EVENT REPORT COVER PAGE

AGREEMENT STATE

EVENT REPORT NO. __ - __ - __

DATE: March 19, 1997

TO:

Deputy Director
Office of State Programs

SUBJECT: ABNORMAL OCCURRENCE REPORT COVERING CONTAMINATION EVENT AT ISOTOPE PRODUCTS LAB (IPL) Involving Am-241 and Cm-244 IN 1995

STATE: CALIFORNIA

Signature and Title: Donald Bunn, Chief

Compliance and Enforcement

Radiologic Health Branch

[redacted]

STATE OF CALIFORNIA—HEALTH AND WELFARE AGENCY PETE WILSON, *Governor*

DEPARTMENT OF HEALTH SERVICES
714/744 P STREET
P.O. BOX 942732
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March 19, 1997

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OSP

Ms. Patricia Larkins
State Agreements Program
Office of State Programs
U. S. Nuclear Regulatory Commission
Washington, D.C. 20555

Dear Ms. Larkins:

Enclosed is our Abnormal Occurrence Report concerning overexposures at Isotope Products Lab.

If you have any further questions, please call me at the number shown above.

Sincerely,

Donald E. Bunn, Chief
Compliance and Enforcement
Radiologic Health Branch

Enclosure

STATE OF CALIFORNIA — HEALTH AND WELFARE AGENCY PETE WILSON, *Governor*

DEPARTMENT OF HEALTH SERVICES
1449 WEST TEMPLE STREET
LOS ANGELES, CA 90026-5698

February 27, 1997

AS 95-5 Multiple Transuranic Exposures to a Worker at Isotope Products Laboratories in Burbank, California

Introduction:

Isotope Products Laboratories was founded in 1967 as a specialist provider of radioactive materials. Initially, its products were used in X- ray astronomy projects and other space based applications. Over the years, the radioactive source product line expanded significantly. Currently the company operates in three markets: environmental and laboratory calibration materials; analytical and process analysis components; and nuclear medical application serving an extensive international market in addition to domestic users of radioactive sources. The current 10,000 sq. ft. laboratory was constructed in 1975 and was modified on a number of occasions to accommodate increased and changed product demands. Over the years, facility improvements and operational techniques were implemented but it is felt they were not sufficient to keep up with some expanded radioactive source production operations, particularly for manufacture of large (millicurie) alpha sources of Am-241 and Cm-244. Containment and laboratory methods that had been utilized for high activity beta-gamma sources, even though made more restrictive for alpha work, apparently did not adequately protect workers when working with the more hazardous alpha materials. This was of particular concern for several special, high level, custom source projects undertaken for DOE laboratories. Recently, more restrictive operational methods and containment devices have been introduced for radioactive material work in general and alpha work in particular, including expanded glovebox facilities, air sampling, bioassay procedures and overall laboratory techniques. These changes have been reflective of a significant improvement in management's and worker's attitudes towards, and implementation of, the radiological safety program.

Date and Place - Between January 1 and December 31, 1995; Isotope Products Laboratories; Burbank, CA.

Nature and Possible Consequences - During 1995, the radiochemist was assigned duties to manufacture transuranic and other types of sources for use mainly by research laboratories.

Among the transuranics utilized were Pu-238/239/240, Am-241 and Cm-244. During January 1995, while making a Cm-244 source, it was discovered that the exhaust fan of the fume hood where a source was being fabricated was not working. Increased room air sampling results confirmed the loss of Cm-244 into the work area.

Fecal and urine samples were provided by the radiochemist. Bioassay results disclosed not only Cm-244 but Am-241 was also detected in the samples. On or about February 18, 1995, their consultant indicated a CDE of 2-3 rem and a CEDE of 1.123 rem.

This incident was discovered on a routine inspection December 5, 1995. It was then an abnormal occurrence was fully confirmed. A follow-up inspection indicated that a Cm-244 incident took place and was significant. Further, other incidents of exposures had occurred indicating the licensee's deficient contamination control program, inability to conduct internal dose assessment and inadequate management oversight.

Dosimetry and radiation protection consultants were hired by the licensee pursuant to regulatory direction. Careful analysis of the bioassay date by these consultants, including dose summation and retrospective time correction of various intakes, suggested that for the year 1995, the radiochemist received greater than 36 rem TEDE and 690 rem CDE to the bone surfaces.

