Estimated Production of Human Lung Cancers by Plutonium from Worldwide Fallout

John W. Gofman

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P.O.B. 11207
San Francisco, California 94101

Foreword

The calculations presented here, and in the other reports of this CNR series, represent a first approximation of the biological hazards from plutonium exposure.

In essence, these are studies of the dosimetry of plutonium exposure. There are certain critical voids in mankind's knowledge of the physical and physiological parameters which determine the dosimetry, and thus we have made necessary assumptions which are all clearly identified.

It is anticipated that as additional data become available, the calculations herein will be updated to take them into account.

Summary of Conclusions

1. Worldwide fallout of plutonium-239 (and other plutonium nuclides) from past atmospheric weapons tests have produced a sizeable, and reasonably well estimated, deposition of plutonium in the lungs of inhabitants of the Northern Hemisphere.

2. Since the lung cancers expected per microgram of plutonium inhaled are available (Reference 1), it is a straightforward matter to estimate how many persons have been irreversibly committed to develop plutonium-induced fatal lung cancer.

3. For the USA alone, it is estimated that 116,000 persons have been committed to plutonium-induced lung cancer. In the entire Northern Hemisphere, the total number is $\sim$ 1,000,000 persons.

4. Since the latent period is over for a sizeable part of the plutonium fallout exposure, many of these estimated lung cancer fatalities must be occurring annually now. Probably in the entire Northern Hemisphere, of the order of 10,000 must be dying annually of plutonium-induced lung cancer.

5. Lung cancers, once induced, do not identify themselves as to cause. This is the reason that the absurd, although common, statement can be made that "cancers due to plutonium haven't been observed".

6. The experience of the small groups of Manhattan Project plutonium workers or the Rocky Flats plutonium workers is totally consistent with the expectations for plutonium-induced lung cancer presented here. By no means can these groups provide any comfort whatever for those hoping for a lesser carcinogenicity of inhaled plutonium.

7. Based upon the data presented here for fatal lung cancers already committed by weapons plutonium fallout in the USA, an estimate can be made for the future lung cancers to be produced by the developing nuclear power industry. If that industry contains its plutonium 99.99% perfectly, it will still be responsible for 500,000 additional fatal lung cancers annually. This would mean increasing the total death rate in the United States by 25% each year, since 2,000,000 persons currently die from all causes combined.

ESTIMATED PRODUCTION OF HUMAN LUNG CANCERS BY PLUTONIUM FROM WORLDWIDE WEAPONS-TEST FALLOUT

John W. Gofman*

Introduction:

Plutonium inhaled in the lung, particularly in the form of such insoluble particulates as plutonium dioxide (PuO_2), is one of the most potent lung cancer-producing agents known. Gofman has recently estimated the carcinogenicity of such particles both for smokers of cigarettes and for non-smokers[1]. The results are best expressed in "lung cancer doses", where one "lung cancer dose" is the reciprocal of the lifetime risk per unit of carcinogen. Thus, as an example, if the lifetime risk of lung cancer per deposited microgram of Pu^{239} is x, then the "lung cancer dose" is $(\frac{1}{x})$ micrograms.

For deposited Pu^{239}, the findings were:

For Cigarette Smokers (males), 0.058 μg. Pu^{239} = one lung cancer dose.
For Non-Smokers (males), 7.3 μg. Pu^{239} = one lung cancer dose.

Plutonium has several nuclides, so that it is important to specify whether pure Pu^{239} is at issue, or some mixture of nuclides. Cohen[14], for example, has estimated reasonably that usual reactor plutonium is 5.4 times as hazardous per microgram deposited in the lung, because of the admixture of shorter-lived plutonium nuclides. A convenient way to deal with unknown mixtures of plutonium nuclides is to determine the alpha particle activity in Curies (or some subunit such as picocuries) of Pu^{239} equivalent, and then convert to micrograms, utilizing

16.3 micrograms Pu^{239} = 1 microcurie Pu^{239}.

