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Contamination Limits for Real and Personal Property

Progress Report

for the Period January to June, 1973

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scientific laboratory
of the University of California
LOS ALAMOS, NEW MEXICO 87544

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by

John W. Healy

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Division of Operational Safety.

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CONTAMINATION LIMITS FOR REAL AND PERSONAL PROPERTY

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ABSTRACT

During the initial 6 month period of this project, attention was focused on several individual problem areas, primarily through literature searches and interpretations of the data found. The first was a review of the information on the effects of radiation doses to the lung with emphasis on the key question of the possible difference in response between a uniformly distributed dose and the same energy delivered to smaller masses of tissue from radioactive particles. The second involved a study of the soil properties of importance to the transfer to man and the factors involved in wind erosion with resulting air concentrations. The third was an attempt to better define the many questions arising from mechanisms of transfer involving localized concentrations of plutonium, either from direct disturbance of the soil or from transport of the contamination to other areas. Finally, calculations were made on the air concentrations expected from wind pickup of particles from one contaminated area for comparison with measurements in this area and to further refine the calculational model.

The work on contamination limits for real and personal property sponsored by the Division of Operational Safety of the U. S. Atomic Energy Commission was first funded in mid-fiscal year 1973. As a result of previous studies, several individual projects were identified as important to assembling information and providing interpretations for use in the derivation of such limits. These projects were carried out by individuals in the Health Physics Group, The Mammalian Radiobiology Group and the Environmental Monitoring Group at Los

Alamos. The results of these studies are given in this first progress report for the project.

1. Particulate Lung Dose Effects

Current standards for limiting lung dose from internal emitters are based upon a calculation of the average dose delivered to the lung by assuming that the radiation absorption is uniform throughout the mass of the tissue. (This is sometimes called the "smear" dose but will be referred to here as the average organ dose). It is

known that this condition does not exist for most "insoluble" radioisotopes which, when deposited in the lung, provide focal spots of high level radiation close to the particle decreasing with distance in a pattern dependent upon the type and energy of the radiation. If one goes through the mathematical exercise of calculating the radiation dose rate at close approaches to an individual particle, it is found that numbers are obtained which appear to be astronomical in relation to those normally considered as permissible. Thus, a one μCi ^{32}P particle which, if the energy were averaged over the 1000 gram lung of the standard man would deliver a dose rate of 0.035 rads/day or a total dose of 0.75 rads, will deliver a dose rate of about $80,000^1$ rads per day or a total dose approaching 2,000,000 rads to the tissue at 100 μm distance from the particle. Similarly, Richmond, et al.,⁸ report an alpha particle dose rate on the order of 10 rads/hour at the surface of a 180 μm ^{238}Pu particle. The photon dose rate at the surface is given as about 500 rads/hour. Doses and dose rates drop off rapidly with distance from the particle due to inverse square and absorption relations so that the total volume of tissue involved is small.

Table I illustrates the same point with relation to the maximum permissible lung burden of plutonium and the number of particles and cells irradiated if the lung burden is divided into uniform particles of various sizes.

It is clear from these illustrations that the physical conditions of irradiation from such particulates are vastly different from the uniform distribution and that the particulate exposure results in a relatively small number of cells irradiated to differing doses ranging from very high to very low.

While one would expect differences in the outcome of irradiation of an organ by these two modes, it is not clear on a priori basis which would be the most damaging since the mechanisms of producing damage are unknown. Certainly, for acute effects occurring shortly after high levels of radiation, limiting the volume of tissue irradiated can greatly ameliorate the outcome. However, adequate data are not available to indicate whether a similar situation exists for the late effects, in particular, carcinogenesis.

It is clear that this problem is a subclass of a more general problem in arriving at radiation protection standards --

TABLE I

RELATION BETWEEN PARTICLE DIAMETER, PARTICLE NUMBER, DISINTEGRATION RATE
AND NUMBER OF CELLS IRRADIATED FOR A LUNG BURDEN OF $0.016 \mu\text{Ci}$ $^{239}\text{PuO}_2$

Diameter (μm)	Number of Particles	Disintegration Rate ($\text{d}\cdot\text{week}^{-1}\cdot\text{particle}^{-1}$)	Number of Cells Irradiated ^a
0.01	5.4×10^{10}	6.7×10^{-3}	1.4×10^{13}
0.1	5.4×10^7	6.7	1.4×10^{10}
1.0	5.4×10^4	6.7×10^3	1.5×10^7

^a For each particle size the number of cells exposed within a 40 μm alpha particle range is estimated. The total number of cells irradiated becomes the product of the number of cells irradiated per particle and the number of particles. A cell volume of $10^3 \mu\text{m}^3$ is assumed.

that of nonhomogeneous dose in any organ. In order to focus more clearly on the important question, we have considered the current limits for uniform radiation to be acceptable and have, then, asked whether there is any evidence which indicates that the nonuniform radiation to an organ, such as occurs in extreme form in the particle problem, is more or less damaging than the homogeneous radiation. Thus, the focus is on the relative, and not the absolute effect. Further, since we assumed that no clear-cut information was available, we went to the literature to see if even a tentative conclusion could be made as to whether the preponderance of the evidence indicated which assumption should be made.

A. Review of the Literature - J. Furchner

A large number of papers and reports on radiation dose to the lung and subsequent damage was reviewed. One problem with much of the work reviewed, and particularly that having to do with individual implanted sources, was the lack of consistency in the

dosimetry. Thus, some groups expressed the dose as the average to the lung while others calculated the dose at some reference distance from the source. While the original intent of this study was to attempt recalculation of the doses on a common basis, this was made difficult in some cases by the lack of data in the published article, the uncertainty of location of the source and the lack of time to complete a job of this magnitude, particularly when the initial appraisal indicated that the results would be uncertain.

For each paper of interest, or potential interest, to the present study a brief abstract emphasizing the actual data presented was prepared. Although such abstracts are of primary usefulness to those who are familiar with the original article, they are presented below as orientation to the data available. In each case, comments by the abstractor are presented in parentheses.

1. IMPLANTED SOURCES

⁶⁰Co Warren and Gates 1960²

Mice 4-6 weeks old. ⁶⁰Co wire implanted through chest wall by trochar. Wire dimension: 2 mm by 0.5 mm. Radioactivity 170-250 μ Ci. Among 190 mice that survived 97 days (time of appearance of first lung cancer) 20 had carcinoma of the lung or bronchus. The last treated mouse died 315 days after implantation. The doses ranged from 90,000 to 460,000 rep. (Trauma to the lung is unavoidable; compare with Richmond et al. 1970).⁸

⁶⁰Co Warren and Gates 1968³

Mice, rats, hamsters, guinea pigs and rabbits. ⁶⁰Co wire, 2 mm long by 0.5 mm were implanted by trochar through the chest wall. The activity ranged from 70-636 μ Ci. Most sources were between 150-200 μ Ci.

Treatment Group (Species)	Number of Animals	Median Duration of Exposure (days)		Mean Total Dose (R x 10 ³)		Malignant Cancer Incidence (%)	
		Lung	Esophagus ^a	Lung	Esophagus	Lung	Esophagus
Mice	286	180	188	262	145	20	15
Rats	20	204	202	353	121	75	30
Hamsters	25	495	368	443	178	8	28
Guinea Pigs	20	416	363	510	454	25	30
Rabbits	12	427	299	909	250	42	25

^aData do not include tumors at other sites or animals with no tumors. (As animal size increases the carcinogenic dose increases.)

¹⁰⁶Ru Laskin et al. 1963⁴

Rats. Hollow platinum cylinder plated with ¹⁰⁶Ru implanted in bronchus. 5 mm long, 1.2 mm diameter, wall thickness 0.2 mm. Hooks on cylinder maintained position in bronchus after implantation by trochar and tracheotomy.

Treatment Group	Number of Animals	Median Survival Time (Days)	Median Time to Cancer (Days)	Number Surviving 143 days ^a	Number with Lung Cancer ^b
μ Ci on implant					
0.008	37	340	430	27	2
0.057	56	310	400	39	6
0.59	57	320	380	40	15
5.0	67	225	325	42	25
13.6	48	190	315	29	20
Pt. control	60	330	---	--	--
Ru Control	22	320	---	--	--

^aTwo rats had cancer before this time.

^bSquamous cell carcinoma.

The authors rearranged the groups on the basis of calculated doses for those animals surviving 143 days.

Average Dose (Rads) ^a	Number of Animals	Squamous Cell Carcinomas	
		Numbers	%
740	6	0	0
3,400	41	3	7.3 ^b
36,000	41	9	21.9
460,000	57	33	57.9
1,600,000	32	21	65.6

^aDose calculated at "target" tissue taken as basal layer of the epithelium of the bronchus in which pellet was implanted - 100 μ m from pellet surface.

^b One tumor at 1400 rads. (There is no mention of the incidence of respiratory infections or causes of earlier deaths. The considerable trauma associated with implantation may be a factor).

¹⁰⁶Ru, ³²P Laskin et al. 1964⁵

Rats. Hollow platinum cylinder plated with ¹⁰⁶Ru implanted in bronchus. 5 mm long, 1.2 mm diameter, wall thickness, 0.2 mm. Hooks on cylinder maintained position in bronchus after implantation by trochar and tracheotomy. A single dose level of ¹⁰⁶Ru (5 μ Ci) was given. The animals were autopsied after spontaneous death and serial sacrifice. Phosphorus-32 pellets were also implanted in rats by this technique.

¹⁰⁶ Ru		Number of Animals		Mean Dose ^a (Rads x 10 ⁵)		Cancer Incidence (%)	
Sacrificed	Mean Time of Death Spontaneous	A	B	A	B	A	B
122	129	10	9	4.2	3.2	0	0
166	158	13	18	4.3	4.5	15.4	11.1
198	193	10	10	5.2	5.2	30.0	20.0
212	233	10	10	5.2	5.5	60.0	50.0
225	300	16	13	5.4	6.9	81.3	84.6
247	347	10	11	6.3	8.0	90.0	81.8
282	378	9	9	6.5	8.6	88.9	88.9
357	424	8	9	7.7	9.2	100.0	100.0

(No cancers before 158 days. Only 4 animals in 31 had lung cancers after doses of 4 x 10⁵ Rads over 160 days. Again no mention of chronic respiratory infection is made).

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Lung Dose^a (Rads)</u>	<u>Lung Cancer %</u>
20.0 μ Ci	18	4×10^5	56
2.0 μ Ci	15	4×10^4	33
0.2 μ Ci	15	4×10^3	0

(The ^{32}P dose response agrees with the ^{106}Ru data from Laskin et al. 1963, better than does the ^{106}Ru data in this paper).

^a See note on Laskin et al. 1963 for meaning of dose calculation.

^{106}Ru Divertie, Titus and Shorter 1967⁶

Rats 150-200 g. Silicone rubber pegs (2.5 mm x 1 mm) impregnated with 50 μ m ceramic spheres containing ^{106}Ru were inserted into a bronchus via tracheotomy. Twelve control animals had inert pegs inserted into a bronchus. Thirteen of 16 rats receiving radioactive pegs had squamous cell carcinomas. None were found in controls. No doses are given.

ANIMALS WITH SQUAMOUS CELL CARCINOMA

<u>Duration of Exposure (wks)</u>	<u>18</u>	<u>19</u>	<u>20</u>	<u>21</u>	<u>23</u>	<u>25</u>	<u>30</u>	<u>34</u>	<u>34</u>	<u>35</u>	<u>36</u>	<u>36</u>	<u>38</u>
μ Ci Dose													
Inserted	15.6	14.0	10.0	13.4	13.4	13.4	11.3	8.1	14.8	12.6	13.4	12.6	14.1
Recovered	11.3	10.1	7.5	10.0	9.6	8.1	7.6	6.0	9.4	7.6	8.1	7.6	8.3

In two of the three experimentals no pegs were recovered. Pneumonitis was usually found distal to the pegs.

^{90}Sr Altmann, Hunstein and Stutz 1961⁷

Rats. Plexiglass capsules containing ^{90}Sr were sewed to the underside of the diaphragm. The activity range was 27-62 μ Ci with most values between 39 and 48 μ Ci.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Carcinoma</u>	<u>Lung Tumors Number Sarcoma</u>	<u>Adenoma</u>
<u>Exposure Time (Months)</u>				
0 - 3	28	0	0	0
3 - 6	21	2	0	0
6 - 9	48	8	0	0
9 - 12	36	30	1	0
over 12	31	26	1	1

The last two groups also had nonpulmonary tumors. (There is no clue to dose data given).

238 PuO₂ Richmond, Langham and Stone 1970⁸

Rats, male, 325 g. Injection of PuO₂ spheres via femoral vein. Spheres 122-200 μ m in diameter. The spheres are trapped in the capillary network of the lung. The animals were sacrificed serially and examined histologically. Note this method involves no surgical trauma to the lung.

Time of Sacrifice (Days)	1	3	7	14	21	30	60	90	120	152	180	211
No. of Animals	3	2	2	2	2	6	10	5	9	6	7	6

The surface dose rates were 10⁸ and 10³ rad/hr for the alpha and gamma radiations respectively. A sphere of cellular debris and collagenous tissue surrounded the spheres. A footnote, added in proof, states that animals at 600 days postinjection show histological changes qualitatively similar to those seen at 90, 120, and 211 days. No tumors were found.

90 Sr Cember and Watson 1958⁹

Rats, male, 286 g. Glass beads with incorporated ⁹⁰Sr were implanted with a hypodermic needle through the chest wall. The beads were 320 $\pm$ 110 μ m in diameter and contained from 1.09 to 59.3 μ Ci. Dose data was given only for tumor bearing rats. The mortality data for the inert Sr and saline control groups was much the same as that for the experimentals and was due to injection trauma.

Dose Rate (Rad/day)	Exposure Time (Days)	Total Dose (Rads x 10 ⁴)	Tumor
160	561	9.0	Lymphosarcoma
160	487	7.8	Squamous cell carcinoma
220	561	12.0	Squamous cell carcinoma
277	169	4.7	Lymphosarcoma
277	545	15.0	Lymphoma
440	581	26.0	Squamous cell carcinoma
660	333	22.0	Squamous cell carcinoma

The first death occurred at 131 days and the last at 575 days post injection. (4 of 23 rats had squamous cell carcinomas at doses of $\sim$ 10⁵ Rads).

2. INHALATION OR INTRATRACHEAL INJECTION (Beta Emitters)

³⁵S Cember et al. 1955¹⁰

Rats, female, 125-200 g. A single intratracheal injection of Ba³⁵SO₄ particles (1.45 μ m $\pm$ 0.40 μ m).

Treatment Group	Number of Animals	Dose to Lung (rep)
4.5 μ Ci	23	58
45 μ Ci	21	3,200
4,500 μ Ci	38	24,000
Controls	25	-

Rats were killed serially in a 9 month period. No tumors were found in any group. (The calculated doses were delivered almost entirely during the first month). Chronic and acute inflammation were common.

³⁵S Cember and Watson 1958¹¹

Rats, female, 244 g. Intratracheal insufflation of 375 μ Ci of Ba³⁵SO₄ once a week for 10 consecutive weeks. Particle size 1.45 μ m $\pm$ 0.40 μ m. Dose estimated to be between 12,000 and 20,000 Rads.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Number Surviving 10 weeks</u>	<u>Number Dead at 500 days</u>	<u>Squamous Cell Carcinoma</u>
Colony control	14	--	2	0
Inert BaSO ₄	24	17	10	0
3,750 μ Ci	24	16	24	2

The tumors were found in rats that died at 312 and 319 days.

¹⁴⁴Ce Cember et al. 1959¹²

Rats, male, 279 g. ¹⁴⁴CeF₃ by intratracheal injection. Particle size 1.0 μ m, Std dev 1.4.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Number of Survivors^a</u>	<u>Days to First Tumor</u>	<u>Lung Dose (Rads)</u>	<u>Number with Squamous Cell Carcinoma</u>
Colony control	20	--	--	0	0
Inert CeF ₃	29	--	--	0	0
5 μ Ci	27	10	178	2,400	1
15 μ Ci	23	21	48	5,100	1
25 μ Ci	28	19	93	10,700	7
50 μ Ci	15	6	83	21,000	4

^aTo observation of first tumor. Severe, acute pneumonia appeared in the two high dose Groups within several days. The first tumor appeared in 48 days. (Mortality of the inert CeF₃ group was not given nor was the duration of the experiment).

¹⁴⁴Ce Cember 1963¹³

Rats, male, 283 g. ¹⁴⁴CeF₃ by oral, intratracheal injection. Particle size 1.0 μ m std dev 1.4.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Days to First Tumor Death</u>	<u>Lung Dose^a (Rads)</u>	<u>Primary Lung Tumors</u>	<u>Squamous Cell Carcinoma</u>	<u>Undifferentiated Carcinoma</u>	<u>Adeno-Carcinoma</u>	<u>Lymph-oma</u>
Inert CeF ₃	29	--	--	0	0	0	0	0
0.5 μ Ci	41	528	600	8	3	3	3	1
1.0 μ Ci	44	367	1,100	6	2	1	3	0
2.0 μ Ci	34	620	2,500	4	2	1	1	2
4.0 μ Ci	42	381	4,400	14	11	1	2	0

^aDose at death with first tumor. Earliest tumor at 361 days. (Experiment lasted at least 1,033 days. No mention of chronic pulmonary disease was made).

