lessons that we have learned from work in the United States. Then, Dr. Marina Degteva will talk about dose reconstruction for the cohort of people living near the Techa River downstream from the Mayak Production Association, which was the first nuclear materials production facility in the former Soviet Union. We will then conclude with an open question/answer/discussion session on dose reconstruction.

As mentioned, the purpose of this presentation is to provide an overview of dose reconstruction with an emphasis on the lessons learned from work in the United States. Several major dose reconstructions have been undertaken in the United States, particularly in reference to Department of Energy (DOE) facilities. Some of these activities have now been completed and these are indicated in the upper part of Table 2. The first major activity took place at the Nevada Test Site (NTS), where researchers have considered several different specific populations. The activities began with an analysis of hypothetical individuals, which was followed by an analysis of the collective dose to all exposed individuals within the surrounding region. Later, the University of Utah undertook some specific epidemiologic studies and calculated doses to specific individuals. The Hanford Environmental Dose Reconstruction Study has completed its results for hypothetical individuals. The Hanford researchers did not report collective dose.

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The lower part of Table 2 indicates dose-reconstruction activities at DOE sites that are currently in progress. As part of an epidemiologic study at Hanford, thyroid doses will be calculated for specific individuals. In addition, a variety of different dose reconstructions are now underway at a number of sites: The Fernald Feed Materials Production Center, the Rocky Flats Plant, the Oak Ridge Site, the Idaho National Engineering Laboratory, and the Savannah River Site. It is important to consider whether or not it is worthwhile to continue to undertake so many dose-reconstruction activities in lieu of lessons learned from those studies already completed.

Table 3 indicates some of the reasons that dose-reconstruction studies might be undertaken. Certainly, the Nevada Test Site was a prime example where there was a known large release from which the possibility of biologic effects could be presumed. The Hanford dose reconstruction falls into the next category, where there was a stunning release of formerly classified information that indicated substantial releases, and there was also at least one deliberate release with public exposure. That situation led to the next category where, at Hanford and elsewhere, issues of social justice arose. Some members of the public felt very strongly that they had been wronged and, in some cases, harmed. In such cases, there are certainly reasons to undertake some kind of a dose-reconstruction study and, perhaps, an epidemiologic study.

Almost all of the studies indicated in the lower part of Table 2 were, in fact, undertaken for social justice reasons. Whether or not this is an adequate reason for conducting such studies, which typically cost a few tens of millions of dollars, is something that should be considered. Certainly, a significant fraction of the public thinks social justice concerns are a valid reason, and they believe that it is reasonable for the government to pay for a preferably independent study in return for the government having allowed a situation to develop where the public has come to question even the good will of its government. Scientists may disagree with the need or the usefulness of such studies, but the desire of the public for more information and reassurance that they are not at significant risk has perhaps been the compelling reason for the implementation of many dose-reconstruction studies in the U.S. Of course, there are other reasons for dose-reconstruction studies to be undertaken, including a desire to advance knowledge of radiation-risk factors, particularly for low dose radiation received at low-to-moderate dose rates, and some

researchers tremendously enjoy the challenge of dose reconstruction. Dose reconstruction is a very difficult activity and sometimes, perhaps, scientists seek to undertake projects because of the challenge and their own interest.

Table 4 indicates some of the methods of dose reconstruction. The methods are arranged hierarchically, with those producing the most credible results at the top. Certainly, if it were possible to examine and extract a sample from every individual of concern, and if such a sample could function as a dosimeter, a more credible dose assessment would result. The next most desirable method of dose reconstruction would be dosimetry based on materials that could be taken from people's homes. A good example of this would be extracting quartz grains from bricks, and then doing a thermoluminescent-dose measurement using the quartz as a dosimeter. This, in fact, has been done in association with dose-reconstruction work for the Japanese survivors of the atomic bombings (Maruyama et al. 1987) and for persons living downwind of the Nevada Test Site (Haskell et al. 1995). It can also be very useful to analyze environmental residues, as such residues may provide a reliable indication of the current or past presence of radioactive materials. Sometimes, if the process that resulted in the exposure occurred a long time ago and if there are no environmental residues, there may be no choice other than to try to reconstruct the releases from process information and to then infer doses using an atmospheric transport model followed by models of the movement of radionuclides through food chains to man and metabolism in man. Sometimes, releases can only be inferred from second-hand information. If only a collective dose is sought, calculations of atmospheric transport are not needed. The lowest order in the hierarchy indicated in Table 4 is simply an inference of releases coupled to the use of global dose factors, such as those published by the IAEA (1985), the UNSCEAR (1982), or the WHO (1983).

Table 5 indicates some of the individual biological dosimeters. It is possible to extract a blood sample and do an analysis of stable translocations within chromosomes of circulating lymphocytes (Lucas et al. 1995). This method has a sensitivity of about 100 mGy. Electron paramagnetic analysis of teeth is another more recently developed possibility (Haskell et al. 1995), and this method is currently receiving very wide interest in Russia and Ukraine. It has not been

used extensively in the United States. Whole-body counting for some materials that remain in the body for a long time, such as ^{90}Sr, is another useful method, as is the analysis of tissues at autopsy or exhumation. Such methods have found widespread application in Russia, and Dr. Degteva will discuss these methods further.

Some of the methods of dose reconstruction involving the analysis of environmental residues are indicated in Table 6; these types of analyses have been very useful in studies in the United States. In cases where there was a large-scale release, such as that at the Nevada Test Site, there are elements in the environment that can still be measured. These include radionuclides such as ^{90}Sr, ^{137}Cs, and $^{239+240}Pu$. A lot can also be learned about the source of plutonium and cesium by measuring the isotopic ratio: ^{240}Pu-to-^{239}Pu (Krey and Beck 1981; Beck and Anspaugh 1991). The Feed Materials Production Center at Fernald, Ohio, was mainly a uranium-processing center. As uranium is a very long-lived radionuclide, the collection and analysis of soil samples has been useful in the reconstruction of material released at that site (Stevenson and Hardy 1993). Iodine-129 is another long-lived radionuclide (half-life of 16,000,000 years) and is sometimes a good tracer for past deposits of its short-lived, but more damaging sibling, ^{131}I. The usefulness of current measurements of ^{129}I in inferring the doses to the thyroid from ^{131}I has been demonstrated for the Chernobyl accident (Straume et al. 1996). In addition, having access to both historical and current measurements of external gamma-exposure rate is very useful, as has been the case for both the Techa River study in Russia and the Nevada Test Site study in the U.S. There is a variety of other measurements that can be made even at times long after the major exposure has occurred, because residues are still in the environment. If the relative abundance of the source term is known, in fact, the deposition of material can be completely reconstructed based upon the measurement of even a single radionuclide (Anspaugh and Daniels 1996).

NEVADA TEST SITE DOSE RECONSTRUCTION

The off-site radiation-review (ORERP) project at the Nevada Test Site was a very large, multi-person, multi-agency, modular study. The names of the Principal Investigators and other key individuals, along with their organizations, are shown in Table 7. As the NTS study was the

first major dose-reconstruction study to be undertaken in the U.S., it is of interest to review why this initial study was started.† An underlying reason was the controversy in the mid seventies over reports that low doses of radiation might be responsible for reputed increases in the incidence of cancer among workers at the Hanford Works (Mancuso et al. 1977) and at the Portsmouth Naval Shipyard (Najarian and Colton 1978). Some more specific reasons are indicated in Table 8; one of the more compelling was that scientists at the Centers for Disease Control and Prevention found a case of leukemia among military personnel present at the NTS during the Smoky nuclear test in 1957, and this sparked the conduct of a larger epidemiologic study (Caldwell et al. 1980, 1983). Another important event was the publication of a study by Lyon et al. (1979) that was widely viewed as indicating that there had been a radiogenic increase in leukemia children exposed to fallout. These reports and other concerns led to Congressional Hearings (e.g., U.S. Congress 1979), demands for the release of all information, and even a Congressional Report with the provocative title of "The Forgotten Guinea Pigs" (U.S. Congress 1980), the cover of which is reproduced in Fig. 1. In addition thousands of claims were filed against the government and eventually two major law suits were tried. These actions renewed old concerns about the correctness of prior estimates of external doses (Dunning 1959) and drew attention to the fact that there had never been an evaluation of internal doses. This latter concern had surfaced before, particularly concerning the dose to the thyroids of infants from the ingestion of ^{131}I with food (Knapp 1963; Mays 1963; Reiss 1963). The U.S. government eventually responded with a commitment to conduct a study, although the initial commitment made on March 28, 1979, was only to collect and disseminate data on fallout and possible health effects (DAAG 1987). A second commitment followed a few months later and was to reconstruct, "...in so far as possible...," estimates of exposures and doses to the offsite people. The commitment to conduct the study resulted in the formation of the ORERP, under the management of the U.S. Department of Energy's Nevada Operations Office.

At the time of the commitment for this dose-reconstruction study, external doses had been estimated and published (e.g., Dunning 1959; historical results are also reviewed extensively by Anspaugh et al. 1990), but had not been extensively peer reviewed. It was not known at that time

† Further perspective is provided in Anspaugh (in press).

if it was possible to reconstruct internal doses. Eventually internal doses were reconstructed (Ng et al. 1990; Whicker et al. 1990, 1996; Kirchner et al. 1996), and the process was carried out in the public arena under the guidance of an academic and political advisory group (DAAG 1987). But it is of historical interest that the commitment was made to perform this study, even though at the time it was not certain that the project could actually be carried out. Several features about the project were unique: One was the determination to make the maximum use of the available measurements, rather than to rely on models; another was to calculate the best estimate of dose along with an expression of uncertainty, this led to the first major implementation of stochastic models for dose reconstruction; another first was to carry out the study under the full awareness of the public through a federal advisory group that included representatives from the governors of four affected states (Nevada, Utah, Arizona, and California); and finally, the study was conducted by a large number of investigators from national laboratories, universities, government agencies including the U.S. Environmental Protection Agency, and private companies. The latter forced the development of a modular approach.

The most important historical measurements that could be used for the dose reconstruction were those of external gamma-exposure rate; such measurements were made after each individual event at the NTS. Fig. 2 indicates the summation of these measurements and their extrapolation with time to provide an overall idea of the extent of the external gamma doses in the nearby communities. These historical estimates of external gamma exposure were confirmed by the later studies of the ORERP. After the ORERP had begun and achieved some initial successes, it became clear that the area of consideration in Fig. 2 was not sufficient to include the majority of people who had received exposures of concern. Thus, consideration was given to broadening the study to include the Phase II region indicated in Fig. 3. Possible expansion into the Phase III area (essentially the remainder of the U.S.) indicated in Fig. 3 was considered. Based upon the analysis of soil samples taken in the areas marked in Fig. 3, however, this expansion was considered by the Dose Assessment Advisory Group not to be necessary (DAAG 1987).

As there had not been comprehensive measurements of external gamma-exposure rate in the Phase II area, a new method of inferring dose was required. The method used was based

upon earlier studies conducted by the U.S. Department of Energy's Environmental Measurements Laboratory (Krey and Beck 1981) and depended upon a variety of historical data and on the collection of soil samples for the analysis of environmental residues. For the contemporary soil samples, the analysis consisted of measuring ^{137}Cs, $^{239+240}Pu$ and the ratio: ^{240}Pu-to-^{239}Pu. The problem was that most of the ^{137}Cs at these locations actually came from global weapons fallout, not from the Nevada Test Site itself. Consequently, a much more elaborate procedure was required to determine how much of it came from the Nevada Test Site. Cesium-137 itself is not of particular interest, but all of the short-lived radionuclides that came with it delivered most of the dose. However, if one knows how much of the currently present ^{137}Cs at a particular location came from a particular test, one can infer the presence of all of the short-lived radionuclides (Hicks 1990; Beck and Anspaugh 1991).

Table 9 presents a summary of the NTS-release characteristics. There were more than one hundred atmospheric tests. The cumulative yield was one megaton, which is not too much considering that one Soviet test, by itself, was 58 megatons. However, this one very large test was conducted at Novaya Zemlya, a very isolated location. The largest test performed by the United States was about 15 megatons, but this was carried out in the Pacific, not in Nevada. The ^{137}Cs released at the NTS was 6 PBq, or 6×10^{15} Bq. That is about ten percent of the amount that was released by the Chernobyl accident (UNSCEAR 1988). Six EBq, or 6×10^{18} Bq, of ^{131}I were released, which is about 20 times more than what was released from Chernobyl. Fortunately, most of the ^{131}I released from the NTS was injected high into the troposphere, where it was extensively diluted and had time to decay before much of it came to earth.

A summary of the local population exposure in Phases I and II is that the collective external dose was about 10^4 Gy, and the collective thyroid dose was about 10^5 Gy. Absorbed doses from the ingestion of fallout-contaminated food were also calculated for 21 organs in addition to the thyroid (Ng et al. 1990; Kirchner et al. 1996; Whicker et al. 1996). These results are summarized in Fig. 4, where are plotted the collective doses to the indicated organs. The highest collective dose is to the thyroid, and the internal dose to this organ is the only one for

which the dose is higher than is the dose from external exposure. Other organs of relatively high dose are those associated with the gastrointestinal tract and the bone (surface and marrow).

Health effects from the exposures of the general population have been studied by a number of groups, more notably in recent times by the University of Utah. In general, it has been extraordinarily difficult to actually detect any biological effects. There is not a detectable increase in leukemia in the general population; but a statistically significant increase has been reported for a defined subgroup of acute leukemias discovered from 1952–1963 among individuals younger than 20 at the time of exposure (Stevens et al. 1990). (This association is based upon five cases of leukemia.) The occurrence of thyroid cancer in children was also studied (Kerber et al. 1993). A statistically significant positive dose-response trend was reported for neoplasms, but not for thyroid nodules or for carcinomas. Thus, whatever health effects have occurred, they are not easily detected by means of epidemiologic study. One significant divergence of the experiences at the NTS and Chernobyl is the high rate of childhood-thyroid cancer that has been observed following the Chernobyl accident (Likhtarev et al. 1994). Based upon published data, it has been estimated that the collective thyroid dose from Chernobyl is 10^6 Gy (Likhtarev et al. 1996); this is about ten times higher than the collective dose to the thyroid from the NTS, even though the release from the NTS was considerably higher. The higher dose from the Chernobyl release is due to the higher population density and the injection of the ^{131}I at a lower altitude.

THE HANFORD WORKS AND OTHER DOSE-RECONSTRUCTION STUDIES

The major effort of the Hanford Environmental Dose Reconstruction (HEDR) has been completed (Farris et al. 1994a,b). The major release of radionuclides was to the atmosphere during the early days of the operation. The monthly releases of ^{131}I are shown graphically in Fig. 5. The total release of ^{131}I is estimated to have been 30 PBq (Heeb 1994), which is about 200 times less than the release from the NTS. As the location at Hanford was also relatively isolated during the mid 1940s, it seems unlikely that any biologic effect might be found here in view of the difficulty in finding any effect as a result of the releases from the NTS. However, a thyroid-health-effects study is underway (Davis et al. 1995), and the results are yet to be reported.

The Hanford Works also released significant quantities of material to the Columbia River, and Fig. 6 shows the releases by year. Most of the radionuclides of concern are short-lived and resulted from neutron activation of materials dissolved in the cooling water, which was once-through water from the River. In general doses from this pathway are substantially less than from the atmospheric pathway (Farris et al. 1994b).

Dose-reconstruction studies at other U.S. sites are still underway, and results are not yet available. However, in comparison to the releases from the NTS, it seems unlikely that any of the other sites indicated in Table 2 would likely have been large enough to have produced epidemiologically detectable health effects.

The results of the U.S. studies might be summarized as follows:

- The dose from virtually any release can be reconstructed (given enough money and time) and used as part of an epidemiologic study,
- The calculated doses may be very uncertain,
- It has been very difficult to detect any biological effects from the large releases from the NTS,
- It is not likely that health effects will be seen from the ^{131}I releases at the Hanford Works, based on a comparison to the NTS results, and
- The scientific value of future studies at U.S. sites is questionable, although the studies will likely continue in order to satisfy social justice.

CONCLUSIONS

The dose-reconstruction efforts for the NTS and the Hanford Works have been completed, and the results are generally well accepted by the scientific community and the public. Any resulting health effects from these exposures have been very difficult to detect by epidemiologic study of the exposed population at the NTS; results for the Hanford thyroid study

are not yet available. In general it seems likely that further dose-reconstruction and epidemiologic studies at U.S. sites will not contribute to scientific knowledge concerning the dose-effect relationship of cancer induction by radiation exposure.

If the problem of dose-effect is to be addressed, particularly as it pertains to doses delivered at low-to-moderate dose rates, meaningful results are much more likely to follow from studies conducted in Russia at sites related to the production of nuclear materials and from studies in Belarus, Ukraine, and Russia related to the Chernobyl accident.

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Whicker, F.W., Kirchner, T.B., Anspaugh, L.R., and Ng, Y.C. Ingestion of Nevada Test Site Fallout: Internal Dose Estimates. *Health Phys.* **71:** 477–486 (1996).

WHO. *Environmental Health Criteria 25. Selected Radionuclides: Tritium, Carbon-14, Krypton-85, Strontium-90, Iodine, Caesium-137, Radon, Plutonium.* World Health Organization, Geneva, 1983.

Table 1. Schedule for Section II on Dose Reconstruction

Presenter	Time	Topic
Lynn Anspaugh	20 min.	Introduction and Overview of Dose Reconstruction: Lessons Learned from Studies in the United States
Marina Degteva	25 min.	Reconstruction of Radiation Doses in Populations near the Techa River
All	15 min.	Open Questions/Discussion on Dose Reconstruction

Table 2 Major dose-reconstruction studies at DOE facilities in the United States

Site	Object of assessment	Reference
	Completed	
Nevada Test Site	Hypothetical individuals	Henderson and Smale (1990)
		Ng et al. (1990)
		Bouville et al. (1990)
	Collective	Henderson and Smale (1992)
		Whicker et al. (1996)
	Specific individuals	Simon et al. (1995)
		Till et al. (1995)
Hanford Works	Hypothetical individuals	Farris et al. (1994a)
		Farris et al. (1994b)
	In process	
Hanford Works	Specific individuals	Davis et al. (1995)
Fernald Feed Materials Production Center		Miller et al. (1994)
Rocky Flats Plant		Miller et al. (1994)
Oak Ridge Site		Miller et al. (1994)
Idaho National Engineering Laboratory		Miller et al. (1994)
Savannah River Site		Miller et al. (1994)

Table 3. Reasons why dose-reconstruction studies might be undertaken

- Known large release that can be presumed to have a biologic effect
- Stunning revelation of formerly classified data
    - Operational releases
    - Deliberate releases with public exposure
- Social justice
    - The public believes it has been harmed.
    - The public believes it has been wronged.
- To advance knowledge of risk factors
- "Because it's there."

Table 4. Methods of dose reconstruction

- Individual biologic analysis
  - Chromosome-translocation analysis of circulating lymphocytes
  - Electron paramagnetic analysis of teeth
- Dosimetry of materials in homes
- Analysis of environmental residues
  - Deposition densities, past or current
  - External gamma-exposure rates (past)
- Known releases, plus atmospheric models
- Inferred releases, plus atmospheric models
- Known or inferred releases

Table 5. Methods of performing dosimetry on individuals themselves

• Chromosome-translocation analysis of circulating lymphocytes
• Electron paramagnetic analysis of teeth
• Whole body counting for long-lived radionuclides
• Analysis of material excreted in urine or feces
• Analysis of tissues collected at autopsy or exhumation

Table 6. Methods of dose reconstruction using the analysis of environmental residues

- Deposition densities, historical or current data
  - Short-lived radionuclides (historical data only)
  - ^{90}Sr
  - ^{129}I
  - ^{137}Cs
  - Uranium
  - $^{239+240}Pu$
  - $^{239+240}Pu$, plus the ratio of ^{240}Pu-to-^{239}Pu
- Historical measurements of external gamma-exposure rate

Table 7. Key individuals and organizations who conducted the Nevada Test Site (NTS) Off-Site Radiation-Review Project (ORERP)

Names	Organization
L. Anspaugh, Y. Ng,[a] H. Hicks[a]	Lawrence Livermore National Laboratory
B. Church, R. Nutley, D. Wheeler, M. Page[a]	U.S. Department of Energy, Nevada Operations Office
W. Whicker, T. Kirchner, M. Otis D. Breshears	Colorado State University
R. Henderson, R. Smale	Los Alamos National Laboratory
H. Beck	U.S. Department of Energy, Environmental Measurements Laboratory
C. Thompson, F. Miller, R. McArthur	University of Nevada, Desert Research Institute
G. Quinn, C. Steadman	U.S. National Oceanic and Atmospheric Administration
F. Grossman	U.S. Environmental Protection Agency
B. Maza, T. Mehas, M. Demarre	Reynolds Electrical and Engineering Co.

[a]Deceased

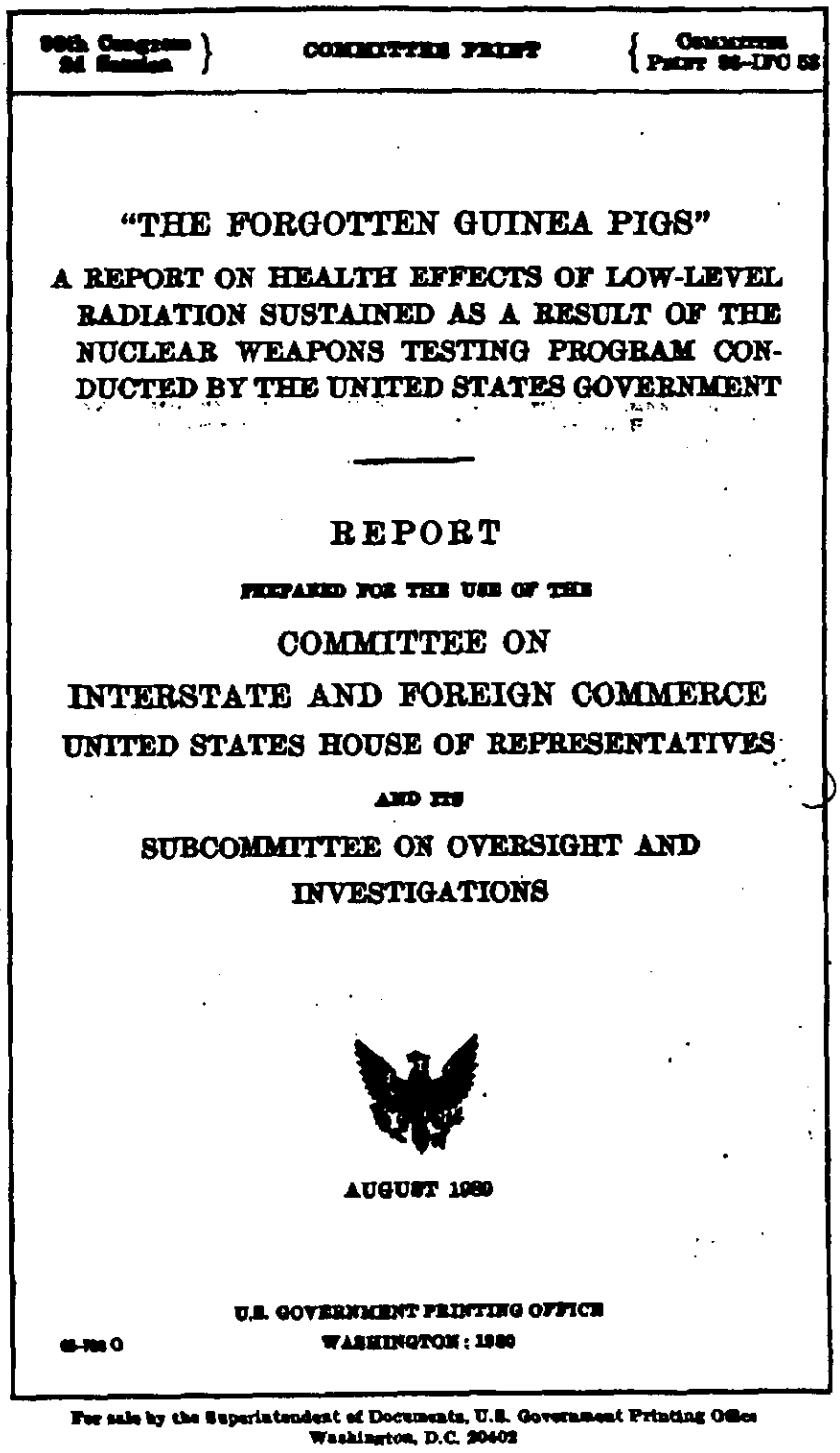
Table 8. Several specific issues and events clarified the need for a dose-reconstruction study for the persons living downwind of the Nevada Test Site during the 1950s and 1960s.

