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Dose Reconstruction
Project for NIOSH

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ACRONYMS AND ABBREVIATIONS

AI	alveolar interstitial
AMAD	activity median aerodynamic diameter
Bq	becquerel
d	day
DOE	U.S. Department of Energy
DTPA	diethylene triamine pentaacetic acid
g	gram
GI	gastrointestinal
HRTM	Human Respiratory Tract Model
ICRP	International Commission on Radiological Protection
IMBA	Integrated Modules for Bioassay Assessment (computer program)
LANL	Los Alamos National Laboratory
LN_{TH}	lymph nodes, thoracic
min	minute
mL	milliliter
NIOSH	National Institute for Occupational Safety and Health
ppm	parts per million
RFP	Rocky Flats Plant
SRS	Savannah River Site
Sv	sievert
TIB	technical information bulletin
Type SS	Super S absorption type
U.S.C.	United States Code
USTUR	United States Transuranium and Uranium Registry
yr	year
µm	micrometer

1.0 INTRODUCTION

Technical information bulletins (TIBs) are not official determinations made by the National Institute for Occupational Safety and Health (NIOSH) but are rather general working documents that provide historic background information and guidance concerning the preparation of dose reconstructions at particular sites or categories of sites. They will be revised in the event additional relevant information is obtained about the affected site(s). TIBs may be used to assist NIOSH staff in the completion of individual dose reconstructions.

In this document, the word "facility" is used as a general term for an area, building, or group of buildings that served a specific purpose at a site. It does not necessarily connote an "atomic weapons employer facility" or a "Department of Energy (DOE) facility" as defined in the Energy Employees Occupational Illness Compensation Program Act of 2000 [42 U.S.C. § 7384l(5) and (12)].

2.0 PURPOSE

The purpose of this TIB is to provide a method for calculating a best estimate (for the purposes of this project) of the annual organ doses for intakes of plutonium that are retained in the lung longer than predicted by the normal absorption Type S model and to describe the conditions for applicability of this method.

Attributions and annotations, indicated by bracketed callouts and used to identify the source, justification, or clarification of the associated information, are presented in Section 6.0.

3.0 BACKGROUND

A body of evidence from animal studies and accidental human intakes has come forth in, approximately, the last 30 years indicating that the lung can retain inhaled plutonium oxides for a very long time. In recognition of this, in 1994 the International Commission on Radiological Protection (ICRP) increased the retention time of insoluble (Type S) plutonium in the ICRP 66 Human Respiratory Tract Model (HRTM) in relation to the retention predicted by the ICRP 30 respiratory tract model (ICRP 1979, 1994, 1997). Nevertheless, a handful of accidental intakes of plutonium oxides at the DOE Rocky Flats Plant (RFP) (Mann 1967), Hanford Site (Carbaugh 1991,2001, Bihl et al 1988), Los Alamos National Laboratory (LANL) (Filipy 2004, James 2005), and Savannah River Site (SRS) (Carbaugh 2001) have exhibited long-term retention of plutonium in the lung exceeding that predicted by the standard Type S model. Recent autopsies on workers exposed to plutonium at the Mayak Production Association (Mayak) in Russia revealed a similar effect. These cases are discussed in Attachment A.

Because the cases discussed in Attachment A are from occupational human intakes rather than controlled animal experiments, information needed to define the circumstances that lead to retention of plutonium in the lung exceeding the Type S model is insufficient [1]. Indeed, the scientific community lacks consensus about whether this phenomenon truly represents another type of material with different lung absorption parameters, a degradation of the anatomical or physiological processes that remove particles from the lung because of damage from smoking or other toxic materials or from the plutonium alpha radiation itself, or is a demonstration of extreme but natural individual human variability in these processes in a few workers [2]. However, it is clear that, with the depletion of the fast-removal components, the rate of removal of plutonium from the lung is slower than that predicted by Type S material for some people under some conditions; as a consequence, the total dose to an organ accumulated over many years is greater. This phenomenon has been popularly referred to as "Type Super S" (or "Type SS" for short), although it is not established that it necessarily is caused only

by slower absorption of the plutonium into the blood (Carbaugh 2003). ICRP publication 66 does allow for the development of material-specific absorption parameters if sufficient information exists. While the absorption parameters in ICRP 66 are controlled by chemical solubility and are thus dependent on chemical form, mechanical clearance from the lungs is considered to be independent of chemical form. In the course of evaluating design cases, it was observed that even when the absorption parameters were set to very long clearance times, the mechanical clearance from the lungs alone was too fast to account for the slow lung clearance observed in the design cases.

As discussed in Attachments A and B, the correction factors in this TIB were developed using nine cases from Rocky Flats and one case from Hanford that had well-defined intakes and exhibited long lung retention times. Individual lung clearance parameters, as well as absorption parameters, were modified for each case in order to match lung counts and urinalyses performed on these individuals. These individual adjustments in themselves are not considered to be appropriate (either as averages or as a distribution of ranges) for application to the general population. By choosing the worst case clearances (i.e., the ones with the largest deviation from Type S), a bounding absorption type can be defined, which is applied to all cases where the default inhalation exposure is to Type SS plutonium.

Therefore, this TIB does not propose a new class of material for general modeling purposes or propose a new variation of the lung model. Rather, to account for the increased organ doses, the TIB analysis developed empirical “dose adjustment factors” from selected cases from RFP and Hanford that exhibited Type SS behavior following intakes of ^{239}Pu mixtures. For intakes calculated from urinary excretion data, a bounding analysis is implemented as an intake adjustment factor rather than a defined change in ICRP model parameters. The basis for this intake adjustment factor is given in Attachment C.

4.0 METHOD

4.1 APPLICATION OF ADJUSTMENT FACTORS

The standard approach adopted in this TIB is to first calculate doses to the organs of interest by applying the standard Type S model to the available bioassay data or air monitoring data. Then, one or more adjustment factors are applied to this dose in order to account for the longer retention of Type SS material in the lungs and, in the case of urine bioassay data, the lower urinary excretion per unit intake of Type SS material compared to Type S material.

Adjustments to both monitored individuals and to coworker data sets are addressed. In general, adjustments made to the *intake* activity are based on dates associated with the bioassay data used to determine the intake rates while adjustments to *doses*, applied to account for the longer retention in the lung of Type SS, are based on the intake periods. In the case of coworker data, adjustment factors are applied independently to each intake period. For example, if four different intake rates are applied throughout an individual’s employment period, four separate sets of intake and dose adjustments would be made.

4.1.1 Doses Calculated from Air Monitoring Data

4.1.1.1 Lung Dose

In cases where the intake is derived from air monitoring, the intake is based on direct measurements. For Type SS material, the annual dose to the lung (including the thoracic lymph nodes) will be underestimated if one assumes a Type S model because of the longer retention time. Therefore, annual lung doses calculated with the Type S model are multiplied by dose adjustment factors. These

factors are given in Attachment D for each year from 1 to 65 for 46 different intake scenarios. The scenarios are based on the period of intake, specifically acute and chronic intake periods from 1 to 65 years in 1-year intervals [3]. Because the dose adjustment factors decrease as the chronic exposure period increases, for chronic intakes for partial years, dose reconstructors should truncate the partial year and use the dose adjustment factor table for the full year; for instance, if the intake period is 4.5 years, use the dose adjustment factors for a 4-year chronic intake [4].

For example, assume a person had a 5-year chronic intake of ^{239}Pu based on air monitoring results and the annual Type SS lung doses to the end of year 10 are needed. First, the Type S lung equivalent doses $H_s(t)$ are calculated for each year based on standard Type S models and methods. These doses are given in column 2 of Table 4-1. The Type SS lung dose is obtained by multiplying the Type S dose by the dose adjustment factors for a 5-year chronic intake from Table D-1 (column 3 of Table 4-1). The Type SS lung doses for each year are given in column 4 of Table 4-1.

Table 4-1. Type S and Type SS lung doses after a 5-year chronic intake of ^{239}Pu.

Year	Type S lung dose (rem)	Dose adjustment factor	Type SS lung dose (rem)
1	29.8	1.6	47.7
2	39.0	1.9	74.1
3	43.5	2.1	91.4
4	46.8	2.4	112.3
5	49.4	2.6	128.4
6	21.2	3.5	74.2
7	13.6	4.5	61.1
8	10.2	5.7	58.3
9	7.9	6.9	54.7
10	6.3	8.2	51.4

The procedure and resulting dose would be the same for any chronic intake period ≥5 years and <6 years because the dose reconstructor truncated the intake period before looking up the dose adjustment factor in Attachment D.

4.1.1.2 Extra-Thoracic Dose

The extra-thoracic retention model is assumed to be the same for both Type S and Type SS material [5]. Therefore, for a given intake, the dose to the extra-thoracic organs (including the lymph nodes) is assumed to be the same for both solubility types. Because the intakes calculated from air monitoring data are the same for both solubility types, doses to this region are calculated assuming that the intake is Type S material [6].

4.1.1.3 GI Tract

For a given intake, the doses to the GI tract organs are assumed to be the same for Type S and Type SS material. Therefore, doses to this region are calculated assuming that the intake is Type S material [7].

4.1.1.4 Systemic Organs

For a given intake, the dose to the systemic organs from a Type Super S material will be less than that from Types M or S because the material will be retained in the lungs longer; the material will be

transported to the systemic organs more slowly. Therefore, the assumption that the dose from Type Super S is equal to that from Type S is favorable to the claimant.

4.1.2 Intakes Calculated from Chest Count Data

4.1.2.1 Lung Dose

To calculate Type SS lung doses from chest count measurements, the dose to the lung is first calculated assuming that Type S material was inhaled. This dose is then adjusted upward with the factors given in Appendix D. However, the application of the adjustment factor will result in an implied Type SS lung content that is inconsistent with the original chest count. To make the observed and predicted chest counts agree, the Type SS lung dose must be adjusted downward by applying the adjustment factor for the year of the chest count used to determine the intake [8]. This also applies to coworker data, where the chest counts used for the derivation of the given intake rate must agree with the predicted values.

This process is best illustrated through example, as shown below.

4.1.2.1.1 Monitored Individual

Given a single measured chest content of 34.7 nCi of ^{239}Pu on day 1,825 (the end of the chronic intake period), an intake rate of 1000 pCi/d is calculated for an intake of Type S material. This yields the annual lung doses in the second column of Table 4-1. The Type SS doses for this intake rate are then obtained by multiplying the Type S dose for a given year by the adjustment factor in Attachment D. For this example, in Table D-1 the “Chronic 5 yr” column would be used. For example, year 5 is calculated by multiplying the 49.4 rem Type S lung dose by 2.6 to obtain a Type SS lung dose of 128.4 rem. However, this adjustment creates an inconsistency; specifically, if one assumes that the lung dose for year 5 is proportional to the measured chest content in year 5, the Type SS dose adjustment implies that the chest content is (34.7 nCi)(2.6) = 90.2 nCi, which is inconsistent with what was measured.

To make the measured and implied lung contents agree (i.e., to make the intake amounts agree), all of the Type SS lung doses must be adjusted downward by the dose adjustment factor for the year of the chest count. In the current example, all Type SS doses are divided by a factor of 2.6, which is shown in Table 4-2. Because this is essentially an intake adjustment factor, it could also be applied directly to the Type S intake prior to dose calculation, thus simplifying the calculations. The year of the chest count should be rounded down to the nearest whole year when selecting the factor from Table D-1. Note that this adjustment makes the Type S and Type SS lung doses agree at the time of the chest count (year 5), which means that the measured and implied lung contents will also agree.

Table 4-2. Type S and Type SS lung doses after a 5-year chronic intake of ^{239}Pu calculated from a chest count.

Year	Type S lung dose (rem)	Dose adjustment factor	Unadjusted Type SS lung dose (rem)	Chest count adjustment factor	Adjusted Type SS lung dose (rem)
1	29.8	1.6	47.7	2.6	18.4
2	39.0	1.9	74.1	2.6	28.5
3	43.5	2.1	91.4	2.6	35.2
4	46.8	2.4	112.3	2.6	43.2
5	49.4	2.6	128.4	2.6	49.4
6	21.2	3.5	74.2	2.6	28.5
7	13.6	4.5	61.1	2.6	23.5

Year	Type S lung dose (rem)	Dose adjustment factor	Unadjusted Type SS lung dose (rem)	Chest count adjustment factor	Adjusted Type SS lung dose (rem)
8	10.2	5.7	58.3	2.6	22.4
9	7.9	6.9	54.7	2.6	21.0
10	6.3	8.2	51.4	2.6	19.8

Alternatively, applying the intake adjustment factor first to simplify the calculations, the adjusted Type S intake is:

$$Adjusted\ Type\ S\ Intake\ Rate = \frac{Type\ S\ Intake\ Rate}{Intake\ Adjustment\ Factor} = \frac{1000\ {}^{pCi}\!/_{d}}{2.6} = 385\ {}^{pCi}\!/_{d}$$

Lung doses are then calculated for this intake rate (385 pCi/d); the annual doses from this intake are in the second column of Table 4-3.

Table 4-3. Type SS lung doses after a 5-year chronic intake of ^{239}Pu calculated from a chest count (simplified method).

Year	Lung dose from adjusted Type S intake (rem)	Dose adjustment factor	Type SS lung dose (rem)
1	11.5	1.6	18.4
2	15.0	1.9	28.5
3	16.8	2.1	35.2
4	18.0	2.4	43.2
5	19.0	2.6	49.4
6	8.2	3.5	28.7
7	5.2	4.5	23.5
8	3.9	5.7	22.5
9	3.1	6.9	21.1
10	2.4	8.2	19.8

4.1.2.1.2 Unmonitored Individual (Coworker Data)

For coworker data, the *intake adjustment* factor is dependent only on the period used for calculating the coworker intake rate; that is, it is independent of the worker to whom the coworker intakes are assigned. For example, given a set of site chest count data from 1974 through 1980 (7 years), a coworker intake rate of 1000 pCi/d is calculated for an intake of Type S material. To determine the coworker intake rate for Type SS material, the intake rate adjustment factor (called the "chest count adjustment factor" in this particular instance) must first be applied to the Type S intake rate. This is a downward adjustment because Type SS material remains in the lungs longer than Type S material. For this example, the last chest count used to fit the data occurred in year 7, so a factor of 3.1 is selected from Table D-1. This factor is applied to the Type S intake rate of 1000 pCi/d to obtain a Type SS intake rate of 323 pCi/d:

$$Adjusted\ Type\ S\ Intake\ Rate = \frac{Type\ S\ Intake\ Rate}{Intake\ Adjustment\ Factor\ (year 7)} = \frac{1000\ {}^{pCi}\!/_{d}}{3.1} = 323\ {}^{pCi}\!/_{d}$$

The annual lung doses, based on Type S, from this intake rate are shown in column 2 of Table 4-4. The *dose adjustment* factor will depend on the intake period assigned to the individual. To continue with the above example, given an unmonitored individual who worked from Jan. 1, 1975 to Dec. 31, 1979, he would have had a 5-year chronic intake and the dose adjustment factors from the "Chronic 5 year" column of Table D-1 would apply. These adjustment factors are shown in the "Dose adjustment factor" column of Table 4-4. The final coworker doses to be assigned to the unmonitored individual are shown in the last column of the table.

Table 4-4. Type SS lung doses after a 5-year chronic intake of ^{239}Pu calculated from 7 years of coworker chest count data.

Year	Lung dose from adjusted Type S intake (rem)	Dose adjustment factor	Type SS lung dose (rem)
1	9.6	1.6	15.4
2	12.6	1.9	23.9
3	14.0	2.1	29.5
4	15.1	2.4	36.2
5	15.9	2.6	41.4
6	6.8	3.5	23.9
7	4.4	4.5	19.7
8	3.3	5.7	18.8
9	2.5	6.9	17.6
10	2.0	8.2	16.6

Note that when a worker's employment spans several coworker periods, each period is addressed separately. Adjustment factors are based only on the employment within the individual coworker periods. For instance, if the worker in the above example worked until 1985, the dose adjustment for the example period would be based on 6 years (1975 to 1980, the year of the last bioassay result used for the coworker intake rate).

4.1.2.2 Extra-Thoracic Dose

The extra-thoracic retention model is assumed to be the same for both Type S and Type SS material [9]. This means that, for a given intake, the dose to the extra-thoracic organs is assumed to be the same for both solubility types. For a given chest count, the intake calculated with the Type S model will be larger than with the Type SS model because the Type S model predicts faster clearance from the thoracic region. Therefore, doses to the extra-thoracic organs should be calculated with the Type S model with no adjustments [10].

4.1.2.3 GI Tract

The GI tract retention model is assumed to be the same for both Type S and Type SS material. This means that, for a given intake, the dose to the GI-tract organs is assumed to be the same for both solubility types. For a given chest count, the intake calculated with the Type S model will be larger than with the Type SS model because the Type S model predicts faster clearance from the thoracic region. Therefore, doses to the GI-tract organs should be calculated with the Type S model with no adjustments [11].

4.1.2.4 Systemic Organs

Doses to systemic organs should be based on urine bioassay data when possible. If it is necessary to calculate these doses from chest count data, the Type S model should be used with no adjustments [12].