¹⁴⁴Ce Cember and Stemmer 1964¹⁴

Rats, male, 258 g. ¹⁴⁴CeCl₃ solution given by intratracheal injection of 0.15 ml by mouth.

Treatment Group	Number of Animals	Number of Survivors ^a	Days to First Lung Tumor	Lung Dose (Rads) ^b	Number of Primary Lung Tumors
Inert CeCl ₃	21	21	--	--	-
10 µCi	68	61	306	14,000	9
15 µCi	55	52	197	19,600	31
30 µCi	58	37	70	25,000	27

^aTwo month survivors

^bDose at death with first tumor

(Dosimetry differs somewhat from Cember et al. 1959, where the dose rate for 5 µCi ¹⁴⁴CeF₃ is given as 59.6 rads/day in a 1.5 g lung. Here the dose rate is given as 28 rads/day for 1 µCi/g. Cember considers all the data for ¹⁴⁴Ce given in Cember et al. 1959,¹² Cember 1963,¹³ and Cember and Stemmer 1964⁴ in Cember 1964.^{15,16}

¹⁴⁴Ce Cember 1964^{15,16}

Values given here are estimated from Fig. 39, Cember 1946a¹⁵ and from Fig. 5, Cember 1964b.¹⁶

<u>1964a</u>		<u>1964b</u>	
Dose (Rads)	Tumor Frequency (%)	Dose (Rads)	Tumor Frequency (%)
660	2.2	650	1.5
1,300	4.0	1,200	2.5
2,500	5.6	4,500	5.0
5,500	9.5	14,000	10.0
15,500	12.5	20,000	12.5
26,000	22.2	41,000	22.5
43,500	29.8	49,500	25.0

¹⁴⁴Ce Hahn et al. 1973.¹⁷

Beagles. By inhalation of ¹⁴⁴Ce fused in clay particles. ~1.4-2.7 A.M.A.D., g std dev 1.5-2.3. To date 15 of 126 beagles are dead of fibrosis and pneumonitis at 143-410 days; and 5 are dead of pulmonary neoplasia at 750-1,318 days.

<u>Initial Lung Burden</u>		<u>Time To Death (Days)</u>	<u>Dose to Lung At Death (Rads)^a</u>	<u>Lung Pathology</u>
µCi/kg	Total µCi			
26	230	765	27,000	Hemangiosarcoma
27	190	1,185	23,000	Hemangiosarcoma
34	330	1,318	36,000	Hemangiosarcoma
35	380	916	34,000	Hemangiosarcoma + bronchiolo-carcinoma
46	470	750	48,000	Hemangiosarcoma + fibrosarcoma
33	320	193		Pneumonitis + fibrosis, no tumors
41	330	185		" " "
51	440	410		" " "
53	440	279		" " "
56	520	273		" " "
65	590	234		" " "
66	470	246		" " "
66	540	257		" " "
68	600	186		" " "
96	740	189		" " "
120	890	171		" " "
180	2,000	182		" " "
190	1,500	173		" " "
190	1,700	181		" " "
210	1,700	143		" " "

^aCalculated by Hahn et al. (It appears that more than 700 days must elapse before tumors are found).

¹⁴⁴Ce Kurshakova and Ivanov 1962¹⁸

Twenty rabbits 2.5 - 3.0 kg were injected with 25 μ Ci of ¹⁴⁴CeF by piercing the anterior wall of the trachea through the skin. The particle size was 0.025 μ m. One rabbit died of bronchopneumonia on the 3rd day. Half of the rabbits died between the 60th and 238th days of sclerosis, bronchiectasis, etc. Tumors were found in 6 of the animals surviving to 238 days. The last tumor was found at 327 days. The doses to the lungs at 238 and 327 days were 51.4 and 68.9 kilorads respectively. There were 5 bronchogenic and alveolar lung cancers and one squamous cell carcinoma of the esophagus.

¹⁰⁶Ru Temple et al. 1960¹⁹

Mice, female. ¹⁰⁶RuO₂ in Tween-80 was injected intratracheally.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Days After Administration</u>	<u>Adenomas %</u>	<u>Number of Malignant Tumors</u>	<u>Dose (Rads)</u>
Colony Control	28	403-470	78	0	0
Inert RuO ₂	21	335-500	9	0	0
3.0 μ Ci	23	350	82	1 Bronchiolar carcinoma	9,000
1.93 μ Ci	11	369-422	90	1 Bronchiolar carcinoma	4,500
0.15 μ Ci	10	340	--	1 lympho sarcoma	300

(The natural incidence of adenomas is a factor of unknown importance to radiation carcinogenesis).

¹⁵²⁻¹⁵⁴Eu Berke and Deitch 1970²⁰

Rats, female, 180-200 g. Rats were made to inhale aerosols of radio-europium chloride for 7 h/day, 5 days/wk for 6 months. The particles were characterized only as "submicronic". The lung dose varied with time and was as much as 6×10^4 rads at 720 days. No animals were free of pulmonary pathology. Severe chronic inflammatory changes and lung abscesses were present in the majority of the animals. There was a complete absence of pulmonary neoplasia.

²⁴Na, ³²P, ⁵⁹Fe, ¹⁹⁸Au Kochetkova et al. 1959²¹

Rats. These isotopes were given by intratracheal injection. Particle size unspecified.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Beta Dose (Rads)</u>	<u>Metaplasia of Bronchial Epithelium</u>		<u>Lung Cancer</u>	
<u>μCi</u>		<u>First Day</u> <u>Total</u>	<u>Number</u>	<u>Months</u>	<u>Number</u>	<u>Months</u>
⁵⁹ Fe ₂ O ₃ 1-27	52	10-20 500-5000	17	6-9	8	6-9
³² P ₄ 40-100	76	300-700 1300-15000	20	2-12	11	6.5-18
¹⁹⁸ Au 100-150	30	1400-2100 5400-8000	4	1-3	3	2.5-12

100-200 μ Ci of ²⁴NaCl in single and multiple doses produced no tumors.

³²P Kochetkova and Avrunina 1957²²

Rats - Intratracheal injection of Cr³²PO₄. No particle size specified.

Treatment Group (μ Ci injected)	Number of Animals	Mean Lung Burden (μ Ci)	Lung Dose (Rep) 24 h	Life Span (Days)	Pathology
270	10	180	2,900	19,000-46,000	3-32 Metaplasia
100	42	81.5	830	10,000-18,000	19-65 Metaplasia
70	34	54	670	8,000-16,000	15-395 3 Squamous Cell Cancers
40	10	38	350	4,500- 7,400	60-451 3 Squamous Cell Cancers

There were no tumors after single and multiple injections of ²⁴NaCl (200-1,900 μ Ci). Of 25 rats that received 320 μ Ci of radiogold all died in 2.5 months. Three of these rats had squamous cell cancer. The doses were in 9,000-9,700 rep range.

3. INHALATION OR INTRATRACHEAL INJECTION (Alpha)

²³⁹Pu Temple et al. 1960¹⁹

Mice (BAF) PuO₂ suspended in Tween-80 or Pluronics for injection. Particle size 0.6-0.06 μ m mean 0.5 μ m.

Treatment Group	Number of Animals	Days After Administration	Dose (Rads)	Lung Tumor
Colony Control	28	400	---	22 Adenomas
0.16 μ Ci	41	100	4,000	1 Bronchiolar carcinoma
0.06 μ Ci	17	400	2,300	2 Squamous cell carcinomas
0.003 μ Ci	21	500	115	1 Fibro sarcoma

At 400 days 78% of the colony controls had adenomas. The fibrosarcoma at the 0.003 level was considered non-radiogenic (the use of surface active agents as vehicles for the particles is a factor of unknown importance).

²¹⁰Po Yuile, et al. 1967²³

Rats, male, exposed once to an aerosol of ²¹⁰Po as the chloride, particle size: geometric mean 0.098 μ m, geometric std dev 1.81.

Treatment Group	Number of Animals	Number of Deaths	Age Range At End (wks)	Lung Dose ^a (Rads)	Primary Lung Tumors	Squamous Cell Carcinomas
NaCl Control	147	88	87-100	0	0	0
0.15 μ Ci	119	119	106	538	22	17
0.05 μ Ci	129	98	95-100	202	15	5
0.02 μ Ci	132	71	89-91	71	4	1

Dose accumulated at 280 days - little increase thereafter. The aerosol was a NaCl solution acidified to a pH of 1. Pulmonary infection was endemic in the colony and an epidemic of acute pneumonia occurred during the second year. The experiment was terminated when the last high-dose animal died at the 96th week.

²³⁹Pu Wager et al. 1955²⁴

Mice (BAF) female - Intratracheal injection with Tween 80. Particle size 0.05 to 0.6 μ m. Of 10 mice that received 0.06 μ Ci of ²³⁹PuO₂, 3 had squamous cell carcinoma at 1 year post-injection.

²¹⁰Po Scott and Thomas 1957²⁵

Rats. Intratracheal injection of ²¹⁰Po nitrate solution. Experiment terminated at 15 months when there were 5 survivors.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Squamous Cell Carcinomas</u>	<u>Time to Tumor (weeks)</u>
10 μ Ci/kg	15	0	----
5 μ Ci/kg	15	2	5, 15

All animals exhibited varying degrees of murine pneumonia.

²¹⁰Po Little et al. 1970²⁶

Syrian golden hamsters. ²¹⁰Po adsorbed on 3 mg of Fe₂O₃ particles (98% < 0.75 μ m): suspended in saline given in 15 consecutive weekly intratracheal injections.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>Number of Dead Animals</u>	<u>Current Week</u>	<u>Tumor Bearing Animals</u>		<u>Total Dose at 2 yr (Rads)</u>
				<u>No.</u>	<u>% First Tumor</u>	
Control	63	52	93	0	0	----
Fe ₂ O ₃ only	32	30	93	0	0	----
0.2 μ Ci/wk	35	35	60	32	91	15th wk
0.01 μ Ci/wk	34	21	59	10	30	40th wk
						4,500
						225

The number of animals consists of the survivors of the 15-week treatment period which were autopsied (60 animals/group at start). The doses given are maxima-carcinogenic doses which are less than 225 rads.

²¹⁰Po Grossman et al. 1971²⁷

A later report on Little et al. 1970²⁶ gives the incidence of bronchogenic tumors as 91% and 43% in the high and low dose groups respectively. Syrian golden hamsters were given intratracheal injections twice weekly for 7 weeks. The doses were given in two separate intratracheal instillations (a and b in table below).

<u>Treatment Group</u>		<u>47 wk Survivors %</u>	<u>Tumors at 27 wk</u>
<u>a</u>	<u>b</u>		
3 mg Fe ₂ O ₃	0.2 μ Ci in saline	2	17
Saline	0.2 μ Ci	6	9
Saline	0.2 μ Ci on 3.0 mg Fe ₂ O ₃	28	7
Saline	0.2 μ Ci on 0.3 mg Fe ₂ O ₃	32	3

²¹⁰Po alone is said to be homogenously distributed in the lung.

²³⁸U Leach et al. 1970²⁸

Monkeys, dogs, and rats were exposed to UO₂ dust (M.M.D. 1.03 μ m, g std dev 2.40) 5 mg/m³ for 6 h/day, 5 days/wk. The rats, after an exposure of 1 year, showed no pathological changes in the lung apart from pigmented macrophages in the alveoli and bronchi. In dogs there were no pathological changes in the lung after 5 years of exposure and estimated radiation doses of 400 rads. Monkeys responded with a patchy hyaline fibrosis that first appeared at 3.6 years after a dose of 500 rads. No tumors were reported in any animals at the end of the 5 year exposure. Despite doses to the tracheo-bronchial lymph nodes of dogs and monkeys that were on the order of 10⁴ rads no pathology other than an occasional necrosis and fibrosis were reported.

²³⁹Pu Antonchenko et al. 1969²⁹

Rats, 140-160 g, were exposed to an aerosol (90% 0.7-1.9 μ m, median diameter 1 μ m) of Pu citrate or ammonium plutonium pentacarbonate. (pH 5 and 8, respectively) for 20 min.

Treatment Group	Number of Animals	Average Life Days	Average Life Dose (rads)	Lung Pathology (%)			
				Carci-noma	Adenoma	Adenoma-like Structures	Epithelial Metaplasia
Controls	248	673	0	---	---	----	---
<u>Citrate</u>							
1.028 μ Ci ^a	25	64	3,820	---	---	----	9.1
0.803 μ Ci	12	69	3,090	---	---	----	8.3
0.511 μ Ci	94	124	2,370	2.2	---	8.9	73.4
<u>Carbonate</u>							
1.460 μ Ci	12	77	7,320	---	---	----	9.09
0.774 μ Ci	23	78	3,900	---	---	4.4	13.0
0.455 μ Ci	69	139	2,780	4.6	3.08	12.0	61.6

^aInitial deposition. (Apparently the short survival time in the higher dose groups precluded the development of the characteristic pathology).

²³⁹Pu Buldakov et al. 30

Rats. Inhalation of soluble Pu compounds: citrate and ammonium pentacarbonate.

	Treatment Group (μ Ci deposited)	Number of Animals	Mean Survival (Days)	Lung Dose (Rads)	Lung Tumors %
Citrate	0.008	157	635	47	5
	0.02	124	585	117	2.5
	0.04	203	545	234	8.4
	0.08	31	546	467	35.5
	0.15	105	464	852	23.8
	0.25	113	416	1,390	23.0
	0.36	39	221	1,740	7.7
	0.51	90	124	2,370	0
	0.80	12	63	3,090	0
	1.03	20	64	3,820	0
Ammonium-	0.004	48	571	41	4.2
Plutonium-	0.007	101	571	80	5.0
Penta-	0.017	91	584	186	13.2
Carbonate	0.045	126	582	497	36.4
	0.15	83	484	1,065	42.7
	0.25	126	361	1,615	26.4
	0.35	22	247	2,140	9.0
	0.46	65	139	2,780	0
	0.77	23	78	3,900	0
	1.46	11	77	7,320	0

The tumors were squamous cell carcinomas, adenocarcinomas and hemangiomas. Note the tumor incidence at low doses and the absence of tumors at high doses.

²³⁹Pu Clark et al. 1964³¹

Dogs inhaled particles (0.5-0.65 μ m). At 855 days 28 were dead. There was one lung tumor at 150 days. Six more died between 855 days and 1,446 days, of these, four had bronchiolo-alveolar tumors. The estimated doses were between 9,000 and 23,000 rads, resulting from burdens of 0.6 to 19 μ Ci.

²³⁹Pu Park et al. 1967³²

Dogs inhaled particles (0.5-0.65 μ m) (continued from above). Of 25 dogs dying or sacrificed between 850 and 2,270 days, 12 had primary pulmonary tumors. The estimated doses to the tumor bearing animals ranged between 3,100 and 13,600 rads, resulting from terminal lung burdens of 0.5-2.7 μ Ci.

²³⁹Pu Park et al. 1972³³

Dogs inhaled PuO₂ particles (0.5-0.65 μ m) (continued from above). Of 65 dogs exposed, 62 are dead, and 24 had pulmonary neoplasia. Between 55 and 1,600 days, 36 died of pulmonary insufficiencies (edema, fibrosis, hyperplasia etc.) Twenty of 21 dogs surviving 1,600 days had lung tumors. Estimated initial lung burdens were 0.2 to 3.3 μ Ci. At 11 years the average dose to the lungs of tumor-bearing animals was in the 2,000-12,000 rads range.

²³⁸Pu Park et al. 1970³⁴

Twelve dogs inhaled ²³⁸PuO₂ particles: CMD 0.05 μ m GSD 1.9.

<u>Terminal Burden</u> <u>(μCi)</u>	<u>Lung Burden</u> <u>(% Terminal Burden)</u>	<u>Survival Time</u> <u>(Days)</u>
261	92	27
167	94	30
168	93	35
112	92	56
74	91	56
140	94	61
84 ^a	90	70
89 ^a	90	76
58	91	77
44 ^a	91	94
17 ^a	80	125
25	77	180

^aanimals with lung tumors: bronchiolo-alveolar carcinoma. Dose range, all animals, 8,000 to 26,000 rads. Almost total necrosis of tracheobronchial, mediastinal, and sternal lymph nodes.

²³⁸Pu C. L. Sanders 1973³⁵

Rats, female, were exposed to an aerosol of crushed ²³⁸PuO₂ microspheres: CMD 0.02, GSD 2.1. The material was considered soluble (72% ultrafilterable). Life-time study (>1,000 days).