- Concern about the implications of a reported increase in the incidence of leukemia in atomic veterans, which was first reported in 1976 by the Centers for Disease Control and Prevention (Caldwell et al. 1980)
- Several hundred personal injury claims from residents in Nevada, Utah, and Arizona
- Congressional Hearings (1979–1980) (e.g., U.S. Congress 1979, 1980)
    - "Atomic Veterans"
    - Forgotten Guinea Pigs"
- The Lyon et al. (1979) epidemiologic study implied an increase in leukemia in "high fallout" locations.
- Concern about the correctness of past analyses of external doses and rekindling of concern about the unquantified doses from internal exposure (from inhalation and ingestion)
- Two legal cases
    - Reopening of Bullock et al. vs. U.S. (sheep deaths)
    - Allen et al. vs. U.S. (human cancer)

Table 9. Characteristics of the releases from the Nevada Test Site

Characteristic	Value
Number of atmospheric tests	>100
Approximate total yield	1×10^6 tonnes
Approximate energy release[a]	1×10^{15} cal
Fission-product atoms created[a]	3×10^{26}
Cesium-137 released[a]	6×10^{15} Bq
Iodine-131 released[a]	6×10^{18} Bq
Collective external dose[b]	1×10^4 Gy
Collective thyroid dose[c]	1×10^5 Gy

[a]These values follow from the 1 Mt total yield and conversions provided in Glasstone and Dolan (1977).
[b]From Henderson and Smale (1992)
[c]From Whicker et al. (1996)



96th Congress 2d Session } COMMITTEE PRINT { Committee Print 96-IFC 88

"THE FORGOTTEN GUINEA PIGS"

A REPORT ON HEALTH EFFECTS OF LOW-LEVEL RADIATION SUSTAINED AS A RESULT OF THE NUCLEAR WEAPONS TESTING PROGRAM CONDUCTED BY THE UNITED STATES GOVERNMENT

REPORT

PREPARED FOR THE USE OF THE

COMMITTEE ON

INTERSTATE AND FOREIGN COMMERCE

UNITED STATES HOUSE OF REPRESENTATIVES

AND ITS

SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

AUGUST 1980

U.S. GOVERNMENT PRINTING OFFICE

63-788 O WASHINGTON : 1980

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402

Fig. 1. Cover of the U.S. Congressional (1980) report on health effects from exposure to radiation from the Nevada Test Site.

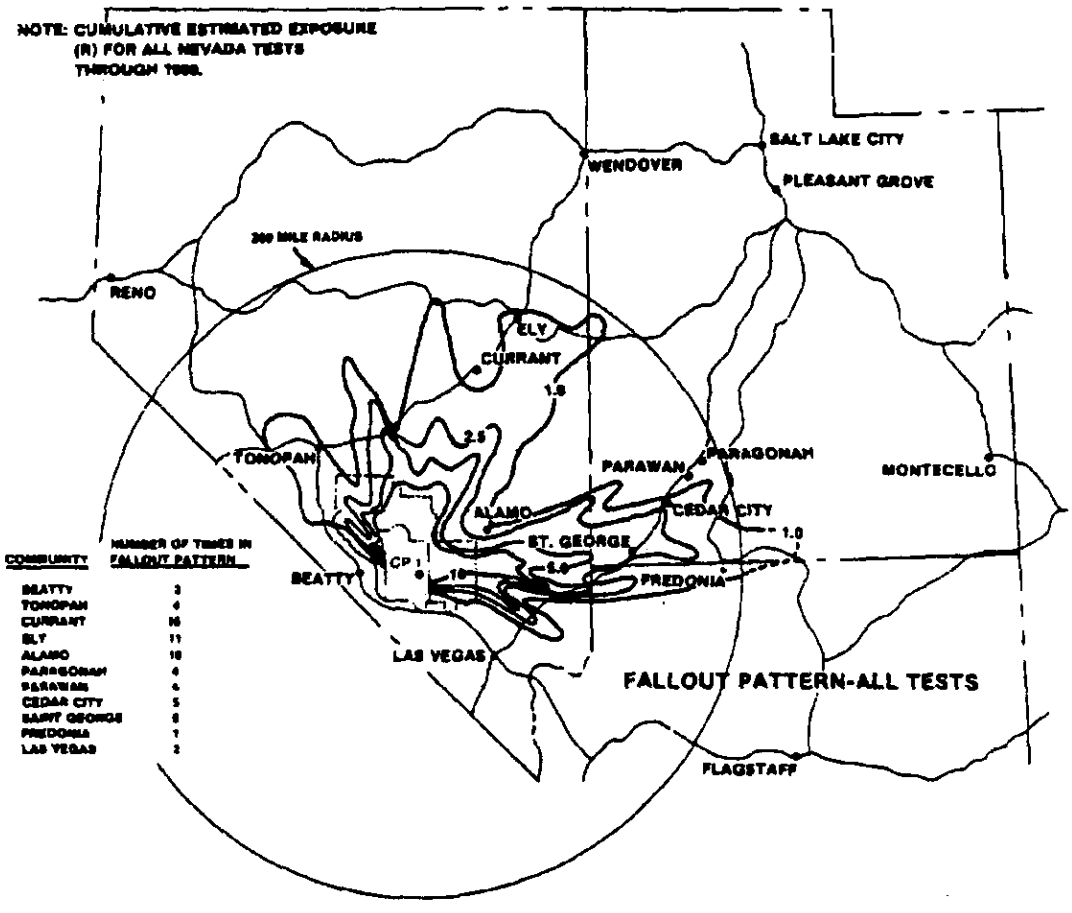


Fig. 2. Isopleths of estimated individual external gamma exposure from the nuclear tests conducted at the Nevada Test Site (from Friesen 1985).

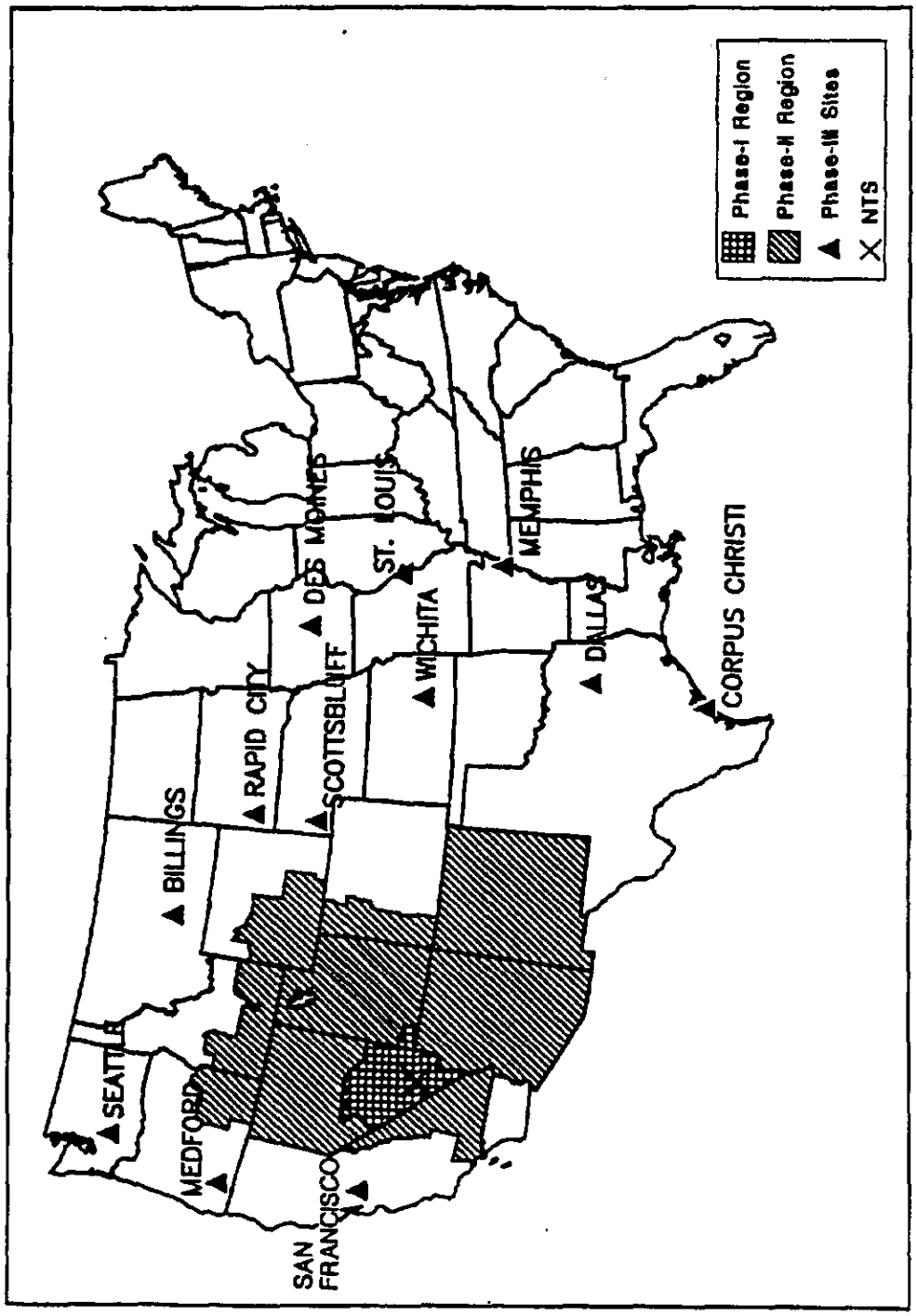


Fig. 3. Study regions for the dose-reconstruction study for the Nevada Test Site (from Beck and Anspaugh 1991).

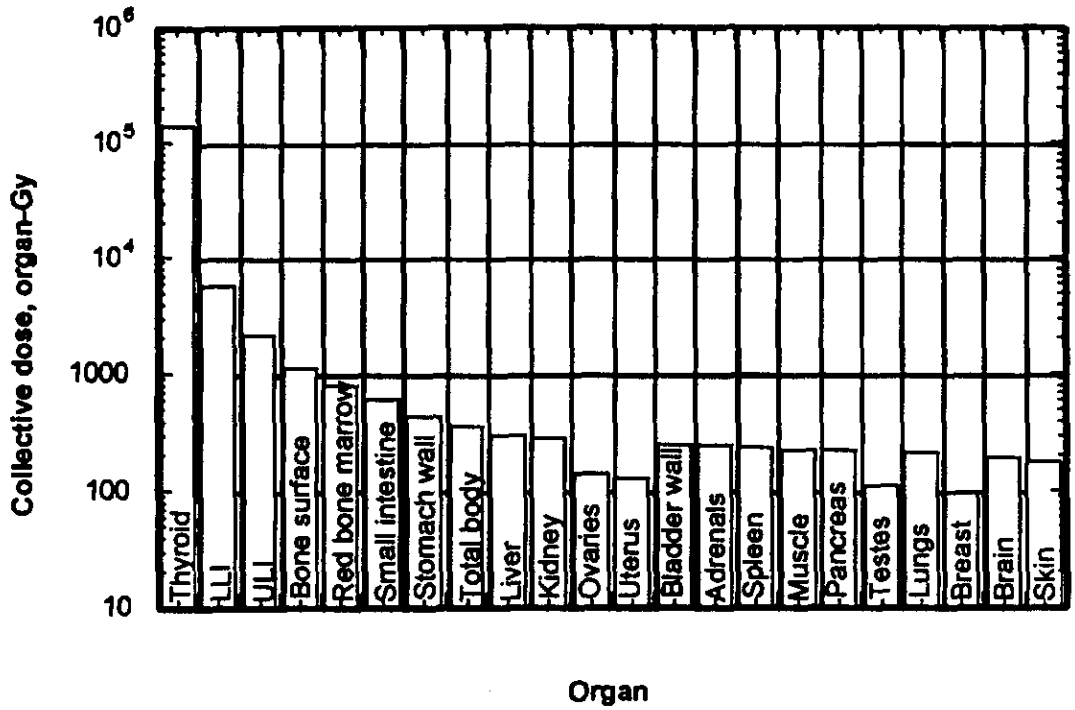


Fig. 4. Collective dose by organ from the ingestion of contaminated food (based on data from Whicker et al. 1996).

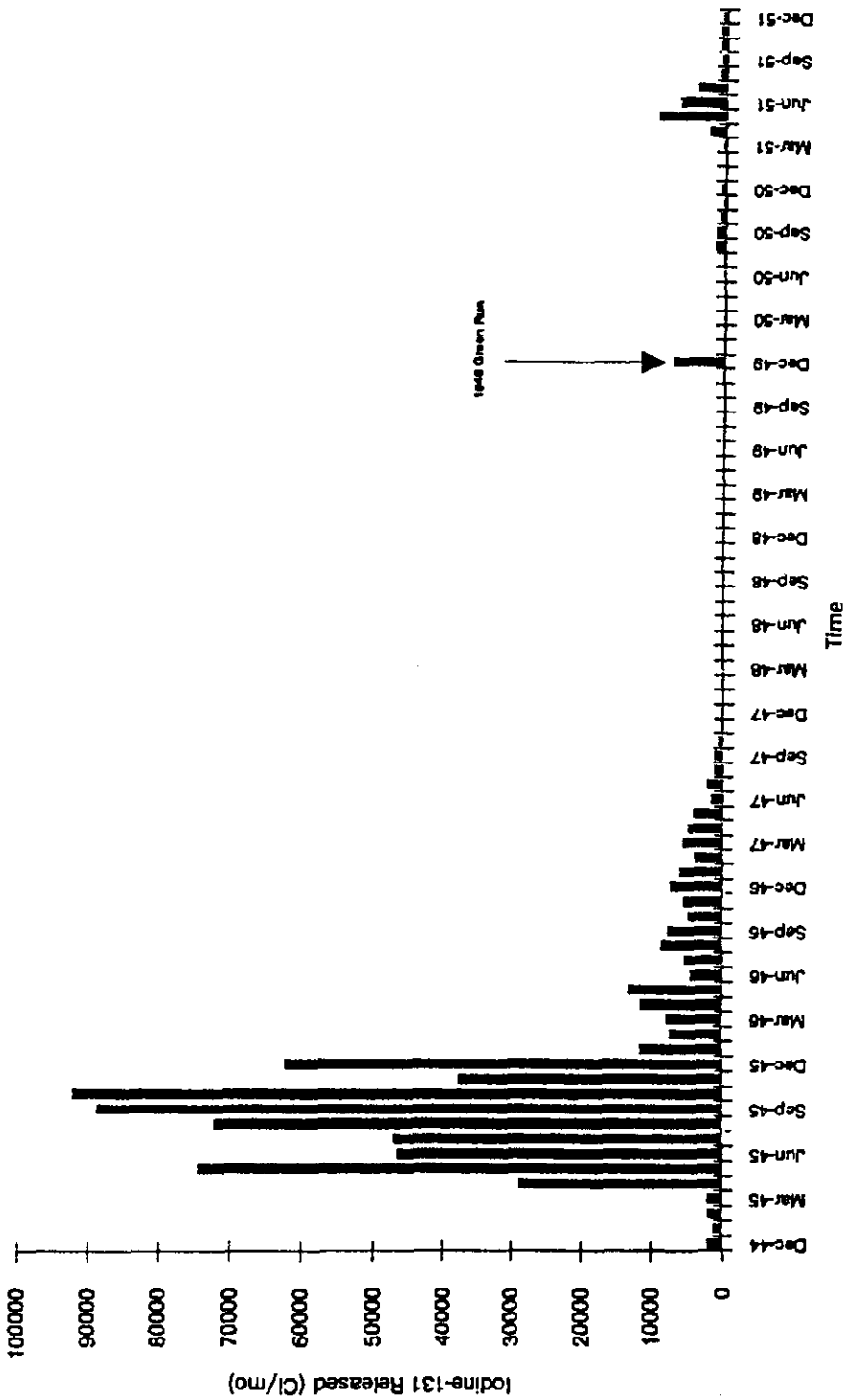


Fig. 5. Monthly releases of ^{131}I to the atmosphere from the Hanford Works (from Farris et al. 1994a).

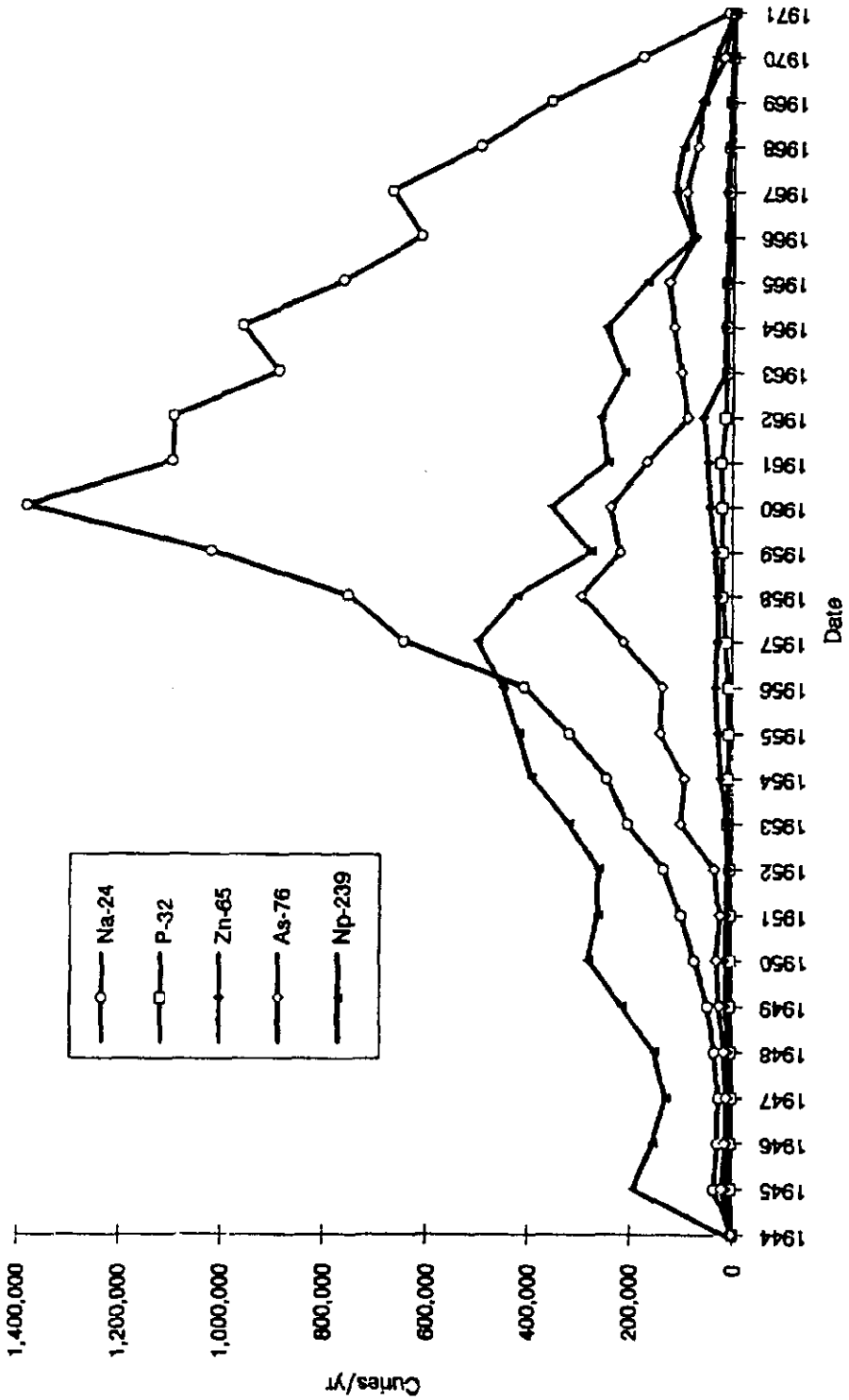


Fig. 6. Yearly releases of radionuclides to the Columbia River from the Hanford Works (from Farris et al. 1994b).