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*John W. Gofman, M.D., Ph.D. is Professor Emeritus of Medical Physics, Division of Medical Physics, University of California, Berkeley, California.

As a result of worldwide fallout of plutonium from weapons tests conducted in the atmosphere, it is estimated that approximately 320,000 Curies of Pu^{239} equivalent received global dispersion and fallout.[2] Some part of this fallout was inhaled by humans, particularly in the Northern Hemisphere, and is now part of the measured body burden of plutonium observed. In view of the extremely high lung cancer potential of plutonium inhalation, it is important to evaluate how many lung cancer fatalities are currently being caused by inhaled fallout plutonium and how many cases are to be expected in the future.

As will become evident in the body of this report, the plutonium inhaled from worldwide weapons test fallout may have already created, irreversibly, one of the prime public health problems of our era.

Analysis of the Lung Cancer Induction by Plutonium Fallout.

The only additional parameter required beyond those cited above concerning micrograms plutonium per lung cancer dose is the average quantity of plutonium inhaled by humans. In an elegant treatment of this problem, Bennett[3] has provided the estimate that the cumulative inhalation intake through 1972 has been approximately 42 picocuries per person. Since so high a fraction of the total inhaled was inhaled during 1962-1964, and since the years before exceeded the years after, an excellent approximation is that 1962 be taken as an average time of inhalation. Bennett pointed out further that the analysis of tissue burdens suggested the fallout plutonium was most likely to behave like PuO_2, such behavior being what ICRP Task Group on Lung Dynamics would refer to as Class Y compounds (highly insoluble particles).[8]

The calculation of expected number of lung cancers will proceed as in Reference (1), followed by two adjustment factors,

(1) an adjustment for the fact that persons inhaled the plutonium in 1962 versus 1975,

(2) an adjustment (minor in nature) for the retention in bronchopulmonary tissue of the 0.4 micron fallout particles versus those considered in Reference (1).

First Step Calculations.

In Reference 1, the conversion of inhalation to deposition is represented by a factor of four. Therefore, 42 picocuries inhaled represents 10.5 picocuries deposited.

Conversion to picograms of Pu^{239} equivalent yields,

(10.5)(16.3) = 171 picograms Pu^{239} equivalent deposited.

Lung Cancer Dose, for cigarette smokers, = 0.058 micrograms deposited.

, for non-smokers, = 7.3 micrograms deposited.

We shall now consider the generation of males in the USA that received the fallout. There was, of course, a spectrum of men, ranging from children through men of advanced age. The treatment of the problem in Reference 1 was for 20-30 year old men. Since the sensitivity of the group under 20 is higher for cancer induction by radiation, and for the group over 30 is lower for cancer induction, a very good approximation is arrived at by considering the entire generation of men to have received the plutonium fallout at the age range 20-30 years.*

Secondly, we shall assume 50% of the men were cigarette smokers; 50%, non-smokers.

At a US population size of $\sim 2 \times 10^8$ people (1962), (approximately ½ men, ½ women), we arrive then at

5×10^7 cigarette smokers (male)
5×10^7 non-smokers (male).

*See Notes 1 and 2 in "Supplemental Notes".

Lung plutonium deposition in each of these groups is

$$(5\times10^{7})\times(171) = 855 \times 10^{7} \text{ picograms.}$$

Conversion to micrograms yields

$$(855\times10^{7})\times(10^{-6}) = 8550 \text{ micrograms } Pu^{239} \text{ equivalent deposited per } 5\times10^{7} \text{ men.}$$

For the smokers,

$$\text{Lung Cancer Doses} = \frac{8550}{0.058} = 147{,}400.$$

For the non-smokers,

$$\text{Lung Cancer Doses} = \frac{8550}{7.3} = 1170.$$

Total Lung Cancer Doses = 147,400 + 1170 = 148,600.

From the definition of the "lung cancer dose", it follows that this calculation means there will occur 148,600 extra lung cancer deaths in the generation of men receiving plutonium fallout.

For women in the population, there are two considerations to make before calculation.