4.1.3 Doses Based on Urinalysis Data

4.1.3.1 Lung and Thoracic Lymph Node Dose

To calculate Type SS lung doses from urinary excretion measurements, the annual dose to the lung for the years of interest is first calculated from urinary excretion data using the standard Type S model. The urinary excretion data can consist of measured results and/or results less than the reporting level. The annual lung doses calculated with the Type S model are then multiplied by the dose adjustment factors in Attachment D. This adjustment accounts for the longer retention of Type SS material in the lung, but it does not address the lower urinary excretion rate expected from Type SS material.

To account for the lower excretion rate expected from Type SS material, the approach adopted here is to apply a single bounding correction factor of 4 (which is derived in Attachment C) to adjust the intake of Type S material upward to an intake of Type SS material. This “intake adjustment” increases the thoracic doses determined from urinalysis with the Type S model by a factor of 4 and is applied in addition to the Attachment D adjustment factors that account for increased retention in the lung.

For example, referring back to Table 4-1, assuming that the Type S chronic intake rate had been calculated based on urinary excretion, the Type SS lung doses are multiplied by the intake adjustment factor of 4 to obtain the final Type SS lung doses (see Table 4-5).

Table 4-5. Type S and Type SS lung doses after a 5-year chronic intake of ^{239}Pu calculated from urinary excretion data.

Year	Type S lung dose (rem)	Dose adjustment factor	Unadjusted Type SS lung dose (rem)	Intake adjustment factor	Adjusted Type SS lung dose (rem)
1	29.8	1.6	47.7	4	190.9
2	39.0	1.9	74.1	4	296.3
3	43.5	2.1	91.4	4	365.6
4	46.8	2.4	112.3	4	449.4
5	49.4	2.6	128.4	4	513.6
6	21.2	3.5	74.2	4	296.8
7	13.6	4.5	61.1	4	244.3
8	10.2	5.7	58.3	4	233.0
9	7.9	6.9	54.7	4	218.6
10	6.3	8.2	51.4	4	205.5

The procedure and resulting dose would be the same for any chronic intake period ≥5 years and <6 years because the dose reconstructor truncated the intake period before looking up the dose adjustment factor in Attachment D.

4.1.3.2 Extra-Thoracic Dose

Extra-thoracic doses should be calculated from urine bioassay data using the Type S model and then multiplied by a factor of 4 to account for the lower excretion rate of Type SS material compared to Type S material [13].

For coworker data, the intake adjustment factor of 4 is applied first and is included in the coworker summary (in the site-specific OTIB or TBD).

4.1.3.3 GI Tract

GI tract doses should be calculated from urine bioassay data using the Type S model and then multiplied by a factor of 4 to account for the lower excretion rate of Type SS material compared to Type S material [14].

For coworker data, the intake adjustment factor of 4 is applied first and is included in the coworker summary (in the site-specific OTIB or TBD).

4.1.3.4 Systemic Organs

Type SS material is absorbed into the blood stream at a slower rate than Type S material, which results in lower levels of material in the systemic organs and in the urine. Assuming that the doses to systemic organs are roughly proportional to the urinary excretion rate, organ doses determined from urine data are the same for Type S and Type SS materials during the period of time that urine data are available. However, for the period of time after the last urinalysis is available, the Type SS model would predict a much slower decrease in urine due to the continuing input to the bloodstream from the material contained in the lungs. Therefore, the predicted integrated urine content (and hence, systemic organ dose) must be adjusted after the time of the last urine bioassay measurement [15].

4.1.3.4.1 Monitored Individual

Consider the annual doses to the liver from a 5-year chronic intake of ^{239}Pu calculated from urine data available for the first 5 years. As shown in Table 4-6, all liver doses from year 6 though year 10 are multiplied by the intake adjustment factor of 4 whereas the doses during years 1 through 5 are not.

Table 4-6. Type S and Type SS liver doses after a 5-year chronic intake of ^{239}Pu calculated from urinary excretion data.

Year	Type S liver dose (rem)	Intake adjustment factor	Type SS liver dose (rem)
1	0.05	1	0.05
2	0.23	1	0.23
3	0.52	1	0.52
4	0.88	1	0.88
5	1.30	1	1.30
6	1.72	4	6.87
7	2.03	4	8.12
8	2.27	4	9.08
9	2.46	4	9.83
10	2.59	4	10.36

In summary, the annual doses to systemic organs should be determined from urine data using the Type S assumption. Annual doses received during the period for which urine data are available should not be adjusted. Annual doses received after the year of the last urine sample used in the determination should be multiplied by a factor of 4.

4.1.3.4.2 Unmonitored Individual (Coworker Data)

Because the adjustment is based on intake rather than dose, the factor is applied to the time following the period used to determine the coworker intake rate rather than the worker's exposure period. For example, given a set of site urinalysis data from 1974 through 1980 (7 years), and an individual who worked from Jan. 1, 1975 to Dec. 31, 1979 (5 years), the adjustment would be applied beginning in year 8.

Table 4-7. Type S and Type SS liver doses after a 5-year chronic intake of ^{239}Pu calculated from urinary excretion data.

Year	Type S liver dose (rem)	Intake adjustment factor	Type SS liver dose (rem)
1	0.05	1	0.05
2	0.23	1	0.23
3	0.52	1	0.52
4	0.88	1	0.88
5	1.30	1	1.30
6	1.72	1	1.72
7	2.03	1	2.03
8	2.27	4	9.08
9	2.46	4	9.83
10	2.59	4	10.36

4.1.4 Doses Based on Fecal Bioassay Data

Fecal samples collected less than 2 months after an acute inhalation intake or less than 2 months after the end of a constant chronic intake (including those collected during the chronic intake) should be evaluated with the standard Type S model. Once the intake is calculated, the adjustments given for evaluating doses from direct measurements (Section 4.1.1, Doses Calculated from Air Monitoring Data) should be followed.

Fecal samples collected more than 2 months after an acute intake or end of a chronic intake should be modeled as if they were urine samples (Section 4.1.3). For example, if the 24-hour fecal sample contained 1 dpm it should be modeled as if it were a 24-hour urine sample that contained 1 dpm. Once the dose to the organ/tissue of interest is calculated, it is adjusted upward by a factor of 3.

The technical basis for these procedures is given in Attachment E.

4.2 PARTICLE SIZE ADJUSTMENTS FOR RFP PLUTONIUM FIRES

Dose adjustment factors are based on the assumption of a 5-µm activity median aerodynamic diameter (AMAD) particle size (ICRP 1994). For the RFP plutonium fires, a particle size of 1 µm AMAD is recommended (ORAUT 2007). The dose adjustment factors underestimate the annual lung doses by a factor of 2.6 for 1 µm AMAD aerosols because the deposition in the alveolar interstitial (AI) region of the lung is 2.6 times greater for 1 µm aerosols than 5 µm aerosols per unit intake. For energy employees involved in a plutonium fire at RFP (or any time the dose reconstructor deems use

of a 1-µm AMAD particle size appropriate), the dose adjustment factors in Attachment D must be multiplied by an additional factor of 2.6. Note that when the assessment is based on chest counts, the adjustment for particle size is not necessary because the lung deposition is directly measured, i.e., the dose would be adjusted upwards by this factor, but in order to get agreement in the Types S and SS predicted chest burdens, it would then need to be adjusted down by the same factor.

4.3 SUMMARY OF DOSE AND INTAKE ADJUSTMENT FACTORS

A summary of the adjustments discussed above is provided in Table 4-8.

Table 4-8. Summary of Type SS adjustments.

	Lung counts	Air concentrations	Urinalysis
Lungs	Table D (normalized to last chest count)	Table D	Factor of 4 followed by Table D adjustment
Extra-thoracic	None	None	Factor of 4
GI tract	No adjustment	None	Factor of 4
Systemic organs	None	None	Prior to last urine sample: none Post last urine sample: factor of 4

Note: For claims involved in high-fired events (e.g., the RFP fires), a particle size adjustment (as described in Section 4.2) of 2.6 should be included.

5.0 APPLICABILITY AND LIMITATIONS

There are a number of restrictions on when and how the adjustment factors can be used:

- Adjustment factors apply only to doses resulting from the intake of plutonium oxide; however, it is favorable to the claimant to apply them if the intake material is unknown and plutonium oxide is a possibility. Considering the uncertainty in the nature of the material, long-term (years) air oxidation of formerly Type M plutonium can be considered to apply [16].

- Adjustment factors apply only to doses resulting from intakes of plutonium for which the activity isotopic ratio of $^{239+240}$Pu to ^{238}Pu is greater than 1. This restriction is based on the observed behavior of relatively pure ^{238}Pu, which tends to be more soluble than ^{239}Pu (Guilmette 1994, Hickman 1995, James 2003). When this condition is met, SS behavior applies to all isotopes in the plutonium mixture.

- Adjustment factors apply to the dose from ^{241}Am in the mixture when the activity ratio of $^{239+240}$Pu to ^{241}Am is greater than 1 [17].

- Adjustment factors do not apply to situations where the plutonium is a minor constituent by mass in another matrix, such as in recycled uranium [18].

- Adjustment factors may be applied to chest count data (using the adjustment discussed in Section 4) except when there are multiple positive chest count results that occur in more than one year. The case should be evaluated to determine if a best fit to the actual data should be performed in such cases [19].

- Because the highest of the various dose adjustment factors was used for Attachment C, no additional uncertainty should be applied to the lung dose calculation; i.e., use the same uncertainty distribution as was applicable to the Type S dose calculation [20].

- The methods described in this TIB can be applied to doses from coworker studies for sites where Type SS absorption is appropriate, but only Type S intakes were assessed. The types of adjustments made to the Type S intakes and doses should be based on the method used to create the coworker study (i.e., whether the intakes are based on urinalysis or chest counts) [21].

The reasons for the restrictions on the use of dose adjustment factors are discussed in detail in Attachments A and B.

6.0 ATTRIBUTIONS AND ANNOTATIONS

Where appropriate in this document, bracketed callouts have been inserted to indicate information, conclusions, and recommendations provided to assist in the process of worker dose reconstruction. These callouts are listed here in the Attributions and Annotations section, with information to identify the source and justification for each associated item. Conventional References, which are provided in the next section of this document, link data, quotations, and other information to documents available for review on the Project's Site Research Database.

[1] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
There are documented cases (see references in first paragraph of Section 3.0) of occupational exposure to plutonium where the plutonium is retained in the lung for longer periods of time than expected for Type S plutonium.

[2] Bihl, Donald E. ORAU Team. Principal Health Physicist. January 2007.
Based on personal discussions on this topic with A. C. James, R. J. Guilmette, F. F. Hahn, W. J. Bair, and others. Also based on peer review on an article on this subject submitted to *Health Physics* that was rejected because of lack of consensus among the reviewers and authors on key issues of the model.

[3] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
The chronic intake periods and dose intervals were selected to provide reasonable increments of the dose factors for all intake scenarios while at the same time limiting the length of the tables.

[4] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
For example, the lung dose adjustment factor for year 7 following a 5-year chronic intake is 4.5 whereas the dose adjustment factor for year 7 following a 4-year chronic intake is 5.2. Thus, if the chronic intake period is 4.5 years, a dose that is favorable to the claimant is assessed by using the value for 4 years.

[5] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
There is no empirical information from the reviewed cases or in the open literature on which to base a modification.

[6] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
Because the doses to the ET region are the same for equal intakes of Type S plutonium and Type SS plutonium, there is no adjustment to be made for Type SS.

[7] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
The dose to the GI tract from an inhalation of plutonium is the result of plutonium that is deposited in and subsequently cleared from the respiratory tract. Because the inhaled Type SS plutonium is retained in the lung for a longer time than Type S material, less is transferred to the GI tract and hence the dose is lower than for an equal intake of Type S plutonium. However, because of uncertainties in the Type SS model parameters, Type S plutonium is recommended for calculating doses to the GI tract in order to be favorable to the claimant.

[8] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
The first factor to account for increased dose to the lung from the Type SS material is due to the longer retention time in the lungs, so for equal intakes of Types S and SS materials, the chest burden from Type SS will be larger than that from Type S at any given time. Because the Type SS adjustment factors are applied to intake calculations based on Type S material, this second factor is applied to make the predicted and measured chest burdens agree for the Type SS material.

[9] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
For example, the lung dose adjustment factor for year 7 following a 5-year chronic intake is 4.5 whereas the dose adjustment factor for year 7 following a 4-year chronic intake is 5.2. Thus, if the chronic intake period is 4.5 years, a dose that is favorable to the claimant is assessed by using the value for 4 years.

[10] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
There is no empirical information from the reviewed cases or in the open literature on which to base a modification.

[11] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
Because the doses to the ET region are the same for equal intakes of Type S plutonium and Type SS plutonium, there is no adjustment to be made for Type SS.

[12] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
The plutonium in the lung has not been taken up into the bloodstream and is therefore not yet available to be deposited in systemic organs and deliver dose. On the other hand, the plutonium in the urine is a good indicator of the levels of plutonium that has been in the bloodstream. For this reason, urine bioassay data are preferred for calculating dose to systemic organs. If the dose to a systemic organ needs to be calculated from the lung content, the more soluble plutonium forms will deliver the largest dose because they will leave the lungs more quickly and deposit in the systemic organs. Therefore, Type S is more favorable to the claimant than Type SS.

[13] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
Because a smaller fraction of a Type SS intake goes to urine, the intake calculated assuming a Type S intake will be too small by up to a factor of 4. To adjust for this, the intake calculated with the Type S model is adjusted upwards by a factor of 4.

[14] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
Because a smaller fraction of a Type SS intake goes to urine, the intake calculated assuming a Type S intake will be too small by up to a factor of 4. To adjust for this, the intake calculated with the Type S model is adjusted upwards by a factor of 4.

[15] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
The systemic dose is not adjusted upwards as long as there are urine bioassay data available because the adjustment would predict a urinary excretion rate that is larger than that actually observed. It is applied after the last bioassay because it is unknown as to whether it decreases at the rate predicted by Type S or by Type SS.

[16] La Bone. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
Plutonium oxide (especially the high-fired variety) is one of the most insoluble forms of plutonium (ICRP 1994, personal discussion with C.W. Sill) typically encountered in the workplace. However, it is not feasible to exclude the possibility that soluble forms of plutonium might become more insoluble over time (La Bone, T. R. and W. M. Findley 1999; J. C. Moody, G. N. Stradling, and A. R. Britcher 1994). Therefore, the TIB is assumed to apply if the form of the plutonium is not known. This is used as an additional possibility of material type; all possibilities are calculated and the type resulting in the largest dose is applied to be favorable to the claimant.

[17] Bihl, Donald E. ORAU Team. Principal Health Physicist. January 2007.
It is standard industry practice to assume the long-term retention of particles in the lung is related to the physical and chemical properties of the particle matrix. For example, if the particle is plutonium oxide with small quantities of a contaminant like ^{241}Am (which is normally much more soluble than plutonium oxide), the ^{241}Am is assumed to be trapped in the particle matrix and exhibit the same retention as the matrix. However, once the ^{241}Am becomes a major component of the particle, it tends to assume its own solubility rather than that of the matrix. Because the point at which this occurs is unknown, it was estimated that the ^{241}Am might behave independently when its mass is about 1% or more of the total mass of the matrix; when converted to activity the 1% mass criterion occurs approximately when the activity ratios of ^{241}Am and ^{239}Pu is equal, so for simplicity the 1:1 activity ratio was used.

[18] Bihl, Donald E. ORAU Team. Principal Health Physicist. January 2007.
It is standard industry practice to assume the long-term retention of particles in the lung is related to the physical and chemical properties of the particle matrix. For example, if the particle is plutonium oxide with small quantities of a contaminant like ^{241}Am (which is normally much more soluble than plutonium oxide), the ^{241}Am is assumed to be trapped in the particle matrix and exhibit the same retention as the matrix. However, once the ^{241}Am becomes a major component of the particle, it tends to assume its own solubility rather than that of the matrix. Because the point at which this occurs is unknown, it was estimated that the ^{241}Am might behave independently when its mass is about 1% or more of the total mass of the matrix; when converted to activity the 1% mass criterion occurs approximately when the activity ratios of ^{241}Am and ^{239}Pu is equal, so for simplicity the 1:1 activity ratio was used.

[19] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
In cases where there are numerous chest counts the preferred approach is to model the data directly rather than use the methods given in this TIB. This approach is preferred because data specific to the individual will provide the most accurate estimate of dose and the Rule prioritizes the use of personal information, when available, over all other information.

[20] Bihl, Donald E. ORAU Team. Principal Health Physicist. January 2007.
Rather than try to determine a distribution of adjustment factors among the various cases, the highest adjustment factor for the given scenario was chosen. Because the dose adjustment factor is the maximum, or upper bound, no additional uncertainty is required.

[21] Bihl, Donald E. ORAU Team. Principal Health Physicist. January 2007.
Coworker data are simply another method of assessing dose to an individual, so the same methods of assessment apply.