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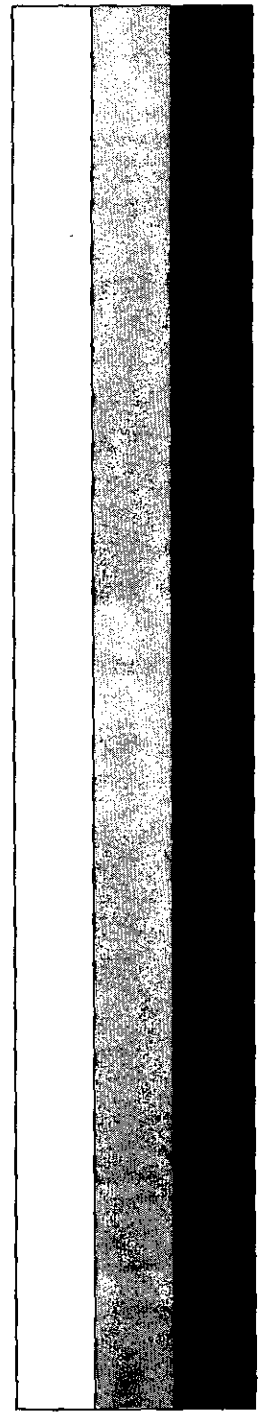
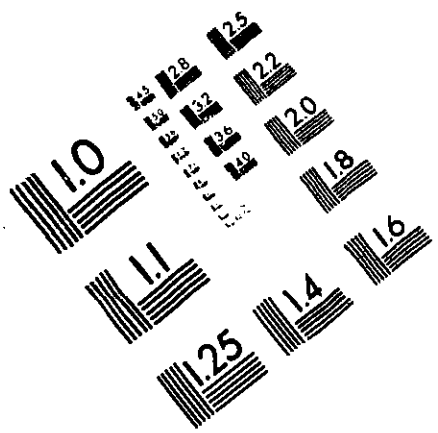


Attachment 20:

Boice, John D., et. al. "A Comprehensive Dose Reconstruction Methodology for Former Rocketdyne/Atomics International Radiation Workers." Health Physics. Vol. 90, No.5, May 2006. 409-430.

Attachment 21:

Layton, David, et. al. Risk Assessment of Soil-Based Exposures to Plutonium at Experimental Sites Located on the Nevada Test Site and Adjoining Areas. California: Lawrence Livermore National Laboratory. UCRL-ID-113605. June 1993.



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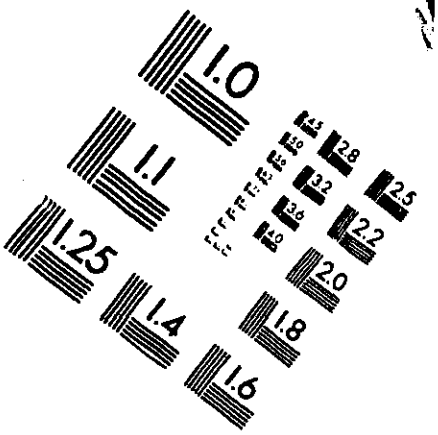

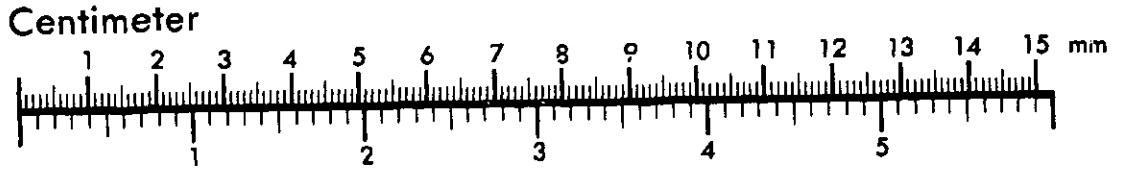

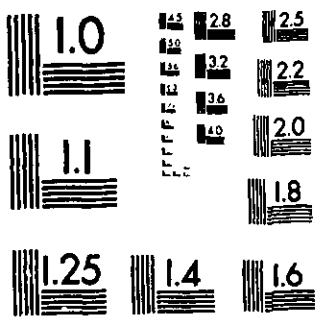

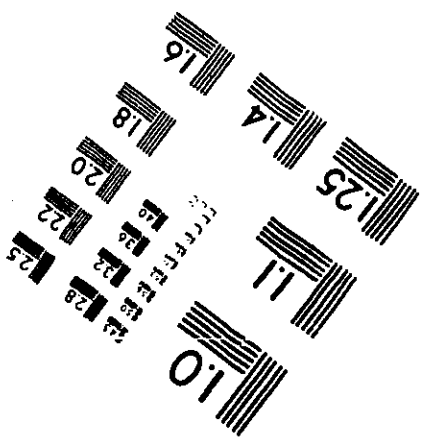

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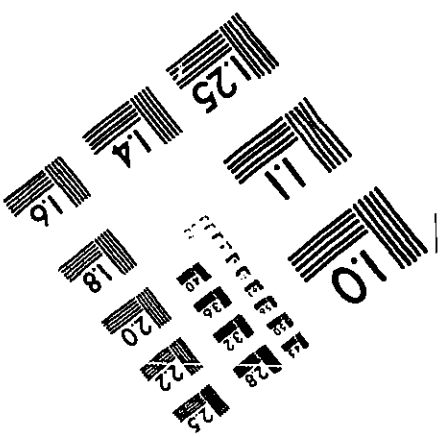

1 of 1

UCRL-ID-112605

Risk Assessment of Soil-Based Exposures to Plutonium at Experimental Sites Located on the Nevada Test Site and Adjoining Areas

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June 1993

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Abstract

In the late 1950s and early 1960s, a series of tests was conducted at or near the Nevada Test Site to study issues involving plutonium-bearing devices. These tests resulted in the dispersal of about 5 TBq of $^{239,240}Pu$ on the surficial soils at the test locations. Access to the sites is strictly controlled; therefore, it does not constitute a threat to human health at the present time. However, because the residual ^{239}Pu decays slowly (half-life of 24,110 y), the sites could indeed represent a long-term hazard if they are not remediated and if institutional controls are lost. To investigate the magnitude of the potential health risks for this no-remediation case, we defined three basic exposure scenarios that could bring individuals in contact with $^{239,240}Pu$ at the sites: (1) a resident living in a subdivision located at a test site, (2) a resident farmer, and (3) a worker at a commercial facility. Our screening analyses indicated that doses to organs are dominated by the internal deposition of Pu via the inhalation pathway, and thus our risk assessment focused on those factors that affect inhalation exposures and associated doses, including inhalation rates, activity patterns, tenure at a residence or occupation, indoor/outdoor air relationships, and resuspension outdoors. Cancer risks were calculated as a function of lifetime cumulative doses to the key target organs (*i.e.*, bone surface, liver, and lungs) and risk factors for those organs. Uncertainties in the predicted cancer risks were analyzed using Monte-Carlo simulations of the probability distributions used to represent assessment parameters. The predicted cancer risks for the resident farmer were more than a factor of three times higher than the suburban resident at the median risk level, and about a factor of ten greater than the reference worker at a commercial facility. At 100 y from the present, the 5, 50, and 95th percentile risks for the resident farmer at the most contaminated site were 4×10^{-6}, 6×10^{-5}, and 5×10^{-4}, respectively. The principal sources of uncertainty in the estimated risks were population mobility, the relationship between indoor and outdoor contaminant levels, and the dose and risk factors for bone, liver, and lung.

Contents

Introduction

Residual plutonium (Pu) in surficial soils at the Nevada Test Site (NTS) is the result of the above-ground testing of nuclear weapons and special experiments involving the detonation of plutonium–bearing devices. The latter experiments were conducted for four basic reasons (Stannard, 1988): (1) to study the behavior of Pu as it was being explosively compressed, (2) to ensure that the accidental detonation of the chemical explosive in a production weapon would not produce a nuclear yield, (3) to evaluate the ability of personnel to handle large-scale Pu dispersal accidents, and (4) to develop criteria for transportation and storage of weapons. The first set of experiments were called "hydrodynamic" or "equation of state" tests. Twenty-two such tests were conducted above ground at the GMX location in Area 5 (see Fig. 1) between December 1954 and February 1956. Many tests were conducted to determine whether nuclear yields could be produced from accidental detonations. The largest ones carried out on the surface were Project 56 in Area 11 (now known as Plutonium Valley); here four devices were detonated in 1956, one with a slight nuclear yield. The final large, surface experiment was conducted as Project 57 in Area 13, just off the NTS. Experiments performed to evaluate cleanup and weapons–handling issues consisted of Operation Roller Coaster, Double Tracks, and Clean Slate I, II, and III. The locations of these detonations off of the NTS are also indicated in Figure 1.

All of the sites where these tests were conducted were contaminated with Pu. At the present time, these sites do not pose a health threat to either workers or the general public because they are under active institutional control. Specifically, inadvertent human intrusion onto the test sites (referred to here as "shot sites") is deterred by the use of fences and warnings, coupled with the fact that the shot sites are within test ranges, all of which have restricted access. However, because the half-lives of ^{239}Pu and ^{240}Pu are 24,110 and 6,560 y, respectively, the residual contamination could indeed pose a long–term health hazard if it were not remediated and industrial, agricultural, or residential land uses emerged following a loss of institutional control. In order to develop relevant cleanup-options for the test sites, it is important to assess the nature and magnitude of the potential health risks associated with naïve uses of the affected lands. Accordingly, the following risk analysis examines the distribution of plutonium in soils at these sites, potential exposures and resulting doses, and finally, incremental cancer risks for those potentially exposed.

1. Methodology Overview

The basic methodology for estimating the potential health risks of residual $^{239,240}Pu$ in the soils of the NTS is shown in Figure 2. The initial task involves the characterization of the concentrations of $^{239,240}Pu$ in surface soils in the vicinity of each test shot. That effort is followed by a conceptualization of how an individual could come in contact with the residual plutonium under various land-use scenarios following loss of institutional control of unremediated lands. Knowledge of route-specific toxicity is also needed to identify potentially important exposure pathways, and hence we have shown a feedback loop between the dose-response assessment and the conceptualization of potentially important exposure pathways.

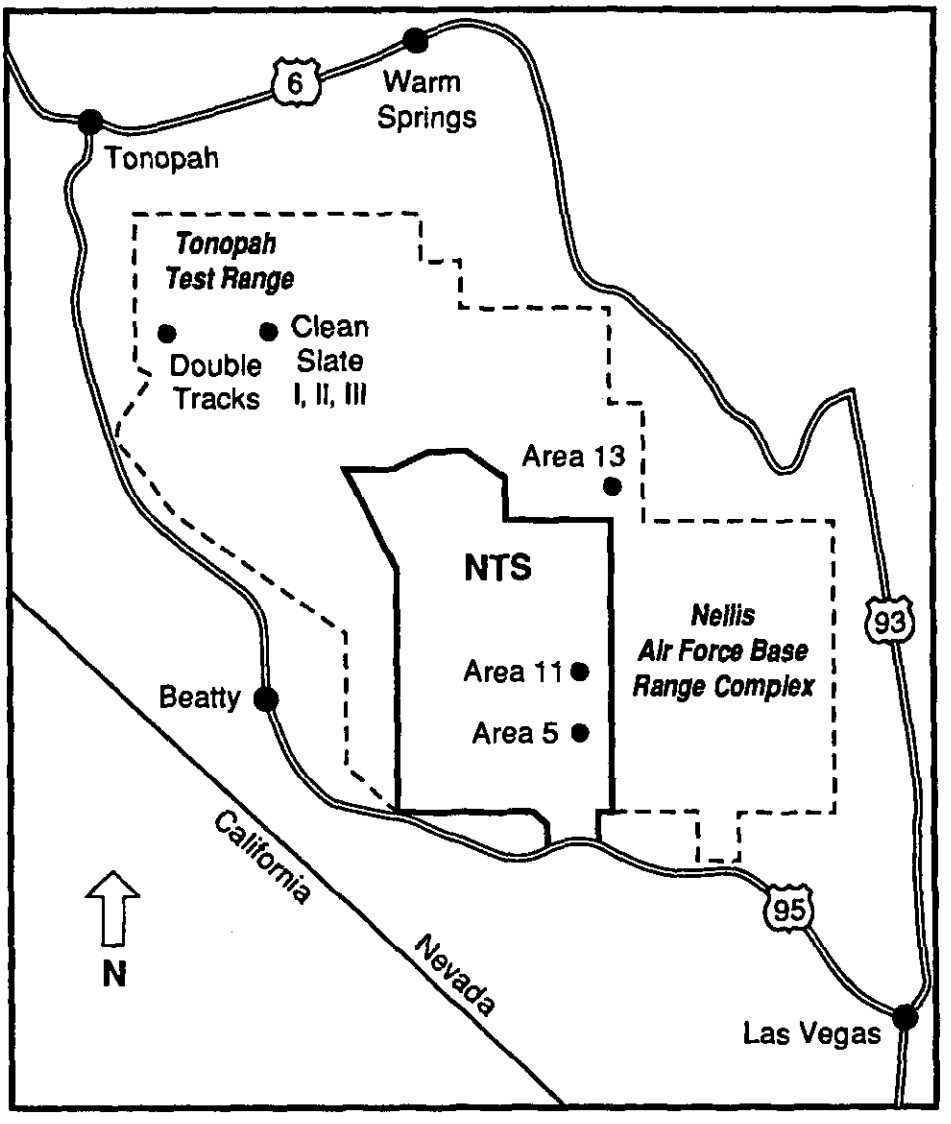

Figure 1. Locations of experiments at the Nevada Test Site and adjoining areas where plutonium-bearing devices were detonated.

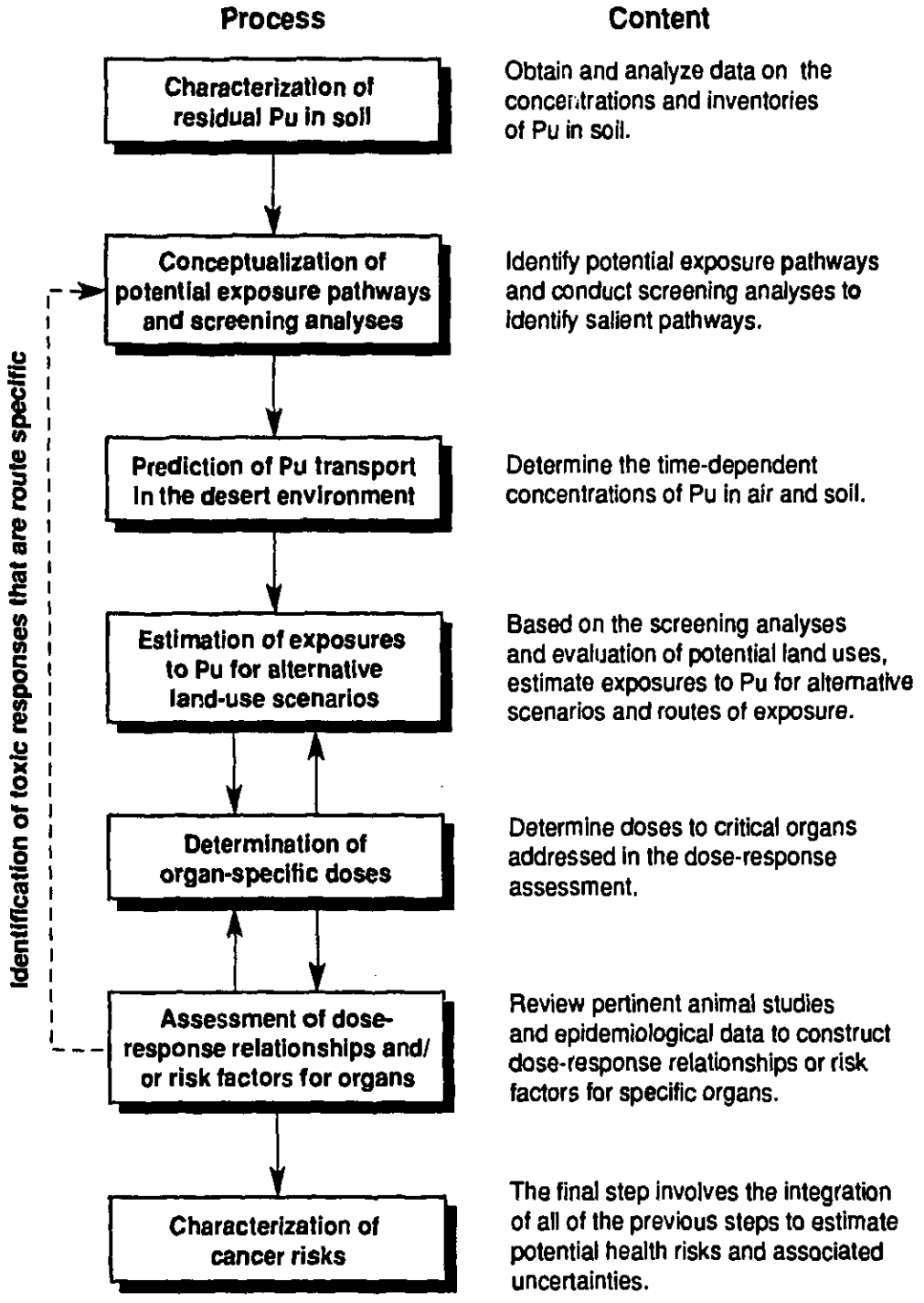

Figure 2. Methodology used to assess the risks associated with residual $^{239,240}Pu$ in soils at the various shot sites.

Before preparing a detailed dose assessment of an environmental contaminant, it is often helpful to prepare a screening-level analysis to identify the exposure pathways that contribute the most to absorbed doses, and conversely, to eliminate from further consideration those pathways that contribute little to absorbed doses. Information from the first two analyses is then used to examine the transport pathways of concern and afterwards, to calculate the doses to key organs.

To relate the internal doses to potential health risks, we next examine dose-response data for estimating the probability of incurring cancer per unit dose to target organs. We have included in Figure 2 additional feedback loops between the dose-response assessment, dose assessment, and exposure assessment tasks to indicate that there is a need to exchange information at each point in the assessment process. The final task in the methodology is to characterize the cancer risks resulting from the exposures to ^{239,240}Pu-contaminated soils/dusts at the different sites along with the uncertainties of the estimated risks. To quantify the overall uncertainty in calculated cancer risks, we use a Monte-Carlo simulation technique to propagate the uncertainties associated with the ^{239,240}Pu source terms (*i.e.*, concentrations in soil), plutonium transport, human exposures, absorbed doses, and dose–response relationships for target organs. Each of the following discussions evaluates separately the uncertainties of key assessment parameters and the concluding section on risk characterization presents the overall results.

2. Residual Plutonium in Soil at Shot Sites

The tests resulting in Pu dispersal were not always conducted in the same manner. A review (U.S. DOE, 1988) indicates that some device detonations occurred on concrete pads and in concrete bunkers. For example, Double Tracks and Clean Slate I were conducted by detonating one or more warheads on concrete pads, while in Clean Slate II and III warheads were exploded within bunkers. Post-shot cleanup operations also varied among the test series. For the Operation Roller Coaster tests, remediation efforts included the scraping of soils and other debris around the ground-zero (GZ) area to a single location where they were covered with clean soil. Decontamination efforts at the Plutonium Valley sites included the excavation of trenches in which contaminated soils and other test-related materials were buried. In contrast, there were no remediation efforts associated with the Project 57 (Area 13) test.

Systematic efforts to characterize the residual ^{239,240}Pu contamination of soils at the various shot sites were completed under the auspices of the Nevada Applied Ecology Group. The concentrations of ^{239,240}Pu in soils at the shot sites and estimates of the associated ^{239,240}Pu inventories are given in Gilbert (1977), but are based in part on earlier work given in Gilbert and Eberhart (1974) and Gilbert *et al.* (1975). The plutonium content of soils at each of the sites was characterized statistically using a stratified, random sampling approach. In this approach the area to be sampled was subdivided into separate strata or sampling areas in such a way that the variability of plutonium concentrations in soil samples taken from within a given stratum is smaller than the variability of the concentrations in samples taken across strata. Strata were defined for each of the shot sites using data obtained from FIDLER (Field Instrument for the Determination of Low-Energy Radiation) surveys. This particular field instrument uses a 5-in. NaI detector to measure 60-keV gamma emissions from the decay of ^{241}Am (432-y half-life), which is a decay product of ^{241}Pu (14.4-y half-life). Although most of the plutonium in nuclear weapons occurs as ^{239}Pu, ^{241}Pu is also present. As a result, ^{241}Am activities in contaminated

soils are expected to provide a reasonable indication of the levels of $^{239,240}Pu$ in soils. FIDLER surveys were conducted across uniform grids for all sites, except GMX where measurements were made on a radial grid. Isopleths of gamma activity (counts per minute of 60-keV gamma rays) based on the survey data were then used to designate strata for subsequent soil sampling to determine concentrations of $^{239,240}Pu$. Figures 3 through 6 present the isopleths of ^{241}Am activity and associated strata for the various areas. The sampling locations within the irregular boundaries of each stratum were determined using a stochastic procedure in which the horizontal and vertical distances on a grid over a site were selected randomly to define a sampling coordinate (*i.e.*, the intersection of the two random distances). The number of soil samples in the strata varied depending on the areal extent of the strata and the expected sample variability for a given stratum. However, at least 10 soil samples were taken in each stratum, regardless of its size. Soil samples were taken to a depth of 5 cm and later 10-gram aliquots of soil were homogenized by ball-milling prior to analysis. Other soil samples were taken to depths of 25 cm in order to evaluate the distribution of $^{239,240}Pu$ concentrations with depth. Gilbert *et al.* (1975) reported that between 68 and 97% of the total plutonium in soil to 25 cm was present in the top 5 cm. Thus, the inventories based on the 5-cm depth may slightly underestimate the total amount of $^{239,240}Pu$ present in the near-surface soils. In addition, the estimated inventories exclude an unknown amount of Pu present in the soils and other shot-related debris that were put in trenches or otherwise covered with clean soil during cleanup operations.

Table 1 summarizes the results of the analyses of the inventories of $^{239,240}Pu$ in soils at the shot sites. An estimated 5.1 TBq of $^{239,240}Pu$ is distributed in the upper 5-cm soils at the sites. The location with the highest inventory is Area 13 (33% of the total), however, the highest levels of contamination of surface soils occur at Area 11 (Plutonium Valley), where as much as 3070 Bq g^{-1} is present in one of the survey strata. The concentrations of $^{239,240}Pu$ in the soils of each of the sites were determined by dividing the estimated inventory for a site by the mass of contaminated soil (based on a density of 1.5 g/cm^3). Appendix A contains the strata–specific data on $^{239,240}Pu$ at each of the shot sites. In later work, scientists associated with the Radionuclide Inventory and Distribution Program (RIDP), which was designed to characterize the spatial distribution and inventory of selected radionuclides at the NTS, made measurements of ^{241}Am at the GMX and Plutonium Valley sites using *in-situ* gamma spectrometry (McArthur, 1991). Those measurements were converted to estimates of $^{239,240}Pu$ activity in soil and then estimates were made of the $^{239,240}Pu$ inventories at those two sites. The resulting RIDP estimates were 0.052 and 1.1 TBq (1.4 and 29 Ci) for GMX and Plutonium Valley, compared with the NAEG estimates of 0.056 and 1.3 TBq (1.5 and 36 Ci), indicating close agreement.