<u>Treatment</u> <u>Group</u>	<u>Number of</u> <u>Animals</u>	<u>Lung Dose^a</u> <u>(Rads)</u>	<u>Lung Tumors</u> <u>%</u>	<u>Median Life</u> <u>Span (days)</u>
Control ^b	92	0	1.1	825
5 nCi ^b	30	9	6.6	~ 650
18 nCi ^b	30	32	23.3	675
230 nCi ^b	30	375	25.0	550

^a mean dose in 2 years

^b mean initial lung deposition

Lung tumor incidence in the 5 nCi group was not significantly different from lung tumor incidence in the control group. Of the 19 pulmonary tumors found, 14 were bronchiolo-alveolar carcinomas, 2 were mixed carcinomas and there was one epidermoid carcinoma, one undifferentiated carcinoma and one lymphosarcoma. The author concludes: " - - - that spreading the Pu dose in the lung, as compared to concentrating in PuO₂ particles, is more carcinogenic due to the greater number of epithelial cells 'hit' by alpha emissions from Pu".

²⁴¹Am Thomas et al. 1972³⁶

Dogs were exposed to an aerosol (AMAD 0.9 μ m, GSD 1.5).

Lung Burden (μ Ci)		Days to Sacrifice	Lung Dose (Rads)	Lung Pathology
Initial	At Sacrifice			
31	2.3	127	3,000	Inflammation
21	1.0	256	3,200	Fibrosis
26	0.71	512	3,800	Fibrosis and mineralization
23	0.38	1,022	5,300	Fibrosis and mineralization

Doses to lung were delivered early; more than 90% of the ²⁴¹Am had left the lung by 127 days. The highest doses were delivered to the tracheobronchial lymph nodes (3,500-17,400 Rads), but the chief pathologies were fibrosis in the medullary areas and depletion of lymphoid elements.

²³⁷Np Levдик et al. 1971³⁷

Rats were injected intratracheally with nitrate and oxalate solutions of ²³⁷Np (pH 2-3 and 5, respectively).

Treatment Group (μ Ci/kg)	Number of Animals	Average Life Span(Days)	Dose (Rads)	Lung Tumor Incidence (%)	
				Malignant	Benign
Control	274	700	0	3.65	0.36
<u>Nitrate</u>					
0.017	50	660	5	16.0	2.0
0.083	44	684	28	20.5	4.65
0.41	48	685	138	12.68	8.35
2.0	49	505	2,500	14.28	2.1
<u>Oxalate</u>					
0.017	85	645	27	10.6	0.0
0.083	89	661	134	9.0	3.36
0.41	89	649	671	28.0	5.6
2.0	81	453	3,220	37.45	3.75

Part of the increased carcinogenicity is attributed to the chemical toxicity of Neptunium.

4. EXTERNAL IRRADIATION

X-ray Koletsy and Gustafson 1955³⁸

220 kV, 15 ma, filters: 1.0 mm Al, 0.5 mm Cu, 60 R/min.

Rats, male, 200 g were exposed to a single total dose of 660 R of whole-body radiation. The 123 rats that survived 6 months or more were autopsied at death.

Treatment Group (Time of Death)	Number of Deaths		Number With Tumors	
	Irrad.	Con.	Irrad.	Con.
6-12 months	46	3	7	1
12-18 months	47	6	32	0
18-24 months	29	14	29	2
over 24 months	1	13	1	5

One rat had primary carcinoma of the lung, an undifferentiated carcinoma in the lower left lobe. The right lung had an adenocarcinoma. The time of appearance is not given.

X-ray Cember et al. 1956³⁹

100 Kv, 4 ma, filters: 1 mm Al. 61.8 R/min.

Rats, female, 270 g; equal doses on 5 consecutive days. Only the thoracic region was exposed. The rats were rotated 4 times during each exposure.

<u>Treatment Group</u>	<u>Number of Animals</u>	<u>First Death (wks)</u>	<u>Median Lethal Time</u>	<u>Last Death</u>	<u>Tumors</u>
Control	20	No losses	---	---	---
5,750	20	6	12 months	6 Sac. @ 15 months	2 lymphoma
11,500	20	3	166 days	11 months	1 lymphoma
17,250	15	3	37 days	6 months	1 lymphoma

The primary loci of the tumors is uncertain because of metastases. Broncho-pneumonia was the most common finding.

X-ray Maisin et al. 1958⁴⁰

250 kV, filters: 1.0 mm Al, 0.25 mm Cu. 90 R/min.

Rats, 145-165 g were exposed to single doses of whole or partial body irradiation. Only animals alive at 6 months post exposure were considered. These survivors were autopsied at death.

<u>Treatment Group</u>	<u>Number Autopsied</u>	<u>Total Number of Cancers</u>	<u>Leuco-Sarcomas</u>	<u>Epitheliomas</u>	<u>Sarcomas</u>	<u>Epitheliomas Sarcomas</u>
Control	460	13	10	2	1	---
300-2,000 R	1,237	100	24	52	21	3
2,000 R left lung	10	5	--	3	2	---

Only one rat (cervico-sternal shield - 600 R) receiving less than 2,000 R had a pulmonary cancer (at 15 months post exposure). Of the 5 cancers in the 2,000 R group, which had the entire body, save the left lung region, shielded, 3 were bronchial epitheliomas appearing at 11, 15, and 23 months. There were 2 sarcomas of the left thoracic wall. No other pulmonary pathologies were reported.

X-ray Castanera et al. 1968⁴¹

200 kV, 15 ma, filters: 1.0 mm Al, 0.5 mm Cu. 27 R/min.

Rats, male, free of epidemic respiratory infection were exposed to single, whole-body irradiation with x-rays or fast neutrons (12 Mev H⁺ on Be).

<u>Treatment Group (Rads)</u>	<u>Number of Animals</u>	<u>Time of Last Death (Days)</u>	<u>Adenomas (%)</u>	<u>Adenocarcinomas (%)</u>
<u>X-ray</u>				
430	88	700-800	16	3
680	107	600-700	11	4
<u>Neutrons</u>				
230	41	500-600	17	7
320	73	500-600	10	6
Control	129	800-900	1	0

Tumor frequency was estimated from chart 5. Operable tumors were removed surgically. There were no other primary pulmonary tumors.

Castanera et al. 1971⁴²

Male rats were given a single whole-body exposure to fast neutrons (12 Mev H⁺ on Be). The rats were free of epidemic respiratory infections.

Treatment Group		Number of Animals	Median Survival Time (Days)	Primary Lung Tumors	
Age (months)	Dose (Rads)			Benign	Malignant
1	215	79	433	13	3
1	0	40	699	5	0
3	230	41	436	17	7
3	0	41	601	0	0
21	215	53	167	8	2
21	0	24	158	4	0

All tumors were bronchiolar in origin. Multiple tumors were found in other organs.

X-ray DeVilliers and Gross 1966⁴³

135 kV, 4 ma, filters: 2.43 mm Al. ~100 R/min.

Male Syrian golden hamsters and male rats were exposed to 5 equal doses of x-rays delivered on 5 consecutive days. A collimated beam was directed at the chest region. Four portals were varied through 90° per day. Hamsters received 4,000 R, rats 3,570 R.

	Time of Sacrifice (Months)	Number of Animals	Tumors	
			Adenomas	Malignant
<u>Rats</u>	4	12	1	---
	8	11	---	---
	12	12	2	1 Adenocarcinoma
	24	12	2	2 - Reticulum cell sarcoma Squamous cell carcinoma

Post Irradiation Time (weeks)

	<u>6</u>	<u>7</u>	<u>10</u>	<u>11</u>	<u>12</u>	<u>13</u>	<u>14</u>	<u>15</u>	<u>16</u>	<u>17</u>	<u>18</u>	<u>19</u>
Hamsters												
Spontaneous Deaths	1	1	2	3	3	13	6	4	2	2	2	3
Squamous Cell Cancer	-	-	-	-	-	2	-	2	1	-	-	-

Fifty-seven hamsters were irradiated, all died spontaneously. Only those dying between 6 and 20 weeks (42) are listed. Pulmonary cancers were not found later than 3 1/2 months, although 4 at 6 months and at 12 months and 13 at 24 months, whereas 7 of the eight tumors found in rats were found at 12 or 24 months.

X-ray Gross et al. 1969⁴⁴

3,000 R, 110 kV, 6.4 ma, filters: 1.83 mm Al. 75 R/min and 4,000 R, 110 kV, 8.6 ma, filters: 1.83 mm Al. 100 R/min.

Rats and hamsters were exposed to a collimated beam of x-rays directed at the chest region. The animals were rotated axially at 7 rpm during exposure which was given in 5 equal doses on 5 consecutive days. Eight weeks after exposure some animals were treated with dimethyl benzantracene (DMBA) and/or jewelers rouge (Fe_2O_3).

<u>Treatment Group (Rats)</u>	<u>Number of Animals</u>	<u>9 Month Survivors</u>	<u>Adeno- carcinoma</u>	<u>Squamous Cell Carcinoma</u>	<u>Fibro- sarcoma</u>	<u>Undiffer- entiated</u>
<u>4,000 R</u>						
DMBA + Fe_2O_3	40	37	15	3	1	1
Fe_2O_3	40	29	7	0	0	1
Radiation only	43	42	18	4	1	1
<u>3,000 R</u>						
DMBA + Fe_2O_3	40	39	14	2	1	0
Fe_2O_3	40	39	16	0	0	0

Data for the appropriate controls are not tabulated. In the unirradiated rats Fe_2O_3 with or without DMBA caused no tumors. DMBA did not increase the prevalence of cancer in irradiated rats.

<u>Treatment Group (Hamsters)</u>	<u>Number of Animals</u>	<u>2.5 Months Survivors</u>	<u>Number of Cancers</u>	<u>Time To Tumor(Months)</u>
<u>4,000 R</u>				
DMBA + Fe_2O_3	40	37	2	2.5, 7.5
Fe_2O_3	38	36	0	---
Radiation only	46	46	1	11
<u>3,000 R</u>				
DMBA + Fe_2O_3	38	38	0	---
Fe_2O_3	38	37	1	14
Radiation only	8	8	0	---
<u>Control</u>				
DMBA + Fe_2O_3	40	35	2	9.7, 18.5
Fe_2O_3	20	19	0	---
No treatment	10	10	0	---

The first appearance of a cancer was the base for the selected survival times (column 3). Only frank malignancies are included, microlesions were not considered. Chronic bronchitis was prevalent. (Note the decreased incidence of cancer among hamsters compared with that of DeVilliers and Gross⁴³1966).

B. Discussion - J. W. Healy

Prior to World War II and the Manhattan Project, radiation exposure limits had been derived for X-rays or radium gamma rays, for radon in the air and for radium as an internal emitter.* The external limits were based on the radiation field to which the individual was exposed with little or no consideration of the distribution of radiation through the body or of the exposure of specific organs.** During the Manhattan Project, the need for considering radiations other than X- or gamma, the presence of varying energies of radiations and the availability of a wide variety of radioactive chemical species resulted in the extrapolation of these limits to the new conditions through the derivation of new concepts (such as the rem) and an increased sophistication in dosimetry as applied to individual organs.

Following the war, considerable attention was given to formalizing these concepts in a manner which could be used by those responsible for guiding radiation protection practices in the vastly increased uses of radiation and radioactive materials resulting from nuclear energy. This work was carried out by the NCRP in consultation with foreign scientists through conferences and informal discussions. In 1954, the NCRP subcommittee on Permissible Internal Emitters published their report⁴⁵ that first expounded on the critical organ concept which has served as

*For an excellent review of the information available on the effects of internal radiation on humans at the time of World War II, the reader is referred to "The Tolerance Dose" MDDC 1100 by C. T. Cantril and H. M. Parker.

**In this statement we are referring to the official limitations adopted by the NCRP and the ICRP. Individuals did concern themselves with these matters in reviewing the data available and in applying the limits.

the basis for the majority of the internal emitter limitations.* Here the critical organs were defined on the basis of experience with external radiation. The skin was chosen as one organ because of the production of skin cancers, usually on the hands from the greater exposure which they received. The increased incidence of leukemia in radiologists led to the designation of the blood-forming organs as one of the more important critical organs, while cataracts produced by high LET radiations resulted in the lens of the eye receiving special designation. Since leukemia was the primary outcome from whole body radiation in the experience available, it was considered " - - safe to assume at present that the blood-forming organs constitute the most critical organs".** Exposure to the more deeply seated organs was then limited to that of the blood-forming organs.

In the 1954 NCRP document, the limits for the blood-forming organs and other organs were established at 0.3 rems per week (if received every week this would be essentially 15 rems per year). This is the limitation used by the Internal Dose Committee⁴⁶ in obtaining their values for organs other than bone. In 1957, the NCRP again revised their recommendations for

*Subcommittee 2 on Permissible Internal Dose published its report in 1953⁴⁶ listing MPC's and maximum permissible body burdens based on the critical organ concept. The dose limitations were those given in the later report of the external dose subcommittee and seem to reflect the NCRP decisions arrived at in the later report.

**Genetic considerations are not pertinent to this review but they were not ignored. "From the point of view of genetic damage manifestable in future generations, the gonads, of course, constitute the critical tissues 'par excellence'." However, the contribution of occupational exposures to the dose to the population as a whole was not considered limiting.

***Bone limits were based upon a biological comparison with radium.

workers to lower the radiation doses to the whole body, head and trunk, active blood-forming organs and gonads to an average of 5 rems per year over the working years beyond age 18.⁴⁷ However, the recommended limits for internal organs other than thyroid, skin, and gonads remained at 15 rems per year. In the same document the NCRP recommended levels of one-tenth of those for workers for individuals outside of the controlled area. The latest report of the NCRP⁴⁸ continues the use of 15 rems per year for organs other than red bone marrow, skin, and gonads for occupational workers, but recommends a limitation of 0.5 rems per year to individual organs for members of the general public.

Thus, it can be seen that the current limitation of 15 rems per year for the lung of workers can be traced to the original critical organ concept and the dose limitations derived from early experience with external radiation. The recent lowering of the recommended limit for the lung of individual members of the public by the NCRP is by a factor of three and is expressly indicated as being " - - based primarily on the desire for numerical simplicity in the standards and not on an established biomedical need." At the same time, the 1971 NCRP recommendations include a concept of "significant volume" over which the dose should be averaged. The implication being that any redistribution of a given dose within this volume would not significantly affect the outcome. The 1971 NCRP report continues, "It is usually assumed that the 'significant volume' should be of the order of one cubic centimeter. This will be grossly conservative

under most circumstances, and in special estimations, use of a larger volume is justified."*

Although the original decision to use the average dose to the lung (or other organs) was made in the early period of the derivation of dose limitations, it should not be inferred that those bodies responsible for such recommendations have ignored the subject. In the Chalk River Tri-Partite Conference with scientists from the U.S., U.K. and Canada,⁴⁹ the statement is made: "In relation to the possible pathological effects of radioactive particulates in the lungs, Dr. Hamilton pointed out that the cells in the immediate neighborhood of a dust particle containing 1 or 2% of plutonium would be subjected to a dose of about 400 r/day. The general opinion which emerged from the discussion was that the carcinogenic effect per unit volume is probably considerably less for the irradiation of small masses of tissue than for large." The ICRP has addressed this general question of non-uniform dose periodically, usually by special groups commissioned by the ICRP to study the question. In its Publication 9 (1966),⁵⁰ the ICRP stated:

"In the case of non-homogeneous distribution of absorbed dose in the lung, an estimate of the Dose Equivalent to the whole lung, determined merely by the product of QF and the mean absorbed dose, may be greatly in error, but our full understanding of this problem must await further experimental evidence. In the meantime there is no clear evidence to show whether,

*The foregoing review has been greatly shortened to indicate the salient points in the derivation of the current lung limitations. At the same time, it has focused on the NCRP recommendations because of their importance in the early days when the present limits were first derived. The ICRP recommendations differ in detail but follow the same general pattern. The reader with interest in this subject is urged to review these documents for further detail.

with a given mean absorbed dose, the biological risk associated with a non-homogeneous distribution is greater or less than the risk resulting from a more diffuse distribution of that dose in the lung." In Publication 14 (1969)⁵¹ prepared by two Task Groups of ICRP Committee 1, the irradiation from radioactive particles was considered specifically. Here, it is stated: "The problems of high local concentration of dose are at their most severe with radioactive particulate material in the tissue, especially with alpha emitters. Here the local dose can reach very high values even though the mean tissue dose may be very low. Certainly it cannot be assumed that linearity of dose and effect will hold at these high doses and dose rates. On the other hand, there may be a great deal of cell death, and particularly with alpha emission, with its short and well-defined range, the number of affected but viable cells may be small compared with the number of killed cells. However, this ratio will depend on the size and activity of the particles, the extent to which they aggregate, and their movement within the tissue, and the movement of the cells past them.