The spontaneous lung cancer rate for women is approximately 0.27 that of men.* While part of that difference may well be accounted for by the difference in cigarette smoking, that is not yet certain, so an intrinsically lower sensitivity will be utilized for women (0.27 x that of men).

Second, we shall divide the female population into 20% cigarette smokers and 80% non-smokers. Therefore,

For 2×10^{7} cigarette smoking women (versus 5×10^{7} smoking men),

$$\text{expected lung cancer doses} = \frac{2\times10^{7}}{5\times10^{7}} \times (0.27)\times147{,}400 = 15{,}900.$$

For 8×10^{7} non-smoking women (versus 5×10^{7} non-smoking men),

$$\text{expected lung cancer doses} = \frac{8\times10^{7}}{5\times10^{7}} \times (0.27) \times 1170 = 500.$$

* In the relative risk method (see Reference 1), all radiation effects are calculated as being proportional to the spontaneous occurrence rate of the particular cancer under consideration.

Adding all groups, we have: 148,600 +15,900 + 500 = 165,000 extra lung cancer deaths from weapons-test plutonium fallout, before making the two adjustments described above. These must now be considered.

Adjustment 1:

Since all radiation effects are calculated relative to the spontaneous rates in operation at the time of dosage, we must use the 1962 spontaneous lung cancer fatality rate rather than the 1975 rate of Reference 1.

From the recent American Cancer Society estimates[4] it appears a best estimate is that the spontaneous lung cancer fatality rate for 1962 was $\frac{38}{62.5}$, or 0.61 times as high as for 1975.

Therefore, the first adjustment leads to,

(165,000)x(0.61) = 100,700 extra lung cancer deaths from plutonium fallout.

Adjustment 2:

In the treatment developed in Reference (1), the initial deposition in lung was taken as

8% to tracheobronchial region

25% to pulmonary region.

This led to an estimate that the radiation source to the cancer-relevant cells of the bronchi was 0.18 times as strong as that for the pulmonary region for cigarette smokers.

Bennett recommends, for the 0.4 micron particles of plutonium fallout, that appropriate values are,

8% to tracheobronchial region

32% to pulmonary region.

Correcting the pulmonary region (32% instead of 25%) leads to the relevant bronchial cells having a source 1.15 times stronger; thus, (1.15) (0.18) = (0.207) times that of the pulmonary region.

Therefore, the adjustment factor is 1.15 for this effect. The final adjustment of the expected lung cancer deaths leads to:

(1.15)x(100,700) ≃ 116,000 extra lung cancer deaths in the U.S. population (men + women combined) as a result of weapons-test plutonium fallout.*

This represents the best estimate within the framework of data and assumptions that appearsto deserve use at this time.

Expected Time Distribution of These Extra Lung Cancer Deaths.

When cancer is induced by ionizing radiation, there is a period of time, the so-called latent period, before any extra cancer deaths appear in the exposed population. That latent period is somewhere in the neighborhood of 10-15 years for many types of cancer (only about 5 years for leukemia). Thereafter, the cases of cancer increase until the maximum effect is observed, generally called the "plateau" effect. This plateau may last 30 years, or even the whole remaining lifespan of the exposed population. But it must also be remembered that plutonium (or other radiation) operates as a multiplier of the "spontaneous" (or "natural") occurrence rate of fatal cancers. Most (though not all) cancers show an increasing rate of occurrence with age in a population. Thus, even if radiation doubles the spontaneous rate, at an early period of life the absolute number of cancers occurring will be low. As the exposed population becomes older, the radiation-induced cases will occur in increasingly large absolute numbers. For lung cancer, we can estimate how the radiation-induced fatalities will occur, once the latent period is passed. The Surgeon General's report on Smoking and Health provides the requisite data for estimating the distribution of cases. Using data from that report (p.138)[5], the following tabulation has been prepared, Table 1.

*See Note 4 in Supplemental Notes.