3. Conceptualization of Potential Exposure Pathways and Screening Analyses

From an exposure-assessment standpoint, one of the most important determinants affecting the nature and magnitude of future contacts with residual contaminants in soils is the type of land use that eventually occurs at a given location. We have identified four plausible land uses for the desert region where the test shots were located: (1) cattle grazing, (2) resident ranching, (3) residential housing, and (4) commercial development. Grazing is currently practiced

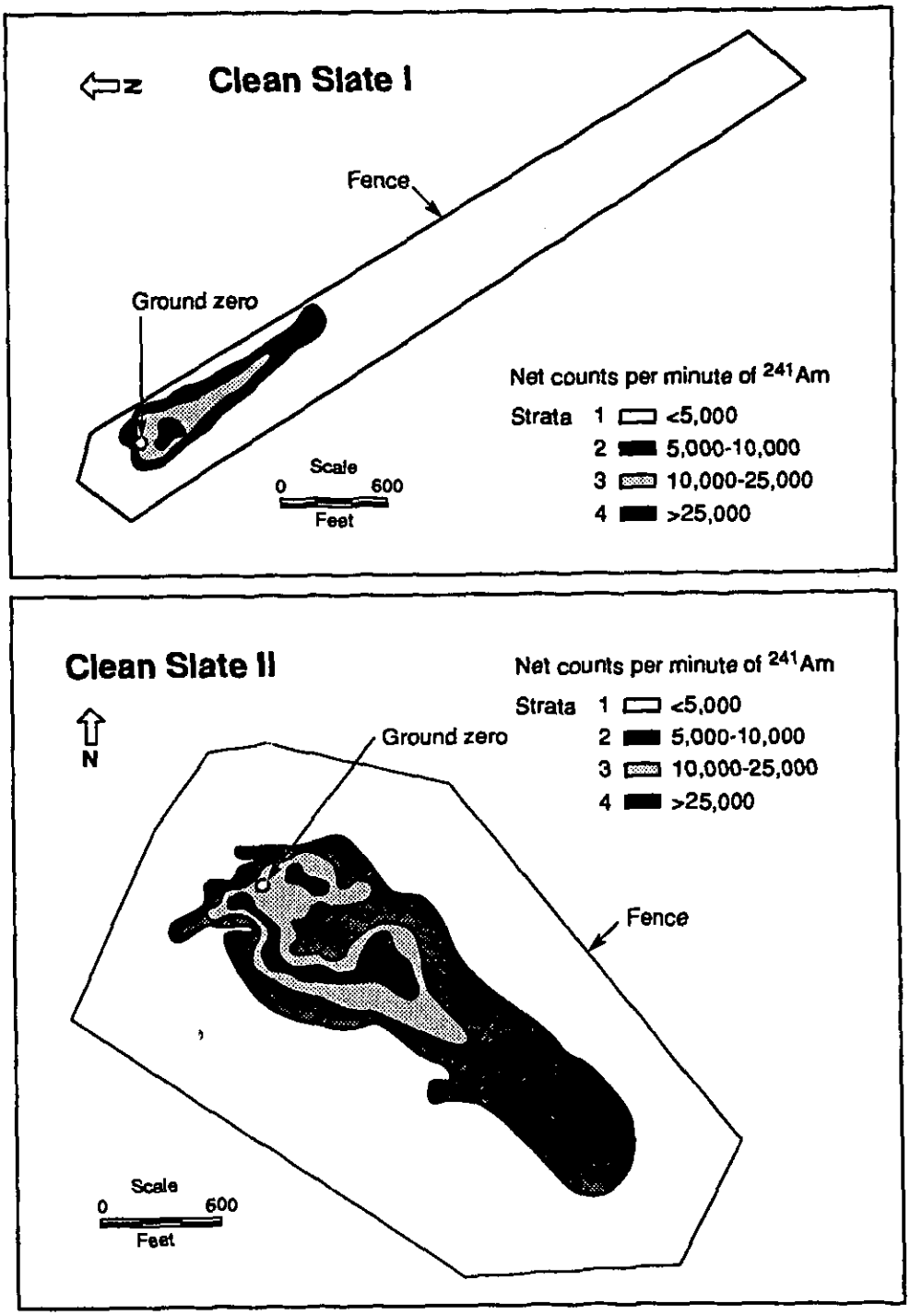

Figure 3. Areal extent of the contamination of ^{241}Am in soils at Clean Slate I and II. The measured ^{241}Am is used as a surrogate for $^{239,240}Pu$ (adapted from Gilbert *et al.*, 1975).

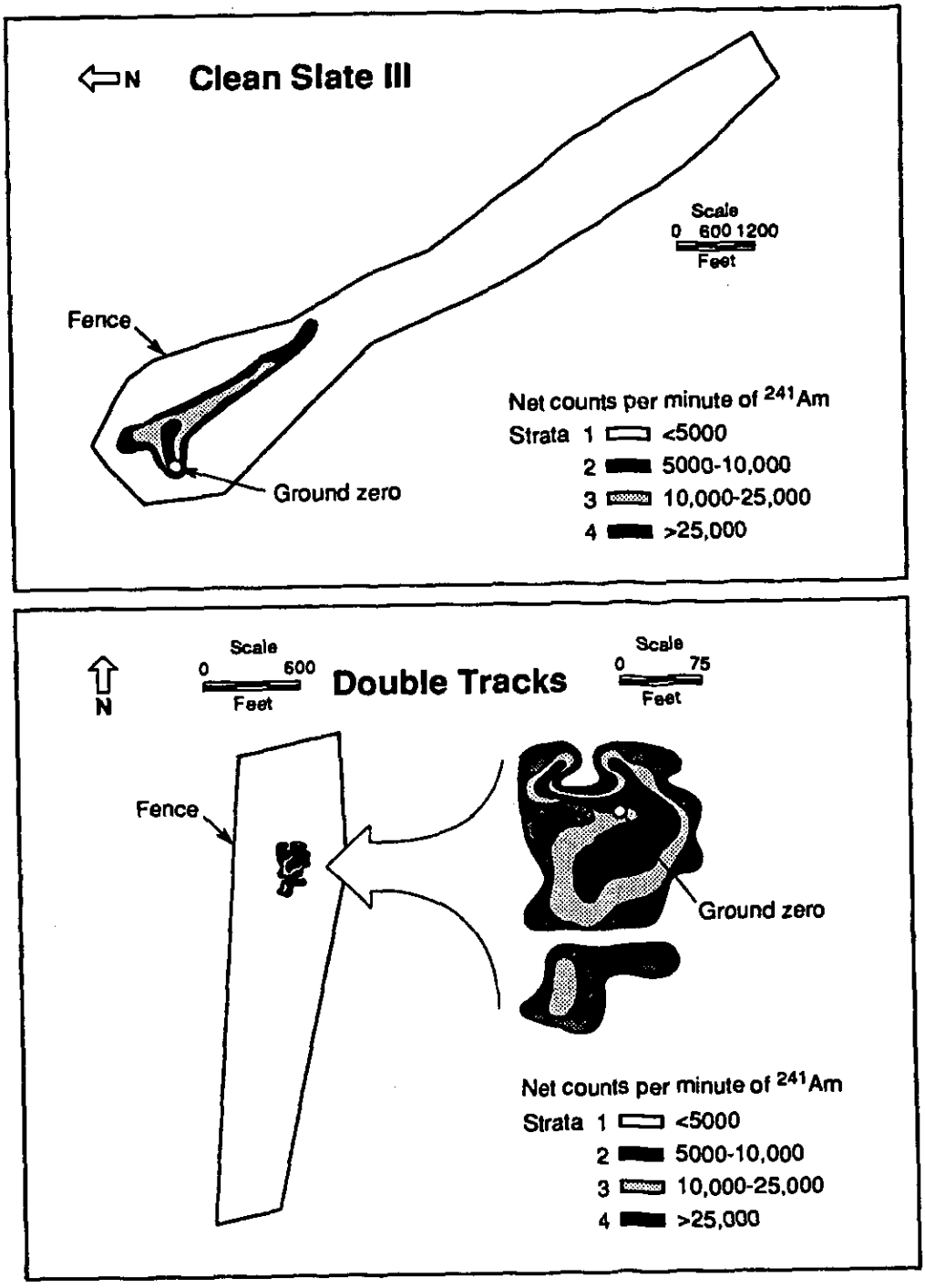

Figure 4. Areal extent of the contamination of ^{241}Am in soils at Clean Slate III and Double Tracks. The measured ^{241}Am is used as a surrogate for $^{239,240}Pu$ (adapted from Gilbert *et al.*, 1975).

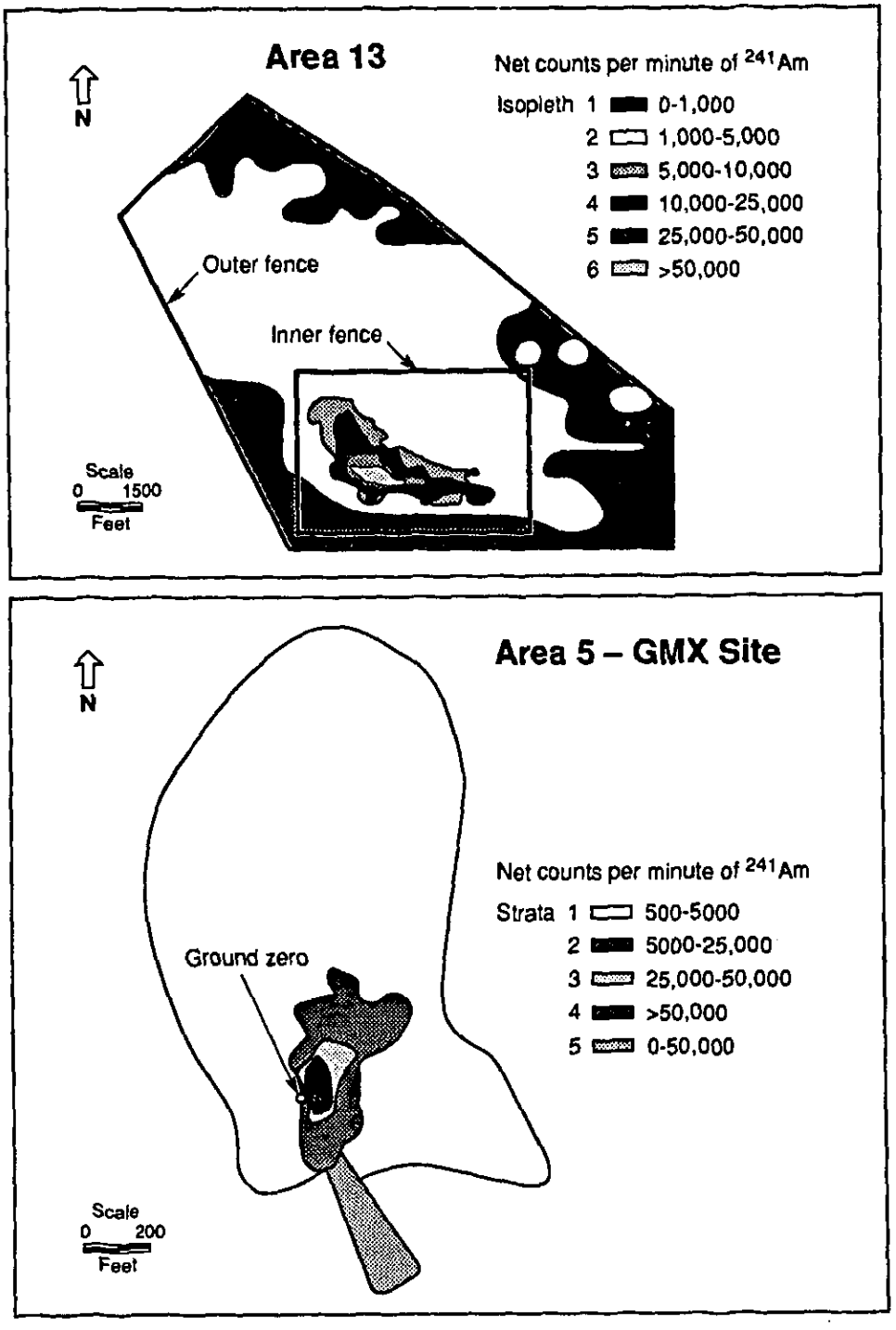

Figure 5. Areal extent of the contamination of ^{241}Am in soils at Area 13 and Area 5 (GMX). The measured ^{241}Am is used as a surrogate for $^{239,240}Pu$ (adapted from Gilbert *et al.*, 1975).

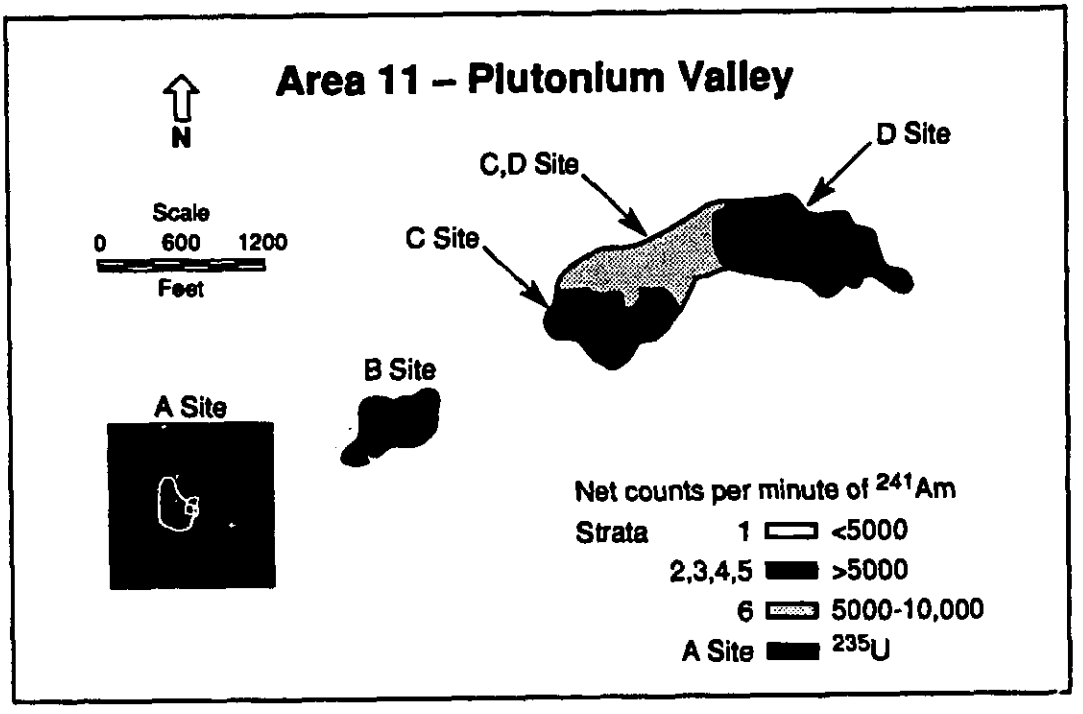

Figure 6. Areal extent of the contamination of ^{241}Am in soils at Area 11 (Plutonium Valley). The measured ^{241}Am is used as a surrogate for $^{239,240}Pu$ (adapted from Gilbert *et al.*, 1975).

Table 1. Summary of the $^{239,240}Pu$ inventories at the shot sites and associated concentrations in soil (from Gilbert, 1977).

		Inventory		Concentration in soil	
Site	Area ha	TBq	±SE	Bq g^{-1}	±SE
Area 13	400	1.7	0.33	5.6	1.1
Area 5 (GMX)	12	0.056	0.007	5.9	0.75
Double Tracks	18	0.13	0.035	9.9	2.6
Clean Slate I	18	0.16	0.044	12	3.3
Clean Slate II	47	0.63	0.137	18	3.9
Clean Slate III	170	1.4	0.20	10	1.5
Area 11[a]					
B site	1.8	0.23	0.041	175	31
C site	2.6	0.29	0.063	149	32
D site	5.3	0.63	0.12	158	30
C/D Overlap	6.2	0.028	0.012	6	2.5

[a] The area encompassing the region where there was less than 5,000 cpm of ^{241}Am contained an additional 0.17 Bq of $^{239,240}Pu$.

throughout the Basin and Range province of Nevada and is therefore likely to occur at the NTS as well. The other three land uses are more dependent on the availability of ground water. The resident rancher, for example, would use well water to support beef cattle and dairy cows as well as a family garden. Residential and commercial developments could obtain potable water from one or more wells supplying multiple users.

The conceptualization of the basic exposure pathways that could bring individuals in contact with the $^{239,240}Pu$ derived from soils at the shot sites is shown in Figure 7. Exposure pathways include ingestion of garden vegetables, consumption of animal products derived from contaminated soils, soil ingestion, and inhalation of airborne particles. Ingestion and inhalation exposures result in the uptake of $^{239,240}Pu$ and subsequent transfer to body organs, which are then irradiated by the emission of alpha particles during the radioactive decay of $^{239,240}Pu$. An integrated exposure and dose model that addresses the pathways shown in Figure 7 was developed specifically for the NTS by the Nevada Applied Ecology Group (NAEG) (see Martin and Bloom, 1980). Kercher and Anspaugh (1991) have performed subsequently a series of sensitivity and uncertainty analyses of the NAEG model. They found that the inhalation pathway produces the greatest doses to individual organs. For example, inhalation of $^{239,240}Pu$-contaminated particles resuspended from soils produced 100% of the dose to the upper-respiratory tract, lungs, and lymph nodes; and 94.9% of the dose to the liver, kidneys, and bone. On a whole-body basis, inhalation represented 94.8% of the total dose and ingestion 5.2%. The principal reason for the difference between the two routes of exposure is the limited uptake of $^{239,240}Pu$ across the GI tract. Plutonium in oxide form has a very low solubility, and since only dissolved species are effectively transported across the epithelial lining of the intestines, the biouptake of Pu is small. After Pu is deposited in the lungs, though, it is slowly translocated to other systemic organs, resulting in higher organ doses than for ingestion exposures.

The analyses that Kercher and Anspaugh performed of the sensitivity of organ doses (*i.e.*, lung, liver, bone, and kidney) to changes in model parameters indicated that doses were most sensitive to changes in the concentration of $^{239,240}Pu$ in soil, the mass-loading factor used to estimate the resuspension of $^{239,240}Pu$ from soil to air, the respiration rate, and dose factors that convert the activity in an organ to the amount of energy deposited. Other less important parameters dealt mainly with biophysical factors such as the fraction of particles deposited in the lungs, clearance rates, and mass of target organs. These screening-level analyses indicate that the inhalation pathway should be the primary focus of the risk assessment for $^{239,240}Pu$ in soils at the NTS. Consequently, our reference land-use scenarios will only consist of residential (suburban) housing, resident farming, and commercial development; cattle grazing is excluded on the basis that the ingestion of $^{239,240}Pu$-contaminated meat products would not constitute a significant radiological health risk.

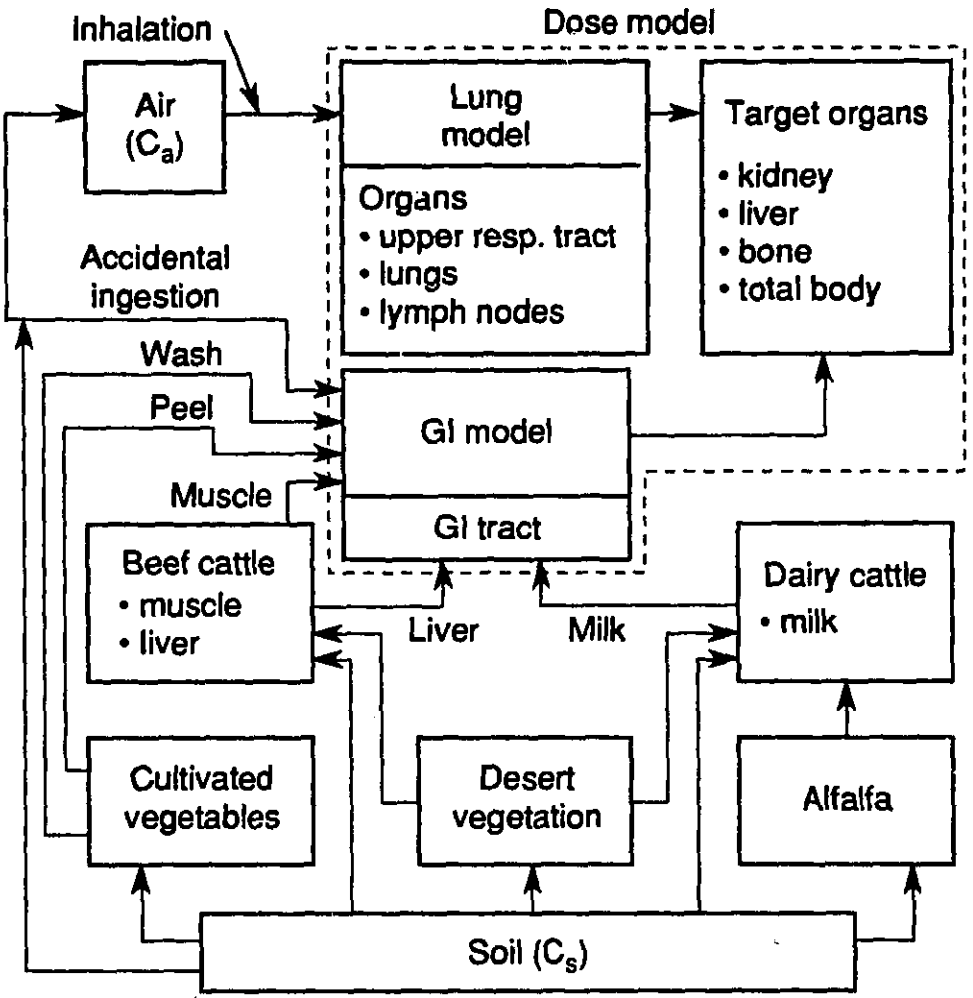

Figure 7. Conceptual diagram of the exposure pathways for ingesting and inhaling $^{239,240}Pu$ in the desert environment of the NTS (adapted from Kercher and Anspaugh, 1991).

4. Transport Processes for Plutonium at Shot Sites

In order to estimate potential inhalation exposures to residual $^{239,240}Pu$ at the shot sites, we must determine the concentrations of Pu in both indoor and outdoor air. People spend routinely more than 90% of their time indoors, and hence differences between the concentrations of $^{239,240}Pu$ in indoor and outdoor air will affect directly estimated inhalation exposures. While outdoors an individual can inhale soil particles containing $^{239,240}Pu$ that have been resuspended into air by wind action. Plutonium-contaminated soil can reach the indoor environment via three principal mechanisms: (1) infiltration of airborne soil particles through the shell of a house, (2) inflow with makeup air drawn into a building by a ventilation system, and (3) tracking of contaminated soil onto floors, followed by resuspension into indoor air by human activities. Suspended particles in indoor air can be removed by filters that are part of air-handling systems in homes and commercial establishments. In the following subsections we estimate concentrations of $^{239,240}Pu$ in outdoor air at the shot sites and then determine the levels indoors for the buildings associated with the residential, farm, and commercial exposure scenarios.