[22] Falk, Roger B. ORAU Team. Senior Life Scientist. January 2007.
The calculations in this section were performed by Roger Falk.

[23] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
These data are unpublished.

[24] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
In other words, the best model is assumed to be the model that predicts lung retention and urinary excretion that best agrees with that observed.

[25] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
The 5 µm AMAD is an ICRP 66 default value. The density of 11.5 g/mL is the value given in the CRC Handbook of Chemistry and Physics for plutonium oxide.

[26] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
Adjustment factors are applied to calculations based on Type S rather than the development of a new model.

[27] Brackett, Elizabeth M. ORAU Team. Principal Internal Dosimetrist. January 2007.
In order to develop adjustment factors, something needs to be calculated on which to apply these factors. The Type S model is used because it is closest to this longer-retained material.

[28] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
The point here is that the doses are being calculated with an empirical adjustment of the standard ICRP models and that these models are not being modified.

[29] LaBone, Thomas R. ORAU Team. Deputy Principal Internal Dosimetrist. January 2007.
Plutonium oxide (especially the high-fired variety) is one of the most insoluble forms of plutonium (ICRP 1994, personal discussion with C.W. Sill) typically encountered in the workplace. However, it is not feasible to exclude the possibility that soluble forms of plutonium might become more insoluble over time (La Bone, T. R. and W. M. Findley 1999; J. C. Moody, G. N. Stradling, and A. R. Britcher 1994). Therefore, the TIB is assumed to apply if the form of the plutonium is not known. This is used as an additional possibility of material type; all possibilities are calculated and the type resulting in the largest dose is applied to be favorable to the claimant.

[30] Allen, David E. NIOSH. Dose Reconstruction Team Leader. January 2007.
The calculations in this section were performed by David E. Allen.

[31] Falk, Roger B. and Bihl, Donald E. ORAU Team. Principal Health Physicists. January 2007.
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A1.0 CASES FROM THE ROCKY FLATS PLANT

The following paragraphs discuss nine cases from RFP [23]. For these cases, the standard Type S model is compared to a custom individual-specific model that modifies mechanical clearance parameters and dissolution parameters. The custom parameters were selected such that reasonable fits to the data were obtained and intakes calculated from urine and chest data were approximately equal [24]. For the custom fits, the particle size was 5 µm AMAD with a density of 11.5 g/mL for all RFP cases (and HAN-1 [25]). For the 1965 plutonium fire cases, custom fits were also determined for a particle size of 1 µm AMAD to assess the impact of varying the particle size. For all RFP cases, the initial activity of ^{241}Pu was assumed based on the general mixture of plutonium handled at Rocky Flats.

RFP cases 101, 207, 825, 872, 934, and 1400 were exposed to plutonium from a fire on October 15, 1965, in the coolant line of a lathe used to machine plutonium metal (Mann and Kirchner 1967). The

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fire started when a maintenance worker was trying to clear plutonium chips clogging the coolant line, first by using carbon tetrachloride to flush the line, and then by using a center punch with force to dislodge the chips. A spark from the center punch ignited the plutonium-carbon tetrachloride mixture, which caused a fire that quickly breached containment and created airborne contamination (particle size = 0.32-µm mass median diameter) that spread throughout Building 776/777. There was no air monitoring alarm system and the building fire alarm was not activated until approximately 15 min after the onset of the fire. The six cases discussed here had no significant previous plutonium intakes and no subsequent intakes. They have modern (post-1990) lung counts and plutonium urinalysis data. Cases 825, 872, and 934 had diethylene triamine pentaacetic acid (DTPA) chelation treatments; cases 101, 207, and 1400 did not.

RFP 101
RFP 101 was an assembler about 50 feet from the origin of the fire, but did not smell the smoke. He was notified of the fire, donned a respirator, and left the area. He did not receive DTPA treatment. Figure A-1 shows plutonium lung activity calculated from ^{241}Am lung count measurements and fitted in the Integrated Modules for Bioassay Assessment (IMBA) computer code for the standard Type S model and the custom fit for which the assessed intake based on lung data was approximately equal to the assessed intake based on urine data.

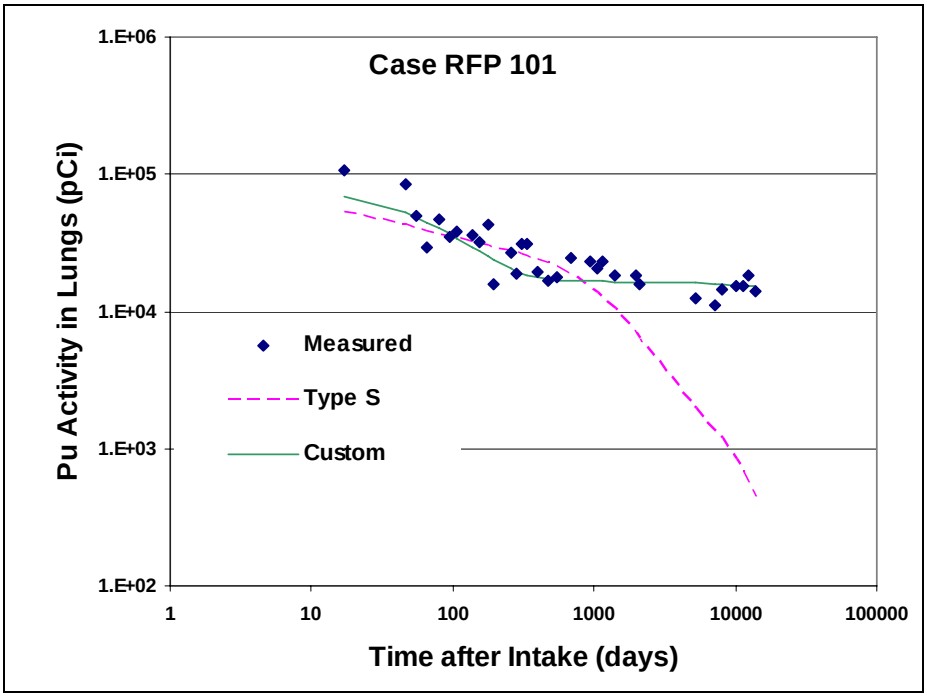

Figure A-1. Comparison of Type S and custom models for Case RFP 101.

RFP 207
RFP 207 was a production machinist in the building (temporarily assigned from depleted uranium operations). He was not near the fire and did not see or smell smoke. He evacuated the building

when notified but did not know why. Later, he was discovered to be contaminated. He did not receive DTPA treatment (see Figure A-2).

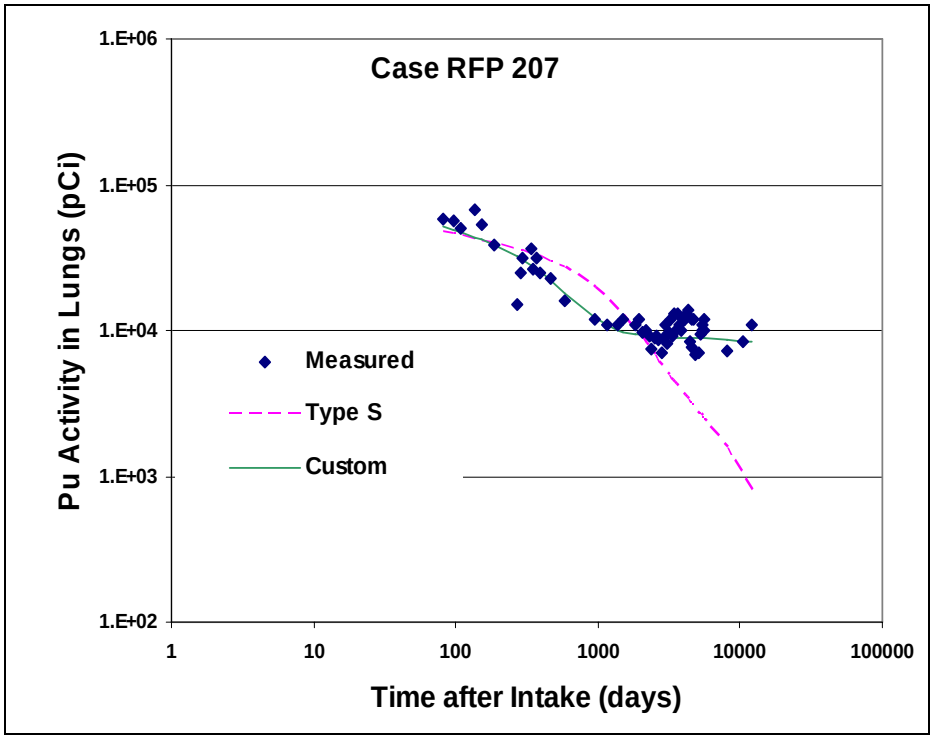

Figure A-2. Comparison of Type S and custom models for Case RFP 207.

RFP 825
RFP 825, a quality control engineer in an office adjacent to the metal production area, smelled the smoke but thought it was welding fumes. He left the office and unknowingly went toward the fire location. When he saw the fire, he donned a respirator and exited the area. Case RFP 825 was administered 4 g of DTPA, starting on October 18, 1965 (see Figure A-3).

RFP 872
RFP 872 was the maintenance supervisor in the vicinity. He smelled smoke for about 2 min, went to the fire, donned a respirator, and exited the area. He was administered 5 g of DTPA starting on October 15, 1965 (see Figure A-4).

RFP 934
RFP 934 was the maintenance worker at the origin of the fire. He was wearing a respirator at the onset of the fire. He was administered 5 g of DTPA starting on October 15, 1965 (see Figure A-5).

RFP 1400
RFP 1400 was an electrician apprentice at a desk in the production area. He did not receive DTPA treatment (see Figure A-6).

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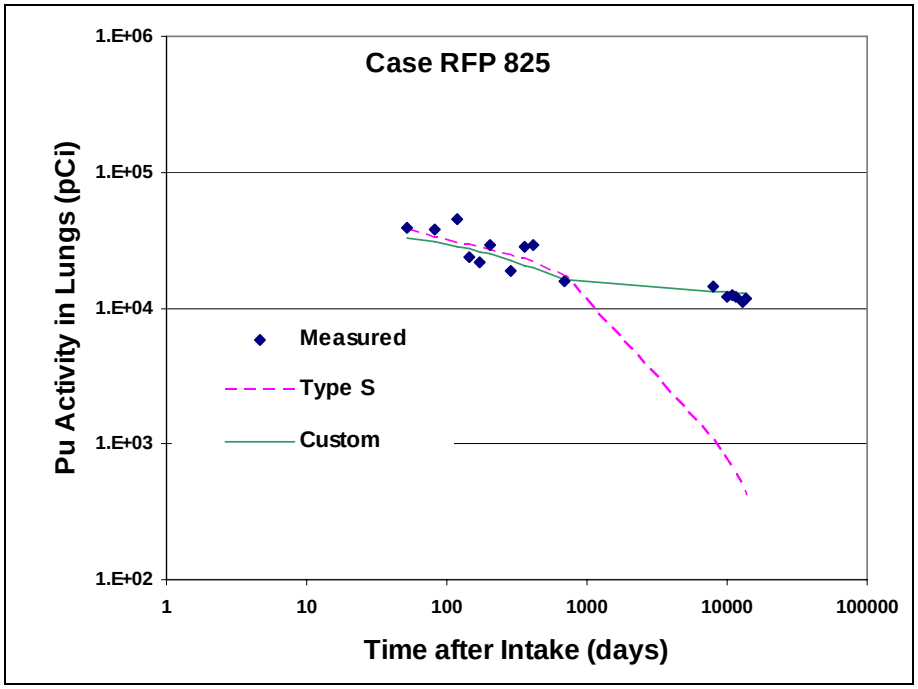

Figure A-3. Comparison of Type S and custom models for Case RFP 825.

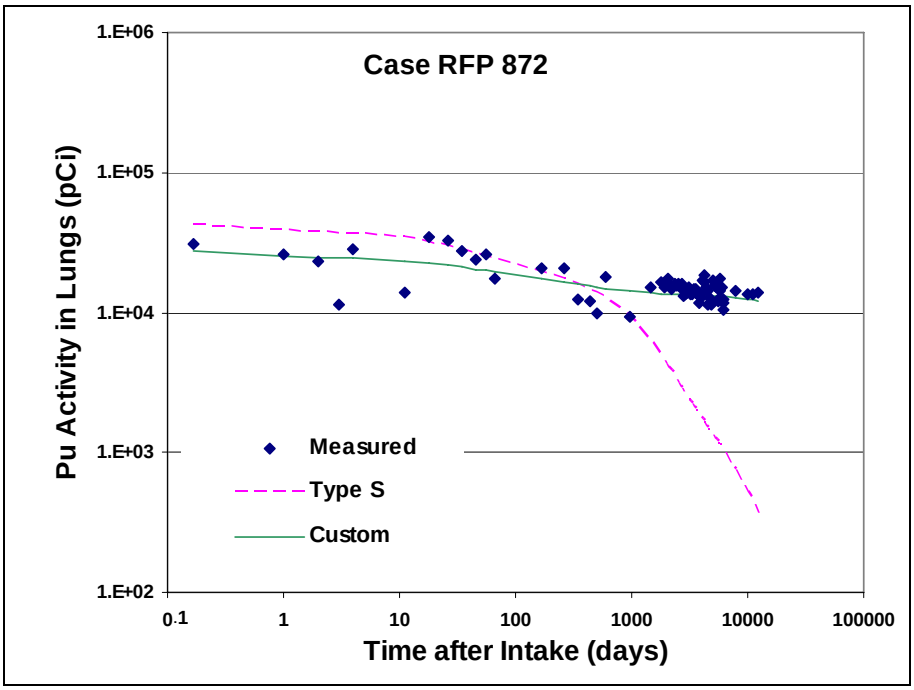

Figure A-4. Comparison of Type S and custom models for Case RFP 872.

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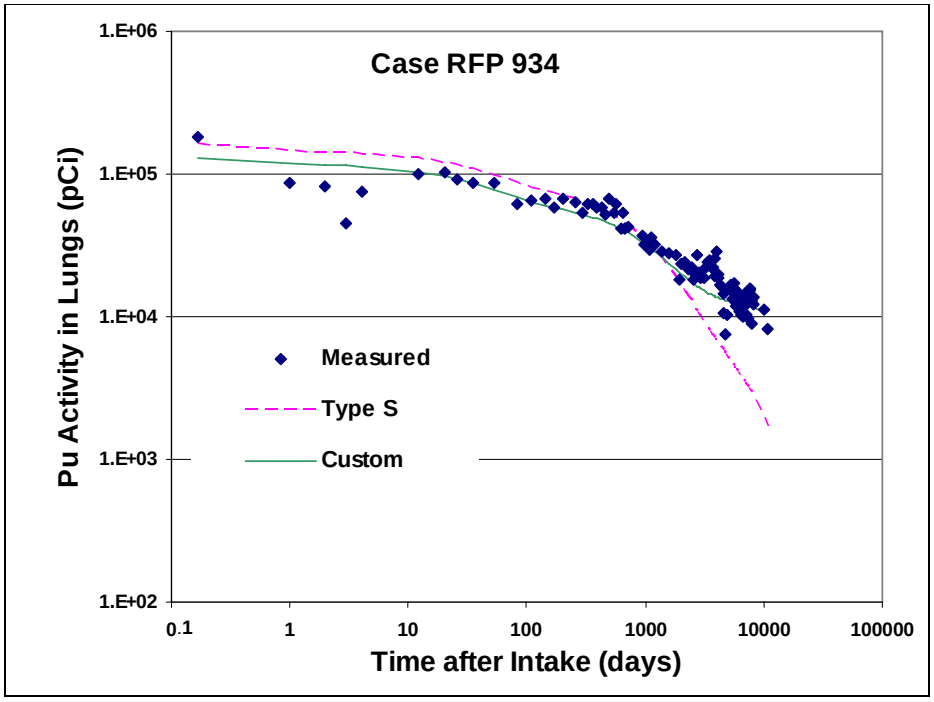

Figure A-5. Comparison of Type S and custom models for Case RFP 934.