Cause or Causes - It was apparent after our investigation that the licensee's radiation protection program was woefully weak, lacking important elements to assure radiation safety to their workers.
These inadequacies include the lack of work permits, glove boxes for certain types of work and procedural controls.

Action Taken to Prevent Recurrence

Licensee - After the consultants conducted their review and comprehensive audit of the licensee's existing program, definitive recommendations were made to assure future compliance with the license and regulations. They then hired a competent Radiation Safety Officer. The radiochemist is now assigned duties not involved in handling or processing radioactive materials.

State Agency - The State has completed its investigation of this abnormal occurrence and is committed to closely tracking the licensee's radiation use and control program to assure continued compliance. For the radiochemist, bioassays will continue to be done although no ciinical evidence after the exposure was indicated nor is expected in the future.

Dose Information for Individual A (radiochemist), 1995

Based on Los Angeles County, Department of Health Services' December 24, 1996 Compliance Letter:

a) Cm-244	CEDE = 27.18 rem
b) Am-241	CEDE = 8.00 rem
c) Pu-238, 239, 240	CEDE = 0.44 rem
d) Total	CEDE = 36.62 rem
e) External radiation	DDE = 2.70 rem
f) Total	TEDE = 38.72 rem

Attachments - For your review, the bioassay results and calculations from two different sources (Battelle and Ronald L. Kathren are attached for all three individuals exposed during this incident.

Battelle
Pacific Northwest Laboratories
Battelle Boulevard
P.O. Box 999
Richland, Washington 99352
Telephone (509) 509 376-6632

October 8, 1996

Mr. Gary Gilmore
Isotope Products Laboratories
1800 North Keystone Street
Burbank, California 91504

Dear Mr. Gilmore:

REVIEW OF IPL INTERNAL DOSE EVALUATIONS AND DATA

A review of two internal dose evaluations and the bioassay data compilations has been completed by Eugene H. Carbaugh of my staff. The scope of the review addressed the data, assumptions and methods, results of intake and dose calculations, and potential directions of impact, but did not extend to a complete reevaluation of the data. Some significant dose calculation errors were identified which would likely change the status of the workers relative to the dose limits of 10CFR20.1201.

If you have any questions concerning this review you may contact me or call Gene Carbaugh directly at (509) 376-6632.

Very truly yours,

Donald Bihl

Donald E. Bihl, Manager
Internal Dosimetry Project
Radiation Protection Services
HEALTH PROTECTION DEPARTMENT

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attachment

BATTELLE REVIEW OF ISOTOPE PRODUCTS LABORATORIES INTERNAL DOSE EVALUATIONS

A review of two internal dose evaluation reports and a compilation of data for several individuals was performed by Eugene H. Carbaugh, CHP, of the Battelle Radiation Protection Services, Internal Dosimetry Unit. The reviewer is the senior internal dosimetrist for the Hanford Site, and has extensive experience in plutonium and americium internal dosimetry. The review addressed the data, methods, and results of intake and dose assessments, but did not extend to complete reevaluation of data and reassessment of dose. Where significant discrepancies or errors were found, corrected values were provided. The potential impact of alternate intake dates (two years earlier) is briefly discussed.

Reference: **Letter dated March 24, 1996; Ronald L. Kathren (WSUTC) to Leonard Hendrickson (IPL)**

Plutonium Intake for Aret Demiral

The intakes of 1.3 nCi $^{239+240}$Pu and 0.11 nCi ^{238}Pu are appropriately calculated for class Y material, based on the fecal sample result and the assumed July 28, 1995 date of intake. An argument could be made that the ^{238}Pu result does not indicate actual detection of ^{238}Pu, because the error term is greater than the result. However, the use of the result does provide a reasonable upper bound on ^{238}Pu intake. Lack of detection of Pu in the March 11, 1996 urine sample is consistent with these intake estimates.

The dose calculational details are not provided; however, it appears the intake-to-dose conversion factors used were those of ICRP-30 Supplement to Part 1 (1979). Use of the factors in ICRP-30 Part 4 (1988) would result in a committed effective dose equivalent about 5% lower, a bone surface committed dose about 15% lower, and a liver dose about 30% lower. The differences would be directly due to the change in bone surface and liver clearance half-times (100 to 50 years and 40 to 20 years, respectively). These differences would not significantly change the standing of assessed dose with regard to occupational [illegible] limits.