4.1. Concentrations of Pu in Surficial Soils and Outdoor Air

The primary loss mechanism of $^{239,240}Pu$ in surficial soils in the dry environs of the NTS is aeolian erosion. In locations where there are no human activities such as vehicular traffic over unpaved roads, excavation activities, etc., the suspension of soil particles is caused by the movement of wind over the land surface. Most of the early work on this transport mechanism for soil contaminants focused on the resuspension of radioactive substances deposited onto surface soils after nuclear testing. There are many environmental and contaminant-specific factors that affect the transfer of a soil contaminant from soil to air, and the complex nature of this transfer process has made it difficult to develop analytical approaches that provide accurate predictions of resuspension (see Nicholson, 1988; Sehmel, 1980; and Smith *et al.*, 1982; for reviews of resuspension models). As an alternative, empirical approaches have been developed to estimate the average concentration of a soil contaminant in air. One commonly used method involves the calculation of a resuspension factor, which is the ratio of a contaminant's concentration in air to its concentration in soil expressed on an area basis (*e.g.*, Bq or $\mu g\ m^{-3}$ ÷ Bq or $\mu g\ m^{-2}$). The resuspension factor (denoted S_f) for a substance initially introduced to a surficial soil as a result of atmospheric deposition or aerial application decreases with time as the material undergoes various weathering processes. Anspaugh *et al.* (1975) proposed this time-dependent relationship for the resuspension factor

$$S_f(t) = 10^{-4} \times \exp(-0.15\sqrt{t})m^{-1} + 10^{-9}m^{-1}, \quad (1)$$

where t is time in days since initial addition to soil. This relationship was based on data from several sites measured at various times post deposition. The first term in Eq. 1 dominates during the early years following a contaminating event to reflect weathering processes, but diminishes with time to the second term, which Anspaugh *et al.* set equal to the resuspension factor

determined for the GMX site at the end of a 17-year period. However, subsequent resuspension studies conducted by Shinn *et al.* (1986) in the early 1980s showed that the resuspension factor for contaminated soils at the GMX site had continued to decline to 2.0×10^{-10} m^{-1}. They also reported that the more erodable soils in Plutonium Valley had a resuspension factor of 6.1×10^{-10} m^{-1}.

An alternative approach for estimating the amount of a surface contaminant in air is termed the mass-loading method. With this method, the concentration of a soil contaminant in air is calculated as the product of the contaminant's concentration in soil and the mass loading of total suspended particles in air, or

$$C_a = C_s \times TSP, \tag{2}$$

where C_a is the concentration in air (Bq or μg m^{-3}), C_s is the concentration in soil (Bq or μg g^{-1}), and TSP refers to the concentration of particles in the atmosphere (g m^{-3}). Note that the value of C_a for a contaminant in particulate form can be computed as the product of its concentration in suspended particles (denoted as C_p) and the mass loading of particles in the atmosphere. Anspaugh *et al.* (1974) found that there was good agreement between measured concentrations of several airborne radionuclides at four sites in the U.S. and concentrations predicted from the mass-loading method, based on measured values of C_s at the sites and an assumed concentration of suspended particles of 100 μg m^{-3}. An important premise of the mass-loading method is that the ratio of C_p to C_s is one. However, this is unlikely to be the case in all instances because the relationship between the concentrations of a contaminant in the different particle-size fractions of air and suspendable soil undoubtedly change from site-to-site, depending on the characteristics of indigenous soils, the physicochemical properties of the contaminant and host soils, ground cover, and climate. An alternative formulation is to calculate C_a as the product of C_s, TSP, and an enhancement factor (EF), that is,

$$C_a = C_s \times TSP \times EF. \tag{3}$$

Since C_a is equal to $C_p \times$ TSP, the enhancement factor is then simply the ratio of the concentrations of a contaminant in suspended particles and in soil. Shinn *et al.* (1986) reported that the enhancement factors for Pu aerosols at the GMX site and Plutonium Valley were 0.87 and 1.04, respectively, while the mass loadings of particles were 1.7×10^{-5} and 4.1×10^{-5} g m^{-3}. These measurements indicate that the enhancement factor is almost unity for the two sites.

Neither the resuspension–factor nor the mass-loading approach for estimating resuspension accounts directly for the wind-induced removal of a soil contaminant at a given site. This loss is estimated by a resuspension rate or the fraction of contaminant activity or mass that is lost per unit time. It is equal to the net emission rate from soil (*i.e.*, mass or activity emitted per unit area and time) divided by the concentration of contaminant on an areal basis (*i.e.*, mass or activity per unit area). This parameter is estimated indirectly using site-specific data on the changes of contaminant concentration with height above the ground surface, wind speed, and friction velocity (Anspaugh *et al.*, 1975). Shinn *et al.* (1986, 1989) estimated a resuspension rate of 3.9×10^{-11} s^{-1} for ^{239,240}Pu in undisturbed soils in Plutonium Valley and 7.9×10^{-13} s^{-1} for

soils at GMX. In earlier experimental work at the GMX site, Anspaugh *et al.* (1976) calculated resuspension rates for $^{239,240}Pu$ ranging from 2.7×10^{-12} to 4.8×10^{-10} s^{-1}. As a comparison, Hartmann *et al.* (1989) reported a resuspension rate of 9×10^{-11} s^{-1} for pre-Chernobyl $^{239,240}Pu$ at rural sites in Germany, using a contamination depth of 1 cm. Their rate would actually be lower, though, if a 5-cm depth was used to represent the amount of $^{239,240}Pu$ available for resuspension. Collectively, these studies indicate that the resuspension rates for the NTS shot sites probably range between 10^{-12} to 10^{-11} s^{-1} (3.15×10^{-5} to 3.15×10^{-4} y^{-1}).

The time-dependent change in the concentration of $^{239,240}Pu$ in soil resulting from resuspension and radioactive decay can be estimated from the initial concentration in soil as

$$C_s(t) = C_s(0) \exp[-(\lambda_d + \lambda_r)\, t]\,, \tag{4}$$

where t is in years, λ_d and λ_r are the rate constants for radioactive decay and resuspension (both rate constants in y^{-1}). If we use a resuspension rate of 5.5×10^{-12} s^{-1} (or 1.7×10^{-4} y^{-1})(the mean of the limiting values 10^{-12} to 10^{-11} s^{-1}) to represent the net loss of $^{239,240}Pu$ from surface soils at the shot sites, the environmental half-life of $^{239,240}Pu$ is 4000 y, or about 17% of the half-life due to radioactive decay alone.

The value of C_a can be calculated as a function of time from C_s using either the mass-loading or resuspension-factor approaches. With the mass-loading method the time-dependent concentration of $^{239,240}Pu$ in air becomes

$$C_a(t) = C_s(0) \exp[-(\lambda_d + \lambda_r)\, t] \times TSP \times EH \tag{5}$$

and using a resuspension factor, it is

$$C_a(t) = C_s(0) \exp[-(\lambda_d + \lambda_r)\, t] \times \eta \times S_f\,, \tag{6}$$

where η is a factor that converts the concentration of Pu in soil on a mass basis to an areal basis, as a function of soil density and depth of contamination. With C_s equal to 1 Bq g^{-1}, the value of C_a is 3×10^{-5} Bq m^{-3}, assuming that the particulate mass loading at the shot sites is approximately 3×10^{-5} g m^{-3} (the average of the two values reported in Shinn *et al.*, 1986) and the enhancement factor is one. For a nominal soil density of 1.5 g cm^{-3} and a contamination depth of 5 cm, the value of η is 7.5×10^{4} g m^{-2}. By setting Eq. 5 equal to Eq. 6, S_f can then be calculated as $(TSP \times EF)/\eta$. With TSP and EH values noted above, S_f equals 4×10^{-10} m^{-1}, a result that is comparable to the resuspension factors reported above.

To quantify the uncertainties in the estimates of C_a at the different shot sites, we will propagate the uncertainties associated with the individual parameters in Eq. 5, using a Monte-Carlo technique (Crystal Ball, v. 2, Market Engineering Corp., 1991). The means and standard errors of the C_s values for the shot sites are represented in Table 1. To avoid the generation of negative concentration values from the Monte-Carlo sampling of the normal distributions for C_s, we converted the mean and standard errors to their lognormal equivalents (*i.e.*, geometric mean and geometric standard error) using the equations given in Ng *et al.* (1990). Resuspension rates are characterized using a triangular distribution with minimum and maximum values of

3.15×10^{-5} to 3.15×10^{-4} y^{-1} (encompassing the range of resuspension rates discussed earlier) and with a mode at 1.7×10^{-4} y^{-1}. We were only able to obtain two measurements of TSP at the NTS (see earlier discussion), and so we completed a statistical analysis of TSP levels in 20 rural locations in the U.S. reported by Shah *et al.* (1986) as a way of characterizing the variability in the particulate mass loadings at the shot locations. The geometric mean of the TSP data was 2.8×10^{-5} g m^{-3} with a GSD of 1.6. The value of EH is set at 1, based on the experimental studies noted above.

Since $^{239,240}Pu$ at the shot sites could pose a radiological health hazard for thousands of years if not remediated, we computed values of C_s for time periods of 100, 10,000, 50,000, and 100,000 y to cover the period of time when residual $^{239,240}Pu$ in soils are likely to constitute a health hazard. At 100 y the 5, 50, and 95% cumulative percentile values of C_s at Area 13 are 5.0, 5.4, and 5.7 Bq g^{-1}, respectively, based on 10,000 random samples (using the Latin hypercube sampling option) of the distributions for C_s and λ_r. However, at 100,000 y the percentile values are 1.8×10^{-6}, 1.8×10^{-5}, and 1.5×10^{-4} Bq g^{-1}. These results indicate that as t increases, the variation in C_s becomes dominated by the variation in λ_r.

4.2. Concentrations of Pu in Indoor Air

The concentrations of certain airborne contaminants indoors will be less than those outdoors, but for others the concentrations indoors may be higher. A primary contributing factor is the source of each specific contaminant. For those contaminants whose primary source is outdoors, as would generally be the case for Pu, the concentration indoors will generally be less than that outdoors. This is due, among other things, to the filtering effect of building shells (see Engelmann, 1992). To determine the levels of $^{239,240}Pu$ in the indoor environment of a house (suburban residence and farmhouse) and a commercial facility as a function of the contamination of soil and outdoor air, we have implemented two simple compartmental models that predict the steady-state concentrations of $^{239,240}Pu$ in the indoor air. As shown in Figure 8a, the transport processes for a house include the infiltration of contaminated outdoor air, exfiltration of building air, resuspension of tracked-in soil particles from floors to indoor air, the gravitational settling of airborne particles to floors, and the filtration of particles by a heating/cooling system. For a commercial facility (Figure 8b) with an air-handling system that exerts a positive air pressure indoors, there is no significant infiltration of outdoor air (and associated airborne particles). Instead, outdoor air is continuously introduced to the air-handling system and exhausted from the building. The concentration of $^{239,240}Pu$ in the indoor air of a house under steady-state conditions (and with no internal sources of $^{239,240}Pu$ other than resuspension) can be estimated from

$$C_{in} = \frac{L_{fl} A_{fl} f_a R + Ach C_a PV}{\left(A_{fl} v_g + Ach V + Q_r f_{on} E_f\right)} \tag{7}$$

where

$$L_{fl} = f_o M_{fl} C_s \tag{8}$$

and

C_{in} = concentration of $^{239,240}Pu$ in indoor air, Bq m^{-3};

L_{fl} = loading of $^{239,240}Pu$ on floor surfaces, Bq m^{-2};

f_o = fraction of indoor floor dust derived from outdoor soil, unitless;

M_{fl} = mass loading of particulate matter on floors, g m^{-2};

C_s = concentration of $^{239,240}Pu$ in soil, Bq g^{-1};

A_{fl} = surface area of floors, m^2;

f_a = fraction of floor surface that is accessible for walking, playing, etc., unitless;

R = resuspension rate from floors, h^{-1};

Ach = air-exchange rate, h^{-1};

C_a = concentration of $^{239,240}Pu$ in outdoor air, Bq m^{-3};

P = fraction of outdoor $^{239,240}Pu$ particles that infiltrate the building shell to indoor air, unitless;

v_g = gravitational settling velocity, m h^{-1};

V = volume of house (*i.e.*, A_{fl} times the ceiling height, h), m^3;

E_f = removal efficiency for particles, unitless;

Q_r = recirculation rate of air through heat/cooling unit, $m^3\ h^{-1}$; and

f_{on} = fraction of a year that a heating/cooling unit is operating, unitless.

For a commercial facility the steady-state estimate of C_{in} is (based on Weschler *et al.*, 1983; Sinclair *et al.*, 1992):

$$C_{in} = \frac{L_{fl}A_{fl}f_aR + Q_r(1-E_f)f_mC_a}{(A_{fl}v_g + Q_rE_f(1-f_m) + Q_rf_m)}, \qquad (9)$$

where

f_m = fraction of the air recirculation rate that is derived from outdoor air, unitless.

Because L_{fl} and C_a are both functions of C_s, Equations 7 and 9 can also be expressed as the ratios of C_{in}/C_s (in units of g m^{-3}), or

$$\frac{C_{in}}{C_s} = \frac{f_oM_{fl}A_{fl}f_aR + \text{Ach TSP EH P V}}{\left(A_{fl}v_g + \text{AchV} + Q_rf_{on}E_f\right)} \quad \text{(for both suburban and farm houses)} \qquad (10)$$

and

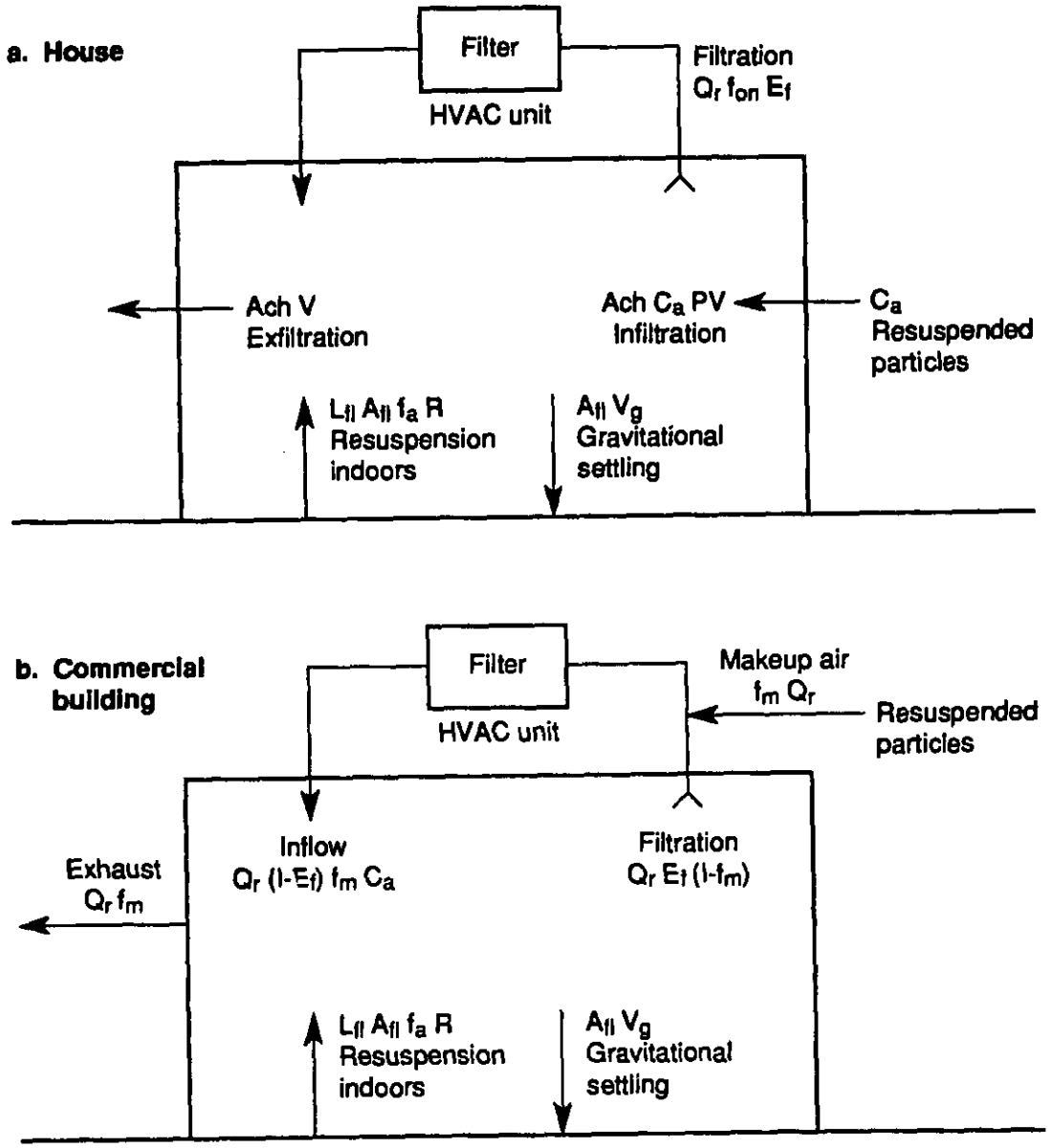

Figure 8. Transport of soil-derived particles in the indoor environment: (a) house with particle filtration in a heating/cooling unit, and (b) a commercial facility with constant filtration of recirculating air. Unlike the house shown in (a), the farm house is assumed to utilize a space heater for heating and so suspended particles are not removed by filtration.

$$\frac{C_{in}}{C_s} = \frac{f_o M_{fl} A_{fl} f_a R + Q_r(1-E_f) f_m \, TSP \, EH}{\left(A_{fl} v_g + Q_r E_f (1-f_m) + Q_r f_m\right)} \quad \text{(for a commercial facility).} \qquad (11)$$

The model parameters can be broken down into two basic groups, one dealing with movement of particles and the other building properties. Particulate-related parameters include the mass loading of soils/dust on floors, the resuspension rate, the settling rate of particles, and the penetration factor. Building properties include the surface area of floors, the air-exchange rate, building volume, the air-recirculation rate, and the particle-removal efficiency of filters in the air-handling system. Many of these parameters are best characterized as statistical distributions, while others can be considered as constants for the purposes of the exposure assessment. In the following discussion we describe the parameter distributions and values for the residential, farm, and commercial buildings.

The loading of $^{239,240}Pu$ on floors (L_{fl}) can be estimated as the product of the concentration of $^{239,240}Pu$ in tracked–in soil, the fraction of indoor dust that is derived from outdoor soil, and the loading of soil/dust on floors. Fergusson *et al.* (1986) estimated that between 45 to 50% of the dust in a house is derived from soil, based on analyses they completed of the elemental compositions of soil and house dust. More recently, Calabrese and Stanek (1992) calculated that 31% of the dust indoors had an outdoor origin, and Allott *et al.* (1992) reported that concentrations of ^{137}Cs in indoor floor dust were between 18 and 48% of the ^{137}Cs concentrations in outdoor soil. Based on these literature values, we have adopted a uniform distribution between 0.2 and 0.5 (a range that encompasses the literature values) to represent the random values of f_o for the three types of buildings. The loadings of soil/dust on floors reported in the literature vary from 0.136 to 0.870 g m^{-2} (see Appendix A). The geometric mean of the mass loadings is 0.42 g m^{-2} with a geometric standard deviation of 1.88. The resuspension rate of particles from floors to indoor air depends largely on human activities, such as walking, playing, etc., that result in the mechanical disturbance of floor surfaces. Healy (1971) estimated a resuspension rate indoors of 5×10^{-4} h^{-1}, using a mix of vigorous, active, moderate, and quiet domestic activities. Murphy and Yocom (1986), in an analysis of the migration of particles into the indoor environment, concluded that the resuspension rate in houses was about 10^{-4} h^{-1}. We will use a uniform distribution between 10^{-4} and 10^{-3} to reflect a range of human activities that resuspend particles from floor surfaces.

Removal of a particulate contaminant by gravitational settling onto floor surfaces is governed by its concentration in air and the average settling velocity of the contaminated particles. The deposition of fine particles onto wall and ceiling surfaces due to diffusion and other mechanisms is estimated using a deposition velocity. Settling/deposition velocities for fine (*i.e.*, < 2.5 μm) to coarse (2.5 to 15 μm) particles suspended in indoor air range from about 0.18 to 25 m h^{-1} (*i.e.*, 0.005 to 0.7 cm s^{-1}) (Sinclair *et al.*, 1992). Diemel *et al.* (1981) made concurrent measurements of airborne lead in homes and the rate of mass accumulation on horizontal collection plates, and from their data we estimate a settling velocity of lead of 1.2 m h^{-1}, which is an intermediate value for fine and coarse particles. In contrast, we estimated that the average settling velocity for dust particles in that study was 2.7 m h^{-1}. Raunemaa *et al.* (1989) reported a deposition velocity of 9.2 m h^{-1} for total coarse particles indoors and 4.9 m h^{-1} for total fine particles. Deposition velocities for individual elements can differ greatly. For the crustal element potassium, Raunemaa *et al.* reported deposition velocities of 15 and 8.8 m h^{-1} for coarse and fine particles,

respectively. They estimated that the average mass–median aerodynamic diameters (MMAD) for all deposited particles was 5 μm. These data suggest that gravitational settling is a more important removal mechanism than deposition, and hence we assume the floor surface is the primary surface of dust accumulation. Settling velocities are represented as a triangular distribution with a mode at 6 m h^{-1} and lower and upper limits of 2 and 10 m h^{-1}.

The median surface area of floors (A_f) in U.S. houses is about 150 m^2 (U.S. Bureau of the Census, 1991), and if we assume that most ceilings are 2.4 m high (8 ft), then the associated volume (V) is 360 m^3. These values will be used to represent both the farm and suburban dwellings. For this assessment we will assume that the commercial facility has 930 m^2 of floor space (*i.e.*, 10,000 ft ^{2}). Since only a portion of floors are actually accessible for walking, playing, etc., we need to adjust the value of A_f downward for the buildings to obtain an effective floor area that contributes to resuspension. We estimate that the fraction of floor space available for resuspension (f_a) is 0.75. Ventilation rates of houses vary according to climatic as well as building characteristics. Nazaroff *et al.* (1985) estimated that the geometric mean air-exchange rate (Ach) was 0.68 h^{-1}, with a GSD of 2.01. The penetration factor, P, can be estimated as the ratio of the indoor-air concentration of a contaminant to its outdoor concentration, provided that there are no significant sources or losses (*e.g.*, filtration by heating and cooling equipment as well as deposition onto interior surfaces) indoors. Penetration factors for contaminants contained in airborne particles generally fall between 0.2 and 0.5, with the highest values associated with substances enriched on fine particles. Cohen and Cohen (1980), for example, reported that the average indoor/outdoor ratios for Ca, Fe, Pb, and Br in homes (which did not have indoor sources of these contaminants) were 0.23, 0.25, 0.53, and 0.38. These penetration estimates correspond directly with the MMAD for the four elements presented in a review by Milford and Davidson (1985) (*i.e.*, 4.64, 3.42, 0.55, and 0.89 μm, respectively). The penetration factor can be estimated using the following power-law fit to the data:

$$P = 0.4\, d^{-0.37}\ (r^2 = 0.96), \tag{12}$$

where d is the MMAD in micrometers. Shinn *et al.* (1989) reported that the activity-median aerodynamic diameter (AMAD) of $^{239,240}Pu$ sampled in the ambient air in Plutonium Valley was 5 μm with a GSD of 8.5. Anspaugh and Phelps (1974) determined an AMAD of 3 μm for airborne particulates collected by high-volume cascade impactors operating at the GMX site. Thus, we would expect that only about 20 to 30% of the resuspended Pu would penetrate a house. In our simulations we will characterize the variation in the AMAD values for resuspended $^{239,240}Pu$ at the sites using a uniform distribution with minimum and maximum values of 3 and 5 μm.