"On the basis of general considerations and some experimental data and clinical experience the Task Group were of the opinion that, for late effects, the same radiation energy absorption might well be less effective when distributed as a series of "hot spots" than when uniformly distributed. Thus, with particulate radioactive sources within a tissue, a mean tissue dose would probably introduce a factor of safety. However, a severe practical problem has now been recognized in connection with the inhalation of plutonium particulates, and is now being considered in detail by a Task Group of Committee 1 of ICRP."

The Task groups also considered the problem of translocation of plutonium to lymph tissue and concluded:

"In the meantime, the Task Group are of the opinion that any immediate change in the dose limit for plutonium on the basis of risk of lymphoid tissue is not warranted."

The potential outcome of an inhalation of radioactive materials can be changed by a number of factors. If, for example, the material is readily translocated from the lung to other organs, the eventual damage to these other organs may well appear earlier than, and overwhelm any lung damage.* Thus, in considering lung dose we are focusing primarily on those materials which will be retained in the lung for reasonably long periods of time. If the quantity in the lung is large enough, death will result at early times due to pulmonary insufficiency resulting from an adema or destruction of functional living tissue. In practice, we are interested in low dose effects which will occur late in life and carcinogenesis would seem to present the end point of greatest interest. Life shortening has been noted in many experiments, particularly at higher levels, and is used as a criterion of damage. The statistical uncertainties in most experiments occasioned by the limited numbers of animals and the variation in death times make this a relatively nonsensitive indicator, even though the argument can be made that a finding of no significant life shortening is of importance since a death is a death, regardless of whether it is caused by a heart attack or a cancer. However, in many experiments in which life-shortening was not significant, the incidence of cancer at the end of life was significant, indicating that radiation effects did occur. As a result, the present studies focused primarily on cancer incidence as being the appropriate end point.

*Of particular interest in this respect is the recent work at Battelle Northwest⁵² which indicates that certain forms of ²³⁸PuO₂ are rapidly translocated from the lung to the bone when inhaled resulting in the production of bone tumors.

In most of the experiments there appears to be a relation between the radiation dose and the time of occurrence of malignancies in animals: In general, the higher the dose (or in case of internal emitters, the dose rate) the shorter the time required for cancer production. This phenomenon is frequently used to invoke the possibility of an "effective threshold" since the time required to permit cancer formation following a low dose will be so great that it exceeds the normal life span even if the induction follows a linear relation with dose. However, in interpreting data, it must be borne in mind that the opposite phenomenon will occur when the dose or dose rate becomes too high. That is, the animal will die from other causes before there is time to induce cancer. This was seen in the results from the dogs at Hanford^{31,33,34} where the early deaths were due to pulmonary insufficiency with cancers eventually appearing only in the animals with lower lung burdens and which had lived most of their life span. Thus, if radiation dose is used as a primary parameter in investigating incidence, it is important not only that the animals live out their normal life span so that the full cancer incidence develops, but that the total dose is not so high that deaths occur from other causes before the cancer can develop. These conflicting trends in causes of death can result in an apparent optimal dose for the production of malignancies. However, even at this optimal dose, the full expression of the malignancies possible per unit dose at lower values will not occur.

Akin to this concept is that of "overkill" of single cells close to the particle. In the case discussed above, the production of early death by causes other than cancer can be regarded as a result of "wasted radiation" in interpretations based upon the narrow concept of carcinogenesis

as an end point.* From this standpoint, doses which lead to death before cancer appears can be considered to be overkill of the organism since the full expression of the carcinogenic effects is not attained. For a single particle in the lung (or other tissue) the dose rates at close approaches to the particle can be high enough so that even a relatively limited time of residence in the tissue will result in the death of cells within a given radius depending upon the activity of the particle and the type of radiation. Such cells will not be able to later reproduce and, regardless of the degree of damage, will not lead to cancer.** From this standpoint, therefore, one would expect that particles which lead to such overkill would be less hazardous than uniform radiation to the overall organ since not all of the radiation is used in attaining the final end point, cancer. In fact, such a concept would lead immediately to the conclusion that the larger the particle (in terms of activity) the less effective it would be in producing cancer since the dose rates close to the particle would increase as the activity increased thereby leading to a greater fraction of radiation wasted on dead cells. One clear cut experiment possibly showing this effect was done by Passonneau^{1,53} using Sr-90 beads on rat skin. Here the same amount of activity was used for the same area of skin but the activity was distributed either as a uniform flat plate, in 50 beads, in 20 beads or in 10 beads. The results given in Table II indicate clearly a decrease in the tumor production efficiency as the activity was

*We have already mentioned that this is an appropriate end-point for consideration of dose limitation since it appears to be the latest effect in time to occur even when other effects are relatively ineffective in shortening the life span.

**However, the presence of dead cells, cellular products or fibrosis may be required before a cellular transformation can express itself as a cancer. This is an interesting possibility which needs more study.

TABLE II

TUMOR PRODUCTION IN RAT SKIN
UPON EXPOSURE TO FLAT PLATE AND POINT SOURCES

Source	Activity	No. of Animals	No. of Tumors	Tumors per μ c	Relative Efficiency
Flat Plate 1000	28.6 μ c/cm ²	71	89	4.94×10^{-4}	1.59
Flat Plate 1500	42.9 μ c/cm ²	73			
50 beads	30 μ c/bead	58	27	3.10×10^{-4}	1.00
20 beads	75 μ c/bead	77	24	2.08×10^{-4}	0.671
10 beads	150 μ c/bead	74	16	1.44×10^{-4}	0.464

subdivided into more active particles. Gamertsfelder, in an analysis of these data,¹ assumed a mid-lethal dose for cells of either 4635 or 9300 rads and a probability of tumor production increasing as the n th power of the dose to the cell. He then calculated the ratio of the number of tumors expected relative to those produced by the 30 bead configuration. The range of the experimental data is not great enough to permit distinguishing between the curves represented by different values of n but within this limited range, the calculations fit the observed trend. It is of interest to note that these calculations indicate a maximum in the relative efficiency of tumor production if n is greater than 1 while if n is equal to one, the curve approaches an asymptote as the activity per particle gets smaller. The value of this asymptote for the assumed median lethal dose of 4650 rads is 3.2 and for 9300 rads is 2.42. Since the condition where the activity per particle becomes very small is essentially that of a uniform, plane source, the comparison between this value and the value of 2.4 noted in the experiment (corrected linearly from the 1000 μ Ci flat plate source data) may be of significance. A somewhat similar

calculation by Langham and Dean⁵⁴ but on an absolute basis, to predict the probability of tumor production from various sizes of plutonium particles, used data derived by Albert⁵⁵ on the production of tumors in rat skin versus dose to the cell. The results of this calculation show a very high probability of tumor production from most particle sizes. However, as the authors indicate, the paper was published to illustrate the method rather than to provide results. The results of this work can be questioned on many grounds including the use of the data on tumors in rat skin for lung tissue, the finding of Albert that the sensitive cells are at the base of the follicle in the rat skin and the fact that the assumed efficiency of production of lung cancer per cell does not conform to the experience with humans in the production of lung tumors from external radiations.

The results of wasted radiation in the production of lung fibrosis at high levels of administration of radioisotopes or the induction of other causes of death before cancer can develop raises the question of the possible effects of such wasted radiation in the particle case. Richmond, et al.⁸ investigated the effects of Pu-238

dioxide particles lodged in the lung vascular following IV injection. These particles averaged about 180 μm in diameter and gave average dose rates to the entire lung of about 3.5 rems per hour with the dose rate in the vicinity of the particle on the order of 10^9 rads per hour. The longest exposure until sacrifice was a group of 6 rats which lived to 600 days. Examination of the lung following these exposures indicated the presence of a microlesion with complete degeneration of the cells close to the particle. However, the evidence indicated that this was not simply a stable type of scar tissue but rather that the lesion was in a dynamic state in which the collagen was renewed constantly with subsequent liquification. Within this time period there was no indication of effects which would be deleterious to the animal's overall well being. It is noteworthy that the energy delivered to the lung, if averaged over the full lung, would be on the order of 2,000,000 rads,* well in excess of those doses which have been shown to produce deaths in relatively short times when more uniformly distributed and considerably above the doses required to produce lung cancers.

One of the uncertainties with such an analysis of overkill of cells is, of course, the possibility of movement of the particles within the lung tissue so that the number of cells at risk becomes much greater and the doses delivered become smaller. In the experiment of Richmond, et al.⁸ quoted above, the particles were relatively firmly held in the blood vessels and, therefore, were not representative of particles

*Richmond, et al.⁸ indicates that Halley has estimated the average dose to a human lung for the same size of particle to be 3.5 rems per hour. Using an RBE of 10 for alpha particles and considering the rat lung to be on the order of 1/500th the mass of the human lung, the dose in 600 days becomes:

$$\frac{3.5}{10} \times 500 \times 24 \times 600 = 2,500,000 \text{ rads.}$$

actually deposited in the alveoli. Movement of such particles is known to occur through ejection with mucus and movement by the cilia and by engulfment by macrophages. Thus, quantitative estimates of the degree of overkill of cells and the fraction of radiation wasted would be uncertain since such movement is difficult to model. However, it would seem that such arguments would be of more interest in the actual quantitative sense than in the conceptual sense. If the particles are large enough so that very high dose rates are encountered in the near vicinity, there still will be a degree of overkill and wasted radiation although it may be considerably lower than would be estimated by the static model.

Additional uncertainty is added by the possible reactions of the cells located at the periphery of the zone of destruction caused by the radiation. This would involve cells receiving radiation doses ranging from just sublethal to essentially zero. If there is attempted repopulation of the volumes of destruction, this could result in rapid proliferation of these cells which have already been damaged. This situation would appear to be the most serious contender for the production of cancer and also one which would be the most difficult to investigate experimentally without an understanding of the basic mechanism of cancer production and the response of individual cells to these conditions in an otherwise normal environment and surrounded by otherwise normal cells. Information on this possibility is limited, but some indication that it is not a predominant problem can be obtained from the experiments of Passonneau⁵³ and Richmond⁸ which did involve just such conditions in several types of tissue.

The outstanding example of increased carcinogenicity of a deposited radioactive material due to localization and nonuniform dose distribution is plutonium in bone.

Here, the classical work of Brues⁵⁶ led to the conclusion that plutonium is about five times as effective for the same energy deposition as is radium, which is, in itself, nonuniformly distributed. Studies of the comparative deposition in bone of these two isotopes have indicated that the radium, being chemically similar to calcium, tends to deposit in the mineralized portions of the bone and eventually is distributed through the bone mineral by remodeling or is covered by new layers of calcified materials. By contrast, the plutonium is deposited on the bone surface in locations where it is adjacent to the regenerative cells and, in remodeling of the bone tends to redeposit on these surfaces. Thus, this represents the case of a very nonhomogeneous organ where the comparative isotope (radium), while not uniformly distributed, is more uniformly distributed than the plutonium. Further, the plutonium is preferentially deposited in the vicinity of the regenerative cells which are presumably more sensitive to the induction of cancer than the mineralized bone. This situation would seem to represent a localization of the radiation dose at cells which present a more sensitive target and therefore, eliminates some of the wasted radiation which occurs with radium in the mineralized portion of the bone. In essence, the bone can be regarded as composed of three regions of differing criticality: the marrow, the proliferating cells on the bone surfaces and the mineralized portion which has minimal metabolic activity and serves primarily as a structural supporting member for the body. In this case, the sensitive tissues are the marrow and the regenerating cells with the regenerating cells of most interest for plutonium as the average dose to the marrow from the poorly penetrating radiations from plutonium is comparatively low. Again, however, some significant dose rates to the marrow on a localized basis can be calculated. These are to a small

fraction of the marrow falling within a few tens of micrometers of the deposited plutonium. The fact that leukemia is a relatively rare outcome in experimental animals given plutonium may serve as an indicator that irradiation of a small portion of an organ (the marrow) to a high dose is not particularly troublesome as long as the average dose is low.

A similar situation may, of course, occur in any organ as a number of different cell types can be present in the same organ and any mechanism which results in preferential irradiation of the more sensitive cell types could, theoretically lead to the same type of result. The high incidence of lung tumors in uranium miners from radon in mine atmospheres is attributed to the deposition of the particulate daughters of radon on the bronchi, particularly at points of division where the turbulence in the air stream produces increased impaction and deposition.

The estimation of the radiation dose to the assumed critical tissue, the bronchial epithelium, is complicated by the uncertainties in the areas of deposition and the thickness of the mucus layer which serves to absorb some of the energy of the radon daughters deposited on the surface. However, in a review of the dosimetry for the Federal Radiation Council⁵⁷ Parker considers, with important reservations, that one working level month corresponds to a dose to the bronchial epithelium of 2.8 rads. The working level for exposure to radon daughters is defined as any combination of radon daughters in one liter of air that will result in the ultimate emission of 1.3×10^5 MeV of potential alpha energy. One working level month, then, is the total exposure resulting from working in such an atmosphere for 170 hours. If we assume that all of the alpha energy associated with the daughter products is released in the lung (i.e. all of the daughters are deposited and none are eliminated before

they decay) the average dose to a 1000 gram lung would be 0.44 rads. This is undoubtedly a maximum estimate since some of the daughters will be exhaled and a portion will be eliminated by ciliary action. However, much of the activity is associated with small particles which are deposited in the bronchi and lower pulmonary regions with relatively high efficiency. The linear velocity of particles moving up the bronchi is 0.25 to 1 cm/min while in the trachea rates can increase to 3 cm/min.¹ Because the longest half-life of the radon daughters of interest is 26.8 minutes, it would appear that a sizeable fraction of the material deposited in the bronchi would decay before elimination and that all of the material deposited below the ciliated region would contribute their full energy. If we apply this estimate of the average lung dose to the estimated exposures of the uranium miners in those exposure ranges where the incidence of lung cancer is high, we find that the dose to the total lung calculated on an average organ basis is, indeed, significant and in the range where animal data would indicate such an outcome to be expected.* Since there is uncertainty about the actual significance of the increase in lung cancer at the lower exposure levels, we will not discuss this phase. However, the dose levels corresponding to the exposure ranges used in the epidemiological study^{57,58} assuming an average dose to the lung of 0.44 rads per WLM are listed in Table III.

*In order to permit a rapid appraisal of the data presented in the abstracts on the incidence of lung cancer at various dose levels, Fig. 1 presents a crude plot of the data for the alpha emitters. No attempt was made in this plot to reevaluate the dose estimates or to correct for experiments in which the incidence was measured before the full life-span of the animals. The five points at the lowest doses were the results of the ²³⁷Np and the ²¹⁰Po administrations. The human data are estimates of doses received by a group of 37 individuals exposed during work with plutonium and represent periods of time ranging from 4 to 24 years after exposure.⁵⁹

TABLE III

AVERAGE LUNG DOSES CORRESPONDING TO LEVELS OF EXPOSURE USED IN THE URANIUM MINER EPIDEMIOLOGICAL STUDY

Exposure WLM	Average Lung Dose-Rads
< 120	< 53
120 - 359	53 - 158
360 - 839	158 - 370
840 - 1799	370 - 792
1800 - 3719	792 - 1636
> 3720	> 1636

An additional argument concerning the present bases for radiation protection standards should be included in this discussion. As a basis for dose limitations, it is normally assumed that the response to a given dose is proportional to the dose received and that there is no threshold. While there is considerable evidence to support the use of this assumption, there is also evidence that the dose rate is an important factor, at least for low LET radiations, with the response decreasing as the dose is protracted, presumably due to the repair of the damage in the intervening time before the full dose is accumulated.* Acceptance of this assumption would indicate that the result of a dose to a small portion of a given tissue would be the same

*We note that the same argument cannot be made for alpha emitters since current evidence indicates that the damage from high LET radiations is not repaired. Thus, the assumption of linearity with dose, regardless of dose rate, would seem to be more appropriate for these materials than for the gamma or x-rays. As an aside, we also note that the amount of repair for gamma radiations appears to be on the order of 90%. If we assume no repair for the alpha radiations, the late result (after repair is over) would be about ten times as great for the alpha radiations as for the gamma. This appears to be about the same as the commonly accepted RBE or Quality Factor for alpha radiations.

Fig. 1 A plot of the crude data for lung tumor incidence versus lung dose. Data have not been screened for length of exposure or accuracy of dose calculations. All doses are expressed as the average to the lung.

Legend: M-mice; R-rats; D-dogs; H-hamsters; S-humans.
 0- ^{210}Po ; 7- ^{237}Np ; 8- ^{238}Pu ; 9- ^{239}Pu .

as if the same amount of energy were distributed over the entire tissue. We have seen that this is not the case in some extreme situations such as in the overkill of cells close to a particle or the induction of more lethal effects at high dose rates. However, acceptance of the assumption of linearity at the more moderate conditions would lead to the conclusion that there should be no difference in outcome regardless of the distribution of the dose throughout the tissue, unless a critical portion of the organ is more sensitive. This would lead to the conclusion that non-uniform distribution of dose could have no greater effect than a uniform dose. Because this is based upon an assumption

which is made in an effort to be conservative and is based upon effects at relatively low doses, we do not believe that this argument is very strong. However, a conclusion of nonlinearity of effect could have a major impact upon current radiations standard setting practices unless it is shown that such nonlinearity occurs only at very high cell doses.