R. L. Kathren identified the possibility of a ^{244}Cm intake based on possible in vivo detection of ^{244}Cm in the liver and results of the January 21, 1995 urinalysis. Urine samples collected subsequent to Kathren's report (March 11, May 14, and July 15, 1996) all appear to show the continued presence of ^{244}Cm suggesting that an intake has probably occurred. An estimate of the magnitude of such an intake requires more information regarding potential intake date and circumstances and is not attempted by this review.

I concur in Kathren's conclusion that fission product intakes were also likely, and that their dosimetric significance is minor relative to the transuranic intakes.

Plutonium Intake for Sayed Taghizadeh

The $^{239+240}Pu$ intake may be overestimated if fecal data are cited correctly. I calculated 0.279 nCi as opposed to Kathren's 0.51 nCi. The fecal excretion fraction I used for day 140 was 1.2E-4. It is possible that Kathren intended to take the average of the two $^{239+240}Pu$ fecal sample results, but in fact used the sum (i.e., did not divide the sum by two); by my calculation that would give a 0.56 nCi intake--close to the Kathren estimate. My ^{239}Pu value of 0.245 nCi is essentially identical to RLK's 0.25 nCi. The lack of detection of Pu in the January 16 urine sample analysis is consistent with these estimates.

Kathren's plutonium dose estimates are consistent with his stated intake estimates. The dose calculational details are not provided; however, it appears the intake-to-dose conversion factors used were those of ICRP-30 Supplement to Part 1 (1979). As noted above, use of the factors in ICRP-30 Part 4 (1988) would result in a committed effective dose equivalent about 5% lower, a bone surface committed dose about 15% lower, and a liver dose about 30% lower. The differences would be directly due to the change in bone surface and liver clearance half-times (100 to 50 years and 40 to 20 years, respectively). These differences would not significantly change the standing of assessed dose with regard to occupational dose limits.

Americium Intake for Sayed Taghizadeh

I concur with the difficulty identified by Kathren in reconciling the ^{241}Am data from in vivo, urine, and feces measurements. If the intake were assumed to have occurred in January or November 1993 (two years earlier than Kathren assumed), the revised intake estimates based on urinalysis data would be about a factor of 1.5 to 2 higher for class W, and for class Y only about 10% higher. Based on in vivo skeleton counting data, the earlier intake would suggest no significant change in magnitude for class W and about half as large an intake for class Y.

Kathren suggested the solubility lies somewhere between Y and W. The use of a nominal 50% W and 50% Y mixture brings the urinalysis-based estimate of intake (26 nCi) and the in vivo chest count-based estimate of intake (5.3 nCi) both within about a factor of two of the in vivo skeleton count-based estimate of intake (12 nCi). If the intake occurred two years earlier (1993) the intake estimates based on urine, chest, and skeleton data would be 63, 11, and 11 nCi, respectively. Further analysis of alternative combinations might bring this data into closer congruence.

Kathren has made a significant math error in calculating the 50-year dose equivalent to the bone surfaces. Using the ICRP-30 Supplement to Part 1 factors, the committed dose equivalent to the bone surfaces should be 370 rem, not 36.7 rem as calculated by Kathren. These results would indicate an overexposure by more than a factor of seven; however, there would be no clinically observed effects at that level.

Reference: Tabulated Summary of Bioassay Data and Lab Data Reports

There is a discrepancy in Aret Demiral's May 14, 1996 ^{244}Cm result as reported in the Lab Data Report (1.9E-1 ± 3.3E-2 pCi/L) and as tabulated in the Summary of Bioassay Data (8.5E-3 ± 8.5E-3 pCi/L). Presumably the lab report is the correct value.

The ^{244}Cm data for Aret Demiral also show one other intriguing characteristic. The January 21, 1995 and March 11, 1996 results are essentially identical, as are the May 14, 1996 and July 15, 1996 results (which are reported as identical). The likelihood that results at 5-to-10 times the MDA would show identical results when the samples are collected at widely separate times seems slim. The data suggest that ^{244}Cm has been readily detectable in urine over a one-year period, and the intake should be evaluated. Alternatively, the data could indicate some kind of quality-related problem with the analysis or reporting of results.