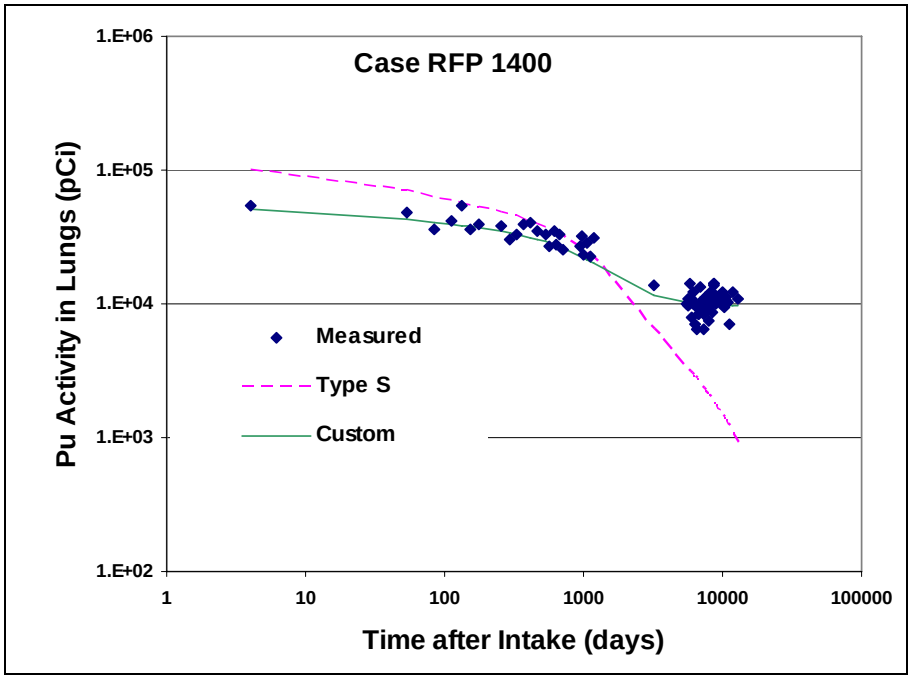

Figure A-6. Comparison of Type S and custom models for Case RFP 1400.

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RFP 700
RFP 700 was a laboratory foreman who received an inhalation intake on August 22, 1971, from the spontaneous combustion of plutonium chips with carbon tetrachloride residue in a sample can in a laboratory in Building 771. The double can pressurized and vented into the laboratory. RFP 700 was a responder to the fire and entered the laboratory wearing a chemical-oxygen respirator to clean up the mess and collect the residue. However, one strap on the respirator was broken, resulting in an ineffective seal. The initial concentration of americium was 480 ppm. Extensive DTPA was administered to the worker starting on August 30, 1971. RFP 700 received a skull count on April 9, 2002, coupled with a lung count. The ratio of normalized skull/lung ^{241}Am counts was 0.15, which indicates that the skeletal contribution to the chest count was reasonably low and did not account for the persistence of ^{241}Am in the chest (see Figure A-7).

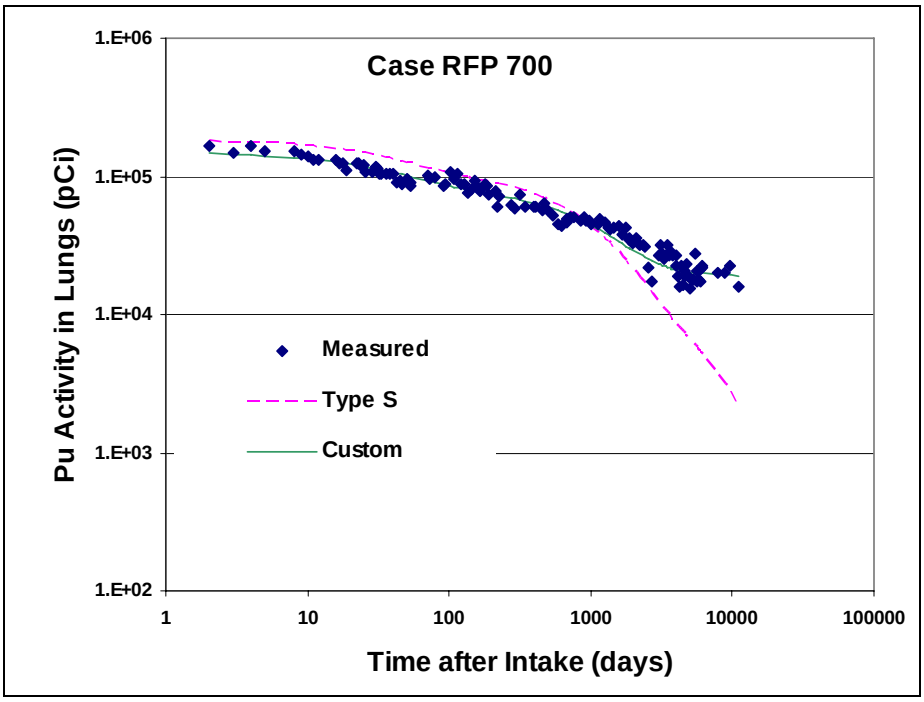

Figure A-7. Comparison of Type S and custom models for Case RFP 700.

RFP 725
RFP 725 was a firefighter who helped control a plutonium fire on May 11, 1969, in Building 76/77. Assigned to the roof of the building, he supervised two crews spraying water on the roof to keep it from breaching. He did not wear a respirator initially. Contamination apparently came out of the plenums without smoke; the roof was not breached. RFP 725 was highly contaminated and received 17 g of DTPA treatments, which had no apparent effect. This indicates that there was essentially no initial soluble content of the plutonium. The particle size is not known. The initial concentration of ^{241}Am was 1,000 ppm; the initial amount of ^{241}Pu was assumed to be 0.5% by weight. He had no previous or subsequent plutonium exposures (see Figure A-8).

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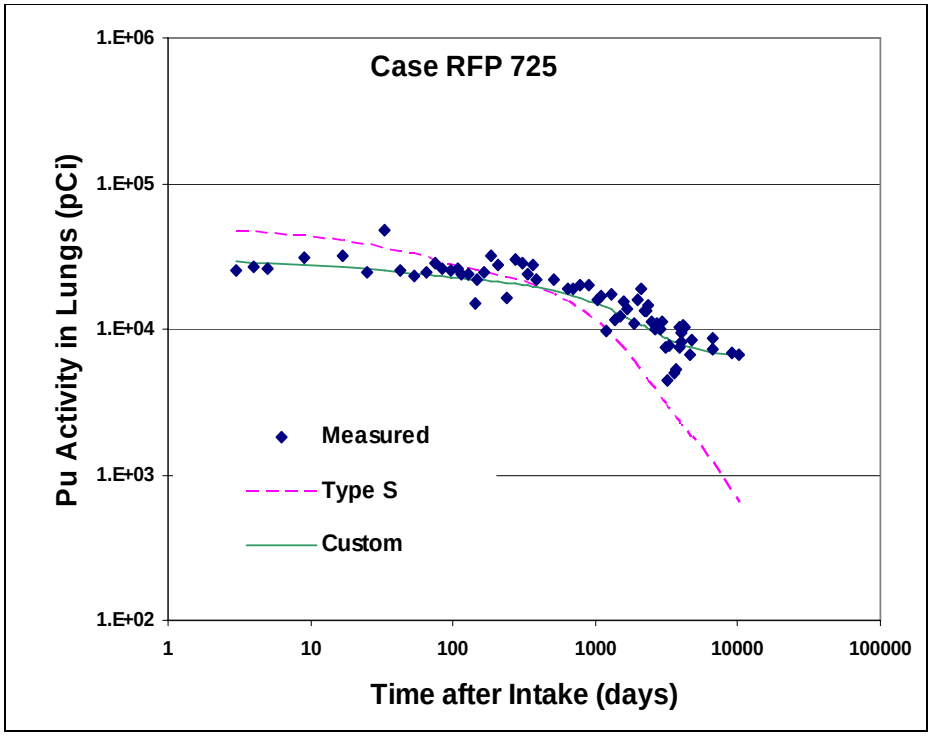

Figure A-8. Comparison of Type S and custom models for Case RFP 725.

RFP 1228
RFP 1228, a process operator in Building 771, was exposed to plutonium, probably oxide, from a glove failure on July 30, 1975. He was not wearing a respirator during the incident. He received 4 g of DTPA treatments. The particle size is not known. The initial concentration of ^{241}Am was 1,050 ppm; the initial amount of ^{241}Pu was assumed to be 0.5% by weight. He had no previous recorded plutonium intakes and no subsequent intakes (see Figure A-9). This case represents avidly retained plutonium in the lung not resulting from a plutonium fire, but probably from slowly oxidized plutonium.

A2.0 CASES FROM OTHER SITES

HAN-1
HAN-1 has been described in detail in Spitz and Robinson (1981), Carbaugh, Bihl, and Sula (1991), and Carbaugh and La Bone (2003). The worker was exposed acutely on May 23, 1978, to plutonium oxide that had been calcined at 600°C. The isotopic composition, including ^{241}Pu, was measured on a sample of the source. Particle size information was not obtained. DTPA was administered on days 0, 2, 3, 7, and 10 after intake, but was determined to be ineffective due to the highly insoluble nature of the plutonium. Numerous chest counts were obtained from day 0 through day 6,639 after intake. The chest counts were corrected for chest wall thickness as measured by ultrasound techniques. Activity in the skeleton, as measured by skull counting, was usually not detectable or just slightly above detection; hence, correction of chest counts for skeletal activity was made only in the last three

measurements. The worker had no previous confirmed intakes of plutonium or ^{241}Am. Figure A-10 shows the plutonium lung content for case HAN-1 as measured by ^{241}Am and custom-fitted in IMBA

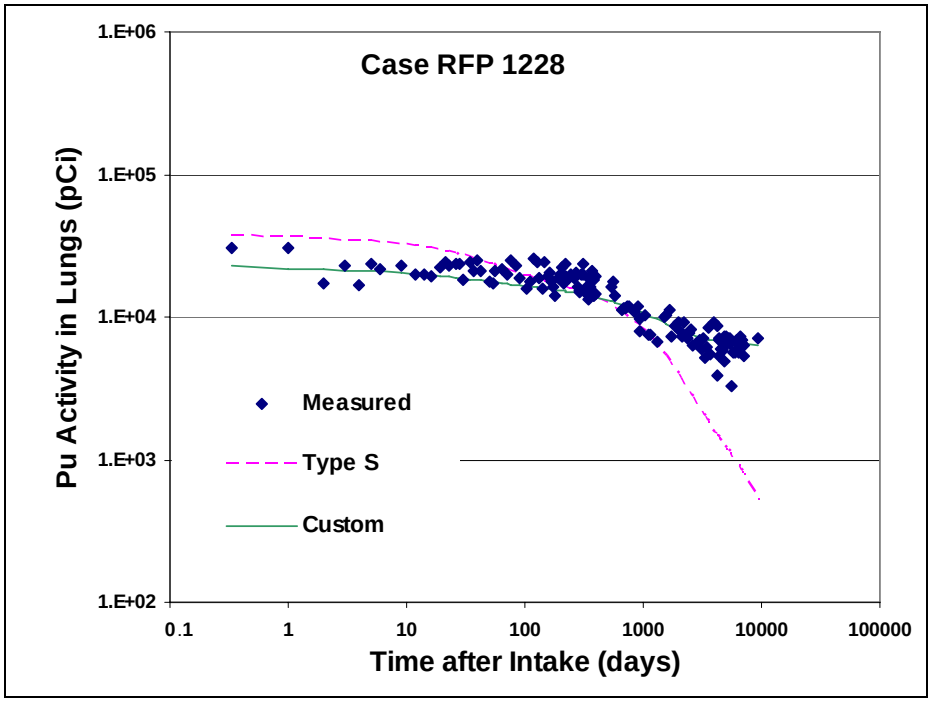

Figure A-9. Comparison of Type S and custom models for Case RFP 1228.

using parameter values in Attachment B. From 1 d after the accident through 18 years, very little plutonium/americium has been cleared. In his 2003 assessment of the case, Carbaugh and La Bone (2003) modeled lung retention as a single component with an 80-year clearance half-time. A. C. James modeled this case as an example for the IMBA user's manual (James 2004). James concluded that for this case both "the absorption characteristics of the plutonium particle matrix and the mechanical elimination rate of particles deposited in the 'deep lung' of this individual worker differ substantially from the standard ICRP default values." James fit the lung data using 0.5-AMAD particle size, a slow absorption rate, S_s, of 2×10^{-5}/d, and reduced mechanical clearance of 0.0001/d for AI1 to bb1 and AI2 to bb1. James retained the insoluble plutonium value of 0.0001 for f_1, but in a later personal discussion agreed that f_1 of 0.00001 would be better for Type SS material. However, to make this fit, James ignored all counts in the last 1,500 d.

SRS 498

SRS 498 has been described in detail in La Bone and Findley (1999) and Carbaugh and La Bone (2003). In September 1999, the worker was exposed to plutonium metal that had oxidized at ambient temperatures for about a year due to a defective weld on the stainless-steel storage container. Only the oxide form of the plutonium had escaped through the hole in the weld and become airborne. One g of DTPA was given shortly after the intake. The isotopic composition of the material, including ^{241}Pu, had been determined by mass spectrometry and was decay-corrected to the time of the intake. These isotopic ratios were verified by alpha spectrometry measurements on material obtained from

the air sampler used to monitor the operation. Particle size information was not obtained; however, the material from the air filter underwent *in vitro* lung solubility analysis at the Lovelace Respiratory Research Institute. Table A-11 (reprinted from Carbaugh and La Bone [2003]) lists results of the *in vitro* solubility analysis and the fit to the *in vivo* ^{241}Am lung counts. The model based on the lung counts also fit well with the urine and fecal samples. As with HAN-1 and most of the RFP cases, the standard Type S absorption model did not fit the long-term lung burden values well.

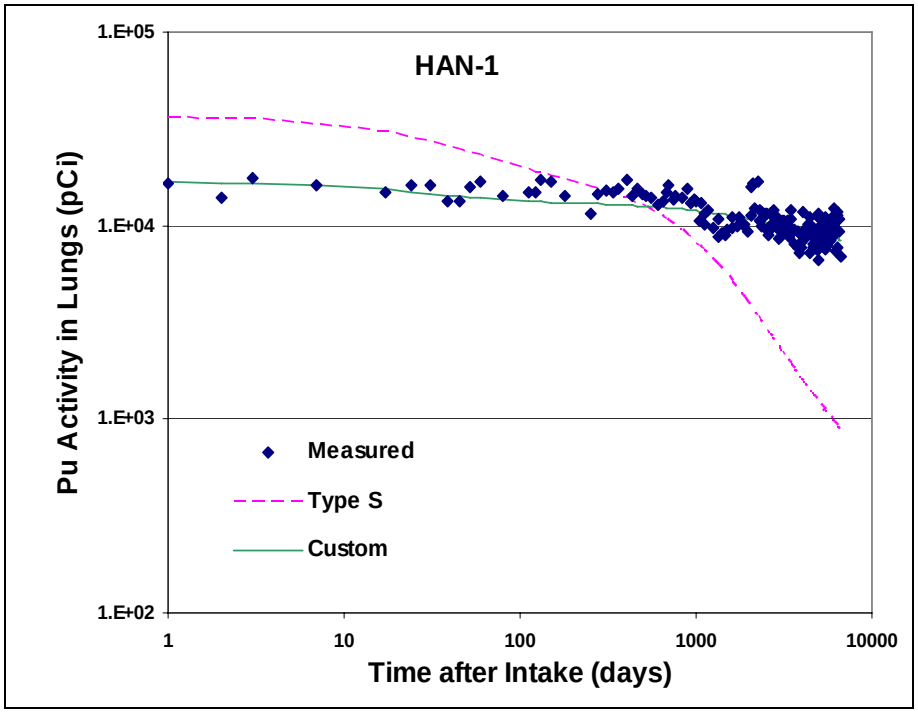

Figure A-10. Comparison of Type S and custom models for Case HAN-1.

Table A-1. Solubility parameter comparison for SRS 498.

Type	Fraction rapidly dissolved, f_r	Rapid solubility rate, s_r (d^{-1})	Slow solubility rate, s_s (d^{-1})
In vitro study of Pu oxide from air sample	0.0007	1.56	1.3×10^{-5}
Parameters inferred from bioassay	0.002	9.9	9.7×10^{-6}
Standard ICRP (1994) Type S parameters	0.001	100	1.0×10^{-4}

A2.1 UNITED STATES TRANSURANIUM AND URANIUM REGISTRY (USTUR) DONOR CASES

All of the following descriptions are from James (2005). USTUR has performed tissue analyses and reported on six whole-body donor cases that demonstrate larger activities in the lung at death than predicted by the Type S absorption model. A brief description of each case follows. Filipy (2004) and James (2005) concluded that all six cases show the inhaled plutonium was substantially less soluble than is assumed by ICRP for its recommended 'Type S' particle absorption behavior. Use of the

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ICRP 'default' absorption rate resulted in predicted lung burdens that were, on average, 18% of the measured burdens at death (range = 2-49%).

TUR 0193
Case TUR 0193 worked at LANL for 37 years. His highest intakes of plutonium occurred by inhalation during his first year of employment while he was converting plutonium oxide to plutonium fluoride in a chemical hood. The original isotopic ratios had to be assumed; the final plutonium activities were, of course, measured directly in lung tissue.

TUR 0208
Case TUR 0208 also worked at LANL for 37 years. His most probable intake of plutonium by inhalation occurred during his first year of employment when he worked in a plutonium reduction and dry chemistry operation. This case was reported by McInroy, Kathren, and Swint (1989).

TUR 0213
Case TUR 0213 worked as a chemist at LANL for 33 years. He had potential for intakes throughout most of his career and had five recorded potential inhalation accidents. The nature of the plutonium for these accidents has not been found (Kathren and McInroy 1991).

TUR 0242
Case TUR 0242 worked as a chemical operator at LANL for 26 years. He was involved in at least six incidents that could have resulted in acute inhalations of plutonium.