No clear cut, overall picture of the relative effects of uniform versus focal dose can be drawn from the present data. It appears, from the $^{233}\text{PuO}_2$ microsphere data and the skin experiments with ^{90}Sr that; in the extreme situation of a single, very active particle, the focal radiation is considerably less damaging. Cember¹⁵ concludes

that the focal source is less damaging for beta emitters than is the uniformly distributed source. The data of Grossman, et al.²⁷ for ^{210}Po on iron oxide particles indicates a seeming decrease in the tumor incidence as well as increased survival for the focal sources. Saunders,³⁵ as a result of his studies with soluble ^{238}Pu derived from crushed microspheres arrives at a conclusion that spreading the dose more uniformly results in an increased cancer incidence due to the greater number of epithelial cells involved. This conclusion was based on the observation of " - a significant incidence of tumors in the lung and in other tissues at radiation doses that have not previously been shown to be carcinogenic in animals". In Figure 1, it is of interest to note that two of these data points are included in the five lowest dose points with the other points being the results of ^{237}Np administration. In both cases, significant numbers of tumors were also noted in locations other than the lung indicating a more general insult to the entire body.

Most of the support for particulates being more hazardous than a uniformly distributed material seems to arise from calculations based upon dose distribution around the particles and an assumed response of individual cells to this dose. In an overall appraisal of the information available, it does not appear that the majority of the data support the hypothesis that the particles are more hazardous than the uniform dose. A reasonable case can be made that they are less hazardous. The conclusion of this work to date, therefore, is that the preponderance of the evidence indicates that the use of an average lung dose is appropriate in limiting exposures and may well be conservative.

II. SOILS CHARACTERISTICS PERTINENT TO PLUTONIUM TRANSFER - J. Nyhan

The first set of proposed standards to be developed under this project are to be for plutonium in soils. It was, therefore, necessary to obtain such information as is available on the properties of soils which are of importance to the many questions which arise in estimating the transfer from soils to humans. In addition, since wind erosion is closely related to the question of resuspension, a brief review of the information available on this phenomena was made to permit an estimate of its application to the question of resuspension.

A. Wind Erosion in the U.S

Although few regions in North America are entirely free from wind erosion, several regions exhibit high activity in this regard. Figure 2 shows that the erosion of soil by wind has been the worst in the Great Plains, as well as the Columbia River plains, and some parts of the Pacific Southwest and the Colorado Basin. Other general regions of interest include the muck and sandy areas in the Great Lakes region and the sands of the Gulf and Atlantic seaboards.⁶⁰ It is useful to consider the three factors influencing the mechanics of wind erosion: wind, soil, and the nature of the eroding surface.

Fig. 2. A generalized map (101) showing the chief large areas of high soil erosion by wind (horizontal shading) or runoff (slanting shading).

Regions in the U.S. with relatively high average surface wind velocities (Figure 3) correspond to areas of high soil erosion by wind (Figure 2). This is not surprising, since one review article indicates that the intensity of wind erosion varies as the cube of wind velocity.⁶¹ The wind factor is centered around the wind velocity pattern near the soil surface, its effect on the initiation of soil movement, and the transportation and deposition of soil particles.

The soil factor involves the influence of soil moisture and soil structure on the severity of wind erosion. Periods of drought, wind erosion and dust storms have occurred simultaneously in the Great Plains, and the intensity of wind erosion varies inversely as the square of either moisture at the soil surface or effective precipitation.⁶¹ Again, a good correlation exists between geographic regions of intense wind erosion (Figure 2) and areas where evaporation is usually in excess of precipitation (Figure 4). These observations are related to the fact that there are very few winds that have sufficient velocities to overcome the very large cohesive forces of moisture films at a tension of 15 atmospheres in an air-dried soil.^{62,63} Soil structure, or the arrangement of individual sand, silt and clay particles with respect to each other into a pattern, also influences soil resuspension. Soil structure is

Fig. 3. Generalized map (101) of average surface wind velocity (mph).

Fig. 4. Evaporation regions:
 1. evaporation much in excess of precipitation
 2. evaporation considerably in excess of precipitation
 3. evaporation usually in excess of precipitation
 4. evaporation in excess of precipitation in warm months
 5. precipitation in excess of evaporation in cooler months
 6. precipitation generally in excess of evaporation (101).

in turn affected by the particle size distribution of the soil, as well as the organic matter and polyvalent cation content of the soil-components important in binding the particles together. Although enormous forces are necessary to break the covalent bonds involved in these soil-cation-organic molecule complexes, winds can move groups of these aggregates in the form of clods less than 840 microns in diameter.⁶⁴ The stability of these structural units is decreased by tillage operations and by freeze-thaw and wetting-drying cycles at the soil surface.

The nature of the eroding surface is another factor which influences the mechanics of wind erosion. Surface conditions which affect wind erosion are the surface roughness and the degree of protection by surface cover. The erosion decreases with increasing surface roughness, when this roughness is due to nonerodible structural units, mainly because of the diminished wind velocity that hits the soil surface. Vegetation not only adds to surface roughness but also provides cover for the soil surface. Regions of the U.S.A. where the

vegetation is minimal (grassland or desert) also exhibit a potential for significant wind erosion (Figure 2) compared with the minimal wind erosion in prairie-forest or forest zones (Figure 5). Vegetation effectiveness as a wind barrier depends primarily upon its density, width, porosity, and orientation with respect to the prevailing winds.⁶⁴

B. Variations in Soil Properties which Influence Erosion

In order to understand the natural variability of soil properties effecting wind erosion, an understanding of some factors in soil formation is helpful. The five factors of soil formation which influence, have influenced or may influence soil genesis are: parent material, organisms, climate, relief (topography), and time (Figure 6). When considering soil variations throughout an area as large as the Great Plains, it is useful to relate the scale of action of these five factors to soil variability within this large region. Climate and organisms tend to be relatively uniform across distances of tens or hundreds of miles, whereas parent materials

Fig. 5. Generalized vegetation map (101).

1. southeastern forest
2. deciduous forest
3. northern evergreen forest
4. prairie-forest transition
5. grassland
6. western conifer forest
7. northwestern conifer forest
- 8a. desert shrubs
- 8b. semidesert shrubs
- 9a. semidesert
- 9b. desert

Fig. 6. The five general factors of soil formation.

are fairly uniform over miles of distance. Almost all of the short-distance changes in soil type are related to the topographic factor.

The five soil formation factors are responsible for general patterns of soils occupying positions on contemporary landscapes within the wind eroded areas in Figure 2. Transects across the northern and southern Great Plains exhibit large areas of entisols and aridisols in the western regions, grading into vast areas of mollisols to the east. These soils are also the major soils in the areas of Washington, Nevada, California, and Arizona where high wind erosion activity exists. Soils belonging to the Entisol order of the U.S. Comprehensive Soil Classification System^{65,66,67} represent the simpler end of the soil continuum relative to soil development and usually occur in mountainous, desert and alluvial floodplain regions. These soils are morphologically similar to their parent materials, partially due to the immobilization of clay illuviation by inert materials, highly siliceous sediments and carbonates. The entisols are similar to soils of the Aridisol order in that they both contain light-colored topsoils with less than 1% organic matter. However, the aridisols, the soils of the arid regions, are more weathered than the entisols and exhibit minimal clay illuviation, maximum carbonate accumulation, and many times a

surface "pavement" of gravel. The mollisols, the darker-colored grassland soils of the prairies, contain greater than 1% organic matter in the top seven inches of their profiles. Several pedogenetic processes are involved in mollisol profile development that are minimized in entisol-aridisol profiles: (1) the process of darkening of soil by organic matter additions, (2) accelerated eluviation and illuviation of clays and (3) loess additions to the topsoils of some mollisols.

Jenny⁶⁸ has advocated a scheme for comparing soils or individual soil properties. This scheme is based on the principle of comparing a given soil property among soils when one soil formation factor is dominant and the other four are ineffective. Jenny took soil samples along a 11° C isotherm in the arid-humid transition belt extending from Colorado through Kansas to western Missouri, and correlated natural variations in soil properties with annual rainfall.⁶⁹ The results (Figure 7) indicated that the depth of the carbonate

Fig. 7. Relative soil property-rainfall curves for some soils in the Great Plains (69).

horizon and hydrogen-ion concentration depend most sensitively on rainfall, with nitrogen content (related to organic matter content) and clay concentrations less influential. Since these soil properties influence soil structure directly, wind erodibility will be inversely related to this climatic factor.

The particle size distribution of a soil is an important factor contributing to soil aggregate formation. The U.S.D.A. has developed a system of classifying soils relative to their texture, i.e. the relative proportions of sand (50-2000 micron diameter particles), silt (2-50 micron diameter particles) and clay (particles with diameters less than 2 microns). These 12 classes are usually presented as a soil-texture triangle (Figure 8). These 12 classes can be further grouped into coarse-, medium- and fine-textured soils as in Table IV.

Natural variations in particle size distributions of surface soils (i.e. top inch of soil) have not received much attention, mainly because the top 6-12 inches of soil is usually the important unit for agricultural and soil classification reasons.

Fig. 8. The soil texture triangle (USDA).

TABLE IV

SOIL-TEXTURE CLASSIFICATION

Coarse-textured soils	1. Sands and loamy sands	Sand
		Loamy sand
	2. Sandy loams	Sandy loam
Fine sandy loam		
Very fine sandy loam		
Medium-textured soils	3. Loamy soils	Loam
		Silt loam
		Silt
4. Moderately heavy soils	Sandy clay loam	
	Clay loam	
	Silty clay loam	
Fine-textured soils	5. Clays and silty clays	Sandy clay
		Silty clay
		Clay

However, it is known that climatic factors have a large influence on the particle size distribution of surface soils, a factor involved in aggregate formation. With increasing aridity, physical processes assume relatively greater significance than chemical forces in the disintegration of rocks. Thus, aridisols bear a closer relation to their parent material than mollisols. However, reduction in size of sand and silt particles is performed largely by physical weathering via differential thermal expansion of minerals, frost action, and by erosion and deposition by water, ice, and wind. In contrast, clay particles have relatively large surface areas which are able to react chemically and relatively fast via chemical reactions, all of which require the presence of water.

In order to observe the net result of physical and chemical weathering processes on parent materials, the particle size distributions of selected textural types of soils is presented in Figures 9-14 and Table V. This compilation of results for over 40 soil types⁷⁰⁻⁸⁴ is presented as summation percentage graphs of percentage of particles (by weight) smaller than a specified particle size, which is plotted on the abscissa. Whenever the data are available, the mechanical composition of "typical soils", as given by one author⁸⁵ is also plotted for comparison (solid lines).

Figures 9-14 indicate that very few soils have appreciable amounts of sand particles greater than 500 microns in diameter. This seems to substantiate the observation

TABLE V
IDENTIFICATION OF SOILS IN FIGURES 9-14 BY TEXTURAL CLASSES

Textural Class	Sample Number In Figure	Soil Type	Sampling Depth (inches)	Location	Comments	Reference
<u>A. Coarse-textured soils</u>						
Sand	1	Average of 5 soil samples from the Bloomfield and Princeton soil series	Varied from 40-200	Indiana	Samples from C horizons of Wabash River transect	70
Sand	2	Average of 7 soil samples from the Bloomfield and Princeton soil series	Varied from 52-136	Indiana	Samples from C horizons of a White River transect	70
Sand	3	Not classified	0-2	Northern Alaska	Samples taken from a tundra soil adjacent to the Meade River	71
Sand	4	Not classified	0-5	Northern Alaska	Samples taken in sand dunes adjacent to the Meade River	71
<u>B. Medium-textured soils</u>						
Sandy loam	1	Scituate sandy loam	0-9	New Hampshire	Sample taken at crest of a drumlin	72
Sandy loam	2	Scituate fine sandy loam	0-7	New Hampshire	Sample taken in middle slope region of drumlin	72
Sandy loam	3	Greenfield fine sandy loam	0-10	California	-	73
Sandy loam	4	Timpahute sandy loam	0-2	Utah	Samples from arid region	74
Sandy loam	5	Escalante fine sandy loam	1-12	Utah	Samples from arid region	74
Sandy loam	6	Average of 7 Ptarmigan sandy loams	0-8	Montana	Located on steep-gentle mountain slopes	75
Sandy loam	7	Musick sandy loam	0-6	California	Podzolic soil	76
Sandy loam	8	Casper sandy loam	0-6	California	Podzolic soil	76
Loam	1	Unclassified but in the Oxbow soil association area	0-7	Saskatchewan	Orthic Black Soil	77
Loam	2	"	0-7	Saskatchewan	Podzolic Orthic Dark Grey Soil	77
Loam	3	Unclassified, but in Whitewood Association	0-4	Saskatchewan	Podzolic Dark Grey Wooded soil	78
Loam	4	Tehama loam	0-8	California	-	73
Loam	5	Kimball loam	0-4	California	-	73
Loam	6	San Joaquin loam	0-6	California	-	73

Loam	7	Barnes loam	0-6	North Dakota	Publication soil # S53ND-2-1 from central North Dakota	78
Loam	8	Barnes loam	0-2	North Dakota	Publication soil # S53ND-11-1 from southeastern North Dakota	78
Loam	9	Barnes loam	0-5	North Dakota	Publication soil #S54ND-10-2 from northeastern North Dakota	78
Loam	10	Aastad loam	0-6	North Dakota	Grassland soil from central-eastern North Dakota	78
Loam	11	Nappanee loam	0-8	Ohio	lake plain sample	79
Loam	12	Nappanee loam	0-8	Michigan	glacial till plain sample	79
Loam	13	Redfield loam	0-6	Utah	alluvial soil in an arid region	74
Loam	14	Uvada loam	0-1	Utah	-	74
Loam	15	Melbourne loam	0-7	California	lateritic soil	76
Silt loam	1	Not classified	0-1	Saskatchewan	Orthic Grey Wooded soil	77
Silt loam	2	Hosmer silt loam	0-8	Illinois	loess-derived podzolic soil	80
Silt loam	3	Antelope Springs silt loam	0-3	Utah	-	74
Silt loam	4	Coropina silt loam	0-6	British Guiana	podzolic coastal soil	81
Silt loam	5	Fairbridge silt loam	1-3	British Columbia	-	82
Silt loam	6	Hopleys silt loam	0-6	Montana	alpine soil	75
Silt	1	Average of 5 soil samples from the Alford, Ragdale and Sylvan series	varied from 40-140	Indiana	Samples from C horizons of a Wabash River transect	70
Silt	2	Average of 4 soil samples from the Alford, Ragdale, and Hosmer series	varied from 52-76	Indiana	Samples from C horizons of a White River transect	70
<u>C. Fine-textured soils</u>						
Clay	1	Frontland clay	0-6	British Guiana	Coastal soil, unleached phase	81
Clay	2	Frontland clay	0-6	British Guiana	leached and cultivated phase	81
Clay	3	Alberni clay	0-2	British Columbia	-	82
Clay	4	Average of 3 Thoeny clays	Unspecified	Montana	Samples from B horizons	83
Clay	5	Princes town clay	0-6	West Indies	-	84
Clay	6	Carron Hall clay	0-6	West Indies	-	84
Clay	7	Fitches clay	0-6	West Indies	-	84

Fig. 9. Summation percent curves for sands (numbers in figure refer to soil sample numbers in Table V).

Fig. 12. Summation percent curves for silt loams (numbers in figure refer to soil sample numbers in Table V).

Fig. 10. Summation percent curves for sandy loams (numbers in figure refer to soil sample numbers in Table V).

Fig. 13. Summation percent curves for silts (number in figure refer to soil sample numbers in Table V).

Fig. 11. Summation percent curves for loams (numbers in figure refer to soil sample numbers in Table V).

Fig. 14. Summation percent curves for clays (numbers in figure refer to soil sample numbers in Table V).

that many soil materials, notably those in glaciated areas where much of agricultural science developed, tend to have a minimum in particle size distribution around 1000 microns.⁸⁶ At the other end of the spectrum, many soils may consist of over 30% by weight of particles smaller than 0.2 microns in diameter; this represents a documented, 5 orders of magnitude difference in soil particle sizes. The results of summation curves for 560 German soils⁸⁷ indicated that: (1) particle size distributions were normally distributed when the log of the particle size was plotted on the abscissa, (2) every particle size range has one maximum size distribution and (3) certain distributions did not occur naturally. Examples of soils that were not found were soils with less than 20% silt and greater than 30% clay and soils with less than 65% sand, less than 5% clay and more than 30% silt.