Reference: Letter dated April 26, 1996; Walter L. Milne (Radiological Safety Services) to Larry Hendrickson (IPL)

A consistent error in calculating dose from intake appears to have been made throughout this report. The report references ICRP and Regulatory Guide methods, but does not fully show the calculations. Apparently the intakes have been divided by the nonstochastic ALI of 10CFR20 Appendix B, Table 1 and then multiplied by the stochastic dose limit conversion factor of 5 rem/ALI. The nonstochastic bone surface dose conversion factor of 50 rem/ALI should have been used in all of the transuranic cases cited to calculate the most limiting committed dose equivalent (bone surface). To obtain the committed effective dose equivalent (DE, as used in the report), the stochastic ALI which is listed parenthetically in 10CFR20 Appendix B Table 1 should have been used and the quotient multiplied by 5 rem/ALI. Results of using the correct dose conversion factors are discussed in the individual cases that follow.

The net impact is that for all the cited intakes and doses, the bone surface committed dose equivalents should be the more limiting with regard to the 50 rem/year dose limit than the committed effective dose equivalents in comparison with the 5 rem/year total effective dose equivalent limit.

<u>Information related to Sayed Taghizadeh</u>

The calculated intakes based on identified air concentrations are correct; however, their conversion to dose is wrong. The correctly calculated committed effective dose equivalents are 0.023 rem, 0.001 rem, 0.098 rem, and .005 rem for ^{244}Cm, ^{252}Cf, ^{239}Pu, and ^{252}Cf, respectively.

The 1993 ^{241}Am doses are also erroneously calculated. The correct values are 7.5 rem for the committed effective dose equivalent (DE, as used in the report) and 125 rem for the committed bone surface dose equivalent (CDE, as used in the report). The report does

not identify the intakes used for the 1995 doses, so a good review of those doses cannot be provided. Based on the erroneous application of the nonstochastic ALI to the stochastic dose limit discussed above, I suspect that these values, too, are wrong.

Information related to Aret Demiral

The range of 5.8 - 12.5 rem should be 3.5 - 7.5 rem for committed effective dose equivalent for the same reasons as cited above. The more limiting regulatory condition of CDE to the bone surfaces would range from 58 to 125 rem.

The ^{244}Cm intake of 2.4 nCi should result in a committed effective dose equivalent of 0.600 rem and a bone surface CDE of 12 rem.

The compiled doses, again, demonstrate the mixing of nonstochastic ALIs with stochastic dose limit conversion factors and stochastic ALIs with nonstochastic dose limit conversion factors. The summations are correspondingly wrong.

Information on Deboleena Roy

The report indicates the DAC is used whereas the intent appears to be to use the measured air concentration. DAC refers specifically to the Derived Air Concentration values of 10CFR20 Appendix B. The intakes are correctly calculated; however, the same dose calculation errors have been made as described above. Corrected values for committed effective dose equivalent should be 0.317 rem (^{241}Am) and 0.159 rem (^{229}Th), with the sum being 0.476 rem. The bone surface CDEs would be 5.28 rem and 3.18 rem, respectively, and their sum is 8.46 rem.

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(509) 375-3316

Diplomate, American Board of Health Physics
Diplomate, American Board of Medical Physics
Diplomate, American Academy of Environmental Engineers

March 14, 1996

Mr. Leonard Hendrickson
Isotope Products Laboratories
1800 North Keystone Street
Burbank, CA 91504

Dear Mr. Hendrickson:

In accordance with our agreement dated January 15, 1996, the following internal dose evaluations are provided for three employees with suspected intakes of radioactivity. Following is a brief discussion of the evaluation made for each employee. Note that since the regulations are based on *intake* rather than ultimate deposition and dose within the various tissues of the body, the primary effort was directed towards determination of a realistic intake estimate, yet one that included an appropriate degree of conservatism, using accepted models and methods consistent with the regulations, even though these models may not reflect the most recent biokinetic parameters and understanding of the biokinetics of the actinides in particular.

Aret Demiral

A fecal sample obtained from Mr. Demiral on December 15, 1995, was reported as containing 0.14 pCi ± 0.038 of $^{239+240}$Pu and 0.012 ± 0.014 pCi of ^{238}Pu. though it is not known when this intake occurred, for conservatism, it was assumed that Mr. Demiral received an acute inhalation intake during the week of July 24, 1995, when a plutonium release occurred in C lab. The likely actual date of exposure was July 28, 1995, and this date was used to back calculate the intake using [illegible] models. The determination of the intake was complicated and rendered unusually difficult by the paucity of data, specifically bioassay data, available and by the rather extraordinary length of time between the likely date of exposure and the bioassay. The initial bioassay sample was not collected until December 15, 140 days post incident, and is assumed to represent a single day's excretion. This was a fecal sample which An initial and preliminary estimate of intake was made using these data and the model put forth in ICRP Publication 30. Since data on the characteristics of the aerosol are unavailable, default values of a Class Y aerosol with AMAD = 1μm were assumed, along with a single acute inhalation. The calculated intake was:

$^{239+240}$Pu: 1.3 nCi (48 Bq)
^{238}Pu: 0.11 nCi (4 Bq)

The above preliminary estimates are not inconsistent with the upper limit (a "most conservative assumption") intake of 8.1 nCi of $^{239+240}$Pu calculated by County of Los Angeles Department of Health Services and reported in their letter to you dated December 26, 1995. However, there is considerable uncertainty in the above preliminary estimates. These include the unknown characteristics of the aerosol, already mentioned as well as individual variability and the not inconsiderable uncertainty associated with calculation of intake on the basis of a single fecal sample taken several months after a suspected inhalation intake. Fecal samples are most useful as indicators of intake of insoluble (Class Y) plutonium compounds during the first few days or weeks after intake when it is reasonably certain that the activity in the feces represents short term particle clearance via the mucociliary pathway. At longer times postintake, the activity in the feces could represent material being cleared via the liver and excreted in the bile, either from the intake in question or earlier or subsequent intakes by the inhalation or percutaneous pathways; a recent exposure via the ingestion pathway; or acute inhalation exposure(s) at other time(s) than the one under consideration.

Given the lack of baseline bioassay data and other exposure history information, it is not possible to rule out these other possibilities with any degree of confidence. Accordingly, a three day simulated 24 h urine sample and subsequently, an in-vivo counting evaluation were recommended. Only the latter was performed and the results of the in-vivo counting analyses were not inconsistent with the preliminary estimate reported above. Thus, the values reported above appear to represent a reasonable estimate of plutonium intake, and include a measure of conservatism.

The 50 year committed dose equivalents from this intake were calculated in accordance with the method put forth in Publication 30 of the International Commission on Radiological Protection, using the assumptions, defaults, and estimated depositions described above for various tissues and organs and are summarized in tabular form below:

50 Year Committed Dose Equivalents (rem) from July 28, 1995
Acute Inhalation Intake of Isotopes of Pu (Aret Demiral)

Tissue or Organ	^{238}Pu	$^{239+240}$Pu	Total
Effective Dose Equivalent	0.03	0.41	0.44
Bone Surfaces	0.33	4.56	4.89
Liver	0.07	1.01	1.08
Lungs	0.13	1.54	1.67

Note: Calculated according to ICRP 30 methodology.

Leonard Hendrickson
March 14, 1996

At my suggestion, Mr. Demiral was evaluated by in-vivo counting at the Pacific Northwest National Laboratory (PNNL). These counts were performed on February 12, 1996, and revealed no detectable ^{239}Pu activity (L_c = 200 nCi) in the lungs. However, 0.39±0.26 nCi of ^{241}Am were measured in the lungs (L_c = 0.26 nCi). The time and circumstances of this intake of ^{241}Am are not known. Discussions with Mssrs. Taghizayed, Demiral and other plant personnel indicate that the plutonium source was relatively pure and hence it is unlikely that the exposure to ^{241}Am was associated with the accidental plutonium intake that occurred during the week of July 24, 1995. It is possible that the intake was incurred some time prior to January 21, 1995 as a urine sample collected from Mr. Demiral that day was reported to contain 0.059±0.28 pCi/L of ^{241}Am (L_c = .011 pCi) which corresponds to a daily excretion of 0.083 pCi. Fecal samples collected on January 22 and 23, 1995, were likewise positive for ^{241}Am. Assuming the exposure to ^{241}Am occurred on January 20, 1995, when a loss of control incident occurred, the estimated intake of this nuclide would be about 0.5 nCi based on the urine data and 0.12 nCi based on the fecal results. This level of intake is not inconsistent with the in-vivo count performed by Helgeson Scientific Services on March 27, 1995, in which no detectable activity was found, but would be too small to yield the amount of ^{241}Am measured by in the lungs by the in-vivo count of February 12, 1996.

A more likely time of intake could have been during mid-May 1995, when air sampling results indicated a loss of control and a release of this nuclide into F lab and possibly elsewhere in the facility. If the intake is assumed to have occurred on May 15, 1995, and, given that lack of specific knowledge regarding the aerosol, using standard conservative default values of solubility Class W with a particle size of 1 μm AMAD, the lung deposition measurements obtained by in-vivo counting are indicative of an intake of approximately 80 nCi. This estimate is considerably greater than estimates made using the results from the January 1996 fecal analysis which indicated a daily excretion of 0.17 pCi±0.11 (MDA = 0.15 pCi) of ^{241}Am. Assuming solubility Class W, this would be equivalent to an intake of only 5 nCi. For solubility Class Y, the corresponding intake is smaller, about 2 nCi.