Airborne particles indoors that are derived from infiltrating air and resuspension from floors can be removed by filters in heating, ventilation, and air-conditioning (HVAC) systems. For the purposes of our assessment, we will assume that the suburban residence is equipped with a central heating/cooling system that recirculates air within the building. The farmhouse, though, is not assumed to have a central heating/cooling unit, and as a result, there is no internal filtration of particles. The air-recirculation rate (Q_r) for a standard heating/cooling unit (*i.e.*, the heating component has a heat input of 80,000 Btu h^{-1}) in a suburban residence is approximately

2400 m^3/h^{-1} (based on a typical air-handling rate of 1400 ft^3 min^{-1}). If the unit operates an average of 30% per year (denoted f_{on}), the effective recirculation rate is 1200 m^3 h^{-1}. The particle-removal efficiency (E_f) of standard fibrous filters in domestic HVACs is approximately 0.5 (Lefcoe and Inculet, 1975). Air recirculation rates in the commercial facility are assumed to be for an office environment, with Q_r defined as the product of the building volume (V) and an air exchange rate (Ach), defined as a uniform distribution between 10 and 20 air-exchanges per hour. The resulting limits of the uniform distribution of Q_r for the commercial building volume of 2200 m^3 (*i.e.*, 930 m^2 × 2.4 m) are 2.2×10^4 and 4.4×10^4 m^3 h^{-1}. The outdoor air makeup (f_m) fraction is defined as a uniform distribution between 0.1 to 0.2 (from Hayes, 1989; Weschler *et al.*, 1983). The filter efficiency for coarse particles is also assumed to be 0.5. Table 2 summarizes the values used to represent the various input parameters to Eqs. 10 and 11.

Monte-Carlo simulations of Eq. 10 using the appropriate parameter values and distributions for a suburban house (with central air conditioning and heating) resulted in a lognormal distribution of the C_{in}/C_s ratios with a GM of 6.8×10^{-6} g m^{-3} and a GSD of 1.89. The GM and GSD of the C_{in}/C_s ratios for the farm house (no central air conditioning or heating) were 9.1×10^{-6} g m^{-3} and 1.95, respectively. The larger ratio for the farm house was due primarily to the lack of indoor filtration of suspended particles by a central air conditioning/heating unit. The commercial facility, which has a continuously operating air ventilation and filtration system, achieved the lowest ratio of C_{in}/C_s, with a GM of 5.3×10^{-6} g m^{-3} and a GSD of 1.56. The geometric means of the ratios of C_{in}/C_a (equivalent to the indoor/outdoor concentration ratios of

Table 2. Summary of the parameter values used to determine the concentrations of $^{239,240}Pu$ in the indoor air of houses and commercial buildings.

Variable	Units	Distribution	Residential	Farm	Commercial
			Statistics		
P	unitless	Uniform	Min. (0.2) Max. (0.3)	Min. (0.2) Max. (0.3)	Min. (0.2) Max. (0.3)
Ach	h^{-1}	Lognormal	GM (0.67) GSD (2.0)	GM (0.67) GSD (2.0)	GM (0.67) GSD (2.0)
v_g	m h^{-1}	Triangular	Mode (6) Min. (2) and Max. (10)	Mode (6) Min. (2) and Max. (10)	Mode (6) Min. (2) and Max. (10)
A_{fl}	m^2	Constant	150	150	930
f_a	unitless	Constant	0.75	0.75	0.75
f_o	unitless	Uniform	Min. (0.2) Max. (0.5)	Min. (0.2) Max. (0.5)	Min. (0.2) Max. (0.5)
M_{fl}	g m^{-2}	Lognormal	GM (0.42) GSD (1.88)	GM (0.42) GSD (1.88)	GM (0.42) GSD (1.88)
h	m	Constant	2.4	2.4	2.4
Q_r	m^3 h^{-1}	Constant	2400	–	–
Q_r	m^3 h^{-1}	Uniform	–	–	Min. (22,000) Max. (44,000)
f_{on}	unitless	Constant	0.3	–	–
E_f	unitless	Constant	0.5	–	0.5
TSP	μg m^{-3}	Lognormal	GM (28) GSD (1.6)	GM (28) GSD (1.6)	GM (28) GSD (1.6)
EH	unitless	Constant	1	1	1
R	h^{-1}	Uniform	10^{-4} to 10^{-3}	10^{-4} to 10^{-3}	10^{-4} to 10^{-3}

airborne $^{239,240}Pu$) for the suburban house, farmhouse, and commercial building were 0.24, 0.32, and 0.19, respectively.

5. Human Factors Describing Exposure Scenarios

To quantify inhalation exposures to $^{239,240}Pu$ from soil–derived aerosols in indoor and outdoor air and the cumulative doses resulting from those exposures, it is necessary to characterize the following parameters: (1) the age-dependent breathing rates for indoor and outdoor locations, (2) the fractions of time spent indoors and outdoors at residential and commercial sites where soil contamination occurs (*i.e.*, the exposure frequency), and (3) the length of time spent at each location (*i.e.*, the exposure duration). The locational aspects of human activities are important because the concentrations of $^{239,240}Pu$ differ between indoor and outdoor air. Moreover, the amount of time people spend at a given location depends on its function (*e.g.*, a residence or place of work). Inhalation rates change from location to location as a function of the physical activities carried out in the different locations. The four basic locations that people occupy are shown in Figure 9. For each location type we will now characterize exposure factors that will be used in the dose assessments for alternative land-use scenarios at the shot sites.

5.1. Activity Patterns

Important exposure factors that must be considered when addressing contacts with soil contaminants at a given location (*i.e.*, nonubiquitous soil contaminants) is the amount of time an individual spends at indoor and outdoor locations at the site and the kinds of activities performed while there. Both of these lifestyle factors changes with age. We have characterized the age-dependent changes in the amount of time an individual spends at indoor and outdoor locations at home using the results of activity surveys of children (< 12 y) and adults (≥ 12 y) in California (Wiley, 1991; Wiley *et al.*, 1991). The random sample of children included 1200 individuals, while the adult sample included 1,762 individuals. Figure 10 shows the fractions of each day that were spent indoors and outdoors at home. As might be expected, there are U-shaped functional relationships between age and the fractions of time spent indoors and outdoors at home. The fraction of time indoors decreases from birth to about age 20 y and then gradually increases into the retirement years. By comparison, the amount of time outdoors at home averages less than 5% of a day for those under 10 y, decreasing to around 2% in middle age, and then increasing again in the retirement years. The amount of sleep time is presented in Figure 11. Children under age 10 sleep more than 10 h per day, and as individuals reach middle age, they sleep an average of eight hours per day. Small increases in sleep time occur after age 50 y. Unfortunately, we were unable to obtain similar age-dependent activity-pattern data for residents of farms. If such data were available, we would expect that farm residents would spend much more time outdoors adjacent to a farm house. We will examine the effect that increased time outdoors has on our dose estimates for the resident farmer in the section dealing with dose estimates.

Indoors at home	Indoors away from home
Outdoors at home	Outdoors away from home

Figure 9. Four basic categorizations of indoor and outdoor locations for use in assessments of exposures to soil-derived contaminants at a given location.

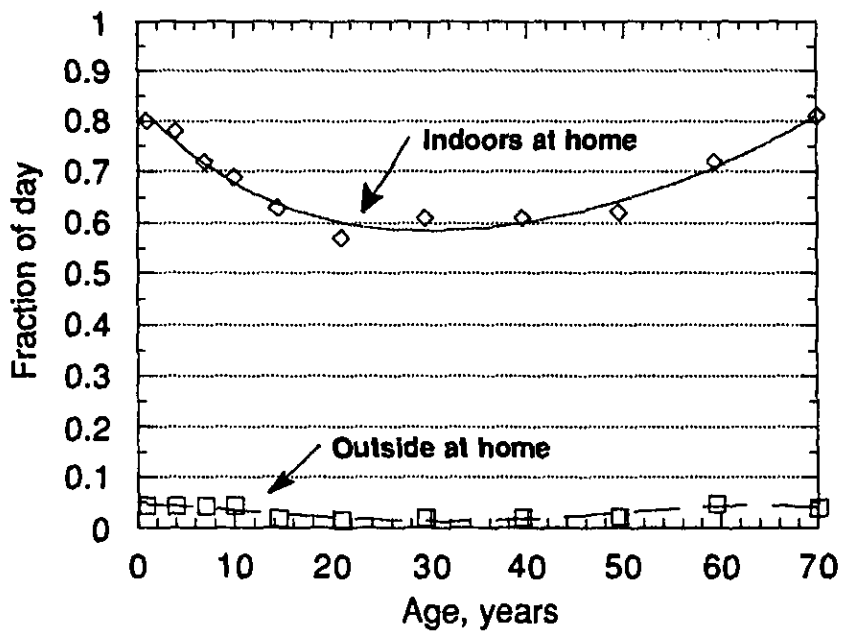

Figure 10. Fractions of time individuals spend indoors at home and outside at home (data are from Wiley, 1991; Wiley *et al.*, 1991).

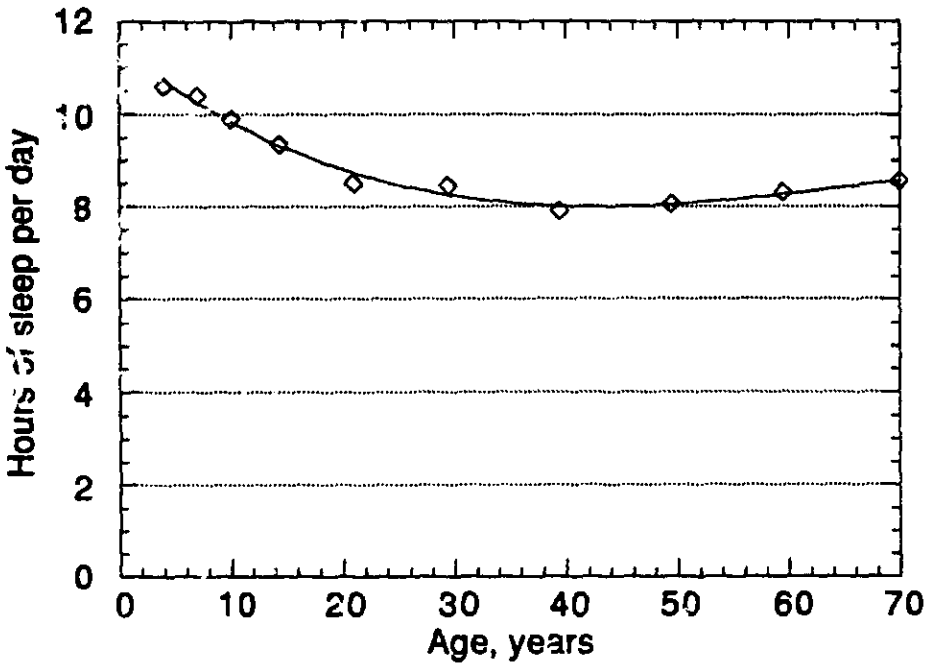

Figure 11. Hours of sleep as a function of age (data from Wiley, 1991; Wiley *et al.*, 1991).

The amount of time spent at a commercial facility depends in part on an individual's employment status (*i.e.*, full- or part-time worker) and the nature of the facility (*e.g.*, retail, office, manufacturing, etc.). Because our commercial land-use scenario is not meant to represent any particular kind of commercial operation, we will use labor statistics for the employed civilian work force in the U.S. to characterize the time spent at an unspecified work location. Employment data (U.S. Bureau of the Census, 1991) show that the work week for wage and salary workers employed in nonagricultural work has averaged between 38 and 39 h since 1970. Expressed on the basis of a seven-day week, this translates to about 23% of an average day (or on an annual basis, 84 d y^{-1}). We were unable to obtain data on the amounts of time that workers spent outdoors at office, retail, and manufacturing locations. For the purposes of our exposure assessment, we have assumed simply that workers in the arid Nevada climate would spend 0.5 h d^{-1} outdoors at their place of employment (or 5.4 d y^{-1}, based on a 52–week working year). One additional consideration in characterizing the worker population is the age span of those employed. Although individuals can enter the workforce at age 16 y, we assume that full-time employment begins at age 19 y and continues until age 64 y. Workers aged 65 y and older and younger than 19 y accounted for less than 10% of the total civilian workforce in 1989 (U.S. Bureau of the Census, 1991).

5.2. Age-dependent inhalation rates

Respiration provides the oxygen needed to metabolize food nutrients and therefore produce the energy needed to sustain our various activities. Inhalation rates can be subdivided into two basic categories, one representing inactive periods of sleep/rest and the other representing the range of various physical activities carried out each day. Thus, the inhalation rate used for individuals indoors at home must reflect the respiration rates associated with both active (*i.e.*, waking hours) and inactive periods (*i.e.*, sleep/rest); at other indoor/outdoor locations breathing rates are not assumed to reflect periods of sleep/rest. The breathing rate indoors at home (designated $BR_{in,h}$) can be expressed as a weighted-average value of the breathing rate for active and inactive hours, or

$$BR_{in,h} = P_r\, BR_r + P_a BR_a, \tag{13}$$

where

$$P_r = S/(T_{in,h} \times 24), \tag{14}$$

$$P_a = 1 - P_r, \tag{15}$$

and

$BR_{in,h}$ = breathing rate while indoors at home, $m^3\ d^{-1}$;

P_r = fraction of time indoors at home that is spent at sleep/rest, unitless;

P_a = fraction of time indoors at home that is spent at various activities, unitless;

BR_r = breathing rate while at rest, $m^3\ d^{-1}$;

BR_a = breathing rate while active, $m^3\ d^{-1}$;

S = sleep time, h; and

$T_{in,h}$ = fraction of time spent indoors at home.

The average age-dependent inhalation rate for periods of sleep/rest (*i.e.*, BR_r) for males and females is shown in Figure 12. This rate is based on a methodology presented by Layton (1993) in which inhalation at rest is determined from the oxygen uptake rate required to support basal metabolism and a ventilatory equivalent, which is the ratio of breathing rate to oxygen uptake. During active hours respiration increases to supply additional oxygen to yield metabolic energy that is equal to the energy expenditures for various activities (*i.e.*, an individual's total daily energy expenditure less energy expended during sleep/rest). The breathing rate for active hours can be expressed as a function of the breathing rate at rest or,

$$BR_a = H \times BR_r, \tag{16}$$

where

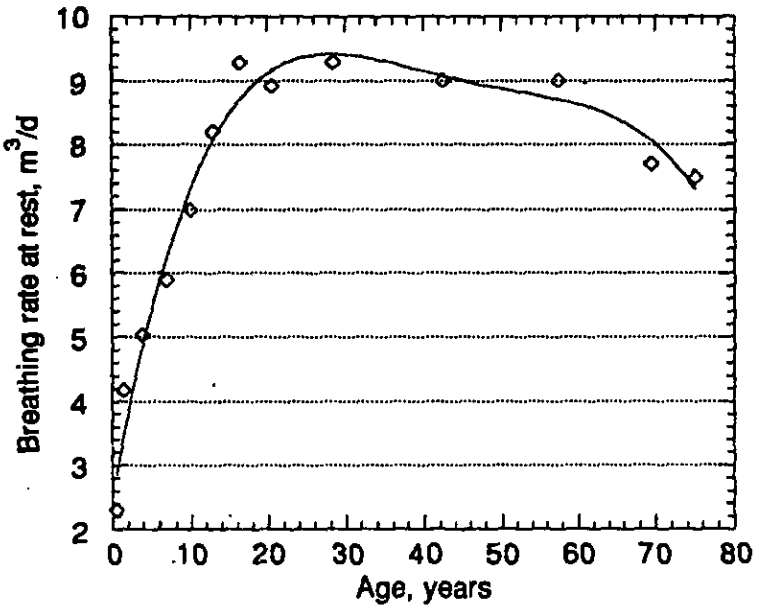

Figure 12. Average inhalation rate at rest for males and females as a function of age (derived from Layton, 1993).

$$^{7}I = (24\ M - S)/(24 - S), \quad (17)$$

and

H = breathing–rate–multiplier used to calculate BR_a as a function of BR_r, unitless; and

M = ratio of total energy expended each day to an individual's basal metabolic rate (BMR), unitless.

In this formulation H represents the factor that BR_r must be increased by to meet the increased metabolic requirements associated with waking hours. The two age-dependent parameters used to calculate H are sleep time (S) and the ratio of total energy intake to BMR (also referred to as the metabolic equivalent, denoted here as M). As sleep time decreases and M increases, the amount of energy expended during active hours increases with respect to the resting inhalation rate, and there is a commensurate increase in the value of H. Figure 13 shows the metabolic equivalents of the total energy expenditures for different ages (from Layton, 1993),

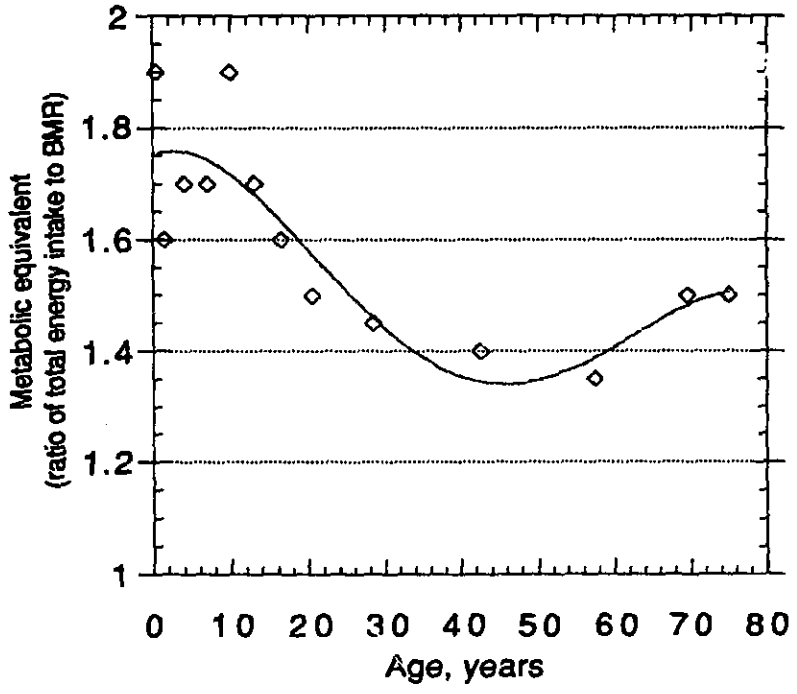

Figure 13. Average energy intakes for males and females expressed as metabolic equivalents (from Layton, 1993).

and a polynomial fit to the data. The breathing rates for sleep/rest, active hours, and indoors at home are depicted in Figure 14. Farmers are expected to have higher average energy expenditures than members of the general public and hence higher breathing rates as well. Riumallo *et al.* (1989), for example, reported that the average energy expenditure (in metabolic equivalents) for six males engaged in light agricultural tasks was about 1.8, which is about 20 to 30% higher than the average values for both males and females depicted in Figure 13. The increased inhalation for a farmer may also be associated with an increase in mouth breathing, which would lead to a larger deposition of particles in the lungs. As a default in our exposure and dose estimates, we will use the age-dependent inhalation values for the general population and estimate the increased exposures for the case of the resident farmer.

5.3. Residential and Occupational Mobility

The cumulative inhalation exposures to airborne $^{239,240}Pu$ at a given residential or commercial location are not only a function of an individual's breathing rate and exposure

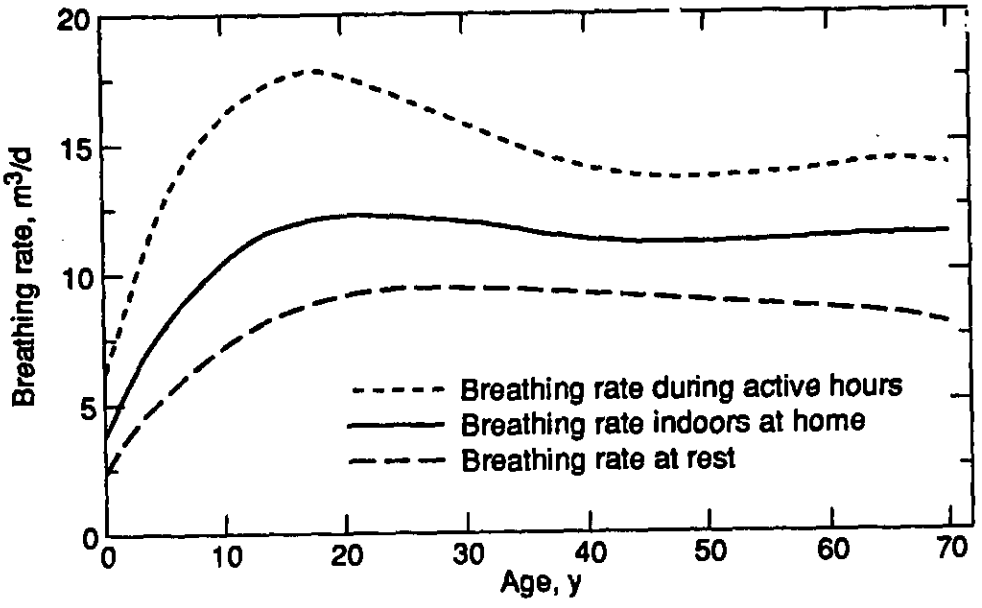

Figure 14. Age-dependent changes in the breathing rates at rest, indoors at home, and during active hours.

frequency for the prescribed location, but also the duration of the exposure. To estimate the length of time spent at residences in different urbanizations (*i.e.*, rural suburban and farm), we have analyzed statistical data from a national survey of U.S. housing in 1989 (U.S. Bureau of the Census, 1991). Figure 15 presents empirical probability distributions of the lengths of time that householders reside at domiciles situated in suburban areas or at farms in rural parts of the U.S. A substantial fraction (*i.e.*, 0.45) of the suburban householders sampled in 1989 spent less than four years at their current residence. The mean residence time was about ten years. As might be expected, those living on farms were far less mobile. They resided an average of 20 years in their homes — twice the mean residence time for those suburbanites living in rural areas. The number of years that employees might work at an unspecified commercial facility can be estimated from survey data on workers from different occupations in the U.S. (U.S. Bureau of the Census, 1991). In 1987 the median occupational tenures for 27 major occupations (defined as those with 650,000 workers or more) ranged from 1.9 to 21.1 y (excluding occupations that are not associated with a single work location, such as truck drivers and carpenters). The geometric mean tenure of the occupations was 6 years and the geometric standard deviation was 1.74 (see Figure 16). These occupational tenures will tend to overestimate the number of years spent at a given commercial location because an individual may change work locations.