C . Classification of Soils Into Wind Erodibility Groups

Eolian materials were described in a recent review⁸⁸ by their pathway of transportation in the atmosphere, persistence in the atmosphere and particle size (Table VI). More specifically, we are interested here in the air concentrations from the soil surface to about 6 feet above the soil surface (the height of a man). Chapil⁸⁹⁻⁹¹

has documented the dynamics of wind movement of arable soils in this zone via surface creep, saltation and suspension, involving progressively smaller soil particles.

After the violent wind erosion in the Great Plains in the 1930's and 1950's, criteria were needed to determine the capability classification of soils in these areas—criteria based on more than land use experience. In 1957 Coover and Moldenhauer⁹² used a formula, which expressed soil loss rates as a function of a soil "erodibility index", plant surface residue, and surface roughness, to quantitatively evaluate the wind erosion in capability classifications for Texas soils. In 1960, Chapil⁹³ developed the more generalized relationship between actual soil loss rates and his soil erodibility index, based on many wind tunnel and field measurements, for Garden City, Kansas. Chapil later developed a wind erosion climatic index for application of the Kansas Experiments to many geographical regions.⁶¹ The final accomplishment came in 1963, when Chapil and Woodruff⁹⁴ published the wind erosion equation, which expressed the many factors involved in soil losses from erosive wind effects:

$$E = f (I C K L V)$$

TABLE VI
CLASSIFICATION OF EOLIAN MATERIALS

Type of Material	Dominant Particle Size µm	Transport		Persistence in Atmosphere
		Pathway	Range(km)	
Dune sand	400-200	At or near ground level	10 ⁻³ to 10 ⁰	Highly transient phase
Loess	50-10	Lower troposphere	10 ⁻¹ to 10 ²	Transient phase
Aerosolic dust	10-1	Middle and upper troposphere	10 ⁴ to 10 ⁶	Residence time depends on precipitation frequency but is thought to be days to weeks
Stratospheric dust	<1	Stratosphere	~10 ⁹	Residence time, years
Extraterrestrial dust	40-0.01	Interplanetary space	~10 ¹³	Undetermined

This equation expresses the annual soil loss in tons per acre (E) in terms of the soil erodibility index (I), the climatic index (C), soil surface roughness (K), the unsheltered field width (L), and the equivalent quantity of vegetative cover (V).

The Soil Conservation Service has recently developed criteria for grouping soils into "wind erodibility groups" as described in Tables VII and VIII.⁹⁵ The procedure is to collect about 4 pounds of dry soil from the surface inch of the field, sieve the soil through a 0.84 mm square-opening sieve in the field and relate the percentage of nonerodible soil aggregates greater than 0.84 mm to the wind erodibility group and soil erodibility index (the I factor in the wind erosion equation). When large areas of land must be classified and sieving is not a feasible procedure, the wind erodibility group can be roughly estimated by the texture by feel method. Once the wind erodibility group has been estimated, site-specific information on the other four factors of the wind erosion equation can be collected to estimate the potential amount of wind erosion for a given field under local climatic conditions.

This work has many direct applications to the problems of concern, especially because the S.C.S. has already collected these data for many soils. The total amount of soil loss per year, as predicted by the wind erosion equation, does consist of about 90% soil loss via saltation (and minimal surface creep) and about 10% soil loss in suspension - on the average.⁹⁶ Thus, many large soil particles would move via saltation close to the soil surface (90% below a height of one foot⁶⁰), and would initiate suspension of smaller soil particles (silt, clay, and aggregates) in the respirable size range. This represents no small amount of soil - estimates of up to 277 million tons per year were found in suspension over the Great Plains in the 1950's.⁹⁶

D. Interception and Retention of Plutonium by Plants

Either vegetation or soil surfaces may be directly contaminated with plutonium as a result of worldwide fallout or local additions of plutonium to the environment. As time progresses the plant plutonium can redistribute in several ways: (1) wind and rain can remove the plutonium from the

TABLE VII

RELATIONSHIP OF DRY SOIL AGGREGATES >0.84 mm
TO SOIL ERODIBILITY INDEX (I) AND WIND
ERODIBILITY GROUP

<u>Wind Erodibility Group (WEG)</u>	<u>Dry Soil Aggregates >0.84 mm-percent</u>	<u>Soil Erodibility Index tons/acre/year</u>
1	0.5-1.5	340-280
2	5.0-15.0	180-117
3	16.0-34.0	113-67
4	15.0-35.0	117-65
4L	15.0-35.0	117-65
5	28.0-52.0	79-33
6	36.0-54.0	63-29
7	36.0-64.0	63-17
8	---	---

TABLE VIII

GUIDE FOR ESTIMATING WIND ERODIBILITY GROUP
FROM APPROXIMATE SOIL TEXTURES

Texture of Surface Inch	Wind Erodibility Group
Very fine sand, fine sand and medium sand	1
Loamy sand, loamy fine sand	2
Very fine sandy loam, fine sandy loam, sandy loam	3
Clay, silty clay, noncalcareous clay loam and silty clay loam with more than 35% clay content	4
Calcareous loam and silt loam; calcareous clay loam and silty clay loam with less than 35% clay content	4L
Noncalcareous loam and silt loam with less than 20% clay content; sandy clay loam, sandy clay	5
Noncalcareous loam and silt loam with more than 20% clay content; noncalcareous clay loam with less than 35% clay content	6
Silt; noncalcareous silty clay loam with less than 35% clay content	7
Very wet or stony; not subject to wind erosion	8

plant surfaces to the soil or to other plants, (2) foliar-absorbed plutonium can be translocated throughout the plant and to the soil, or (3) the plant may die and eventually be mineralized in the soil. The plutonium in the soil may be physiologically taken up by the plants or may be deposited on plant surfaces by the wind or rain. The result of these redistribution mechanisms will affect the inventory and concentrations of plutonium in soils.

Physiological uptake of plutonium by plants in the laboratory is usually minimal, resulting in uptake rates of 10^{-4} per cent of the added plutonium.⁹⁷ Thus, the net redistribution process to be evaluated may be simplified to plant interception of fallout and resuspended plutonium-soil particles and transfer of these intercepted

particles to the soil pending further investigation of the possibility of increased plutonium uptake through the roots after aging processes in the soils.

A review of the contamination of vegetation indicated that clover and wheat plants near ground zero intercepted about 0.2% of the fallout activity, whereas plants 106 miles away intercepted a maximum of 25% of the fallout.⁹⁸ The amount of fallout intercepted by the vegetation is also a function of plant population density. However, the wind erosion research discussed previously indicates that when a certain level of ground cover is achieved (about 4000 pounds of vegetation/acre), resuspension is negligible.

The concentration of radionuclides on fallout-contaminated foliage as a function

of time was characterized in a recent review,⁹⁹ but similar retention curves have not been characterized for plutonium. If plutonium acts like other fallout particles or simulated fallout particles, the exponential decrease in amounts of plutonium retained may be expressed as:

$$C(t) = C(0) \exp(-\lambda_p t). \quad (1)$$

The concentration of radionuclide at time t [$C(t)$] is expressed as a function of the initial concentration on the foliage [$C(0)$], and the effective elimination constant, λ_p . The latter is defined as:

$$\lambda_p = \lambda_r + \lambda_w \quad (2)$$

where λ_r is the radioactive decay constant and λ_w is the weathering elimination constant. The effective half-lives of 44-88 or 88-175 micron fallout particles on 5 different plant species averaged about 7 days, with weathering half-lives of 16-26 days.⁹⁹

These data indicate that plutonium fallout particles or resuspended plutonium-soil particles may have a short half life (7 days) on vegetation. The major exposure problem over periods of time greater than this would seem to be associated with the plutonium on the soil surface. However, if the vegetation consisted of a tall, dense stand of mature deciduous trees, the half-life of the suspended particles falling to the soil surface could be much longer than one week. This situation might represent a greater exposure to man due to the longer effective half-life if very large concentrations of nuclides were retained by the trees.

E. A Possible Approach to Sampling of Soils for Plutonium

Very few studies have been made of sampling error involved in estimating the distribution of heavy metals in soils. Without the results of experimental data such as

these, there is very little information on which to base sound sampling procedures.

In the absence of prior knowledge related to the amounts and distribution of plutonium in the soil, the first step to follow would be to systematically sample a geographical area for inventory purposes. The second step would involve following Soil Conservation Service soil sampling procedures for classifying soils into wind erodibility groups. Thus, a series of soil samples would be taken from the surface inch of soil, which would then be sieved through an 0.84 mm square-opening sieve in the field. This second set of soil samples should be collected in such a way that each sample represents a relatively uniform region within a contaminated area and the area should be observed for variations in soil properties, topography, degree of soil erosion, and other factors influencing suspension of soil particles.

The percentage of weight of nonerodible soil aggregates (greater than 0.84 mm) will be determined in the laboratory and used to estimate annual soil losses from a given region within a contaminated area. Approximately 10% of this total soil loss represents suspended-particle movement for the Great Plains and only a fraction of this suspended soil is in the respirable particle size range. Thus, plutonium determinations should be performed on two types of soil plutonium samples: (1) the respirable size fraction, and (2) the nonrespirable size fraction. Thus, we now have a very rough estimate of the amount of potential annual soil movement of respirable soil particles and a measure of the plutonium in the respirable and nonrespirable soil fractions.

Standards for plutonium concentrations should consider that soil weathering processes may result in smaller topsoil particles with time. Although specific information has not been collected relative to the

weathering rate of these larger aggregates to aggregates and particles in the smaller size ranges, there is information on the general soil formation factor of time. A recent review of rates of soil formation¹⁰⁰ indicates that it took approximately 30-208 years to form one inch of topsoil in several regions of Iowa and that longer time periods were necessary in arid regions. Therefore, depending on the stage of development of the soil and the factors of soil formation, significant increases in the respirable particle range may occur within 30-1000 years.

III. RESUSPENSION CALCULATIONS - J.W. Healy

Previous recommendations on limits for plutonium on the ground have been based on an estimated air concentration resulting from the resuspension of the plutonium from the ground using the concept of the resuspension factor: i.e. the ratio of the concentration in the air to the concentration on the ground. This method of calculation is a gross oversimplification of the real situation which might work in well-defined situations but does not provide the detail necessary to permit estimation of the effect of changing variables nor does it permit investigation of the effect of size of contaminated area or atmospheric conditions. A model for calculating air concentrations resulting from resuspension from the ground due to either wind pickup or mechanical disturbance has been under study for several years. This model derives from work done earlier in estimating the wind pickup of particles and the probability of impaction on a person downwind^{102,103} and is based upon describing the source term as a rate of injection of the material into the atmosphere and accounting for the dispersion and deposition downwind to arrive at a final concentration at the location of the receptor. Although this work has been used in draft form in studies such as those at the Nevada Test Site, the detailed model has not as yet been published. A brief

description is, therefore, included here to serve as a background for the calculational work done during this period.

If we consider a point source of material on the ground which is subject to resuspension, the concentration in the air, X , at a distance downwind can be given as:

$$X = K \Omega D' D'' \quad (3)$$

In Eq. (3), Ω represents the quantity of material on the ground which is subject to resuspension, K is the resuspension rate or the fraction of the material which enters the atmosphere per unit time, D' is the dispersion which occurs as a result of atmospheric turbulence and D'' is the fraction of the material originally resuspended which is not deposited between the source and the receptor. In atmospheric dispersion terms, the product $K\Omega$ is the source term, i.e. the rate at which the material is injected into the atmosphere. For a contaminated area, then, the air concentration can be obtained by integrating Eq. (3), for the specific distribution of material in the source.

$$X = \int_{x_1}^{x_2} \int_{y_1}^{y_2} K \Omega D' D'' \, dy \, dx \quad (4)$$

In application of Eq. (3), or (4), it is possible to measure X and Ω and use dispersion and deposition relations to estimate the value of K for the existing conditions of the measurement. With a sufficient number of such measurements under different conditions of terrain, wind speed, turbulence, etc. it should be possible to obtain correlations with the various parameters of importance.

The dispersion downwind from the source can be estimated from the experimental correlations available from the numerous micro-meteorological studies in the past.¹⁰⁴ These are not completely satisfactory in

three respects: (1) The data are stratified into arbitrary classifications of stability while the atmosphere behaves as a continuum; (2) the data for the growth of a plume in the vertical are particularly uncertain and the deposition correction is strongly dependent upon this parameter; and (3) the time dependence of the dispersion parameters is only vaguely defined with most values selected as the average for unspecified periods of measurement. However, for purposes of this model and taking into account the other associated uncertainties in present information, they will provide a reasonable estimate and can be used to investigate the effect of changing some of the conditions. After considerable review, we have chosen to use Sutton's formulation¹⁰⁵ which, in spite of its early derivation, provides answers comparable to most of the other formulations if the coefficients are chosen properly. In addition these expressions can be integrated analytically for many of the area sources thereby eliminating some of the numerical integrations which would otherwise be necessary. Note, however, that any of the dispersion correlations can be used, but with greater necessity for numerical solutions. The dispersion term from a point source is, then, given by:

$$D' = \frac{R}{\bar{u} \pi C_y C_z x^{2-n}} \exp \left[- \left(\frac{y^2}{C_y^2 x^{2-n}} + \frac{z^2}{C_z^2 x^{2-n}} \right) \right] \quad (5)$$

where x is the distance of the receptor downwind from the source, y is the horizontal cross wind direction, z is the height of the receptor, C_y , C_z and n are Sutton's parameters which vary with stability of the atmosphere, and R is a reflection factor normally taken as 2 to account for the increase in concentration by reflection of the plume from the ground.

The deposition correction was obtained by the method of Chamberlain¹⁰⁶ by integrating the deposition between the source and receptor and subtracting this quantity

from the quantity emitted. While there are many difficulties with this technique, such as the correction for the quantity remaining in the cloud through the full height of the cloud rather than only in the portion adjacent to the ground, it provides answers which correspond to the meager information available, at least for the order of magnitude. From Eq. (5), this deposition correction can be obtained as:

$$D'' = \exp \left[- \frac{2 R x^{n/2} V_d}{\sqrt{\pi} C_z \bar{u}} \right] \quad (6)$$

where V_d is the deposition velocity and the other terms are as given earlier. V_d is defined as the ratio of the deposition rate on the surface (C_i/m^2 per second) to the concentration in the air at a reference height (C_i/m^3).

In providing a model for the velocity of deposition and its variation with particle size, the transfer of mass across the boundary layer in the atmosphere was taken to be equal to the transfer of momentum. This leads to an expression for the turbulent transfer of:

$$v_t = \frac{(u^*)^2}{u_z} \quad (7)$$

where V_t is the transfer velocity due to turbulence, u^* is the friction velocity and u_z is the wind velocity at a reference height x . For a neutral atmosphere where a logarithmic wind profile in fully rough flow can be assumed, u^* can be evaluated from the profile and the Von Karman constant, k . For other stabilities, the power law profile of Deacon¹⁰⁷ can be used. These lead to values for the turbulent transfer velocity of:

$$v_t = \left[\frac{k}{\ln \left(\frac{z}{z_0} \right)} \right]^2 \bar{u}_z \quad (\text{neutral atmosphere}) \quad (8)$$

$$v_t = \left[\frac{k(1-\beta)}{\left(\frac{z}{z_0}\right)^{(1-\beta)} - 1} \right]^2 \bar{u}_z \quad (9)$$

(unstable or stable atmosphere).

Here, β is the power law coefficient of the change in wind velocity with height, z is the reference height, u_z is the wind speed at the reference height and z_0 is a constant characteristic of the surface, the so-called roughness parameter.

It was noted from field data that the actual deposition velocity differed for different materials. The ^{131}I vapor, for example, had a deposition velocity which appeared to be about the same order of magnitude as that given by equation (7) as did particles with aerodynamic diameters on the order of 1-2 μm . However, smaller particles, such as those produced in an electric arc, had smaller deposition velocities. From this, it was reasoned that the measured deposition velocity is a combination of the turbulent transfer velocity and the retention of the material on the ground once it was brought to the surface. Thus, it was considered that the ground acted as a "perfect sink" for the larger particles and for iodine vapor with a fraction of the smaller particles reflected from the ground and, therefore, not contributing to the measured deposition velocity.

From this basis and meager data on the measured deposition velocity, a very crude model for the effect of particle size was derived. This assumes that the turbulence transfer across the boundary layer is the limiting mechanism for deposition with variations from this transfer rate caused by changes in the efficiency of retention on the ground. From the data available, particles of less than 0.1 μm were arbitrarily assigned a retention of 3% and particles greater than 1.5 μm a retention of 100%. Since no evidence was available on the

shape of the curve between these sizes, a linear relation was chosen as the simplest although it is likely that a sigmoid shape would be more realistic. The actual deposition velocity from a given size particle is, then, obtained by multiplying the turbulent transfer velocity by this retention. Above 1.5 μm , the deposition velocity remains constant at the value of the turbulent transfer velocity until gravitational settling becomes the predominant mechanism. This will occur at differing particle sizes dependent upon the wind speed and stability of the atmosphere. It is again emphasized that this model for the deposition requires considerable refinement before it truly reflects the real world. Not only is the shape of the assumed curve naive, but intuition would tell us that the terrain features may well affect the retention on the surface as well as the turbulent transfer. However, until better data on these factors are available, we will use this model.