Table 1

Expected Distribution of Lung Cancer Fatalities by Age Group, After the Latent Period is Over

Age Group	Percent of Ultimate Number of Cases
Under 40 years of Age	0.2%
Under 50 years of Age	2.2%
50-55 years of Age	3.2%
55-60 years of Age	6.8%
60-65 years of Age	11.3%
65-70 years of Age	17.6%
Between 70-80 years of Age	58.8%

In 1975, some 13 years after our "average" time of receiving the plutonium dose, the latent period is just about over, so the lung cancer cases should be starting to occur. However, the largest proportion of the persons who received plutonium fallout were under 35 years of age in 1962. Thus, when these individuals reach 50 years of age, the data of Table 1 suggest that only about 2.2% of the total number of radiation induced lung cancer fatalities will have occurred. So, by approximately 1977, the extra lung cancer fatalities should be (0.022)x(116,000), or 2550 deaths.

The expected rate will then climb fairly rapidly. For example, when the individuals are in the 60-65 year age bracket, the data of Table 1 indicate that 11.3% of the total number of plutonium-induced cancers will occur, and (0.113)x(116,000) = 13,100 deaths. Similar calculations can be made for any age bracket. Thus, our existing epidemic of fatal lung cancers will become materially increased from plutonium fallout already received, even if all other factors productive of lung cancer remain constant.

There is a special reason for appreciation of the age distribution of expected cases. In the community of nuclear energy proponents there seems to exist the expectation that all the cases

will occur in a very short time. When the full 116,000 lung cancer deaths don't materialize immediately, we can probably count upon nuclear proponents to say, "See, plutonium isn't all that bad".

The number of weapons-test plutonium-induced lung cancer deaths occurring right now is probably of the order of 1,000 cases per year in the USA, since the latent period is just about over. Over the next couple of decades this number will rise steadily in annual rate. Worldwide, the now-occurring plutonium-induced lung cancer deaths must be of the order of 10,000 cases per year.

Worldwide Lung Cancer Production From Plutonium Fallout.

The plutonium fallout from atmospheric weapons testing is worldwide in scope, with the Northern Hemisphere receiving most of the fallout. While Bennett's calculation of 42 picocuries was derived from New York data, there is no reason to doubt that this is a reasonable approximation worldwide (Northern Hemisphere).

Based upon World Health Statistics[4], the spontaneous lung cancer death rates, age adjusted (1968-69), and averaged over 33 countries of the Northern Hemisphere is 33.3 per 100,000 compared with 44.0 per 100,000 in the USA for the same time period.

Since the relative risk method relates radiation to spontaneous cases, the worldwide (Northern Hemisphere) rate, for plutonium fallout, must be adjusted downward by the factor $\frac{33.3}{44.0}$, or 0.76.

As a first approximation, the Northern Hemisphere population, which received the fallout, was some 10 to 15 times that of the USA. Let us use 10x, to allow for possible differences in fallout received (possibly an underestimate).

Therefore, estimated worldwide (outside USA) cases of fatal lung cancer induced by plutonium fallout is

(116,000)x(0.76)x(10), or 882,000 extra deaths.

Combining USA + outside USA, the total = 998,000 extra deaths. Probably some 10,000 extra deaths are occurring annually right now.

Life Expectancy Considerations.

There have been some nuclear advocates who have pointed out that radiation-induced cancers tend to occur late in life, say 60 years of age and later, and that the problem is therefore not serious. What these individuals fail to realize is that the life expectancy at 60 years of age, without benefit of plutonium poisoning, is about 15 years. Would the 60 year olds appreciate losing 15 years of life from plutonium-induced lung cancer?

Are The Estimates Consistent With Experience?

There are few specified population samples with known documented exposure to plutonium deposition in the lung. Two exceedingly small groups are known. The first is represented by 25 Manhattan Project workers who had been discovered to excrete plutonium in their urine, and who, as a result, have been under surveillance. Hempelmann and co-workers[6] have reported on the results of such surveillance. The second is represented by 25 workers who received significant lung burdens in the course of the Rocky Flats fire in 1965.