Given an estimated acute inhalation intake of ^{241}Am of 80 nCi, as suggested by the in-vivo data for the lung, it is somewhat puzzling that ^{241}Am was not detected in the skeleton. Assuming solubility Class W, AMAD of 1 μm, and intake 9 months previously, the expected skeletal deposition would be on the order of several nCi, well above the lower limit detectable by in-vivo counting. And, recent publications by the United States Transuranium and Uranium Registries have shown that at long times after intake, virtually all of the systemic ^{241}Am is in the skeleton, and the clearance from the liver is considerably more rapid ($T_{1/2}$ = 2.5 y) than predicted by the ICRP ($T_{1/2}$ = 20 y).

The observations from the initial in-vivo measurements are thus more indicative of an earlier smaller intake of ^{241}Am as suggested by the excreta data. Alternatively, the in-vivo results could be interpreted as an intake of a solubility Class Y compound of ^{241}Am during May 1995. Note that if Class Y is assumed, the intake would be significantly lower, viz. about 4 nCi, which is consistent with the estimates derived from the fecal data. However, compounds of ^{241}Am are typically are cleared more rapidly from the lung and thus are considered Class W. Accordingly, additional bioassay measurements are recommended to gain additional information from which more definitive and reliable estimates of intake can be made. As a minimum, these should be an additional in-vivo counting series and a three day simulated 24 hour urine sample. Fecal sampling could also provide useful information. The excreta samples should be specifically radiochemically analyzed for $^{239+240}$Pu,

^{241}Am and ^{244}Cm.

In the interim, however, until additional bioassay data are obtained, based on the very limited information available at this time, the estimated intake of 80 nCi for ^{241}Am should be considered as representing an *upper limit*, which may well be subject to downward revision on the basis of additional bioassay data and more detailed examination of the likely circumstances of the exposure. Based on the currently available data, and in particular the excretion data, a more realistic 'best estimate of intake', would be on the order of 20 nCi, and this value is recommended as a provisional estimate of intake. An intake of 20 nCi corresponds to a 50 year committed effective dose equivalent of 8 rem and a 50 year committed dose equivalent to the bone surfaces of 18 rem, assuming solubility Class W. The corresponding doses for solubility Class Y would be somewhat lower.

The in-vivo count of Mr. Demiral revealed a possible deposition of ^{244}Cm in the liver. The results are entirely consistent with excreta analysis results obtained during January 1995 which were positive for ^{244}Cm and indicative of an intake of this radionuclide but unfortunately were not followed up or confirmed until the in-vivo counts performed by PNNL more than a year later. Using the combined results of the two 50 minute liver counts taken by PNNL, the liver deposition of ^{244}Cm was estimated 260±73 nCi, well above the reported L_1 of 47 nCi. However, there is considerable uncertainty surrounding the evaluation of the two individual 50 minute liver counts taken on Mr. Demiral. While it is proper and scientifically valid to combine the data from the two counts, the 18 keV L x-ray peaks used to quantify ^{244}Cm were absent in one count, although clearly apparent in the other. Given the possibility of interferences from the Pu and other x-rays as well as other factors that might affect the in vivo counting results, it would seem to be prudent to obtain additional in-vivo counts and a urine bioassay before making an estimate of intake for this nuclide. However, given the liver deposition estimate resulting from the in vivo counting results, the potential seriousness of the possible magnitude of the intake of this nuclide should not be underestimated, and the importance of a radiochemical validation for this element cannot be too strongly stressed.

In addition to the actinide elements discussed above, several fission product radionuclides were detected in Mr. Demiral by the in-vivo counts, most significantly 19±1.8 nCi of ^{137}Cs and 6.1±1.3 nCi of ^{138}Ba. The levels of both of these nuclides were sufficiently great as to preclude weapons test fallout or other environmental sources as a source. Since Mr. Demiral works with these nuclides periodically, it is assumed that these levels are indicative of a more or less chronic occupational exposure situation, and, given the levels detected, that the doses attributable to these nuclides are therefore likely to be relatively small. However, it is recommended that a more in depth assessment be made of these exposures, with an eye to both assignment of dose and to determining effective control strategies to obviate future exposures. Also measured was 120 nCi of the naturally occurring radionuclide ^{40}K, a normal quantity for a man of Mr. Demiral's size.