Calculation of the deposition velocities for several conditions of particle size, terrain and stability lead to the values given in Table IX. In this table, R_i represents the Richardson's Number which is a measure of the stability.

As a result of this model for deposition, it can be seen that the reflection factor in Eq. (5), can be written to account for the material which is retained on the ground on the basis that if it is retained it is not reflected. Thus, if we use f as the fraction retained, R becomes:

$$R = 2 - f \quad (10)$$

This provides a reflection factor of 2 if the retention is zero and 1 if the retention is 100%.

In reviewing the above equations, there are several points of interest which have a bearing on the form of the calculations. From equations (8) and (9) the turbulent

TABLE IX

APPROXIMATIONS OF DEPOSITION VELOCITIES

(for 1 m/sec wind)

		(for 1 m/sec wind)		
Particle Size	f	Short Grass z = 0.1 cm	Thick Grass	
			10 cm z ₀ = 2.3 cm	50 cm z ₀ = 9 cm
Neutral, Ri = 0				
>1.5 μm	1.0	0.0028	0.0080	0.017
~1 μm	0.7	0.0020	0.0056	0.012
~0.5 μm	0.3	0.00084	0.0024	0.0051
<0.1 μm	0.03	0.00008	0.0002	0.0005
Unstable, Ri = 0.02				
>1.5 μm	1.0	0.0093	0.017	0.028
~1 μm	0.7	0.0065	0.012	0.020
~0.5 μm	0.3	0.0028	0.0051	0.0084
<0.1 μm	0.03	0.0003	0.0005	0.0008
Stable, Ri = 0.08				
>1.5 μm	1.0	0.00046	0.0029	0.0084
~1 μm	0.7	0.00032	0.0020	0.0059
~0.5 μm	0.3	0.00014	0.0009	0.0025
<0.1 μm	0.03	0.00001	0.00009	0.0003

transfer velocity (and, hence, the deposition velocity for a given particle size) is directly proportional to the wind speed. Thus, the ratio of V_d/u is a constant for a given particle size and stability. As this ratio appears directly in equation (6) it is not necessary to provide separate calculations for different wind speeds. Because the deposition rate at a given distance is given by xV_d ; the dispersion varies inversely as the wind speed (equa-

upon the stability, particle size and terrain but not on the wind speed). These factors permit the calculations to be made for a parameter $\chi u/K\Omega$ which permits using various wind speeds, ground concentrations and pickup rates as appropriate once the primary solutions are available. Combining the above equations, we obtain for a point source:

$$\frac{\chi \bar{u}}{K\Omega} = \frac{(2-f)}{\pi C_y C_z x^{(2-n)}} \exp \left[- \left(\frac{y^2}{C_y^2 x^{(2-n)}} + \frac{z^2}{C_z^2 x^{(2-n)}} + \frac{2(2-f)x^{\frac{n}{2}} V_d}{\sqrt{\pi} C_z n \bar{u}} \right) \right] \quad (11)$$

tion 5); and the deposition velocity is proportional to the wind speed, the actual rate of deposition at a given distance will be independent of the wind speed (i.e. the deposition at a given distance will depend

In using the equations, it is necessary to choose parameters which are representative of the conditions to be expected. For the following work, we have chosen three atmospheric conditions: unstable, neutral,

and stable. Sutton's parameters were chosen to give a reasonable fit to Pasquill's classes B, D, and F¹⁰⁴ and values of V_d/u were taken from Table IX. It should be noted that this choice involves a mixing of concepts since the degree of stability represented by the Pasquill classes may or may not conform to the degree of stability represented by the Richardson numbers in Table IX. This, however, is one of the inherent difficulties in the present classifications of the dispersion data. The parameters which have been chosen to represent these three atmospheric conditions are given in Table X.

It must also be recognized that the values of C_y and C_z represent an unspecified time of sampling which for the above values is reasonably long, perhaps one to several hours. For a shorter period, the values, particularly for C_y , would be expected to be smaller.

During the first half of 1973, attention was given to obtaining calculational methods and values for various area sources. Equation (11) can be integrated directly for simple configurations such as an infinite line source or an infinite plane source with, however, the assumption that

$z=0$. In practice we are usually concerned with the concentration at some distance above the ground (usually one meter) as this is where most measurements are made and it is a reasonable representation of the height of concern. In order to obtain the concentration at such a height, it is necessary to perform numerical calculations, at least for those source elements which are close enough so that a significant difference will occur. Solutions for various uniform and Gaussian distributed sources were investigated before it was decided to look at an actual deposition pattern and estimate the influence of wind direction, size of pattern, and stability for this more realistic situation. The GMX area in Nevada was chosen since there has been relatively intensive work on this area in the past few years and experiments on air concentrations are now in progress by the Lawrence Livermore Laboratory.

This area was surveyed with the FIDLER in June 1971¹⁰⁸ using the 60 keV photons from ²⁴¹Am. The isopleths from this survey are given in Figure 15 with the coordinate system located at ground zero. By assuming that the pattern represented in Figure 15 was Gaussian in the cross-pattern direction

TABLE X

CALCULATIONAL PARAMETERS

	<u>Unstable</u>		<u>Neutral</u>		<u>Stable</u>	
	$z_0=0.1$ cm	$z_0=2.3$ cm	$z_0=0.1$ cm	$z_0=2.3$ cm	$z_0=0.1$ cm	$z_0=2.3$ cm
C_y		0.45		0.2		0.3
C_z		0.3		0.1		0.07
n		0.2		0.25		0.5
<u>>1.5 μm particulates</u>						
(2-f)		1.0		1.0		1.0
V_d/u	0.0093	0.017	0.0028	0.0080	0.00046	0.0029
<u><0.1 μm particulates</u>						
(2-f)		1.97		1.97		1.97
V_d/u	0.0003	0.0005	0.00008	0.0002	0.00001	0.00009

Fig. 15. Results of FIDLER survey at GMX area. Solid lines are measured isopleths while dotted are fitted by the method described in the text.

Fig. 16. Ground readings for the centerline of the GMX pattern.

with the standard deviation increasing exponentially with distance from GZ and with a peak deposition at the centerline of the pattern as given in Figure 16, it was possible to obtain the calculated isopleths given by the dotted lines in Figure 15. The standard deviation of the crosswind pattern is given by:

$$y = 37.3 \exp(0.00335a) \quad (12)$$

Where y is the standard deviation in meters at a distance a meters from GZ. It would be desirable to obtain a similar mathematical relation for the peak deposition on the centerline, but none was found.

Since the samplers are planned to be located on the pattern centerline, the initial problem was to estimate the value of $Xu/K\Omega$ for various wind directions so that

the data obtained from these samplers and the associated wind data could be used to estimate the overall value of K for this pattern. The basic approach was to calculate the expected concentration from line sources of Gaussian shape at various distances from the sampling station with winds ranging from 0° (directly down-pattern) to 180° (directly up-pattern). The overall concentration resulting from these winds was, then, obtained by numerical or graphical integration after weighting the Gaussian line values by the appropriate centerline deposition given in Figure 16.

For a Gaussian line source with a center deposition of $\Omega_p \text{ Ci/m}^2$, a standard deviation of A meters, and a wind direction of θ , the concentration-pickup parameter becomes:

$$\frac{\chi_u}{K \Omega_p} = \frac{(2-f)}{\pi C_y C_z} \sum_{y=-4A}^{y=4A} \frac{\Delta}{(a \cos \theta + y \sin \theta)^{2-n}} \exp \left[- \left\{ \frac{1}{(a \cos \theta + y \sin \theta)^{2-n}} \left(\frac{(a \sin \theta - y \cos \theta)^2}{C_y^2} + \frac{z^2}{C_z^2} \right) + \frac{2(2-f)Vd}{\sqrt{\pi} C_z n \bar{u}} \left(a \cos \theta + y \sin \theta \right)^{\frac{n}{2}} + \frac{y^2}{2A^2} \right\} \right] \quad (13)$$

In the special case where $\theta = 0^\circ$ this can be solved analytically to give:

$$\frac{\chi_u}{K \Omega_p} = \frac{\sqrt{2} (2-f) A}{\sqrt{\pi} C_z x^{\frac{2-n}{2}} \sqrt{2A^2 + C_y^2 x^{2-n}}} \exp \left[- \left(\frac{z^2}{C_z^2 x^{2-n}} + \frac{2(2-f)x^{\frac{n}{2}} Vd}{\sqrt{\pi} C_z n \bar{u}} \right) \right] \quad (14)$$

A program was written for the Wang calculator to solve Eq. (13), by Simpson's rule accounting for the change in A by using Eq. (8). This program provided the values for the Gaussian Line source with the peak deposition in each case taken as one so that the weighting for Figure 16 could be done later and the area integration performed manually. This has proven to be a sizeable task and consideration is being given to revising the program to provide the weighting and numerical integration directly. Limits of integration were generally chosen as four standard deviations for the line chosen. For close approaches to the receptor (<20m) it was found necessary to use 1000 steps in Simpson's rule to attain reasonable accuracy although at further distances as few as 25-50 were needed.

Results for the station at GZ with the sampler at one meter height are given for the stable and unstable conditions and for wind directions at 22.5° intervals in Figures 17 and 18. It may be noted that the patterns and values for angles between 180° and 360° may be obtained from this figure by symmetry. These values have been corrected for the variation in centerline deposition and, therefore, represent χ_u/K .

If the resuspension rate is known for a mechanical disturbance, the figures can be used directly to estimate the air concentration at the ground zero station due to a vehicle driving across the pattern at right angles to the centerline with the wind in the given direction. These curves along with the similar ones for the neutral condition were integrated numerically to give the total concentration at this station due to a uniform pickup rate with winds in the various directions. These results are given in Figure 19. There is little dependence with wind direction up to 90° but sizeable increase is noted as the wind direction approaches the down-pattern direction (180°). The increased concentration in the stable condition may well not represent real conditions since wind speeds are normally low under these conditions and we have no information on the possibility that pickup by winds may be lower in this condition because of the decreased turbulence.

In the application of these calculations, it is necessary to relate K, the pickup rate, to the quantity of material on the ground which is in a form and position to be susceptible to resuspension. The

Fig. 17. Values of X_u/K for line sources weighted for centerline deposition with sampler at ground zero of the GMX area. Ω expressed as cnts/min on the FIDLER. Stable conditions.

Fig. 18. Values of X_u/K for line sources weighted for centerline deposition with sampler at ground zero of the GMX area. Ω expressed as cnts/min on the FIDLER. Unstable conditions.

Fig. 19. Values of X_u/K for sampler at ground zero in GMX area. Expressed as cnts/min on FIDLER.

shape of the pattern used for the GMX area was determined by FIDLER surveys using the 60 keV photons from ^{241}Am . Eberhardt and Gilbert¹⁰⁸ provided an initial statistical analysis of data from this area. Here FIDLER readings were made followed by sampling in three 5-inch circles to a three centimeter depth for chemical analysis for plutonium. The number of samples taken for this study was limited and no correlation was found in measurements taken from the two lower isopleths. However, in the >5000 c/m isopleth six samples showed an apparent correlation between the FIDLER reading and the sample analyses. From the data presented, this relation indicated about 0.3 dis/min of plutonium per gram of soil per cnt/min on the FIDLER.

In assessing this correlation, the difference in the character of the two measurements must be considered along with the needs of the resuspension calculation. The soil sample to a depth of 3 cm averages the profile over this full depth and, if converted to a unit such as Ci/m^2 , may include plutonium at such a depth that it is not in the region where the winds can act upon it to produce resuspension. In addition, if the sensitivity of the FIDLER is considered to extend over a radius of one meter, the soil samples represent only about 0.05% of the total area so that any nonhomogeneity in the distribution over this area could affect the results. In order to better assess the meaning of the FIDLER readings,

a rough calibration was calculated, assuming uniform distribution in the soil from data on moving a point source of ^{241}Am across the area to a radius of 65 cm. Using an absorption coefficient of $0.29 \text{ cm}^2/\text{gm}$ and a soil density of 2 g/cc , the FIDLER sensitivity decreases to about 50% of the surface value for a plane source buried about 8 mm deep and to 10% at a depth of 2.5 cm. The FIDLER, therefore, has its maximum sensitivity for the material in the uppermost layers although some response will occur for materials buried as deep as three centimeters.

As can be seen, there are many details on the measurement of the area and the interpretation of the results which are lacking at this time making further interpretation difficult and uncertain. It is believed that some of these uncertainties will be resolved when the full data on the survey are available. In the meantime, a preliminary appraisal was made assuming that the FIDLER readings correspond to a plutonium concentration of 0.3 dis/min per gram with the full depth of 3 cm used in the sampling used to convert this value to Ci/m^2 . This provides a conversion factor of about $1 \times 10^{-8} \text{ Ci}/\text{m}^2$ per cnt/min on the FIDLER. Obviously, this will overestimate the quantity of plutonium which is available for wind pickup since it is not expected that the influence of the wind will reach to a three centimeter depth, but, without further information on the actual profile with depth in the soil, the fraction in the top layer cannot be evaluated.

Some very preliminary data have been obtained in tests of the Lawrence Livermore Laboratory high volume air sampler¹⁰⁹ which was designed and built for purposes of studying resuspension in areas such as this over relatively short periods of time. The sampler, for these tests, was located at a point about 250 feet (76 m) north of ground zero which places it somewhat off of the centerline of the pattern and close to the

area of maximum deposition. If, however, we apply the integrations for the ground zero location to this sampler, we obtain pickup rates ranging from 10^{-12} to 6×10^{-11} per second with the ratio of K/u^2 ranging from 10^{-13} to 2×10^{-11} .

These data were taken over a four hour period with a total of only three samples and wind fluctuations in direction of $50-100^\circ$ as indicated by hourly readings at one meter height. Thus, the correspondence of the coefficients used to the actual conditions is not particularly good with a high probability of more spread in the horizontal than the coefficients would account for. The order of magnitude of these results can be compared with the values for K/u^2 from 10^{-9} to 4×10^{-8} as obtained with fresh zinc sulphide deposits.¹⁰² The uncertainties in the GMX values are large, with many of them tending to minimize the calculated value, but it does seem that they are lower than the ZnS values, perhaps due to weathering and aging of the deposits. The range of the GMX values is also disappointingly large and only further studies will indicate whether this is due to the calculations or some as yet unsuspected factor.

These preliminary calculations have been useful in addressing the complicated problem of accounting for the large number of variables inherent in a satisfactory solution to the resuspension problem and have indicated one approach to modeling and interpreting data from studies in a known area. As has been indicated above, much work remains to satisfactorily account for many of the variables before a truly satisfactory relation can be obtained which will permit assessment of the importance of each of the factors involved.

IV. DIRECT RESUSPENSION - E. Storm, A. Blackstock, J. W. Healy

One of the unsolved problems in assessing the intake of plutonium by individuals living or moving around in an area where

soils are contaminated with plutonium (or other materials) is the possibility of significant exposure through more direct pathways than are accounted for by the more usual considerations of resuspension resulting in a general region of atmospheric contamination such as was discussed in the last sections. In general, these mechanisms involve situations where direct transfer from the soils to the body or high localized concentrations in the breathing zone may result. They range, for example, from the localized concentrations of dust resulting from mechanical disturbance of the soils through working (or even rolling) in the area to such direct mechanisms as the ingestion of soils by children. While some of these possibilities have been investigated for specific situations, they still remain as largely undefined in a quantitative sense so that it is difficult to assess their relative importance. During this period, a review of the literature to obtain information available on such phenomena was made and an attempt was made to further define the situations of interest. However, it is apparent that a more detailed and definitive study will be needed to define the parameters of interest so that an experimental program can be devised to obtain the necessary information.

A possible indication of the importance of such mechanisms is given by the elevated blood lead concentrations in children less than eight years old in an area near a smelter at El Paso.¹¹⁰ Here, the streets and yards were covered with dusty soil and it is speculated that the young children might have ingested enough dirt and inhaled enough soil to cause a problem if the soil were contaminated. Testing of the soils indicated that they contained lead generally in the range of 0.5% and two samples showed greater than 1%. This situation is not clear-cut because there was old paint in some homes with high lead content, there

was improperly glazed foodware in use and the area bordered on a busy highway.

As an illustration of the potential mechanisms, a partial listing of some applicable to people living or working in a contaminated area is given below.