TUR 0425
Case TUR 0425 worked at RFP (but is not one of the RFP cases discussed above) for 24 years and was involved in several incidents with airborne plutonium, personal contamination, and minor wounds during his first decade of employment. A detailed review of the case was reported by Filipy (2004).

TUR 0744
Case TUR 0744 worked at RFP (but is not one of the RFP cases discussed above) for 32 years and was involved in as many as 17 incidents, including two plutonium-contaminated wounds and exposure to a plutonium fire. A detailed review of the case was reported by Filipy (2004).

ATTACHMENT B
DERIVATION AND USE OF DOSE ADJUSTMENT FACTORS

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Once inhaled, an aerosol can be removed from the respiratory tract by mechanical transport and by dissolution and subsequent absorption into the bloodstream. These removal processes are independent and competitive (i.e., a particle of material can be dissolving as it is being moved in the respiratory tract). It has long been known that some inhaled aerosols, like those of high-fired plutonium oxide, can be tenaciously retained in portions of the respiratory tract. The ICRP Publication 66 HRTM attempts to account for the retention of such aerosols by decreasing the dissolution rate of the plutonium (ICRP 1994). In the absence of case-specific information, Type S dissolution parameters ("S" for slow dissolution) will result in the longest retention of material in the respiratory tract. Note that the ICRP has not recommended generic changes in the mechanical transport parameters of the HRTM to account for increased retention time.

In the last few years, a number of papers have described the retention of plutonium in the respiratory tract that is not adequately modeled with Type S dissolution parameters. Carbaugh and La Bone (2003) describe occupational intakes of ^{239}Pu at Hanford and SRS that were retained in the respiratory tract much longer than predicted by the standard Type S parameters. A series of recent papers by researchers from Russia and the United States discussed the retention of plutonium in the respiratory tract of workers from the Mayak Production Association that is much longer than that expected for Type S plutonium (Hahn et al. 2003; Hahn et al. 2004; Romanov et al. 2003; Khokhryakov et al. 2005). Finally, a number of workers occupationally exposed to ^{239}Pu at RFP exhibited unexpectedly long-term retention in the respiratory tract (Mann and Kirchner 1967). For convenience, plutonium that displays this type of behavior is referred to as "Type SS" plutonium.

To calculate the equivalent dose to the respiratory tract and systemic organs resulting from an intake of Type SS plutonium, one must use a biokinetic model that accurately depicts the transport and retention of the plutonium in the tissues. Because the ICRP has not published recommendations on how to modify the HRTM to account for Type SS behavior, the authors of the papers cited above have used the following types of modifications to model Type SS behavior.

- The dissolution rate of the material is decreased beyond that of Type S plutonium. This general approach, which can be based on *in vitro* dissolution studies and animal studies, is endorsed by the ICRP even though it has not provided specific parameters for Type SS plutonium (ICRP 2002).

- The mechanical transport rate constants are reduced, which slows the mechanical clearance of particles from the respiratory tract.

- The *bound state* of the HRTM, which is somewhat of a vestigial organ in the model,[1] is used to prolong retention of dissolved plutonium in the respiratory tract by delaying its absorption into the bloodstream.

During the development of this document, various combinations of these techniques were used in an attempt to achieve an acceptable fit to plutonium activities derived from chest count data to plutonium activities derived from nine RFP cases and one Hanford case (referred to as the "design cases") that displayed Type SS behavior. Details of these efforts are presented in Attachment A. The original goal was to propose parameters for a generic Type SS model, but several difficulties were encountered that made this impractical. First, bioassay data from the design cases could not be adequately fit simply by decreasing the dissolution rate of the plutonium. However, combining the slow dissolution rate observed in cases at SRS with extremely slow mechanical clearance (e.g., the clearance half-life in the AI3 compartment is increased by a factor of 100) resulted in acceptable fits to the design cases. The problem with this approach is that a basic assumption of the HRTM is that mechanical transport rates are independent of the solubility of the material. In other words, there is no technical basis for decreasing mechanical transport rates across the board for a specific chemical form of plutonium.[2] Researchers analyzing bioassay data from Mayak workers used the bound state of the HRTM to achieve acceptable fits, but there are conceptual problems associated with requiring the plutonium to dissolve before it can be strongly retained. The researchers acknowledged this by stating, "So, although the present approach has shown adequate mathematical flexibility to be able to handle the new data, it is probably not the best way to interpret the mechanisms by which these particles are handled in the lung" (Romanov et al. 2003). In the end, we concluded that there is insufficient information available to recommend a generic modification to the HRTM suitable for evaluating cases that exhibit Type SS retention. For this reason, this TIB recommends an alternate approach to modeling Type SS plutonium cases, referred to as the "Dose Adjustment Factor." This approach, which is discussed below, enables the evaluation of Type SS plutonium cases without explicitly making generic changes to the HRTM [26].

The Dose Adjustment Factor

For a given acute inhalation intake of Type S ^{239}Pu, there is an initial deposition $q_s(0)$ in the lung and a lung content $q_s(t)$ at some later time t. The fraction $r_s(t)$ of the initial deposition in the lung is given by:

$$r_s(t) = \frac{q_s(t)}{q_s(0)}$$

Given an appropriate biokinetic model (see below), a similar function $r_{ss}(t)$ can be derived for Type SS ^{239}Pu. It is assumed that $q_s(0) = q_{ss}(0)$ for a given aerosol (i.e., the pattern of initial deposition in the compartments of the respiratory tract is the same for aerosols of both materials). Now, assume that the annual equivalent dose $H_s(t)$ to the lung from an inhalation intake of Type S ^{239}Pu from t-1 year to t year is proportional[3] to the lung content $q_s(t)$ at t year:

1. In the sense that it was defined in ICRP (1994) but never really used.
2. While modifying the mechanical transport parameters is acceptable in an appropriate individual case, it is another situation altogether to propose a generic model with those same modifications.
3. The dose is actually proportional to the area under the retention curve. This is a good assumption if the retention curve for the lung is fairly flat over the period in question (1 yr), which it is for Types S and SS plutonium.

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$$H_s(t) = kq_s(t)$$

where *k* is a time-independent constant specific to ^{239}Pu. The dose adjustment factor *F(t)* is defined as follows:

$$F(t) = \left[\frac{q_s(0)}{q_s(t)}\right]\left[\frac{q_{ss}(t)}{q_{ss}(0)}\right] = \frac{r_{ss}(t)}{r_s(t)}$$

To adjust $H_s(t)$ to get $H_{ss}(t)$ one simply multiplies by the dose adjustment factor:

$$H_{ss}(t) = H_s(t)F(t) = kq_s(t)\left[\frac{q_s(0)}{q_s(t)}\right]\left[\frac{q_{ss}(t)}{q_{ss}(0)}\right] = kq_{ss}(t)$$

The implicit assumption is that equal lung contents of Types S and SS ^{239}Pu at a given time produce the same equivalent dose rate to the lung at that time.

If the Type S lung dose was calculated from a chest count, the application of the adjustment factor will increase the implied Type SS lung content so that it is inconsistent with the original chest count. If a chest count at time *T* must be held constant, the basis of the dose adjustment factor must be converted from equal intakes at *t=0* to equal chest burdens at *t=T*:

$$H_{ss}(t) = kq_s(t)\left[\frac{q_s(0)}{q_s(t)}\right]\left[\frac{q_{ss}(t)}{q_{ss}(0)}\right]\left[\frac{q_s(T)}{q_s(0)}\right]\left[\frac{q_{ss}(0)}{q_{ss}(T)}\right] = kq_{ss}(t)$$

$$H_{ss}(t) = kq_s(t)\left[\frac{q_s(T)}{q_s(t)}\right]\left[\frac{q_{ss}(t)}{q_{ss}(T)}\right] = kq_{ss}(t)$$

$$H_{ss}(t) = kq_s(t)\frac{F(t)}{F(T)} = kq_{ss}(t)$$

Thus, to make the observed and predicted chest counts agree, the Type SS lung dose must be adjusted downward by the adjustment factor for the year of the chest count used to determine the intake. To ensure that the doses to the lung are not underestimated, the year of the chest count should be rounded down to the nearest whole year when selecting the factor.

Derivation of the Dose Adjustment Factor
The derivation of lung dose adjustment factors is based on an empirical comparison of the plutonium retained in the lungs for 10 well-documented cases involving acute intakes of plutonium (nine from RFP and one from Hanford) in relation to the amount projected for each case using the default Type S model for the same intake. These cases are discussed in Attachment A.

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The data for the design cases were custom modeled in the IMBA computer code to get a curve fit to plutonium lung data that could be used to generate, analytically, the plutonium retention in the lungs at any time and for any intake scenario using the IMBA Intake-to-Bioassay feature. For the given intake scenario and the same intake, the plutonium lung retention was calculated for the default ICRP Type S model [27]. The annual dose adjustment factors are the ratios of the plutonium lung retentions projected annually for the actual case to those projected for the default Type S model. Projections of the retained plutonium lung content are shown in Figure B-1 for the 10 design cases, for an acute intake of 1 Bq plutonium with a particle size of 5 µm AMAD. For reference, the theoretical curve for Type S plutonium is also shown. In relation to Type S, the design cases tend to exhibit a higher retention of plutonium in the lungs, especially after the first several years, with a similar flatness of the retention curves after 10 years. Two cases represent a similar upper bound, one from RFP (RFP 872) and one from Hanford (HAN-1).

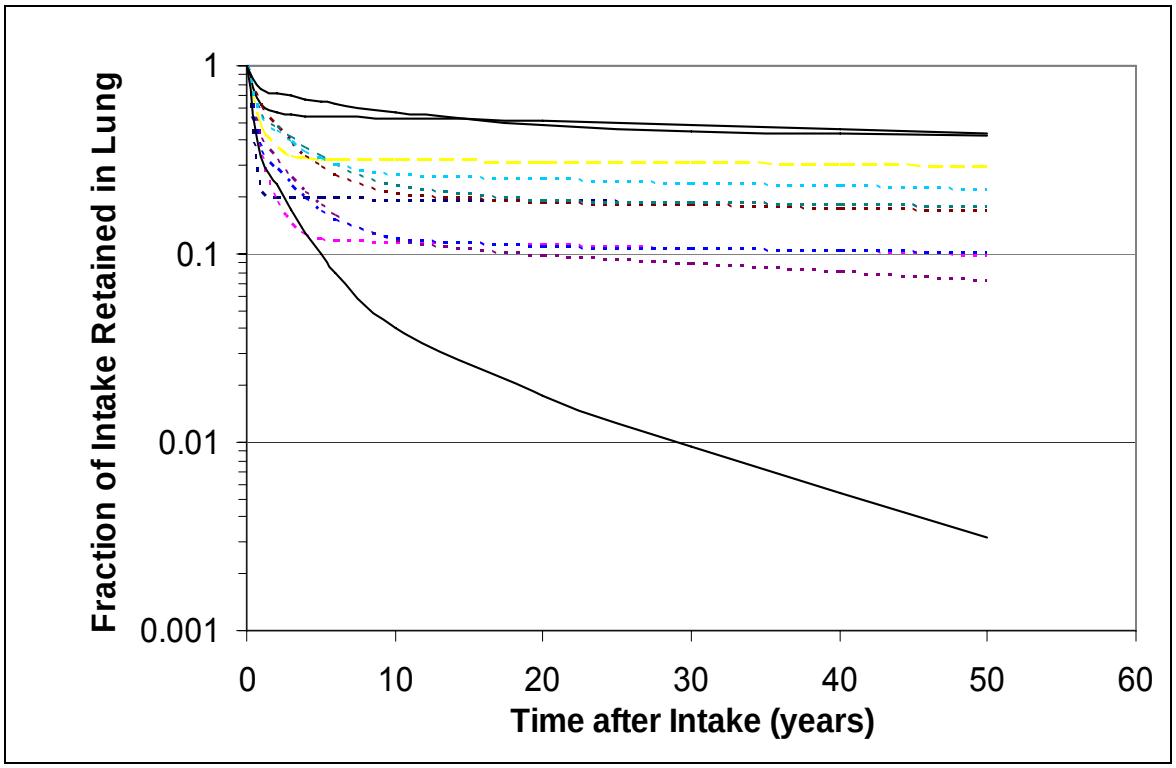

Figure B-1. Comparison of projected plutonium lung content for 10 design cases and standard Type S plutonium for acute intake of 1 Bq and particle size of 5 µm AMAD. The two highest solid lines are HAN-1 and RFP 872 and the lowest solid line is standard Type S material.

The dose adjustment factor is the ratio of the plutonium retention for the highest of the design cases (RFP 872 or HAN-1) and the plutonium retention predicted by the default Type S model any year after an acute intake or start of a chronic intake. Figures B-2 and B-3 show these ratios for the 10 design cases for acute and 30-year chronic intakes, respectively. Cases RFP 872 and HAN-1 consistently represented the upper bound for the design cases.

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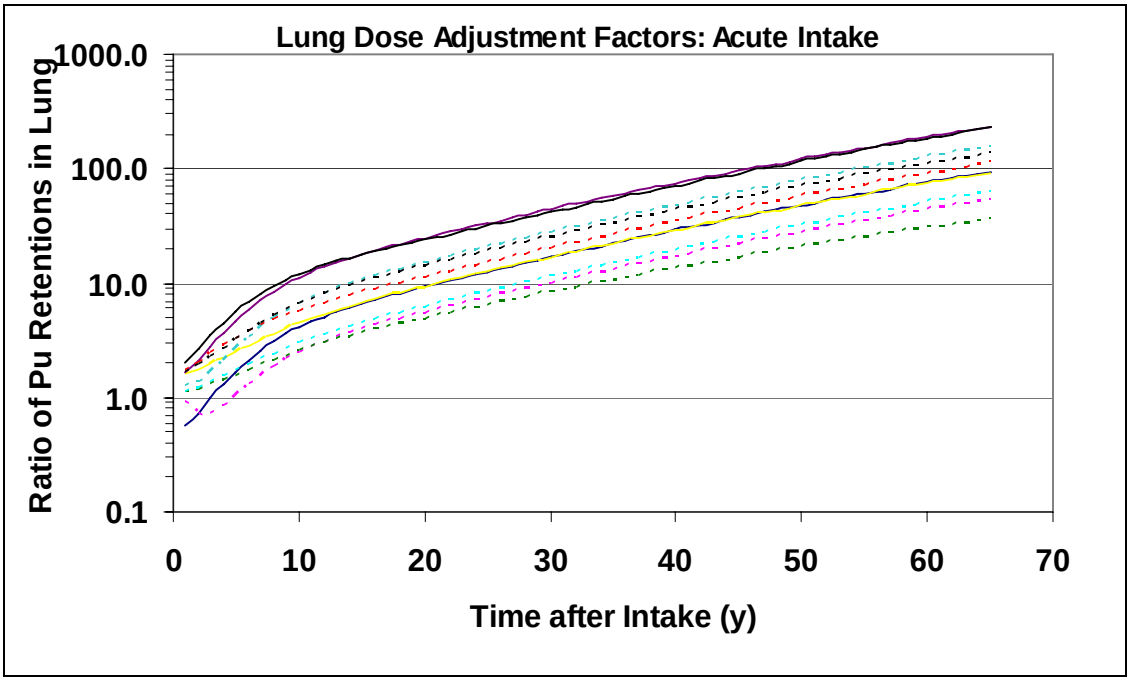

Figure B-2. Lung dose adjustment factors for design cases for acute intake. The two highest solid lines are HAN-1 and RFP 872.

For the derivation of lung dose adjustment factors in Attachment D, Standard HRTM Type S parameters for a 5-µm AMAD aerosol were used except for those listed below for the two bounding cases, HAN-1 and RFP 872:

HAN-1

Particle Density	11.5 g/mL
Particle Size	5 µm AMAD
Particle Transport	AI2->bb1 = 0.0003
	AI3->bb1 = 1×10^{-6}
	AI3->LN_{TH} = 1×10^{-6}
	AI2/AI = 0.4
	AI3/AI = 0.6
Absorption	Sp = 0.16
	St = 5×10^{-6}

RFP 872

Particle Density	11.5 g/mL
Particle Size	5 µm AMAD

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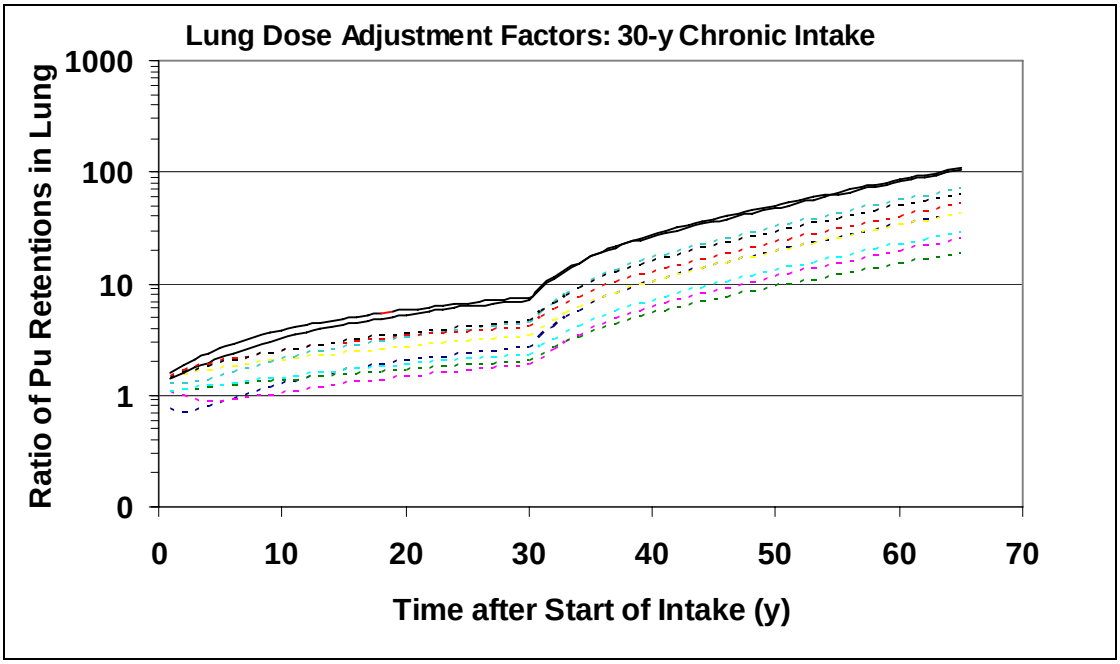

Figure B-3. Lung dose adjustment factors for design cases for 30-year chronic intake. The two highest solid lines are HAN-1 and RFP 872.