Without any meaningful quantitative approach, a number of observers have suggested that the non-occurrence of lung cancer to date in these two groups means a relatively low lung carcinogenicity for plutonium. Bair, for example,[7] has suggested this. Non-quantitative approaches can lead not only to absurd, irrelevant

conclusions, but also to very serious underestimations of extremely crucial cancer hazards. It behooves us, therefore, to ascertain here whether the experience to date for the Manhattan Project workers or the Rocky Flats workers is or is not consistent with the estimates presented above for the lung cancer of plutonium inhalation.

The Manhattan Project Workers.

At the outset it must be emphasized that the lung inhalation of plutonium by these 25 workers is exceedingly poorly known. This group cannot be treated as in the treatment above, simply because no inhalation data are available. However, some rough estimates can be made for these workers based upon body burdens measured many years after the exposure had occurred. The problem of estimating initial lung deposition from body burden measured 10-27 years after the exposure is severe. Therefore, at best it would be foolish for anyone to base serious conclusions about plutonium carcinogenicity on the tenuous data for these Manhattan Project workers. However, as a rough effort to ascertain order of magnitude consistency with prediction, it is worthwhile to look at this plutonium exposure experience.

There is every reason to consider that inhalation, rather than ingestion, represents the source of the ultimate body burden of the Manhattan Project workers. Thus, if we really knew the body burden, it would be possible to state that originally this burden had been in the bronchopulmonary system. The difficult problems are to know the body burden at a time of decades beyond exposure, to know how to correct this burden back in time (which involves knowing accurately the fraction of plutonium lost from lung via the gastrointestinal tract), and lastly, but extremely importantly, to know the

degree of solubility of the initial plutonium deposited in the lungs. All of these factors are subject to serious error for these workers, which accounts for the statement above concerning the foolishness of serious conclusions based upon the experience of this group of workers.

Hempelmann and co-workers[6] recently reported on several estimates of the "current" body burden, measured at several times, between 1953 and 1972. These authors suggest that their 1972 estimates are probably their best estimates. However, the excretion curve they utilize for periods beyond ~ a few thousand days, based upon relatively short-term measurements of Langham (for periods shorter than 1500 days), are grossly at variance with estimates that the ICRP model suggests for liver and skeleton clearance or that Bennett uses. The nature of the difference is such as to lead Hempelmann and co-workers to <u>overestimate</u> the body burden of these workers by a large factor.

The ICRP model suggests (see Bennett)[3]

For liver, $T_{\frac{1}{2}} = 40$ years, for man. (40 years = 14,600 days).

For bone, $T_{\frac{1}{2}} = 100$ years, for man. (100 years = 36,500 days).

Therefore, for liver clearance,

daily elimination fraction = 0.693/14600, or $\underline{4.7 \times 10^{-5}/\text{day}}$

and, for skeleton clearance,

daily elimination fraction = 0.693/36500, or $\underline{1.9 \times 10^{-5}/\text{day}}$.

If, as the ICRP model suggests, the liver and skeletal reservoirs are equal in size, then overall excretion would be,

$$\text{daily elimination rate} = \tfrac{1}{2}\,(4.7 \times 10^{-5}) + \tfrac{1}{2}\,(1.9 \times 10^{-5})$$

$$= (2.35 + 0.95) \times 10^{-5}$$

$$= 3.3 \times 10^{-5}/\text{day}.$$

The body burden estimates of Hempelmann and co-workers, for their 1972 evaluation (which they prefer) are based upon an excretion fraction at 27 years (9855 days) of $\sim 2.4 \times 10^{-6}$/day. Their estimate is at variance with what the ICRP model suggests, what ICRP itself suggests[8], and the $T_{\frac{1}{2}}$ values for liver and skeleton calculated above.

The body burden estimated by Hempelmann and co-workers should be reduced by this corrected factor for excretion, which is factor of $\frac{2.4 \times 10^{-6}}{3.3 \times 10^{-5}}$, or $\underline{0.073}$.

For the 25 Manhattan Project workers, the 1972 cumulative body burden (all individuals combined) = 2.44 microcuries Pu^{239} equivalent. (per Hempelmann et al).