Sayed Taghizadeh

According to information provided during my visit, Mr. Taghizadeh was working with a 1 mCi plated plutonium source both inside and outside a hood in C lab during the week of July 24, 1995. Based on a reconstruction of his activities, the likely actual date of exposure was July 28, 1995, and this date was used to back calculate the intake using suitable models. As is true of Mr. Demiral, the

determination of the intake was complicated and rendered unusually difficult by the paucity of data, specifically bioassay data, available and by the rather extraordinary length of time between the likely date of exposure and the bioassay. Fecal samples were collected on December 14 and 19, 1995, approximately 140 days post incident, and was reported as containing 0.036 pCi ± 0.022 and 0.031 ± 0.021 of $^{239+240}$Pu and 0.040 ± 0.023 and 0.021 ± pCi of ^{238}Pu, referenced to 2σ. Each sample was assumed to represent a single day's excretion. The reported uncertainties are relatively large. An estimate of intake was made using these data and the model put forth in ICRP Publication 30 in the same manner as was done for Mr. Demiral. Since data on the characteristics of the aerosol are unavailable, default values of a Class Y aerosol with AMAD = 1μm were assumed, along with a single acute inhalation. The calculated intake was :

$^{239+240}$Pu: 0.51 nCi (19 Bq)
^{238}Pu: 0.25 nCi (10 Bq)

The above preliminary estimates are considerably lower than the upper limit (ie "most conservative assumption") intake of 8.1 nCi of $^{239+240}$Pu calculated by County of Los Angeles Department of Health Services and reported in their letter to you dated December 26, 1995.

The 50 year committed dose equivalents from this intake were calculated in accordance with the method put forth in Publication 30 of the International Commission on Radiological Protection, using the assumptions, defaults, and estimated depositions described above for various tissues and organs and are summarized in tabular form below:

50 Year Committed Dose Equivalents (rem) from July 28, 1995 Acute Inhalation Intake of Isotopes of Pu (Ferred Taghizadeh)

Tissue or Organ	^{238}Pu	$^{239+240}$Pu	Total
Effective Dose Equivalent	0.07	0.16	0.23
Bone Surfaces	0.68	1.78	2.46
Liver	0.16	0.37	0.53
Lungs	0.29	0.60	0.89

Note: Calculated according to ICRP 30 methodology.

At my suggestion, Mr. Taghizadeh was evaluated by in-vivo counting at the Pacific Northwest National Laboratory (PNNL). These counts were performed on February 12, 1996, and revealed no detectable ^{239}Pu activity (L_c = 81 nCi) in the lungs.

This employee was potentially exposed to ^{241}Am during the above mentioned incident in F lab in May 1995. A three day simulated 24 hour urine sample was obtained in January 1996 and Mr. Taghizadeh

underwent in-vivo counting at PNNL on February 12, 1996. The in-vivo counting results revealed depositions in the whole body of a normal level of the naturally occurring radionuclide ^{40}K for a person of Mr. Taghizadeh's size, and levels of ^{137}Cs and ^{10}Be about an order of magnitude lower than in Mr. Demiral. A skeletal deposition of 0.33±0.32 nCi of ^{241}Am (L_c = 0.19 nCi) and lung deposition of 0.31±0.13 (L_c = 0.11 nCi) were measured. Assuming that the exposure to ^{241}Am occurred during the May 15 incident in F lab, and the same default values used above, the estimated acute inhalation intake for Mr. Taghizadeh is 64 nCi which indicates a skeletal deposition estimated at about 4 nCi . If the aerosol were Class Y, the estimated intake is only about 3 nCi, with an indicated skeletal deposition of only a few tens of pCi, below the detection limit of the in-vivo assay. Given the in-vivo results as reported by PNNL, the data suggest that the aerosol solubility class lies somewhere between Y and W, and hence between 4 and 64 nCi.

The urinalysis results obtained from the January 1996 sample revealed (054±0.021 pCi/L of ^{241}Am, which, using the ICRP Publication 23 Reference Man value of 1.4 L/d for urinary excretion corresponds to a daily excretion of 0.076 pCi. This corresponds to an initial acute inhalation intake of about 36 nCi for solubility Class Y material and about 13 nCi for solubility Class W material using the ICRP 30 method and associated biokinetic parameters. These results, taken in conjunction with the calculations based on the in-vivo results, again are strongly suggestive that the solubility class lies between W and Y.