Particle Transport	AI2->bb1 = 0.003
	AI3->bb1 = 1×10^{-6}
	AI3->LN_{TH} = 1×10^{-6}
	AI2/AI = 0.3
	AI3/AI = 0.7
Absorption	Sp = 0.27
	St = 1×10^{-5}

These parameters were used in IMBA to generate $r_{ss}(t)$ at various times after intake for HAN-1 and RFP 872. It must be emphasized that these biokinetic models were selected simply to match empirical lung contents and urinary excretion and were not used to calculate equivalent doses to the lung [28].

Finally, IMBA was used to calculate $r_s(t)$ for standard Type S material, and then $F(t)$ was calculated for both cases. The higher of the two values of $F(t)$ for any given time and intake pattern was selected as the dose adjustment factor. This process was repeated for various chronic intake scenarios to create the tables in Attachment D.

Discussion of Applicability and Limitations

The slower removal of plutonium from the lung could be related to the activity or mass of plutonium in the lungs. The Mayak autoradiography studies (Hahn et al. 2003) show a correlation between radiation-induced pulmonary scars and the longer retention of the plutonium, which would be

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consistent with an activity-threshold effect.[4] Occupational exposures in U.S. cases generally resulted in smaller intakes than those observed for the Mayak workers, but evidence has been found for pneumosclerosis in some U.S. workers with lung doses over ~10 Sv (Newman et al. 2005). Because the existence of an activity or mass threshold effect is uncertain, it is favorable to the claimant to assume that the phenomenon applies to workers with smaller intakes of plutonium for whom bioassay data are insufficient for the dose reconstructor to create individual-specific models. All the U.S. human cases involve plutonium oxide, with the exception of USTUR cases, for which the chemical form of intakes was often not known. The plutonium usually had been exposed to high temperatures, but the SRS case and one of the RFP cases involved plutonium that had been oxidized under ambient temperatures. In mice, retention in the lung was not significantly different for $^{239}PuO_2$ fired at 550°C, 750°C, 1,000°C, or 1,250°C (Morgan et al. 1988). Conversely, not all intakes of plutonium oxide have shown lung retention inconsistent with the standard Type S model. Nevertheless, because of the uncertainty in the understanding of the nature of the material that results in SS behavior in the lung, it was considered favorable to the claimant to assume that all plutonium oxide could produce SS behavior.

In mice, in the same experiment mentioned above, a direct comparison was made between $^{238}PuO_2$ and $^{239}PuO_2$ fired at the same temperatures. The slow removal lung component in the two-component model was significantly faster and retained quantities in the liver and skeleton were greater for $^{238}PuO_2$ for all temperature groups (Morgan 1988). The authors are not aware of any intakes of $^{238}PuO_2$ that demonstrate very-long-term retention in the lung comparable to the cases discussed in Attachment A.

Because the ^{241}Am chest-count data in the design cases show similar results to the plutonium-in-lung autopsy data, it is assumed that when ^{241}Am is a small component by mass of the inhaled mixture or grows in from ^{241}Pu while in the lung, it behaves in the lung in the same manner as the plutonium. Stated another way, when the mixture of plutonium isotopes and ^{241}Am is mostly ^{239}Pu by mass, the Type SS dose adjustment factors apply to all components of the mixture [29].

4. During personal communications with Dr. Raymond Guilmette, he speculated that scar tissue caused by agents other than radiation could also become sites where particles might be sequestered.

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DERIVATION OF THE URINALYSIS ADJUSTMENT FACTOR [30]
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C-3	Comparison of urinary excretion following a chronic intake	43

Type SS material is absorbed into the blood stream at a slower rate than Type S material. This causes less material to be deposited in the systemic organs, as well as less plutonium being eliminated through the urine. Per unit intake, the difference between predicted urine content for Type S and Type SS varies considerably. This makes determining a correction factor difficult because the value of the correction factor is dependent on the time after intake and the length of exposure for each urine sample result. Therefore, the approach adopted here is to determine a single bounding correction factor to correct intakes determined from urinalysis. This correction factor is based on the clearance parameters developed using the slowest clearing design cases (HAN-1 and RFP 872). The intakes per unit excretion in the urine at various times using the Type S model, the HAN-1 case, and the RFP 872 case were then determined and compared.

First, a constant chronic excretion of activity per day was assumed to occur for 50 years. For this scenario, the largest difference between the Type S, the HAN-1 case, and the RFP 872 case occurred at approximately 6.5 years after the beginning of the intake. This difference, when rounded up, was a factor of 4.0. Figure C-1 provides a graph of the daily plutonium intake per unit excretion for Type S, HAN-1, RFP 872, and the adjusted Type S intake. It can be seen in the figure, that the intakes based on Type S clearance parameters are well below those of the intakes predicted using HAN-1 for the entire intake period. When the Type S intakes are adjusted upward by the factor of 4, the new intake projection figure shows that the estimated intakes over all time periods are equal to or greater than those of HAN-1.

The intake adjustment was also evaluated to determine the effect on an acute intake scenario. The results of this analysis are provided in Figure C-2. While the chronic exposure scenario produced adjustment factors that are fairly consistent throughout the duration of exposure, the acute exposure scenario does not. In fact, the acute scenario produces a correction factor greater than 4 for a short period of time after the acute intake. However, the factor is below 4 up to 214 d post-intake (Figure C-3).

It is unlikely that a known acute exposure incident would be detected by urinalysis 6 months after the intake. For incidents identified in the field, it is common practice that urine samples would be collected shortly after exposure.

ATTACHMENT C
DERIVATION OF THE URINALYSIS ADJUSTMENT FACTOR [30]

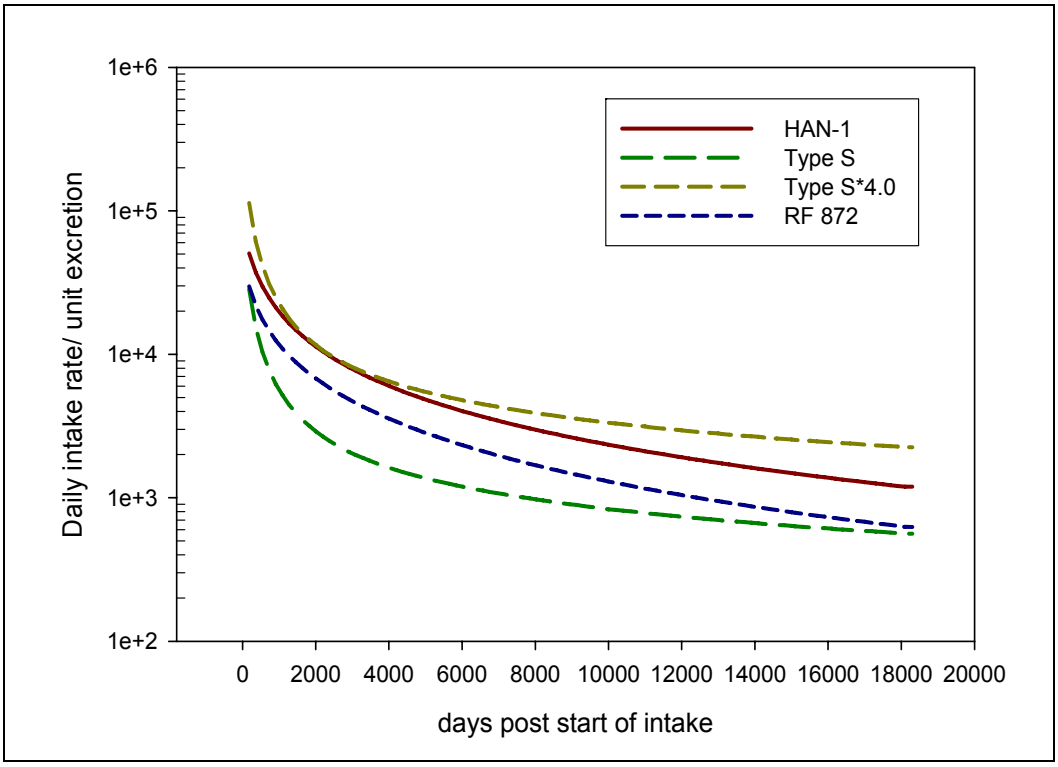

Figure C-1. Comparison of urinary excretion for a 50-year chronic intake.

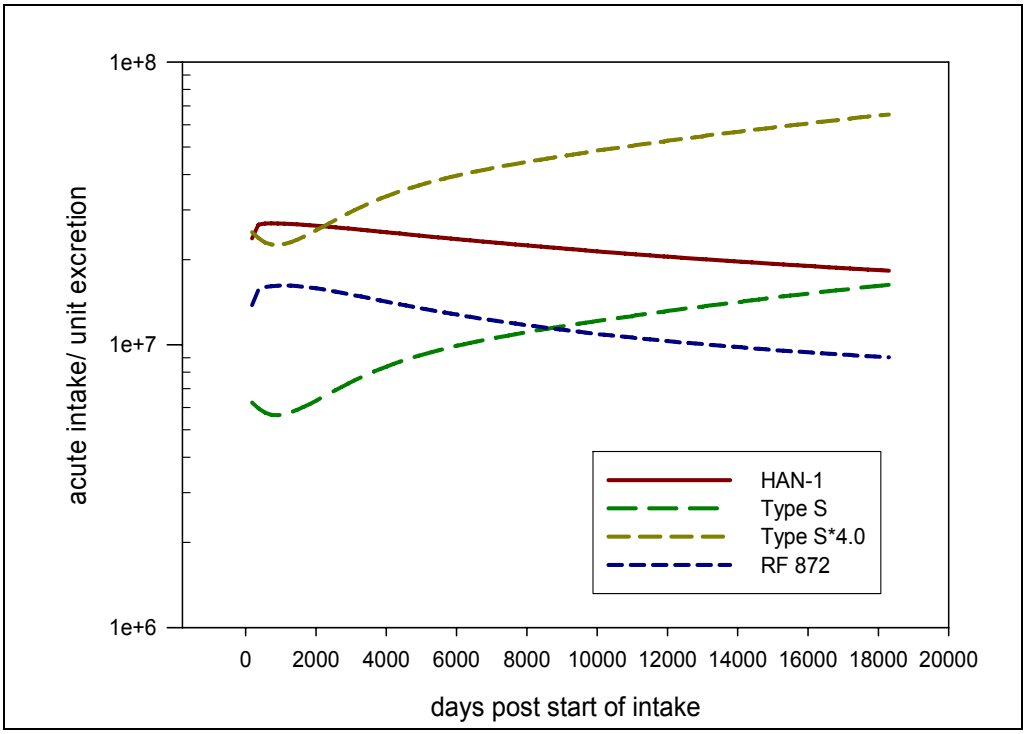

Figure C-2. Comparison of urinary excretion following an acute intake.

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DERIVATION OF THE URINALYSIS ADJUSTMENT FACTOR [30]
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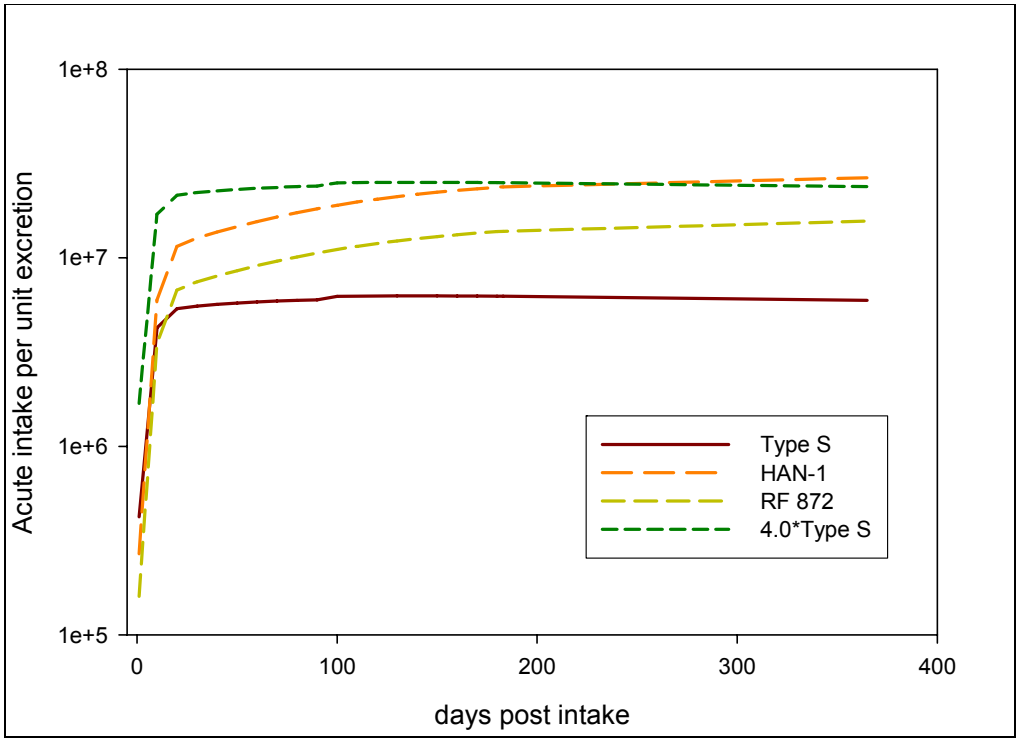

Figure C-3. Comparison of urinary excretion following a chronic intake.

In order to independently evaluate if the adjustment factors discussed above provide plausible bounding results, autopsy and bioassay data from USTUR were obtained for a number of Rocky Flats Plant workers with confirmed plutonium intakes. Seven cases were selected that had detectable values for both lung and urine bioassay measurements. These cases included three from the October 15, 1965, fire. One of these cases also had several wound uptakes and received DTPA treatments. One of the other cases from the fire received DTPA treatments while the third did not. The cases also included two additional wound cases with no indication of significant airborne exposure. One of these cases received DTPA treatment. The last two cases did not receive DTPA treatment and were exposed to airborne incidents other than the 1965 fire. One was a discrete incident while the other case involved several incidents.

All cases were evaluated as if little were known about the case. That is, they were assumed to be exposed to a constant chronic intake for the duration of their employment. Urine samples less than 0.2 dpm/d were excluded from the evaluation as being below the detection limit. In one case, an additional injection intake was assumed. This was necessary since no inhalation scenario could fit the urine data and the individual had a very large and well-documented injection incident (18 µg resulting in approximately 2000 dpm/d in the urine for only a short time).

Once the intakes were estimated, the expected lung content and liver content at autopsy was estimated using standard Type S parameters. The lung content was corrected using the adjustments discussed above; the liver content was not adjusted. These estimated contents were compared to autopsy data to verify that the adjustments are bounding. The results of this evaluation are provided in Table C-1.

ATTACHMENT C
DERIVATION OF THE URINALYSIS ADJUSTMENT FACTOR [30]

The first column of Table C-1 shows that the lung content is overestimated in every case. This can be accounted for by realizing that these are well-defined incident cases associated with the registry and so are really acute intakes. Figure C-2 shows that the factor of 4 intake correction would likely cause the intake to be overestimated following an acute intake.

Table C-1. Comparison of lung and liver estimates to autopsy data.