1. Inhalation or ingestion of more localized air concentration due to walking across area or doing light work in area, e.g., gardening, mowing, etc.
2. Transfer of contamination to clothing or skin followed by inhalation or ingestion of material from clothing or skin.
3. Transfer to tools, equipment, vehicles, toys, etc., from which contamination is inhaled or ingested.
4. Transfer to animals (pets or livestock), then inhaled, ingested, or transferred to home. (Note child hugging the pet)
5. Transfer from clothing, skin or contaminated objects, or by wind, into home or place of work where it is inhaled or ingested by anyone present.
6. Direct transfer to food products or water which is ingested.
7. Direct ingestion of soil, e.g., by small children.
8. Transfer either externally or internally to game animals and then to a hunter by inhalation or ingestion.
9. Transfer by produce from garden or farm to home or market and then to people.
10. For material transferred to home or place of work
 - a. direct inhalation or ingestion from clothing or body,
 - b. transfer or inhalation while changing clothes, and
 - c. resuspension and inhalation by anyone present of that deposited on floor, furniture, etc.

A review of the literature was made to obtain any available information which could be useful in assessing these pathways of exposure. Most of the experiments have been directed towards finding a quantitative correlation between the surface deposition and the air concentration, after mechanically disturbing the contaminated surface. This correlation has been expressed in terms of the "resuspension factor," K' , which has units of reciprocal length and is defined by the ratio,

$$K' = \frac{\text{air concentration } (\mu\text{Ci}/\text{m}^3)}{\text{surface deposition } (\mu\text{Ci}/\text{m}^2)} = (\text{m}^{-1}).$$

The earliest measurements appear to be those of Chamberlain and Stanbury¹¹¹ who used ¹³¹I as a tracer during a civil defense study and obtained resuspension factors of $4 \times 10^{-5} \text{ m}^{-1}$ for dirty operations in a confined space and $2 \times 10^{-6} \text{ m}^{-1}$ for similar operations in an open area. Bailey and Rohr¹¹² measured resuspension factors at the Oak Ridge K-25 plant of 1.3×10^{-3} to $1.5 \times 10^{-4} \text{ m}^{-1}$ when fans alone were blown on the floor and an average value of $2 \times 10^{-3} \text{ m}^{-1}$ when both fans and vibration were used. Langham and Harris¹¹³ measured resuspension factors of $7 \times 10^{-5} \text{ m}^{-1}$ after vehicles were driven through a plutonium-contaminated area in Nevada.

These early efforts were followed by a succession of experiments attempting to pinpoint the resuspension value within an order of magnitude or two in a variety of experimental conditions. These efforts have been reviewed by Mishima,¹¹⁴ Stewart,¹¹⁵ Kathren,¹¹⁶ Healy,¹¹⁷ and Langham.¹¹³ The reviewers all point to the wide variation in the measured resuspension factors which range from 10^{-2} to 10^{-13} m^{-1} , and then, inevitably, recommend an "average" value. Mishima suggests 10^{-5} to 10^{-6} m^{-1} . Stewart proposes 10^{-5} to 10^{-4} m^{-1} for outdoors with moderate activity and 10^{-6} m^{-1} for quiescent conditions. Kathren

offers 10^{-4} m^{-1} as a "conservative value." More realistically, Healy proposes values which vary with the degree of activity in the area. Langham feels "intuitively" that a factor of 10^{-6} m^{-1} is a reasonable value to use in estimating the potential hazard of occupying a plutonium contaminated area.

It is instructive to study some of the experimental papers in detail, since they point to many of the reasons for the wide variation in the resuspension factor.

Fish, et al.,¹¹⁸ at Oak Ridge introduced powdered ZnS (MMD = $3 \mu\text{m}$) through a hose into an 8 by 12 by 8 ft high unventilated room. Fans were used to obtain a "more homogeneous" concentration and dust was allowed to settle for two days. A man wearing protective clothing entered the room and performed as follows: (1) vigorous work including sweeping for 10 min, $K' = 1.9 \times 10^{-4} \text{ m}^{-1}$; (2) vigorous walking for 20 min, $K' = 3.9 \times 10^{-5} \text{ m}^{-1}$; and (3) sample collection for 40 min, $K' = 9.4 \times 10^{-6} \text{ m}^{-1}$. A similar experiment with CuO (MMD = $2 \mu\text{m}$) and a man doing light sweeping and sample collection for 90 min gave $K' = 7.1 \times 10^{-4} \text{ m}^{-1}$. The experiment indicates that the resuspension factor varies with the degree of activity, increasing as the activity becomes more vigorous. The degree of activity, however, is described in a qualitative fashion.

Glauberman, et al.,¹¹⁹ at HASL measured resuspension factors at (1) a discontinued plutonium-recovery plant where contamination was uniformly distributed in two unventilated rooms over a 160 ft^2 painted concrete floor, room height 16 ft; and (2) an operating uranium-processing plant with contamination uniformly spread over a $280 \text{ by } 100 \text{ ft}$ concrete floor, room height 50 ft, but two floor areas comparable to the plutonium area was used. They made measurements lasting 30 to 60 min for each room with (1) no movement ($K' = 4.4$ to $16 \times 10^{-7} \text{ m}^{-1}$ for Pu and $K' = 2$ to $19 \times 10^{-7} \text{ m}^{-1}$ for U),

(2) with fans 1 ft above the floor blowing parallel to the floor ($K' = 1.3 \times 10^{-3} \text{ m}^{-1}$ for Pu and $1.3 \times 10^{-4} \text{ m}^{-1}$ for U), and (3) fans plus a dolly moved continuously along the length of the test area ($K' = 1.0 \times 10^{-2} \text{ m}^{-1}$ for Pu and $1.5 \times 10^{-4} \text{ m}^{-1}$ for U). Again K' increased with the degree of the mechanical disturbance. However, the most significant observation is the order of magnitude differences between the Pu and U facility. The differences are attributed to the different physical and chemical characteristics of each nuclide, and to the differences in the relative size of the rooms. The Pu was in small rooms with confined air, whereas the U was in a large industrial building in which air contamination was free to move from the area.

Glauberman, et al.,¹¹⁹ also made air and surface measurements during the regular daytime shifts at the uranium plant. They plot surface contamination as a function of air concentration, and for a given surface value, the air value varies by two orders of magnitude. They obtain a correlation of 0.12 which at the 5% level of significance indicates "airborne dust and surface contamination are independent of one another." They conclude that "surface contamination is not an index of occupational exposure," and that the "data that have been presented here indicate that air-surface contamination relationships derived at one facility will not be valid at another facility, except by coincidence."

These conclusions may be contrasted with those of Schultz and Becher¹²⁰ at the Oak Ridge diffusion plant, who remarkably found a "positive correlation between the average uranium-alpha activity on the work surface and the average urinary-excretion rate": a similar relationship "between the general air and the urinalysis data was not statistically significant at the 95% confidence level."

Brunskill¹²¹ at Windscale Works conducted three experiments in change rooms. In the first one, four to six people walked around a highly contaminated change room 8.5 ft in height, ventilated by nine air changes/hour. Air samples were taken 1.5 and 4.5 ft above the floor, but show no consistent trend with height. At one location the 1.5 ft activity was a factor of four higher than at 4.5 ft, whereas the ratio is reversed at another location. An average value of $K' = 2 \times 10^{-4}$ to 10^{-3} m^{-1} was based on an estimate of the "loose" activity on the floor. A smaller value of K' was obtained with a waxed linoleum floor. After movement ceased, the material settled with a half-life of 20 min.

The second experiment was performed in a 20.5 by 11.5 by 10 ft high room, unventilated except for the free flow of air from an adjacent room. The floor was covered with waxed paper and two people made eight changes of lightly contaminated coveralls denoted as "clean" and then with "dirty" coveralls. The purpose was to determine the relationship between clothing and air contamination. The changing of clean coveralls increased the air activity by factors of 7 to 140 over background. Further changes with dirty coveralls increased air activity by factors which varied from 0.5 to 25. Static air samples gave an average floor-to-air ratio of $3.8 \times 10^{-3} \text{ m}^{-1}$ and an average coverall-to-air ratio of $2.8 \times 10^{-3} \text{ m}^{-1}$. However, the static air samplers differed from personal samplers by ratios which varied from 0.28 to 6.3. Coveralls-to-air ratios measured by personal air samplers averaged $6.4 \times 10^{-4} \text{ m}^{-1}$ but varied from 2.5 to $11 \times 10^{-4} \text{ m}^{-1}$.

In the third experiment, air samples were taken at heights of 2 and 4 ft as people walked through the change rooms during normal working hours. A positive correlation between throughput of personnel and airborne activity was found. However, a plot of air activity as a function of the number of personnel walking through in

a given period shows no correlation. In addition, the ratio of readings at the two heights varied from 0.1 to 10. Of interest is the following remark: on one run "an unusually high level of activity was shown by one sample. An autoradiograph of the sample showed that almost all the activity was associated with one large particle."

Jones and Pond¹²² at Harwell utilized a laboratory with filtered air ($540 \text{ m}^3/\text{h}$) having a floor area 15.7 m^2 and a height of 3.15 m. Fifteen air samplers were placed at five different heights above the floor and the results averaged. Plutonium oxide (particle size <0.4 to $60 \mu\text{m}$) in a water suspension was dropped by pipette evenly over the floor. In a separate experiment, plutonium nitrate in a diluted acid solution was dropped evenly over the floor. The material was left to dry for 16 h. Four different floor coverings (paper, PVC sheeting, waxed and polished linoleum, unwaxed linoleum) were tested. Four different types of activity were carried out: (K'_1) no movement, (K'_2) operator monitors floor for 60 min at 14 steps/min, (K'_3) operator monitors floor for 60 min at 36 steps/min, and (K'_4) two operators walk at 200 steps/min carrying hair dryers aimed at the floor. The results for all observations gave values of K' which ranged from 10^{-7} to 10^{-3} m^{-1} . The value of K' is "markedly affected" by the form of contaminant ($K'_3 = 6 \times 10^{-5} \text{ m}^{-1}$ for Pu oxide and $K'_3 = 5 \times 10^{-6} \text{ m}^{-1}$ for Pu nitrate), the type of floor surface (linoleum gave higher value of K' than paper or PVC), the condition of the surface (unwaxed linoleum gave higher values of K' than waxed linoleum), and movement of the operator ($K'_4 \sim K'_3 > K'_2 > K'_1$). Since each run was followed by a more active experiment, more of the activity became progressively fixed to the floor. Consequently, they obtained the unexpected result that the values of K'_4 were about the same as K'_3 , indicating that the resuspension factor depends upon the type of activity that has occurred prior

to the measurement. Autoradiographic results showed that "measurements of air concentration were biased by a few highly active large ($>5\mu\text{m}$) particles," and that "in practice an even spread of contamination over the whole floor is rarely met."

Butterworth and Donoghue¹²³ at the uranium fuel-element facility at the Springfield Works measured greater air contamination with personal air samplers than with fixed samplers in the working area. They attribute the difference to activity released by contaminated clothing which is collected by the personal sampler but not by the static sampler. In their experiments, increasing the contamination on the clothing tended to increase the air contamination, but beyond a certain level no further increase occurred.

Shapiro¹²⁴ at the Harvard School of Public Health, conducted a number of interesting laboratory experiments. He studied particle dispersion by a variety of mechanisms including air entrainment, particle production following combustion, abrasion, and release to the air from surfaces after contact, collision or rubbing on other surfaces. He also made some field studies with plutonium and americium. He contaminated 1- by 3-in. slides of various materials (lucite, steel, rubber) by suspending them in air in contaminated dryboxes. After contamination from the air in the box, they were removed to another box, vacuumed to remove loose contamination, and fastened to the upper surface of a jig which allowed it to come in contact with a lower similar slide. A series of 50 blows was given to the lower plate by the top plate. The air flowing between them was sampled and the activity on each slide measured. The results varied so widely that he was unable to deduce whether there was any difference between Pu and Am or between the various materials used as slides. In several runs the tests were repeated on the same slide and in all cases the activity released decreased as the experiments were repeated.

Autoradiographs showed that the dispersed activity was carried on a relatively small number of particles with most of the tracks emanating from a few highly radioactive particles. With air flowing between the slides he estimates a resuspension factor of $3 \times 10^{-5} \text{m}^{-1}$ and compares this to Jones and Pond's value of $K'_1 = 2 \times 10^{-8} \text{m}^{-1}$. The repeated contact and separation experiment gave him a K' of $2.3 \times 10^{-4} \text{m}^{-1}$, which he compares with Jones and Pond's value of $K'_3 = 10^{-5}$ to 10^{-6}m^{-1} . He believes that much of the contamination dispersed by the contact-separation test would redeposit rapidly on the floor and only a small fraction would remain airborne. He concludes by saying that his data is applicable only under the conditions in which it was obtained and should be regarded as an example of what can be obtained.

Spangler and Willis¹²⁵ state that the equilibrium airborne concentration from a surface contamination concentration is given by

$$\bar{P} = \frac{C_s R}{D_{rs} D_{ac}} \quad (15)$$

where $\bar{P}$ is the airborne concentration ($\mu\text{Ci}/\text{m}^3$), C_s is the surface contamination concentration ($\mu\text{Ci}/\text{m}^2$), R is the release rate or fraction of the surface contamination which becomes airborne each minute (min^{-1}), D_{rs} is the room-size dilution factor or volume/surface area (m), and D_{ac} is the number of air changes per minute (min^{-1}). If only floor contamination is considered, D_{rs} is given by the room height, h , and the resuspension factor, K' , in terms of these variables is

$$K' = \frac{\bar{P} (\mu\text{Ci}/\text{m}^3)}{C_s (\mu\text{Ci}/\text{m}^2)} = \frac{R}{h D_{ac}} \quad (16)$$

A somewhat more sophisticated approach was employed by Shapiro.¹²⁴ He considers an enclosure with vertical walls, a contaminated lower surface, and sufficient circulating air currents to provide uniform mixing of airborne activity. The change in

particle concentration is given by

$$V \frac{dN_v}{dt} = \lambda_d N_s A - AN_v v_s - \lambda_a N_v V \quad (17)$$

where $\lambda_d N_s A$ is the source term, $AN_v v_s$ is the loss due to settling, and $\lambda_a N_v V$ the loss due to ventilation, N_s is the average number of particles/cm² on the surface, N_v the average number of particles/cm³ in the air, λ_d is the dispersion factor or fraction of the particles on the surface dispersed per unit time, v_s is the particle settling velocity, λ_a the fraction of the air cleared per unit time by ventilation, V the volume, A the area, and $h = V/A$ the height of the enclosure. The buildup of particles due to the dispersion process is

$$N_v = \frac{\lambda_d N_s}{v_s + h \lambda_a} \left[1 - e^{-(\lambda_a + v_s/h)t} \right] \quad (18)$$

The steady-state solution is essentially the formula given by Spangler and Willis. N_v corresponds to F , N_s to C_s , λ_d to R , and $h \lambda_a$ to $D_{rs} D_{ac}$. The addition of the settling velocity in the denominator removes the mathematical embarrassment encountered in the Spangler equation when there is no ventilation. Shapiro tabulates values of v as a function of particle size and demonstrates that under usual conditions of room ventilation, the ventilation effect dominates over the settling velocity term. The exponential term, of course, represents the decay of the airborne contaminant. For the steady-state condition, the resuspension factor, K' , is given by

$$K' = \frac{N_v}{N_s} = \frac{\lambda_d}{v_s + h \lambda_a} \quad (19)$$

In this approach, λ_a and v_s can be estimated, but the basic problem of evaluating the dispersion factor, λ_d , remains. The wide variability observed by Shapiro in his attempts to evaluate λ_d has already been discussed. Shapiro points out that for a

nonuniform distribution, there is no term for activity per unit area, so that the concept of resuspension factor cannot be utilized.

The experiments indicate that the dispersion coefficient depends on (1) the chemical and physical properties of the contaminant. The type (Pu, Am) and form (nitrate, oxide) of the nuclide, the particle-size distribution, the shape of the particle, the surface charge, the amount of coagulation, adsorption, cohesion, and condensation all effect the dispersion. The coefficient also depends on (2) the method of dispersing the contaminant. Introduction of the material by a single event or a continuous series of events, by dropping or settling or a jet stream, and the use of an open or enclosed area with natural ventilation or fans, will effect the uniformity with which the contaminant is spread and the degree to which it lies loosely or is impacted into the surface. The coefficient also depends on (3) the type of floor covering and its treatment. The physical, chemical, and electrical properties of the surface will determine how much contamination is dispersed and these vary with the material (linoleum or concrete), how it is treated (waxed or painted), as well as cleanup schedules and techniques. The coefficient also clearly varies (4) with the degree and kind of activity occurring in the past as well as in the present. The number of people in the area, the clothing worn, the type of play or work, the tools and equipment used, and a myriad of other factors will influence the dispersion.

It is apparent from the experiments that the dispersion coefficient is dependent on numerous interrelated variables which vary with time as well as place, and any empirically derived value applies only to the conditions prevailing at that time.

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