Applying the correction factor, 0.073, for excretion, We have

Cumulative body burden (1972) = (0.073)(2.44) = 0.178 microcuries.

We presume, since inhalation was the prime route of access for the plutonium, that all this body burden was originally in the lung. But we must allow, additionally, for the loss of plutonium from the lung via the gastrointestinal tract. Of lung deposited plutonium, the ICRP Task Group model suggests:[8]

40% rapidly lost via gastrointestinal tract

40% lost with $T_{\frac{1}{2}}$ = 500 days via gastrointestinal tract

20% cleared to (lymph + blood).

Therefore, at times long compared with lung clearance, the body burden should be 1/5 of the <u>initial</u> lung deposit, <u>if</u> the gastrointestinal clearance fraction is correct. Bennett has suggested the ICRP model <u>may</u> overestimate the g.i. tract loss. In any case, use of the factor of 5 to convert from current body burden to

initial lung deposit cannot underestimate the initial lung deposit, since it credits gastrointestinal excretion maximally.

Therefore, conversion of body burden, cumulative, for 25 workers, to initial lung deposit, cumulative, yields

$(5) \times (0.178) \simeq 0.89$ microcuries Pu^{239} equivalent.

In micrograms,

$(0.89) \times (16.3) \simeq 14.5$ μgs Pu^{239} as cumulative initial lung deposit.

The smoking history is not available for these men, so we can assume they may have been comparable with the population-at-large, ½ smokers, ½ non-smokers.

Therefore, 7.25 μgs Pu^{239} is cumulative deposition in smokers

7.25 μgs Pu^{239} is cumulative deposition in non-smokers.

Estimation of Lung Cancer Doses, Cumulative, in Manhattan Project Workers.

Before calculation of expected lung cancer doses in the Manhattan Project workers, there are two adjustment factors required:

(a) Exposure was in 1945. From Vital Statistics data, the spontaneous lung cancer rate in 1945 was 0.22 times that of 1975.

(b) Exposure was, in all probability, to relatively soluble compounds of plutonium, from the nature of the work described for the men. Indeed, Hempelmann and co-workers refer to just 2 of the men as "most likely received exposure to plutonium oxide".

We can, therefore, reasonably assign 90% of the cumulative exposure to Class W compounds; 10% to Class Y compounds.

This would represent an average clearance $T_{\frac{1}{2}}$ of

$(0.9)\ (50) + (0.1)\ (500) \simeq 95$ days.

This would require lung exposures to be corrected by $\frac{95}{500}$, or a factor of (0.19), since all the dosimetry calculations are based upon $T_{\frac{1}{2}} \simeq 500$ days for PuO_2 type aerosols.

Finally, therefore, the lung cancer doses, to be applicable to this group, can be corrected for (a) 1945 exposure, and (b) 90% Class W compounds.

Therefore, for the Manhattan Project workers,

for smokers, lung cancer dose $(0.058) \times (\frac{1}{0.22}) \times (\frac{1}{0.19}) = 1.39$ µgs. Pu^{239}

for non-smokers," " " $(7.3) \times (\frac{1}{0.22}) \times (\frac{1}{0.19}) = 175$ µgs. Pu^{239}

Among the cigarette smokers, cumulative initial lung deposit = 7.25 µgs.

so there were $\frac{7.25}{1.39} = 5.2$ lung cancer doses.

Among the non-smokers, cumulative initial lung deposit = 7.25 µgs.

so there were $\frac{7.25}{175} = 0.04$ lung cancer doses.

The total, cumulative among the 25 workers, is 5.24 lung cancers, as a lifetime expectation.

Hempelmann and co-workers describe these men as "in their early 50s". Examination of Table 1 indicates that by the early 50s, the men should have developed approximately 3.5% of their lifetime expectation in lung cancers,

or $(0.035) \times (5.24) \cong 0.2$ lung cancer cases.

Since lung cancer cases can't be fractional, we can say there are 4 chances out of 5 that at the "early 50s" we will observe zero cases; 1 chance out of 5 that one case would have been observed.