Ratio of lung estimate to autopsy measurement	Ratio of liver estimate to autopsy measurement	Type of intakes	DTPA
4.2	9.2	Fire	No
33.8	3.3	Wound	No
8.4	3.6	Other air	No
53.4	1.0	Fire/wound	Yes
8.6	2.9	Fire	Yes
30.2	1.1	Various air	No
123.5	3.7	Wound	Yes

Even without the factor-of-4 intake adjustment, the lung content is overestimated in every case. This could be caused by several factors. The largest factor is that some of these cases had uptakes to the bloodstream other than through inhalation, most notably, through wounds. The three highest overestimates of lung content are associated with wound cases. Another contributing factor could be that some of the plutonium may not be Type SS material. For cases with the next highest overestimate of lung content (ratio of 30.2 in Table C-1), the Type S assumption alone produces an estimated lung content greater than all but one of the 17 lung counts performed on the individual. The 16 over-predicted measurements range from a factor of just 1 to just under 3. Meanwhile, the Type SS adjustments overestimate all 17 lung counts by an average factor of 20. This indicates that this case is actually a Type S inhalation exposure due to the relatively close agreement of intakes determined using lung data and urine data when Type S is assumed.

The liver content was overestimated in all but one case. It is important to realize, however, that this is based on Type S parameters and not Type SS parameters. Therefore, for these cases, the Type S assumption alone is sufficient to overestimate the liver dose. This validates the assumption that the integrated urine content is proportional to the systemic organ dose. The overestimate is likely a result of the constant chronic intake assumption, which tends to overestimate the integrated urine content when discrete intakes occur.

ATTACHMENT D
LUNG DOSE ADJUSTMENT FACTORS [31]

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ATTACHMENT D
LUNG DOSE ADJUSTMENT FACTORS [31]

Table D-1. Lung dose adjustment factors, acute intake and chronic intakes, 1–7 years.

Year after acute intake or start of chronic intakes	Lung dose adjustment factor for intake periods							
	Acute	Chronic 1 yr	Chronic 2 yr	Chronic 3 yr	Chronic 4 yr	Chronic 5 yr	Chronic 6 yr	Chronic 7 yr
1	2.0	1.6	1.6	1.6	1.6	1.6	1.6	1.6
2	2.6	2.3	1.9	1.9	1.9	1.9	1.9	1.9
3	3.5	3.0	2.6	2.1	2.1	2.1	2.1	2.1
4	4.4	3.9	3.4	2.9	2.4	2.4	2.4	2.4
5	5.5	5.0	4.4	3.8	3.2	2.6	2.6	2.6
6	6.8	6.1	5.5	4.8	4.2	3.5	2.9	2.9
7	8.1	7.4	6.7	6.0	5.2	4.5	3.8	3.1
8	9.4	8.7	8.0	7.2	6.5	5.7	4.9	4.1
9	11	10	9.3	8.5	7.7	6.9	6.1	5.2
10	12	11	11	9.9	9.1	8.2	7.4	6.5
11	13	13	12	11	10	9.6	8.7	7.8
12	14	14	13	12	12	11	10	9.1
13	15	15	14	14	13	12	11	11
14	17	16	15	15	14	13	13	12
15	18	17	17	16	15	15	14	13
16	19	18	18	17	16	16	15	14
17	21	20	19	18	18	17	16	16
18	22	21	20	20	19	18	18	17
19	23	23	22	21	20	20	19	18
20	25	24	23	23	22	21	20	19
21	27	26	25	24	23	23	22	21
22	28	27	26	26	25	24	23	22
23	30	29	28	27	26	26	25	24
24	32	31	30	29	28	27	26	25
25	34	33	32	31	30	29	28	27
26	36	35	34	33	32	31	30	29
27	38	37	36	35	34	33	32	31
28	40	39	38	37	36	35	33	32
29	42	41	40	39	38	37	35	34
30	45	44	42	41	40	39	38	36
31	47	46	45	43	42	41	40	39
32	50	49	47	46	45	43	42	41
33	53	51	50	49	47	46	45	43
34	56	54	53	51	50	48	47	46
35	59	57	56	54	53	51	50	48
36	62	60	59	57	56	54	52	51
37	65	64	62	60	59	57	55	54
38	69	67	65	63	62	60	58	57
39	72	71	69	67	65	63	62	60
40	76	74	72	70	69	67	65	63
41	80	78	76	74	72	70	68	66
42	84	82	80	78	76	74	72	70
43	88	86	84	82	80	78	76	74
44	93	91	88	86	84	82	80	78
45	98	95	93	91	88	86	84	82

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Year after acute intake or start of chronic intakes	Lung dose adjustment factor for intake periods							
	Acute	Chronic 1 yr	Chronic 2 yr	Chronic 3 yr	Chronic 4 yr	Chronic 5 yr	Chronic 6 yr	Chronic 7 yr
46	100	100	97	95	93	90	88	86
47	110	110	100	100	97	95	92	90
48	110	110	110	110	100	100	97	95
49	120	120	110	110	110	110	100	99
50	120	120	120	120	110	110	110	100
51	130	130	120	120	120	120	110	110
52	140	130	130	130	120	120	120	110
53	140	140	140	130	130	130	120	120
54	150	150	140	140	140	130	130	130
55	160	150	150	150	140	140	140	130
56	160	160	160	150	150	140	140	140
57	170	170	160	160	160	150	150	140
58	180	170	170	170	160	160	150	150
59	180	180	180	170	170	170	160	160
60	190	190	180	180	180	170	170	170
61	200	200	190	190	180	180	180	170
62	210	210	200	200	190	190	180	180
63	220	210	210	210	200	200	190	190
64	230	220	220	210	210	200	200	200
65	240	230	230	220	220	210	210	200

ATTACHMENT D
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Table D-2. Lung dose adjustment factors, chronic intakes, 8–15 years.

Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 8 yr	Chronic 9 yr	Chronic 10 yr	Chronic 11 yr	Chronic 12 yr	Chronic 13 yr	Chronic 14 yr	Chronic 15 yr
1	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
2	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.9
3	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
5	2.6	2.6	2.6	2.6	2.6	2.6	2.6	2.6
6	2.9	2.9	2.9	2.9	2.9	2.9	2.9	2.9
7	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
8	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
9	4.4	3.6	3.6	3.6	3.6	3.6	3.6	3.6
10	5.6	4.7	3.8	3.8	3.8	3.8	3.8	3.8
11	6.8	5.9	5.0	4.0	4.0	4.0	4.0	4.0
12	8.2	7.2	6.2	5.3	4.2	4.2	4.2	4.2
13	9.6	8.6	7.6	6.5	5.5	4.5	4.5	4.5
14	11	10	9.0	7.9	6.8	5.8	4.7	4.7
15	12	11	10	9.3	8.2	7.1	6.0	4.9
16	14	13	12	11	9.7	8.5	7.4	6.2
17	15	14	13	12	11	10	8.9	7.7
18	16	15	14	14	13	12	10	9.2
19	17	17	16	15	14	13	12	11
20	19	18	17	16	15	14	13	12
21	20	19	18	17	17	16	15	14
22	22	21	20	19	18	17	16	15
23	23	22	21	20	19	19	18	17
24	25	24	23	22	21	20	19	18
25	26	25	24	23	23	22	21	20
26	28	27	26	25	24	23	22	21
27	30	29	28	27	26	25	24	23
28	31	30	29	28	27	26	25	24
29	33	32	31	30	29	28	27	26
30	35	34	33	32	31	30	29	28
31	37	36	35	34	33	32	31	29
32	40	38	37	36	35	34	32	31
33	42	41	39	38	37	36	34	33
34	44	43	42	40	39	38	37	35
35	47	45	44	43	41	40	39	37
36	49	48	47	45	44	42	41	40
37	52	51	49	48	46	45	43	42
38	55	54	52	50	49	47	46	44
39	58	56	55	53	52	50	48	47
40	61	60	58	56	54	53	51	50
41	65	63	61	59	57	56	54	52
42	68	66	64	62	61	59	57	55
43	72	70	68	66	64	62	60	58
44	75	73	71	69	67	65	63	61
45	79	77	75	73	71	69	67	65

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Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 8 yr	Chronic 9 yr	Chronic 10 yr	Chronic 11 yr	Chronic 12 yr	Chronic 13 yr	Chronic 14 yr	Chronic 15 yr
46	83	81	79	77	75	72	70	68
47	88	85	83	81	78	76	74	72
48	92	90	87	85	83	80	78	76
49	97	94	92	89	87	84	82	80
50	100	99	96	94	91	89	86	84
51	110	100	100	98	96	93	90	88
52	110	110	110	100	100	98	95	92
53	120	110	110	110	110	100	100	97
54	120	120	120	110	110	110	110	100
55	130	130	120	120	120	110	110	110
56	130	130	130	130	120	120	120	110
57	140	140	130	130	130	120	120	120
58	150	140	140	140	130	130	130	120
59	150	150	150	140	140	140	130	130
60	160	160	150	150	150	140	140	140
61	170	160	160	160	150	150	150	140
62	180	170	170	160	160	160	150	150
63	180	180	180	170	170	160	160	150
64	190	190	180	180	170	170	170	160
65	200	200	190	190	180	180	170	170

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Table D-3. Lung dose adjustment factors, chronic intakes, 16–23 years.

Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 16 yr	Chronic 17 yr	Chronic 18 yr	Chronic 19 yr	Chronic 20 yr	Chronic 21 yr	Chronic 22 yr	Chronic 23 yr
1	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
2	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.9
3	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
5	2.6	2.6	2.6	2.6	2.6	2.6	2.6	2.6
6	2.9	2.9	2.9	2.9	2.9	2.9	2.9	2.9
7	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
8	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
9	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.6
10	3.8	3.8	3.8	3.8	3.8	3.8	3.8	3.8
11	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
12	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2
13	4.5	4.5	4.5	4.5	4.5	4.5	4.5	4.5
14	4.7	4.7	4.7	4.7	4.7	4.7	4.7	4.7
15	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9
16	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
17	6.5	5.2	5.2	5.2	5.2	5.2	5.2	5.2
18	7.9	6.7	5.4	5.4	5.4	5.4	5.4	5.4
19	9.5	8.2	6.9	5.6	5.6	5.6	5.6	5.6
20	11	9.8	8.5	7.2	5.8	5.8	5.8	5.8
21	13	11	10	8.7	7.4	6.0	6.0	6.0
22	14	13	12	10	9.0	7.6	6.2	6.2
23	16	14	13	12	11	9.2	7.8	6.3
24	17	16	15	14	12	11	9.5	8.0
25	19	17	16	15	14	13	11	9.7
26	20	19	18	17	16	14	13	11
27	22	21	20	18	17	16	15	13
28	23	22	21	20	19	18	16	15
29	25	24	23	22	21	19	18	17
30	27	26	25	23	22	21	20	19
31	28	27	26	25	24	23	22	20
32	30	29	28	27	26	25	23	22
33	32	31	30	29	27	26	25	24
34	34	33	32	30	29	28	27	26
35	36	35	34	32	31	30	29	28
36	38	37	36	34	33	32	31	29
37	41	39	38	36	35	34	33	31
38	43	41	40	39	37	36	35	33
39	45	44	42	41	40	38	37	35
40	48	46	45	43	42	40	39	38
41	51	49	47	46	44	43	41	40
42	53	52	50	48	47	45	44	42
43	56	55	53	51	50	48	46	45
44	60	58	56	54	52	51	49	47
45	63	61	59	57	55	53	52	50

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Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 16 yr	Chronic 17 yr	Chronic 18 yr	Chronic 19 yr	Chronic 20 yr	Chronic 21 yr	Chronic 22 yr	Chronic 23 yr
46	66	64	62	60	58	56	55	53
47	70	68	66	63	61	59	58	56
48	73	71	69	67	65	63	61	59
49	77	75	73	70	68	66	64	62
50	81	79	76	74	72	70	67	65
51	85	83	80	78	76	73	71	69
52	90	87	85	82	80	77	75	72
53	94	92	89	86	84	81	79	76
54	99	96	93	91	88	85	83	80
55	100	100	98	95	93	90	87	84
56	110	110	100	100	97	94	91	89
57	110	110	110	110	100	99	96	93
58	120	120	110	110	110	100	100	98
59	130	120	120	120	110	110	110	100
60	130	130	120	120	120	110	110	110
61	140	130	130	130	120	120	120	110
62	140	140	140	130	130	130	120	120
63	150	150	140	140	140	130	130	120
64	160	150	150	150	140	140	130	130
65	160	160	160	150	150	140	140	140

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Table D-4. Lung dose adjustment factors, chronic intakes, 24–31 years.

Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 24 yr	Chronic 25 yr	Chronic 26 yr	Chronic 27 yr	Chronic 28 yr	Chronic 29 yr	Chronic 30 yr	Chronic 31 yr
1	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
2	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.9
3	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
5	2.6	2.6	2.6	2.6	2.6	2.6	2.6	2.6
6	2.9	2.9	2.9	2.9	2.9	2.9	2.9	2.9
7	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
8	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
9	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.6
10	3.8	3.8	3.8	3.8	3.8	3.8	3.8	3.8
11	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
12	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2
13	4.5	4.5	4.5	4.5	4.5	4.5	4.5	4.5
14	4.7	4.7	4.7	4.7	4.7	4.7	4.7	4.7
15	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9
16	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
17	5.2	5.2	5.2	5.2	5.2	5.2	5.2	5.2
18	5.4	5.4	5.4	5.4	5.4	5.4	5.4	5.4
19	5.6	5.6	5.6	5.6	5.6	5.6	5.6	5.6
20	5.8	5.8	5.8	5.8	5.8	5.8	5.8	5.8
21	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0
22	6.2	6.2	6.2	6.2	6.2	6.2	6.2	6.2
23	6.3	6.3	6.3	6.3	6.3	6.3	6.3	6.3
24	6.5	6.5	6.5	6.5	6.5	6.5	6.5	6.5
25	8.2	6.7	6.7	6.7	6.7	6.7	6.7	6.7
26	10	8.5	6.9	6.9	6.9	6.9	6.9	6.9
27	12	10	8.7	7.0	7.0	7.0	7.0	7.0
28	14	12	10	8.9	7.2	7.2	7.2	7.2
29	15	14	12	11	9.1	7.4	7.4	7.4
30	17	16	14	13	11	9.3	7.5	7.5
31	19	18	16	14	13	11	9.5	7.7
32	21	19	18	16	15	13	11	9.7
33	23	21	20	18	17	15	13	12
34	24	23	22	20	19	17	15	14
35	26	25	24	22	21	19	18	16
36	28	27	26	24	23	21	20	18
37	30	29	27	26	25	23	22	20
38	32	31	29	28	27	25	24	22
39	34	33	31	30	29	27	26	24
40	36	35	33	32	31	29	28	26
41	38	37	35	34	33	31	30	28
42	41	39	38	36	35	33	32	30
43	43	41	40	38	37	35	34	32
44	46	44	42	41	39	38	36	35
45	48	46	45	43	42	40	38	37

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Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 24 yr	Chronic 25 yr	Chronic 26 yr	Chronic 27 yr	Chronic 28 yr	Chronic 29 yr	Chronic 30 yr	Chronic 31 yr
46	51	49	47	46	44	42	41	39
47	54	52	50	48	47	45	43	41
48	57	55	53	51	49	47	46	44
49	60	58	56	54	52	50	48	47
50	63	61	59	57	55	53	51	49
51	67	64	62	60	58	56	54	52
52	70	68	66	63	61	59	57	55
53	74	71	69	67	65	62	60	58
54	78	75	73	70	68	66	63	61
55	82	79	77	74	72	69	67	65
56	86	83	81	78	76	73	71	68
57	90	88	85	82	79	77	74	72
58	95	92	89	86	84	81	78	76
59	100	97	94	91	88	85	82	80
60	110	100	98	95	92	89	87	84
61	110	110	100	100	97	94	91	88
62	120	110	110	110	100	99	96	93
63	120	120	110	110	110	100	100	97
64	130	120	120	120	110	110	110	100
65	130	130	130	120	120	110	110	110

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Table D-5. Lung dose adjustment factors, chronic intakes, 32–39 years.

Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 32 yr	Chronic 33 yr	Chronic 34 yr	Chronic 35 yr	Chronic 36 yr	Chronic 37 yr	Chronic 38 yr	Chronic 39 yr
1	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
2	1.9	1.9	1.9	1.9	1.9	1.9	1.9	1.9
3	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4
5	2.6	2.6	2.6	2.6	2.6	2.6	2.6	2.6
6	2.9	2.9	2.9	2.9	2.9	2.9	2.9	2.9
7	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
8	3.4	3.4	3.4	3.4	3.4	3.4	3.4	3.4
9	3.6	3.6	3.6	3.6	3.6	3.6	3.6	3.6
10	3.8	3.8	3.8	3.8	3.8	3.8	3.8	3.8
11	4.0	4.0	4.0	4.0	4.0	4.0	4.0	4.0
12	4.2	4.2	4.2	4.2	4.2	4.2	4.2	4.2
13	4.5	4.5	4.5	4.5	4.5	4.5	4.5	4.5
14	4.7	4.7	4.7	4.7	4.7	4.7	4.7	4.7
15	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9
16	5.0	5.0	5.0	5.0	5.0	5.0	5.0	5.0
17	5.2	5.2	5.2	5.2	5.2	5.2	5.2	5.2
18	5.4	5.4	5.4	5.4	5.4	5.4	5.4	5.4
19	5.6	5.6	5.6	5.6	5.6	5.6	5.6	5.6
20	5.8	5.8	5.8	5.8	5.8	5.8	5.8	5.8
21	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0
22	6.2	6.2	6.2	6.2	6.2	6.2	6.2	6.2
23	6.3	6.3	6.3	6.3	6.3	6.3	6.3	6.3
24	6.5	6.5	6.5	6.5	6.5	6.5	6.5	6.5
25	6.7	6.7	6.7	6.7	6.7	6.7	6.7	6.7
26	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9
27	7.0	7.0	7.0	7.0	7.0	7.0	7.0	7.0
28	7.2	7.2	7.2	7.2	7.2	7.2	7.2	7.2
29	7.4	7.4	7.4	7.4	7.4	7.4	7.4	7.4
30	7.5	7.5	7.5	7.5	7.5	7.5	7.5	7.5
31	7.7	7.7	7.7	7.7	7.7	7.7	7.7	7.7
32	7.9	7.9	7.9	7.9	7.9	7.9	7.9	7.9
33	9.9	8.0	8.0	8.0	8.0	8.0	8.0	8.0
34	12	10	8.2	8.2	8.2	8.2	8.2	8.2
35	14	12	10	8.4	8.4	8.4	8.4	8.4
36	16	14	12	11	8.5	8.5	8.5	8.5
37	18	16	15	13	11	8.7	8.7	8.7
38	20	19	17	15	13	11	8.9	8.9
39	23	21	19	17	15	13	11	9.1
40	25	23	21	19	17	15	13	11
41	27	25	24	22	20	18	16	14
42	29	27	26	24	22	20	18	16
43	31	29	28	26	24	23	21	19
44	33	32	30	28	27	25	23	21
45	35	34	32	31	29	27	25	23

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Year after start of chronic intakes	Lung dose adjustment factor for intake periods							
	Chronic 32 yr	Chronic 33 yr	Chronic 34 yr	Chronic 35 yr	Chronic 36 yr	Chronic 37 yr	Chronic 38 yr	Chronic 39 yr
46	37	36	34	33	31	29	28	26
47	40	38	37	35	33	32	30	28
48	42	41	39	37	36	34	32	30
49	45	43	41	40	38	36	34	33
50	47	46	44	42	40	39	37	35
51	50	48	46	45	43	41	39	37
52	53	51	49	47	45	44	42	40
53	56	54	52	50	48	46	44	42
54	59	57	55	53	51	49	47	45
55	62	60	58	56	54	52	50	48
56	66	63	61	59	57	55	53	50
57	69	67	65	62	60	58	56	53
58	73	71	68	66	63	61	59	56
59	77	74	72	69	67	64	62	60
60	81	78	76	73	70	68	65	63
61	85	82	80	77	74	72	69	66
62	90	87	84	81	78	75	73	70
63	94	91	88	85	82	79	77	74
64	99	96	93	90	86	84	81	78
65	100	100	97	94	91	88	85	82

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LUNG DOSE ADJUSTMENT FACTORS [31]

Table D-6. Lung dose adjustment factors, chronic intakes, 40–45 years.

Year after start of chronic intakes	Lung dose adjustment factor for intake periods					
	Chronic 40 yr	Chronic 41 yr	Chronic 42 yr	Chronic 43 yr	Chronic 44 yr	Chronic 45 yr
1	1.6	1.6	1.6	1.6	1.6	1.6
2	1.9	1.9	1.9	1.9	1.9	1.9
3	2.1	2.1	2.1	2.1	2.1	2.1
4	2.4	2.4	2.4	2.4	2.4	2.4
5	2.6	2.6	2.6	2.6	2.6	2.6
6	2.9	2.9	2.9	2.9	2.9	2.9
7	3.1	3.1	3.1	3.1	3.1	3.1
8	3.4	3.4	3.4	3.4	3.4	3.4
9	3.6	3.6	3.6	3.6	3.6	3.6
10	3.8	3.8	3.8	3.8	3.8	3.8
11	4.0	4.0	4.0	4.0	4.0	4.0
12	4.2	4.2	4.2	4.2	4.2	4.2
13	4.5	4.5	4.5	4.5	4.5	4.5
14	4.7	4.7	4.7	4.7	4.7	4.7
15	4.9	4.9	4.9	4.9	4.9	4.9
16	5.0	5.0	5.0	5.0	5.0	5.0
17	5.2	5.2	5.2	5.2	5.2	5.2
18	5.4	5.4	5.4	5.4	5.4	5.4
19	5.6	5.6	5.6	5.6	5.6	5.6
20	5.8	5.8	5.8	5.8	5.8	5.8
21	6.0	6.0	6.0	6.0	6.0	6.0
22	6.2	6.2	6.2	6.2	6.2	6.2
23	6.3	6.3	6.3	6.3	6.3	6.3
24	6.5	6.5	6.5	6.5	6.5	6.5
25	6.7	6.7	6.7	6.7	6.7	6.7
26	6.9	6.9	6.9	6.9	6.9	6.9
27	7.0	7.0	7.0	7.0	7.0	7.0
28	7.2	7.2	7.2	7.2	7.2	7.2
29	7.4	7.4	7.4	7.4	7.4	7.4
30	7.5	7.5	7.5	7.5	7.5	7.5
31	7.7	7.7	7.7	7.7	7.7	7.7
32	7.9	7.9	7.9	7.9	7.9	7.9
33	8.0	8.0	8.0	8.0	8.0	8.0
34	8.2	8.2	8.2	8.2	8.2	8.2
35	8.4	8.4	8.4	8.4	8.4	8.4
36	8.5	8.5	8.5	8.5	8.5	8.5
37	8.7	8.7	8.7	8.7	8.7	8.7
38	8.9	8.9	8.9	8.9	8.9	8.9
39	9.1	9.1	9.1	9.1	9.1	9.1
40	9.2	9.2	9.2	9.2	9.2	9.2
41	12	9.4	9.4	9.4	9.4	9.4
42	14	12	9.6	9.6	9.6	9.6
43	16	14	12	9.7	9.7	9.7
44	19	17	14	12	9.9	9.9
45	21	19	17	15	12	10

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Year after start of chronic intakes	Lung dose adjustment factor for intake periods					
	Chronic 40 yr	Chronic 41 yr	Chronic 42 yr	Chronic 43 yr	Chronic 44 yr	Chronic 45 yr
46	24	22	20	17	15	13
47	26	24	22	20	18	15
48	29	27	25	23	20	18
49	31	29	27	25	23	21
50	33	31	30	28	26	23
51	36	34	32	30	28	26
52	38	36	34	33	31	29
53	41	39	37	35	33	31
54	43	41	39	37	36	34
55	46	44	42	40	38	36
56	48	46	45	43	41	39
57	51	49	47	45	43	41
58	54	52	50	48	46	44
59	57	55	53	51	49	47
60	61	58	56	54	52	49
61	64	62	59	57	55	52
62	67	65	63	60	58	55
63	71	69	66	63	61	59
64	75	72	70	67	64	62
65	79	76	73	71	68	65

ATTACHMENT E
EVALUATION OF FECAL SAMPLES

The plutonium activity in fecal samples collected during the first month or two after an acute inhalation intake comes predominately from the rapidly clearing compartments of the respiratory tract. Because of the short timeframe, the removal of material from these compartments is dominated by mechanical clearance. As shown in Figure E-1, fecal samples collected during this timeframe will contain a fraction of the intake that is essentially independent of the dissolution type of the inhaled material.

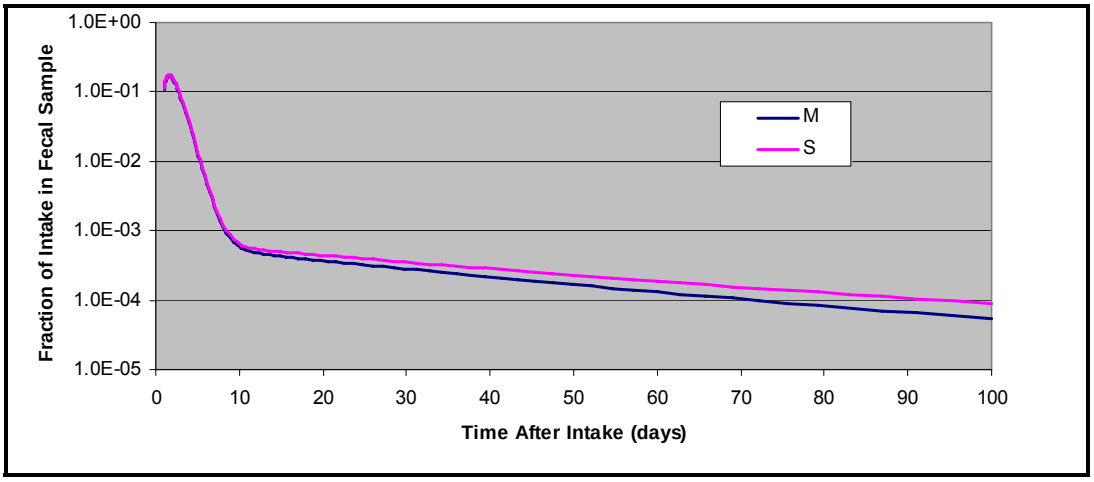

Figure E-1. Comparison of fecal excretion following an acute inhalation intake of ^{239}Pu .

This is the basis of the recommendation to evaluate fecal samples collected shortly after acute intakes of Type SS ^{239}Pu with the standard Type S models. A chronic intake is simply a large number of acute intakes. Thus, Types M, S, and SS plutonium have essentially the same fecal excretion fraction during the intake period, which is shown in Figure E-2. So, if the fecal sample is assumed to be collected <u>during</u> a constant chronic intake, it should also be evaluated with the standard Type S ^{239}Pu model.

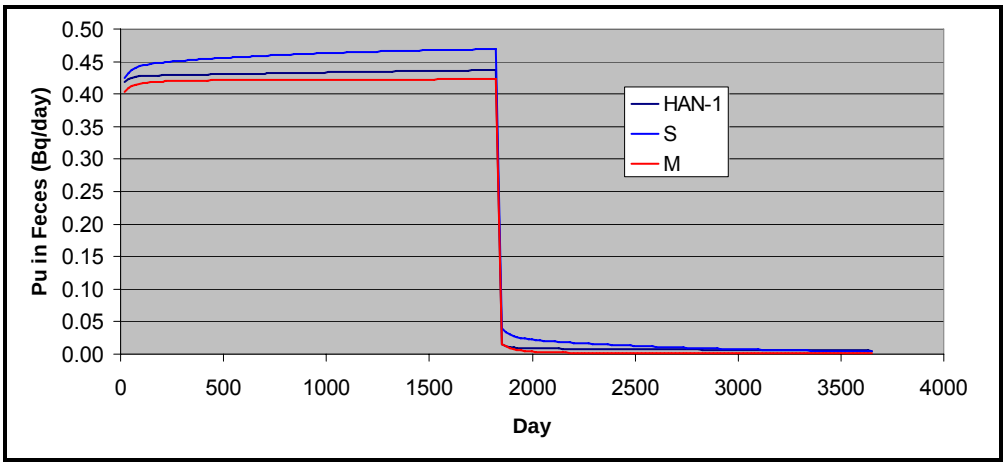

Figure E-2. Fecal excretion from 1,825-day chronic intake of 1 Bq/day.

ATTACHMENT E
EVALUATION OF FECAL SAMPLES

Once the intake is calculated the instructions given in this OTIB for evaluating doses from direct measurements (Section 4.1.1, Doses Calculated from Air Monitoring Data) should be followed.

The methods discussed thus far apply to fecal samples collected at times less than a few months after either an acute inhalation intake or the end of a constant chronic intake. However, there are problems with interpreting fecal samples at long times after an intake of Type SS ^{239}Pu because there is no model for long-term fecal excretion of this material.

A bounding solution to this problem is based on the fact that part of the plutonium in the feces comes from material that was mechanically cleared from the lungs and part from material that dissolved, was absorbed into the bloodstream, and subsequently excreted directly into the GI tract via the bile. If we assume that for Type SS plutonium there is absolutely no mechanical clearance from the respiratory tract, then all of the plutonium that is in a fecal sample came from systemic excretion. As will be shown below, the systemic excretion of plutonium to the feces is closely related to the systemic excretion of plutonium to the urine, addressed in Section 4.1.3. Thus, the basic approach is to model the activity in the fecal sample as if it were a urine sample using the methods given in Section 4.1.3 and then adjust the resulting intake to account for the fact that it was a fecal sample. The only task remaining is to derive an acceptable adjustment factor.

The urine and fecal excretion curves in Figure E-3 are for an injection intake of Pu-239. After a month or two the relationship between the two curves settles down and becomes fairly constant. This is shown more clearly in Figure E-4, which shows the ratio of the urine activity to the fecal activity. As can be seen in Figure E-4, the urine activity ranges from about 2 to 3 times the fecal activity for the timeframe of interest. An upper estimate of an intake calculated from a fecal sample following an inhalation intake of Type SS plutonium is therefore taken to be 3 times the intake estimated assuming that the fecal sample was a urine sample and applying the methodology in this OTIB for a urine sample.

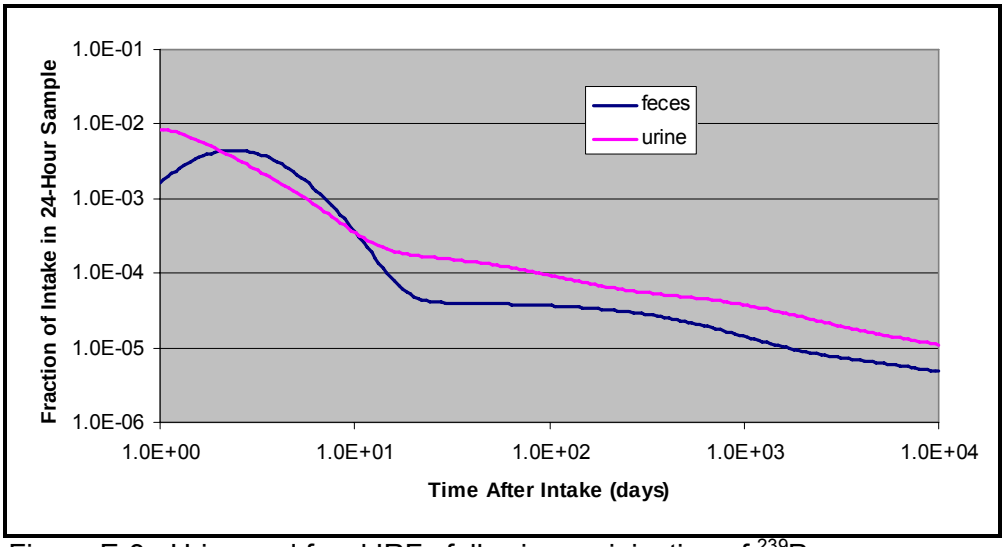

Figure E-3. Urine and fecal IRFs following an injection of ^{239}Pu .

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EVALUATION OF FECAL SAMPLES
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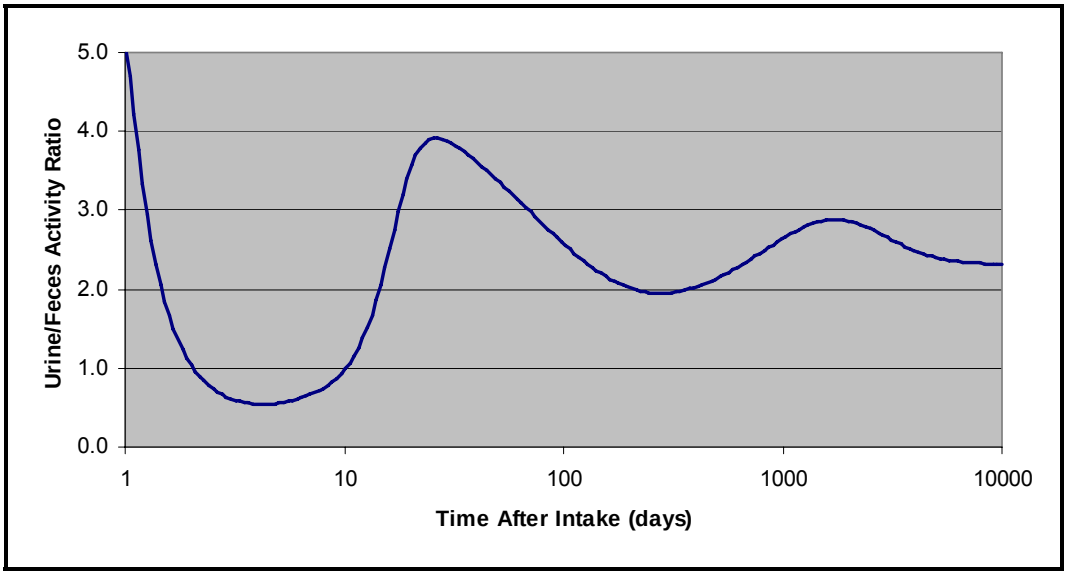

Figure E-4. Ratio of urine IRF to fecal IRF.