Taken together, the urinalysis results and the in-vivo counting results suggest that the ^{241}Am intake was in the range of a few tens of nCi, and likely did not exceed 40 nCi. This is consistent with results obtained with the lung model recently put forth in ICRP Publication 66, as well as with the analysis performed for Mr. Demiral. On this basis, 40 nCi is put forth as the provisional *upper limit* estimate of ^{241}Am intake for Mr. Taghizadeh, with the 'best estimate' set somewhat lower, at perhaps 15 nCi. The 50 year committed effective dose equivalent for a 40 nCi acute inhalation intake of solubility Class W ^{241}Am is 16 rem and the corresponding 50 year dose equivalent to the bone surfaces is 36.7 rem; doses for a 15 nCi intake would be proportionately lower. Additional bioassay are recommended with an eye towards validation or refinement of this initial estimate, including an additional in-vivo assessment and urine bioassay. Also recommended is a review of operations to determine more precisely when an acute exposure might have taken place.

Debolanna Roy

Levels of ^{241}Am in a urine sample submitted by Ms. Roy during January 1996 were below the stated detection limit of 0.062 pCi. It is not know why the laboratory reported a detection level for Ms. Roy five times greater than that for Mr. Taghizadeh, although presumably a smaller sample was submitted which could account for a part of the difference. If the below detection limit value reported by the laboratory for Ms. Roy were used as a basis for estimation of intake, then her intake would be estimated as 18 nCi by analogy with Mr. Taghizadeh. However, it must be remembered that statistically speaking, there is no basis for assigning any intake to Ms. Roy as the single urine sample results were below the detection limit. But, given the high detection limit, it is recommended that the urine bioassay be repeated using a three day simulated 24 hour sample so that a more definitive estimate of intake can be made; in-vivo counting is also recommended.

In summary, the intake estimates and associated doses represent conservative estimates and are based upon very limited data obtained long after the intake. The uncertainty in these estimates is large; therefore the dose estimates provided above represent conservative estimates which are in some cases upper limits. Additional data are essential if more reliable estimates are desired. It is strongly recommended that additional bioassay be performed on all three employees. Specifically, in-vivo whole body, lung and liver counts and three day simulated 24 hour urine samples with specific isotopic analysis for $^{239+240}Pu$, ^{241}Am, and ^{244}Cm should be performed and that these data be taken with an eye towards validation or refinement of the provisional estimates provided above. Additionally, fecal analyses for these specific nuclides could provide additional information useful in assessing dose, as would retrospective assessment of operations and incidents to gain further understanding and better define the times and details of the exposures. Note that the relatively long elapsed period between the likely time of exposure and the present time adds considerably to the difficulty and uncertainty of the intake estimates, and underscores the need for immediate implementation of a suitable routine bioassay program as well as an appropriate plan for bioassay and other health physics followup in the event of loss of control or other potential acute accidental exposure situations. The specifics of this program will depend on the type of work, quantities and forms of the specific nuclides used. However, on the basis of the brief observation of the your facility and subsequent discussions with you, as a minimum, the routine bioassay program should include routine quarterly alpha isotopic urine analyses for all staff working with plutonium, americium, curium and other actinides. To assure proper detection and control of potential exposures, the detection limits for plutonium and americium in urine should be $\leq$ 0.02 dpm (ie 0.01 pCi) per sample. An annual in-vivo lung and liver count is also recommended for actinide workers. For staff working with fission products, routine quarterly urinalyses are also recommended. Depending on the nuclides involved, gross beta analyses and gamma scans might be suitable. However, the detection levels for the nuclides under consideration should be sufficient as a minimum to detect $\leq$ 10 per cent of the applicable ALI. It is suggested that the urine sampling be carried out away from your facility, and a three day simulated 24 hour sample is recommended. In addition, provision should be made for prompt excreta sampling and analysis following suspected or potential accidental intakes. Note that the above recommendations are only part of an overall bioassay monitoring program, which in turn is but a part of an inclusive radiation protection program for internal emitters.

Hopefully, the evaluation provided above is clear, but, given the paucity of available data and the large uncertainties, it is inevitable that questions will arise. If so, please do not hesitate to call on me.

Sincerely yours,

Ronald L. Kathren