The observation of zero cases is directly in accord with the calculations above that indicate the very high probability (4/5) of observing zero cases.

Finally, the conclusion is reached that the Manhattan Project experience is totally consistent with the plutonium lung cancer expectations of this report and of Reference 1. No comfort whatever can be drawn from these Manhattan Project experiences concerning any hoped-for lowering of the lung cancer hazard of plutonium inhalation.

The Rocky Flats Workers.

For this group of plutonium-exposed workers the data are much better than for the Manhattan Project workers. First, measurements by body counting were made within a very short period after the inhalation exposure. Second, Mann and Kirchner[9] reported that the exposure was to PuO_2 particles, so we know that Class Y behavior, with a $T_{\frac{1}{2}} = 500$ days for lung clearance, should be applicable.

The data for the individual exposures were recently provided by Rocky Flats Management.* The mean value for the deposition, expressed by Rocky Flats as a time-weighted-average over the 12 months following exposure, for the 25 workers was 31.6 nanocuries, or 0.032 microcuries. This time-weighted average should closely approximate the lung deposition.* The smoking habits of the workers at exposure remains unknown, so we shall approximate this as ½ cigarette smokers, ½ non-smokers. The average age at exposure was 43.6 years.

For 0.032 microcuries, the lung deposition would have been (0.032) (16.3), or 0.51 micrograms per worker. For 25 workers, the aggregate dose = 25x0.51, or 12.8 micrograms of Pu^{239} equivalent.

Therefore, for the cigarette smokers, dose = ½x12.8 = 6.4 micrograms,

for non-smokers, dose = ½x12.8 = 6.4 micrograms.

Estimation of Lung Cancer Doses in the Rocky Flats Workers.

(a) The exposure occurred in 1965. From Vital Statistics data[4], the spontaneous lung cancer death rate in 1965 was 0.69 times that for 1975.

(b) Mann-Kirchner's evidence indicates that the exposure, in all probability, was to PuO_2, so Class Y (insoluble) behavior is expected.

*Supplemental Note(3) provides the individual case data.

For the cigarette smokers,

1 Lung cancer dose is, therefore, $\frac{0.058}{0.69}$, or 0.084 micrograms Pu^{239}

For the non-smokers,

1 Lung cancer dose is, therefore, $\frac{7.3}{0.69}$, or 10.6 micrograms Pu^{239}.

Therefore, for the cigarette-smoking Rocky Flats workers, the lifetime expectation is $\frac{6.4}{0.084}$, or 76.2 lung cancer doses.

For the non-smokers, the lifetime expectation is $\frac{6.4}{10.6}$, or 0.6 lung cancer doses.

Adding these two groups, the lifetime expectation for the Rocky Flats workers is $\sim$77 lung cancer doses, provided the workers were at a mean age of 25 years at exposure. But since the mean age at exposure was 43.6 years, this expectation must be reduced approximately for the lower risk associated with exposure at ages beyond 25 years (see Supplemental Note 1). From Table IV of the Supplemental Note, it is calculated that for exposure at 43.6 years of age, the risk per rad (or rem) is $\frac{1}{4}$ that for exposure at 25 years of age. Therefore, $\frac{1}{4} \times 77 \simeq 19.3$ lung cancer doses as the final corrected lifetime expectation for the Rocky Flats workers.

In order to maximize the expectation, we shall assume that by 1975, ten years after exposure, the latent period for cancer development is over. From Table 1, it is estimated that for men at 53.6 years (43.6 + 10), approximately 3.5% of the lifetime expectation should have occurred.

Therefore (0.035)x(19.3), or 0.68 lung cancers should have occurred. For an expectation of 0.68 cases, the probability is about 0.5 that zero cases will have been observed. And even this is conservative, since the period to reach the full plateau is quite likely to be greater than 10 years. Thus, the non-occurrence of lung

cancers in this small group of workers by 1975 is totally consistent with the lung cancer potential for PuO_2 exposure derived here and in Reference (1). In no way is a lesser carcinogenicity of plutonium suggested by the Rocky Flats experience.