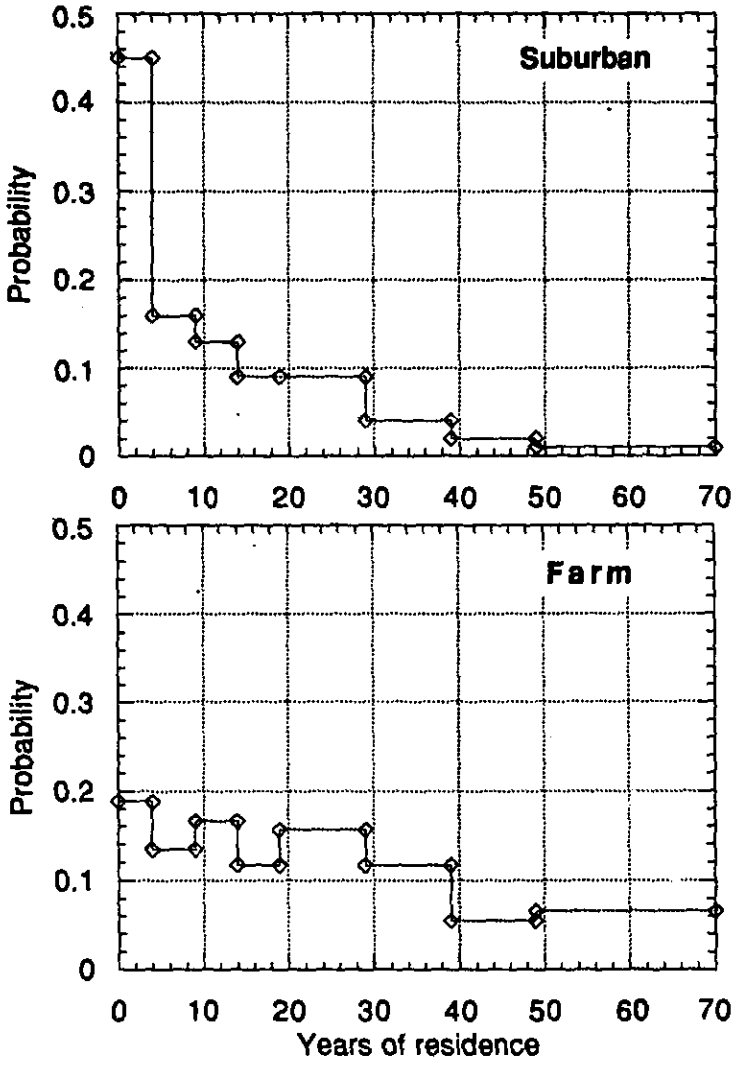

Figure 15. Mobility of persons living in rural suburban residences and farms (derived from U.S. Bureau of the Census, 1991).

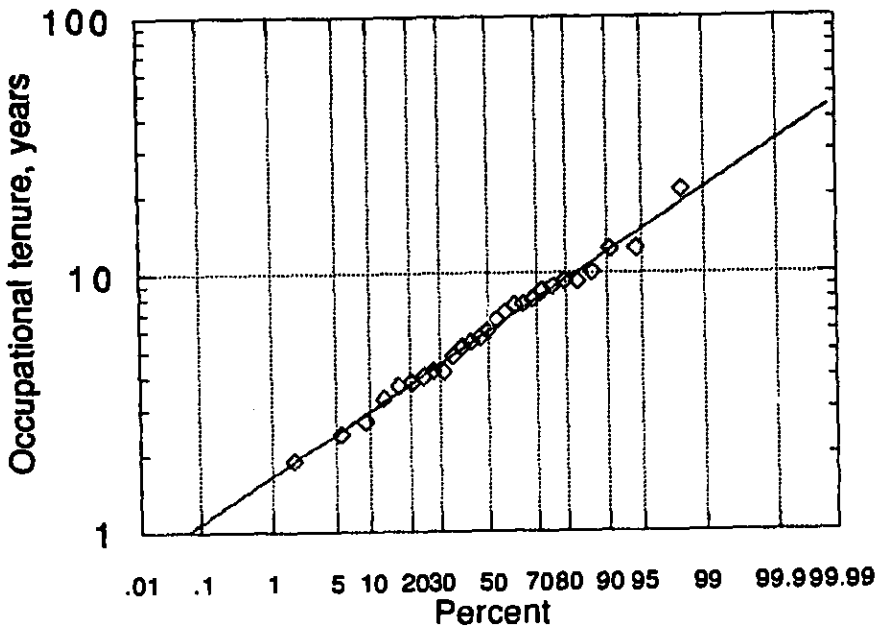

Figure 16. Logprobability plot of the median occupational tenures for 27 major, nonfarm occupations in 1987 (data from U.S. Bureau of the Census, 1991). The geometric mean is 6 y and the geometric standard deviation is 1.74.

6. Organ–Specific Doses and Risk Factors

The cancer risk associated with the inhalation of $^{239,240}Pu$ in environmental media is governed by the doses to various organs and the risk factor (*i.e.*, the incremental probability of cancer per unit dose) for each organ. Accordingly, we have developed estimates of organ-specific doses and risks for the exposure pathways discussed in the previous section. The basic objective of the dose assessment is to determine the cumulative doses of $^{239,240}Pu$ to critical organs in individuals as a function of the concentrations of $^{239,240}Pu$ in environmental media, human factors (*e.g.*, land use, inhalation rates, activity patterns, mobility, and diet), and the age-dependent retention, translocation, and excretion of plutonium.

The cancer risk of exposures to $^{239,240}Pu$ can be expressed as

$$R = \sum_{i=1}^{n} D_i^{70} \times R_i \qquad (18)$$

where

R = the incremental risk of incurring cancer, unitless;

D_i^{70} = the cumulative lifetime dose to age 70 y for the ith organ of n critical organs, Gy; and

R_i = the linear risk factor for the ith organ, Gy^{-1}.

With this methodology, cancer risk is calculated directly from the quantities of energy deposited in key organs resulting from the decay of retained $^{239,240}Pu$ and the dose-response relationship between deposited energy and the probability of cancer for those organs. In contrast with this approach, ICRP (1991) methodology relies on the determination of equivalent doses to individual organs (*i.e.*, doses that are adjusted to account for the biological effectiveness of different radiations, in units of sieverts, Sv) and the use of tissue-weighting factors, which reflect the different probabilities of developing cancer in various organs of a body uniformly irradiated by external gamma radiation (based largely on cancer-mortality data for A-Bomb survivors). In the sections below we present our estimates of organ-specific doses resulting from the inhalation exposures to $^{239,240}Pu$ derived from contaminated soils associated with the alternative land uses discussed earlier, and we also assess risk factors for estimating the probability of cancer for internally irradiated organs.

6.1. Organ-Specific Doses from Inhalation Exposures

Doses to specific organs are calculated from the exposure conditions used to define the frequency and duration of an individual's inhalation of airborne $^{239,240}Pu$ and its concentrations in the indoor and outdoor environments. The lifetime cumulative dose to age 70 y for a given organ resulting from an inhalation exposure over a specified period of time (defined by the integration limits a and b) can be calculated from

$$D_i^{70} = \int_a^b C_{out}(t)\, BR_{out}(t)\, T_{out}(t)\, DC_i^{70}(t)\, dt + \int_a^b C_{in}(t)\, BR_{in}(t)\, T_{in}(t)\, DC_i^{70}(t)\, dt \qquad (19)$$

where

D_i^{70} = lifetime cumulative dose to age 70 y for the ith organ resulting from inhalation exposures to $^{239,240}Pu$ at a given location over an exposure period defined by the integration limits a and b, μGy;

$C_{in}(t)$, $C_{out}(t)$ = time-varying concentration of $^{239,240}Pu$ in indoor or outdoor air, Bq m^{-3};

BR_{in}, BR_{out} = age-dependent breathing rate while indoors or outdoors, m^3 d^{-1};

T_{in}, T_{out} = age-dependent amount of time spent indoors or outdoors at a given location, d y^{-1};

$DC_i^{70}(t)$ = cumulative lifetime dose to age 70 y for the ith organ for inhalation exposure at time t, μGy Bq^{-1}.

One difficulty with the formulation of Eq. 19 is that it cannot be used easily to simulate the residential and occupational mobility of individuals, that is, the lengths of time spent at a given location as a function of age (as defined by the integration limits). A simpler formulation can be derived by treating the indoor/outdoor concentrations of $^{239,240}Pu$ as constants over exposure periods and by assuming that the residence times of individuals at a given location are distributed randomly with age. The revised formulation then becomes

$$D_i^{70} = M\left[C_{out}\bar{F}_{i,\,out} + C_{in}\bar{F}_{i,\,in}\right] \tag{20}$$

where

$$\bar{F}_{i,\,out} = \frac{1}{(b-a)}\int_a^b BR_{out}(t)\,T_{out}(t)\,DC_i^{70}(t)\,dt, \tag{21}$$

$$\bar{F}_{i,\,in} = \frac{1}{(b-a)}\int_a^b BR_{in}(t)\,T_{in}(t)\,DC_i^{70}(t)\,dt, \text{ and} \tag{22}$$

where

M = the residence time at a given residential, farm, or occupational location, y; and

$\bar{F}_{i,\,in}$ and $\bar{F}_{i,\,out}$ = exposure-normalized dose-conversion factors for indoor and outdoor inhalation exposures over the period defined by the integration limits a and b, m^3–μGy y^{-1}–Bq^{-1};

For workplace exposures to Pu aerosols, the parameters T_{out} and T_{in} are treated as constants (*i.e.*, 5.4 and 84 d y^{-1}, respectively) and the breathing rate for active hours is used as the effective inhalation rate for both indoor and outdoor locations.

The age-dependent dosimetry for $^{239,240}Pu$ is based on a model that was developed by Leggett (1985) and later modified for use in ICRP Publication 56 (ICRP, 1990). Our implementation of the modified model (illustrated schematically in Figure 17) utilizes the age-specific physiologic and biokinetic parameter values given in Tables 1.-1 and A-1 of ICRP Publication 56, respectively. In addition, our model includes a modified version of the ICRP (1979) lung model, as shown in Figure 17. In the respiratory portion of the model, inhaled $^{239,240}Pu$ aerosols (*i.e.*, Class Y aerosols with an AMAD of 1 μm) are deposited on the surfaces of the nasal passages (denoted NP) and the trachea and bronchial tree (denoted TB) and are transferred instantaneously to blood and the GI tract via lung subcompartments a, b, c, and d. The standard ICRP lung model integrates the dose to these subcompartments; however, because they are cleared rapidly, little accuracy is lost in omitting them. The parameters f_a, f_b, f_c, and f_d indicate the fractions of the deposited $^{239,240}Pu$ transferred to blood or lungs (see Fig. 17). Aerosols reaching the pulmonary region that are deposited (determined from the deposition fraction f_p) are transferred to lung e, h, and g subcompartments (the quickly cleared f subcompartment is omitted in our model). The lung subcompartment h clears to the pulmonary lymph nodes, while the e and g subcompartments clear to blood and the GI tract, respectively. Once in the blood, some $^{239,240}Pu$ is distributed to the systemic organs shown in Figure 17, but most is translocated to skeleton and liver. The Leggett model divides the skeleton into cortical bone and trabecular bone. These bone types are subdivided into three biologically–relevant compartments for estimating doses: (1) bone surface, (2) bone volume, and (3) bone marrow. The liver is treated as a single compartment in the modified Leggett model.

The portion of the model used dealing with bone dosimetry is based entirely on the bone-recirculation approach of Leggett (1985) as implemented in ICRP Publication 56. In this approach, specified fractions of all Pu circulating in blood continuously enter compartments representing cortical-bone and trabecular-bone surfaces, respectively, and the Pu is then lost (with first-order kinetics) to the volume and marrow portions of those types of bone. The cortical and trabecular marrow also receives $^{239,240}Pu$ directly from blood, again through first-order processes. Integrated $^{239,240}Pu$ α-particle dose to bone is calculated as summarized in Table 7.4 of ICRP Publication 30 (ICRP, 1979). According to this dosimetry model for α-emitters in bone, all bone surfaces plus all trabecular (*i.e.*, red) bone marrow comprise the biologically relevant set of bone-related target organs. Thus integrated dose to bone surfaces includes (a) dose delivered to that surface from $^{239,240}Pu$ situated within cortical and trabecular surfaces plus (b) the doses delivered from $^{239,240}Pu$ situated within cortical and trabecular bone volumes, appropriately discounted for the limited range of $^{239,240}Pu$ α-particles and the geometric properties of the source and target compartments involved. Similarly, integrated dose to red marrow includes 100% of the $^{239,240}Pu$ dose contained within trabecular marrow plus those doses (again, appropriately adjusted) delivered to that marrow from trabecular-bone surface and volume compartments.

Four target organs (liver, lung, bone surface, and red bone marrow) were selected for analysis based on a preliminary review of dose-response data on those organs. The uptake, translocation, deposition, and excretion of Pu through the various compartments and corresponding integrated doses to the target organs was calculated using a system of ordinary differential equations, which we have solved numerically using *Mathematica* (Version 2.0, Wolfram, 1991). These equations included time-dependent parameters, as noted above. The continuous relationships between age

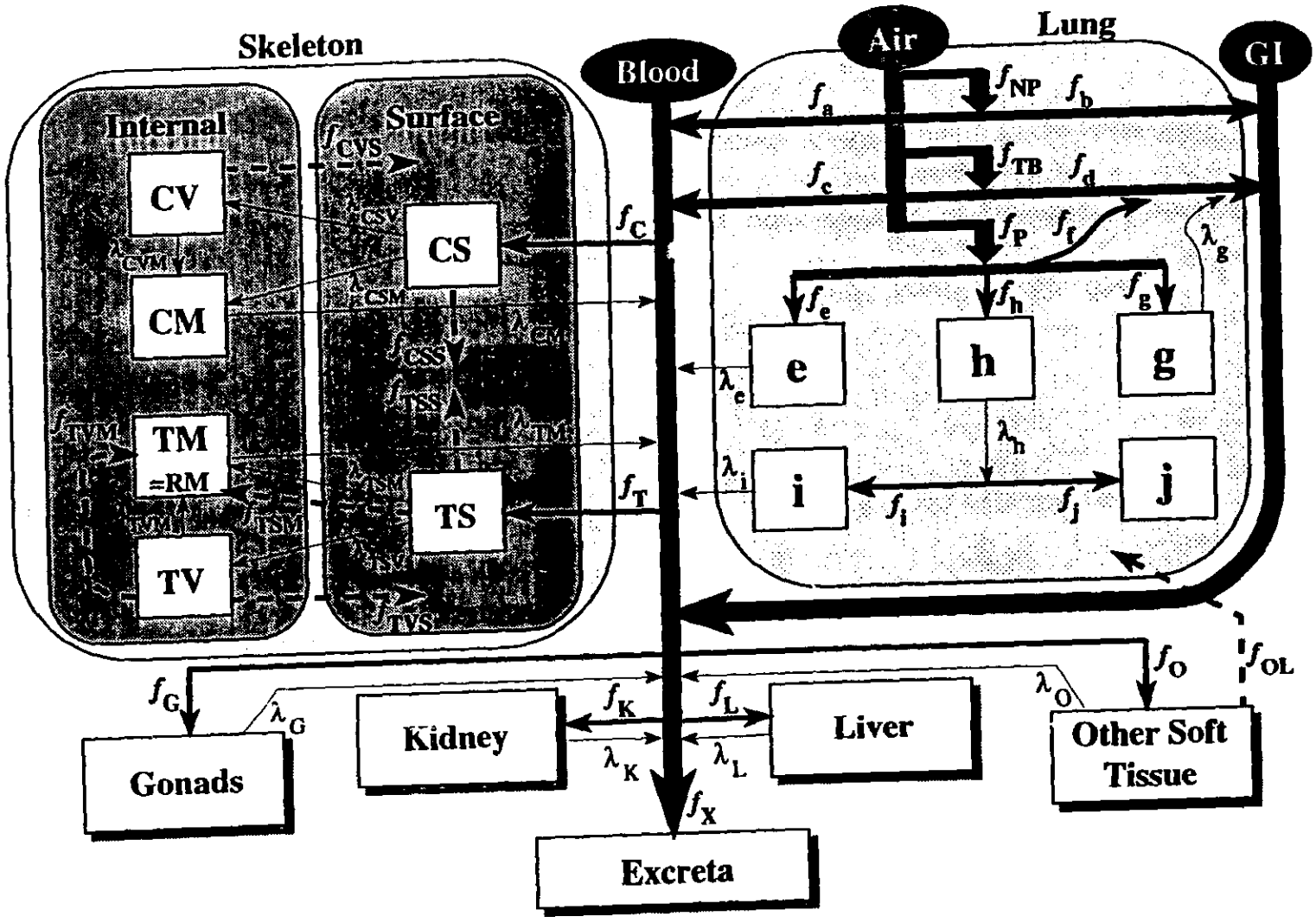

Figure 17. Modified Legget-ICRP dosimetry model for inhaled and/or ingested plutonium. C = cortical, T = trabecular, R = Red, M = marrow, S = surface, V = volume, e-j = ICRP lung compartments, f (λ) = fraction deposited via (or loss constant for) processes presumed instant (or 1st-order exponential, indicated by thinnest arrows and typeface). Dashed arrow signifies a deposition of radiation dose without mass transfer. See text for further details.

at exposure and cumulative dose to age 70 y to target organs per unit respiratory intake ($DC_i^{70}(t)$ in μGy Bq^{-1}) are illustrated in Figure 18. The cumulative doses to the four organs decrease with age at exposure as the period of integration decreases. Nonmonotonic changes in the shape of the curves are due to corresponding age-dependent changes in organ weights, organ-specific transfer fractions, and excretion rates. The curves are in the form of interpolating functions for cumulative doses determined at various ages of exposure. Our cumulative dose estimates for each of the target organs are all within a few percent of the inhalation dose coefficients (in Sv Bq^{-1}) for ^{239}Pu given in ICRP Publication 56 for six ages when exposure is assumed to occur [*i.e.*, 3 mo, 1 y, 5 y, 10 y, 15 y, and adult (> 17 y)].

For the residential and farm exposure scenarios, the integration period (a – b) encompasses the ages of 0 to 70 y, whereas for a commercial land use the integration period encompasses the age span of the worker population (*i.e.*, 19 to 65 y). Table 3 contains the average dose-conversion factors for the two types of domestic exposure scenarios as well as the commercial exposure scenario. For a unit concentration of $^{239,240}Pu$ in air and exposure period, bone surfaces receive the greatest doses, followed by the lungs, liver, and bone marrow. The bone marrow receives less than 10% of the dose to the other organs. Differences between the $\bar{F}_{i,in}$ and $\bar{F}_{i,out}$ variables are due to breathing rates used for indoor/outdoor locations and the amounts of time spent indoors and outdoors. The values of $\bar{F}_{i,out}$ for a resident farmer would increase by a factor of approximately five if the value of T_{out} were equivalent to 3 h d^{-1} (12.5% of a day) spent outside in the immediate vicinity of a farmhouse. An increased breathing rate associated with a metabolic equivalent energy expenditure of 1.8 would only increase the active breathing rate by about 25%.

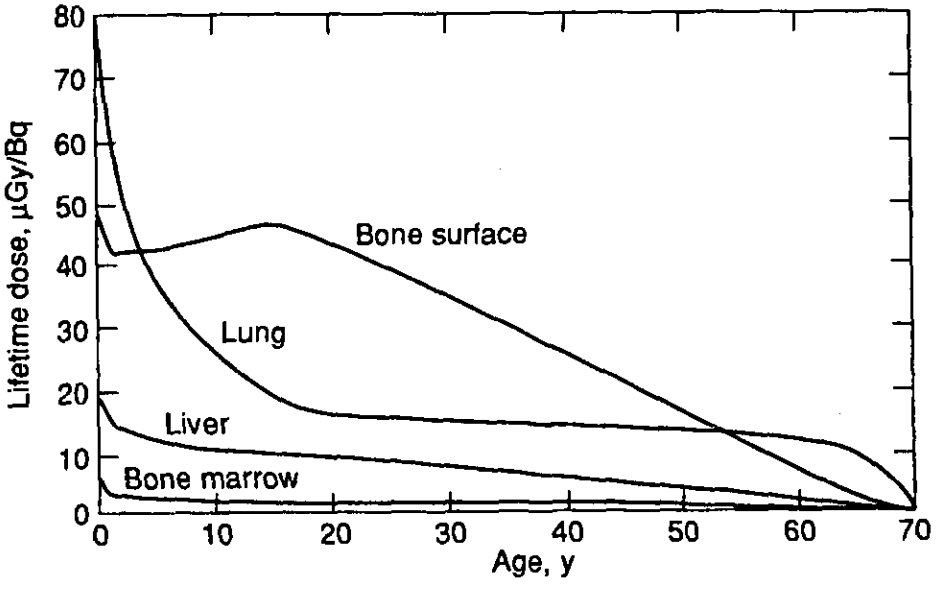

Figure 18. Cumulative lifetime doses to age 70 y for selected body organs based on instantaneous respiratory exposures at different ages.

Table 3. Exposure-normalized dose-conversion factors calculated for different exposure scenarios and organs.

Exposure scenario/tissue	Exposure-normalized dose-conversion factors $\bar{F}_{i,in}$	$\bar{F}_{i,out}$
	$m^3 - \mu Gy\ y^{-1} - Bq^{-1}$	
Residential/Farm		
Bone surfaces	6.7×10^4	3.9×10^3
Liver	1.6×10^4	9.4×10^2
Lung	4.1×10^4	2.7×10^3
Bone marrow	3.8×10^3	2.3×10^2
Commercial		
Bone surfaces	3.1×10^4	2.0×10^3
Liver	6.7×10^3	4.3×10^2
Lung	1.7×10^4	1.1×10^3
Bone marrow	1.7×10^3	1.1×10^2

Variability in the $\bar{F}_{i,in}$ and $\bar{F}_{i,out}$ parameters between individuals will be due to interindividual variations in breathing rates, time spent indoor/outdoors, and biodosimetric parameters such as the fractions of inhaled Pu deposited in different tissues, residence times in organs, etc. As a means of characterizing the variability of the exposure-weighted dose-conversion factors, we reviewed data from various studies that included measurements of the burdens of $^{239,240}Pu$ in autopsy samples of livers, lungs, and bone from people exposed to fallout Pu. Table 4 summarizes the geometric standard deviations of the tissue analyses. Most of the researchers found that burdens of $^{239,240}Pu$ in organs were lognormally distributed, with GSD values ranging from 1.48 to 2.66. We have chosen a GSD value of 2 to represent the variability in our dose-conversion factors for those organs, as this represents an intermediate value of the GSDs listed in Table 4. Because the $^{239,240}Pu$ burdens in organs are unlikely to be independent, we must also quantify correlations between the doses estimated for the different organs. Bunzl and Kracke (1983), for example, reported positive correlations between $^{239,240}Pu$ in 30 autopsy samples of livers and lungs (based on the log-transformed burdens) from individuals who lived in Germany. However, neither they nor Takizawa *et al.* (1987) found a statistically significant correlation between age and burden. To explore further the relationship between the $^{239,240}Pu$ burdens in lungs, liver, and bone, we calculated the linear correlation coefficients between $^{239,240}Pu$ burdens in autopsied organs from 28 individuals who had been exposed to $^{239,240}Pu$ fallout in Great Britain (Popplewell *et al.*, 1985). The linear correlation coefficient between the log-transformed $^{239,240}Pu$ burdens in lung and liver was 0.88, between lung and bone (femur) it was 0.77, and between bone and liver it was 0.81. We will use these correlations when generating correlated random variables representing the $\bar{F}_{i,in}$ and $\bar{F}_{i,out}$ parameters in the Monte-Carlo simulations.

Table 4. Summary of the geometric standard deviations of the $^{239,240}Pu$ burdens (*i.e.*, activity/unit mass of tissue) in autopsy samples of livers, lungs, and bone tissue analyzed by various researchers.

Organ	Sample size	Geometric standard deviation	Ref.
Liver	171 (male)	2.03	Griffith and Guilmette (1991)
Liver	139 (female)	1.91	Griffith and Guilmette (1991)
Liver	10	1.93	Kawamura and Tanaka (1983)
Liver	59	2.5	Takizawa *et al.* (1987)
Liver	10[a]	2.5	Singh *et al.* (1983)
Liver	10[b]	2.3	Singh *et al.* (1983)
Liver	28	2.04	Popplewell *et al.* (1985)
Lung	10[a]	1.5	Singh *et al.* (1983)
Lung	10[b]	1.9	Singh *et al.* (1983)
Lung	10	1.48	Kawamura and Tanaka (1983)
Lung	66	2.1	Takizawa *et al.* (1987)
Lung	28	2.66	Popplewell *et al.* (1985)
Lung	25	1.48	Irlweck *et al.* (1980)
Bone	7[a]	1.3	Singh *et al.* (1983)
Bone	12[b]	1.7	Singh *et al.* (1983)
Vertebrae	16	2.0	Takizawa *et al.* (1987)
Femur	28	1.63	Popplewell *et al.* (1985)
Vertebrae	6	2.30	Kawamura and Tanaka (1983)

[a] Autopsy samples from Washington, DC.

[b] Autopsy samples from Grand Junction, CO.

6.2. Organ-Specific Risk Factors

Several major reviews have been published recently concerning the effectiveness of various forms of radiation to induce cancer in humans; several of these studies are of direct interest here. Broad general reviews have been published by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1988), the U.S. National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiation (BEIR) (USNAS, 1990), and the International Commission on Radiological Protection (ICRP, 1991). Other reviews, which specifically consider the effects of alpha radiation, are of more specific interest. These include ICRP Publication 50 (ICRP, 1987), which considers the lung-cancer risk from exposure to radon decay products, and a prior report from the USNAS (1988), which also considered the risk from radon and other internally deposited alpha-emitting radionuclides, specifically including Pu.

It is well established that most injected Pu ends up in the skeleton and liver (ICRP, 1986), and the latest ICRP metabolic model contains the assumption that of the total amount of Pu

reaching the blood stream 50% will be retained by the skeleton and 30% by the liver. Of the remainder, most goes to the muscle and skin. The same situation pertains for inhalation, except that most of the Pu will be swallowed and pass through the gastrointestinal tract without being absorbed and that which is retained in pulmonary tissue and which will enter the blood has a removal half-time of 500 days (ICRP, 1986).

Thus, the tissues at risk seem clearly to be the lung, liver, and bone. This is borne out by many animal studies, with most cancers occurring in lung and bone. The types of bone-related cancers observed do not include leukemia, but are typically bone sarcomas. This is compatible with the observation that Pu does concentrate on the bone surfaces, and not in the marrow (USNAS, 1988). A group of 26 white male early Pu workers at Los Alamos with known significant inhalation exposure to Pu have now been followed up for 42 years (Voelz and Lawrence, 1991). Of the 26, four have died of cancer. Three died of lung cancer, and were heavy smokers. One died of an osteosarcoma that is most probably a result of his exposure to Pu.

The risk factor to the lung from exposure to alpha particles has received a great deal of attention lately because of concern about radon. The most recent review of data associated with radon exposures is that published by the ICRP (1991). On the basis of this review, the ICRP concluded that existing studies indicate a lifetime risk of $(1–4) \times 10^{-4}$ per Working Level Month (WLM) of exposure for members of an average population (the risk for non-smokers is less, perhaps by a factor of 4). Based on various dosimetry models of the lung and other assumptions, the calculated dose to the tracheobronchial region per unit of exposure varies from 4–13 mGy WLM^{-1} (ICRP, 1991). Using the geometric mean of both numbers, we calculate the risk factor to be 280×10^{-4} Gy^{-1}. Further, if we take the two ranges above to represent the 90% range, the geometric standard deviation (GSD) is calculated to be 1.74. This risk factor is broadly consistent with that recently published by Puskin (1992), who calculated a lifetime risk of 2.24×10^{-4} WLM^{-1} with a 90% range of 1.4×10^{-4} to 5.7×10^{-4} WLM^{-1}. From data in Puskin's paper we calculate a geometric standard deviation of 1.53, whereas the equivalent number for the range of $(1–4) \times 10^{-4}$ WLM^{-1} is 1.52.

There have not been as many attempts to derive a risk factor for bone surfaces. The UNSCEAR (1988) stated that the data would not support the derivation of a risk factor. The later report of the ICRP (1991), however, supported the use of a value from USNAS (1988, p. 208), which is derived from a life-table analysis of patients injected with ^{224}Ra. The value is $(133 \pm 36) \times 10^{-4}$ Gy^{-1}, which the ICRP corrected for a lethality factor of 0.7. To match our desire to use lognormal functions, this was converted to a geometric mean of 90×10^{-4} Gy^{-1} with a GSD of 1.31. Puskin *et al.* (1992) have recently pointed out that the original value of $(133 \pm 36) \times 10^{-4}$ Gy^{-1} was based on the average skeletal dose, not the dose to the bone surface. As the dose to the tissue at risk, the bone surface, is 7.5 times higher than the average skeletal dose, they argue that the risk factor should be divided by 7.5, if it is to be applied to the calculated dose to the bone surface. This concept has been cautiously endorsed by Bair and Sinclair (1992). This would result in a risk factor of about 10^{-3} lethalities Gy^{-1}. However, as mentioned above, there is one case of bone sarcoma amongst the 26 early Pu workers at Los Alamos (Voelz and Lawrence, 1991), and there seems to be little doubt that this rare cancer is a radiogenic sarcoma. From the Voelz and Lawrence paper we have estimated that the collective dose to the bone surface of the 26 workers was ~20 person–Gy. Thus, the risk factor calculated from this study would be $\sim 500 \times 10^{-4}$ Gy^{-1}, but the uncertainty (90% range) is from

26×10^{-4} Gy to 2120×10^{-4} Gy^{-1}. While the associated uncertainty is large, one would have a difficult time arguing that the actual risk from chronic exposure is near zero or far less than the value of 90×10^{-4} derived above. Also, the surviving 22 individuals are now elderly and therefore it is not anticipated that they will accumulate a greatly increased collective dose. At the same time, however, it is quite possible that yet more bone sarcomas will be found.

Lloyd *et al.* (1993) have recently published the results of a lifespan study of beagles injected with Pu. They extrapolate to man a risk factor of 548×10^{-4} Gy^{-1} for the average dose to the skeleton. This is equivalent to a risk factor of 70×10^{-4} Gy^{-1} for dose to the bone surfaces. Thus, we have elected to use the value of 90×10^{-4} Gy^{-1}, but it seems clear that the GSD value of 1.31 is too small. This is especially true with consideration of the reported wide variation in the amount of Pu that does end up in the skeleton (Kathren *et al.*, 1988). Thus, the GSD (1.74) for the lung-risk factor has been used as a more likely default value for the GSD for the bone-surface-risk factor.

Even fewer data are available for the induction of liver cancer by radiation. The only solid body of evidence concerns the many individuals who were injected with ^{232}Th in the form of "Thorotrast." Again, the ICRP (1991) refers to the analysis of lifetime excess risk done by the USNAS (1988). The calculated risk coefficient for three major studies varies from $(260–300) \times 10^{-4}$ Gy^{-1}. For our calculations, we will use the value of 280×10^{-4} Gy^{-1} and also use the GSD of 1.74 for the lung-risk factor as the default value for the GSD for the liver-risk factor.

7. Predicted Cancer Risks and Associated Uncertainties

The cancer risks for individuals inhabiting a suburban residence, living in a farm house, or working at a commercial facility at the shot locations were calculated for periods of 100, 50,000, and 100,000 y in order to evaluate the long-term risks associated with the possible loss of institutional controls at the NTS. As a cautionary note, we stress that our risk estimates for such lengthy time frames are predicated on the existence of exposure scenarios that are consistent with our previous characterizations (*i.e.*, activity patterns, housing properties, etc.). Results of the site-specific risk assessments are shown in Table 5 as the 5, 50, and 95% percentiles of incremental cancer risk from the cumulative probability distributions generated by the Monte-Carlo simulations. The resident farmer is shown to have the highest risk of the three exposure scenarios. Principal factors contributing to that risk are long tenure and higher concentrations of ^{239,240}Pu in indoor air due to the lack of indoor filtration afforded by a central heating/cooling unit. At the median risk level, the resident farmer's risk is more than a factor of three higher than the suburban resident and about a factor of ten higher than the worker at a commercial facility. The resident farmer's estimated risk, though, could be a factor of five to ten higher if additional assumptions were made regarding time spent outside adjacent to a farmhouse and an increased inhalation rate associated with physical labor. These results demonstrate that exposure scenarios incorporating lifestyle factors such as mobility and indoor/outdoor relationships for soil-derived contaminants can result in significant differences in the levels of cancer risk predicted for individuals whose lifestyles match the reference scenarios.

Table 5. Results of Monte-Carlo simulations of the cancer risks associated with three different land uses at three shot sites at the NTS.

Site	Year	Land use	Cumulative cancer risk 5%	50%	95%
Area 13	100	Residential	7×10^{-8}	7×10^{-7}	7×10^{-6}
		Farm	1×10^{-7}	2×10^{-6}	2×10^{-5}
		Commercial	2×10^{-8}	3×10^{-7}	3×10^{-6}
	10,000	Residential	8×10^{-9}	9×10^{-8}	1×10^{-6}
		Farm	2×10^{-8}	3×10^{-7}	3×10^{-6}
		Commercial	5×10^{-9}	5×10^{-8}	5×10^{-7}
	50,000	Residential	6×10^{-11}	6×10^{-10}	6×10^{-9}
		Farm	2×10^{-10}	2×10^{-9}	2×10^{-8}
		Commercial	9×10^{-11}	9×10^{-10}	3×10^{-9}
	100,000	Residential	3×10^{-12}	3×10^{-11}	7×10^{-11}
		Farm	4×10^{-12}	4×10^{-11}	8×10^{-11}
		Commercial	2×10^{-12}	2×10^{-11}	5×10^{-11}
Clean Slate II	100	Residential	2×10^{-7}	2×10^{-6}	2×10^{-5}
		Farm	5×10^{-7}	7×10^{-6}	5×10^{-5}
		Commercial	8×10^{-8}	1×10^{-6}	1×10^{-5}
	10,000	Residential	3×10^{-8}	3×10^{-7}	4×10^{-6}
		Farm	9×10^{-8}	9×10^{-7}	9×10^{-6}
		Commercial	2×10^{-8}	2×10^{-7}	2×10^{-6}
	50,000	Residential	2×10^{-10}	2×10^{-9}	2×10^{-8}
		Farm	8×10^{-10}	8×10^{-9}	6×10^{-8}
		Commercial	3×10^{-10}	3×10^{-9}	9×10^{-9}
	100,000	Residential	1×10^{-11}	1×10^{-10}	2×10^{-10}
		Farm	2×10^{-11}	2×10^{-10}	4×10^{-10}
		Commercial	8×10^{-12}	8×10^{-11}	2×10^{-10}
Area 11 D site	100	Residential	2×10^{-6}	2×10^{-5}	2×10^{-4}
		Farm	4×10^{-6}	6×10^{-5}	5×10^{-4}
		Commercial	6×10^{-7}	8×10^{-6}	9×10^{-5}
	10,000	Residential	2×10^{-7}	3×10^{-6}	3×10^{-5}
		Farm	7×10^{-7}	8×10^{-6}	8×10^{-5}
		Commercial	1×10^{-7}	1×10^{-6}	1×10^{-5}
	50,000	Residential	2×10^{-9}	2×10^{-8}	2×10^{-7}
		Farm	1×10^{-8}	1×10^{-7}	5×10^{-7}
		Commercial	3×10^{-9}	3×10^{-8}	8×10^{-8}
	100,000	Residential	1×10^{-10}	1×10^{-9}	2×10^{-9}
		Farm	3×10^{-10}	3×10^{-9}	5×10^{-9}
		Commercial	7×10^{-11}	7×10^{-10}	1×10^{-9}

A survey of the predicted cancer risks for the various shot sites given in Table 5 indicates that the highest risks are associated with Area 11 or Plutonium Valley. The resident farmer's cancer risk, for example, exceeds 10^{-4} at the 95th cumulative percentile level calculated for an assumed loss of institutional control at 100 years from the present. At 50,000 and 100,000 y in the future, the predicted cancer risks are all below 10^{-6}, which indicates that we have covered the principal time domain during which future adverse effects might occur.

To gain additional insights to the sources of uncertainty in the cancer risks, we analyzed how much of the variance in our risk estimates was derived from each of the following parameters: resuspension rates, concentrations of $^{239,240}Pu$ in soil, indoor/outdoor concentrations of $^{239,240}Pu$, mobility, and organ-specific risk factors for cancer. For this analysis, we completed a series of Monte-Carlo simulations in which only one parameter was allowed to vary randomly, while the other parameters were held constant at their mean value. Because the calculated risks are determined essentially from a multiplicative model, the sum of the individual log variances in the various input parameters should equal (approximately) the log variance in cancer risk. The fraction of total variance in estimated risk that is attributable to a given parameter is determined simply as the ratio of the log variance in cancer risk due solely to the variation in a given parameter and the total log variance in risk from all the random variations in the model parameters. In Figure 19 we show the contributions to total variance in the predicted cancer risks for the residential scenario for Area 13 at 100 y from the present. The principal source of variance in cancer risk is the mobility of residents (68.6%), followed by the exposure-weighted dose factors (17.1%), the ratio C_{in}/C_s used to relate levels of $^{239,240}Pu$ in outdoor soil to concentrations in indoor air (9.4%), and the cancer-risk factors (4.9%). Variation in the resuspension rate and the concentration of $^{239,240}Pu$ in soil contributed little to the total variance. However, at 50,000 y the resuspension rate becomes the principal source of variation in predicted risks. This is because the resuspension rate appears within an exponential term as the negative product of time (see Eq. 5), and so as time increases, the value of the resulting exponential term eventually becomes the primary determinant of the concentration of $^{239,240}Pu$ in air and hence cancer risk as well. Thus, for any given exposure scenario occurring over a person's lifetime, the resuspension rate will contribute little to overall risk, but when risks are predicted far into the future, the resuspension rate can become an important parameter.

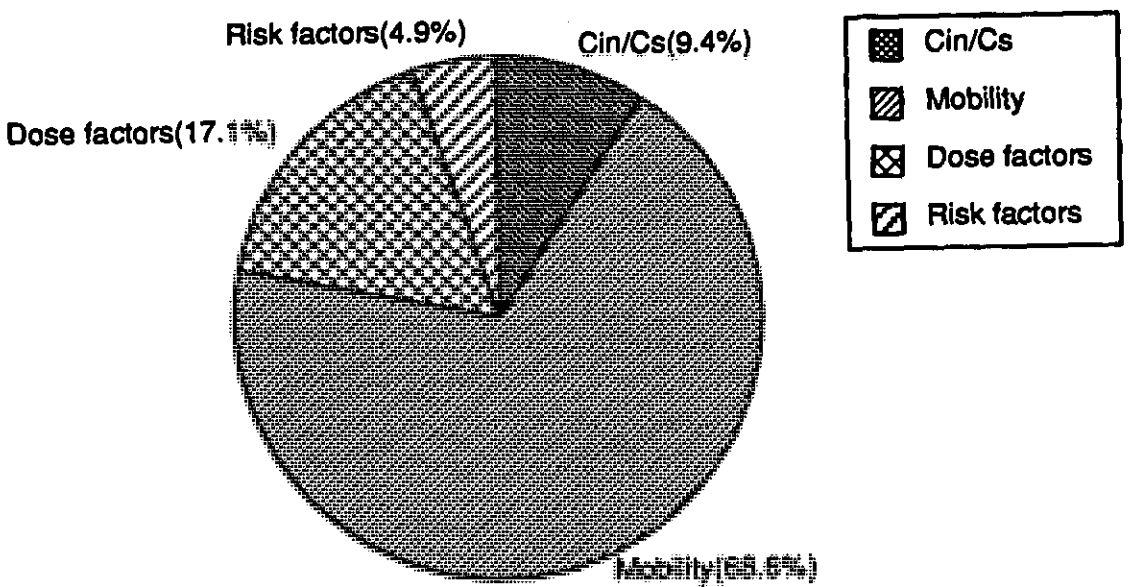

Figure 19. Sources of variance in the estimated cancer risks for the residential exposure scenario for Area 13 at 100 y.

Appendix A
Supporting Data

Table A–1. Summary of the residual concentrations and inventories of $^{239,240}Pu$ in soils at shot sites at the NTS (from Gilbert, 1977).

Site	Survey strata	Strata Area m^2	Mean Concentration $\mu Ci\ m^{-2}$	(±SE)	Estimated Inventory Ci	(±SE)
13	1	1,245,000	1.9 ±	0.34	2.4 ±	0.42
	2	2,547,000	5.8 ±	1.4	15 ±	3.6
	3	108,00	23 ±	4.3	2.5 ±	0.46
	4	74,000	54 ±	8.8	4.0 ±	0.65
	5	19,000	110 ±	19	2.1 ±	0.36
	6	24,000	820 ±	340	20 ±	8.2
	Total	4,017,000			46 ±	9.0
5	1	111,300	3.1 ±	0.66	0.35 ±	0.073
(GMX)	2	8,400	42 ±	9.1	0.35 ±	0.076
	3	800	270 ±	64	0.22 ±	0.051
	4	1,000	530 ±	150	0.53 ±	0.15
	5	3,800	4.6 ±	1.6	0.02 ±	0.006
	Total	125,300	1.5 ±	0.19	1.5 ±	0.19
Double	1	176,000	6.7 ±	3.5	1.2 ±	0.62
Tracks	2	1,600	350 ±	250	0.56 ±	0.40
	3	800	190 ±	59	0.15 ±	0.047
	4	600	4.6 ±	1.6	1.7 ±	0.60
	Total	179,000			3.6 ±	0.95
Clean	1	157,000	15 ±	7.0	2.4 ±	1.1
Slate I	2	10,000	64 ±	22	0.64 ±	0.22
	3	8,400	110 ±	35	0.92 ±	0.29
	4	1,700	120 ±	39	0.20 ±	0.066
	Total	177,100			4.2 ±	1.2
Clean	1	351,000	4.1 ±	1.3	1.4 ±	0.46
Slate II	2	82,300	73 ±	30	6.0 ±	2.5
	3	26,200	270 ±	99	7.1 ±	2.6
	4	11,000	260 ±	65	2.9 ±	0.72
	Total	470,500			17 ±	3.7
Clean	1	1,615,000	12 ±	2.2	19.4 ±	3.6
Slate III	2	61,000	58 ±	16	3.5 ±	0.98
	3	40,000	210 ±	63	8.4 ±	2.5
	4	16,000	370 ±	190	5.9 ±	3.0
	Total	1,732,000			37 ±	5.4

Table A–1. (Continued)

Site	Survey strata	Strata Area m^2	Mean Concentration $\mu Ci\ m^{-2}$	(±SE)	Estimated Inventory Ci	(±SE)
11 B Site	2	8,200	30	± 18	0.25	± 0.15
	3	6,000	220	± 55	1.3	± 0.33
	4	3,300	1,400	± 300	4.6	± 0.99
	Total	17,500			6.2	± 1.1
11 C Site	2	16,400	34	± 22	0.56	± 0.36
	3	13,300	88	± 25	0.49	± 0.14
	4	3,500	1,400	± 390	4.9	± 1.4
	5	300	6,200	± 2,800	1.9	± 0.84
	Total	25,800			7.8	± 1.7
11 D Site	2	32,300	46	± 9.5	1.5	± 0.31
	3	13,300	220	± 86	2.9	± 1.1
	4	4,900	990	± 370	4.9	± 1.8
	5	2,900	2,700	± 840	7.8	± 2.4
	Total	53,400			17.1	± 3.2
CD Overlap	6	62,200	12	± 5.2	0.75	± 5.2

Table A-2. Summary of data on lead and dust in the indoor environment.

Location	Number of dwellings	Pb in dust $\mu g\ g^{-1}$	Loadings on floor/carpets $\mu g\ Pb\ m^{-2}$	$mg\ Dust\ m^{-2}$	References
Christchurch, NZ	65	515	423	804	Fergusson and Schroeder, 1985
	22	590	349	525	
Edinburgh, Scotland	10	381	52	136	Laxen *et al.*, 1988
Birmingham, UK	97	336	60	179	Davies *et al.*, 1990
Cincinnati, OH[a]					
Public	12	350	200	571	Clark *et al.*, 1985
Rehabilitated	46	623	370	594	
Private	33	1410	750	532	
Champaign-Urban, IL	12	500	440	870	Solomon and Hartford, 1976
Arnhem, The Netherlands	107	282 (coarse)	123	255	Diemel *et al.*, 1981
		957 (fine)			

[a] Excludes deteriorating and dilapidated housing

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