The time to observe the Rocky Flats workers will be in the next five to ten years. These workers did receive exposure to PuO_2 in a respirable particle size and did receive appreciable doses. Their lung cancer death rate some 10 years beyond 1975 will be of great importance. We can hope, for the sake of the workers, that fewer than 50% were cigarette smokers at exposure. Also, since the lung cancer risk is diminished in ex-smokers, it is to be hoped that the workers were advised to cease cigarette smoking after plutonium exposure.

GENERAL DISCUSSION

The calculations presented indicate that at least 998,000 premature lung cancer deaths can be expected to have been irreversibly committed throughout the Northern Hemisphere as a result of plutonium weapons-test fallout.* It is also expected that, worldwide, these must by now be yielding some 10,000 or more lung cancer fatalities per year. But since the lung cancer cases caused by plutonium exposure do not carry any flag that tells us that these particular cases are the ones caused by plutonium exposure, the absurd statement is possible that "I don't know anybody that's died as a result of exposure to plutonium, do you?"(11)

Perhaps biology will evolve, in time, to accomodate the proponents of nuclear energy, by having each cancer sprout a flag indicating each origin. Until that time, we will have to resort to public health science to derive rational understanding of such problems as

*See Note 2 in "Supplemental Notes".

The effort to downgrade plutonium carcinogenicity by pointing to non-occurrence of lung cancers in the small groups of Manhattan Project and Rocky Flats workers is here shown to be a vain effort. The non-occurrence at this early time is in excellent accord with expectations.

It is the documented history of the promotion of nuclear energy that the cancer hazard of radiation has been underestimated on virtually every possible occasion. When the full story became evident with the passage of sufficient time for the radiation-induced cancers to develop, the authoritative bodies responsible for radiation protection have revised their estimates upward. Thus, it was possible for the National Committee on Radiation Protection to state in 1954[12] that 36,000 millirems would be without effect upon humans, while the BEIR Committee in 1972 estimated that 100 millirems per year (3000 millirems in 30 years) might be anticipated to cause 3500 additional cancer deaths per year.[13] (p. 90-BEIR report).

Bair has recently stated,

> "There has been no recorded instance of cancer in man resulting from the internal deposition of any plutonium isotope in the more than three decades that plutonium has been used. The excellent record has resulted from extremely effective control methods."

There is no reasonable framework in which the Bair statement can be defended. It may even be supposed that Bair may wish to reject all the calculations of this report and of Reference 1. In that event, Bair would be forced to examine his own published data on lung cancer induction by PuO_2 in the beagle dog. The maximum difference between his beagle data and these calculations for humans is a factor of 3.7 fold.[1] Therefore, instead of 998,000 lung cancer

fatalities irreversibly committed by plutonium exposure, Bair would have to estimate at least 270000 fatal lung cancers irreversibly committed. This is a long way from the suggestion above of no cancers from plutonium exposure.

Bair would be correct that the plutonium-induced cancers are not "recorded". But that is only because human cancers have not evolved to the point of printing out a label indicating which of the various carcinogens caused the particular case in point.

Some Implications of the Lung Cancer-Plutonium Fallout Estimates for the Developing Nuclear Power Industry.

The current estimates indicate the number of fatal lung cancers produced for a known fallout intensity. It becomes possible, therefore, to estimate, for various degrees of containment achieved, what the expected number of lung cancers will be from the nuclear power industry. It cannot be assured that the nature of fallout particles from releases in the nuclear power industry will be identical with that for weapons testing. The situation could be worse, equal, or better. The best estimate, within current knowledge, is that the fallout will be similar in character. The calculations will proceed from an estimate of the amount of weapons-test plutonium fallout over the USA to an estimate of the amount, in comparison, that would fall out at various levels of containment in the nuclear power industry. The lung cancer consequences are then directly available by comparison with the results of this report for weapons-test plutonium fallout.

A first approximation to the total plutonium deposition in the 50 states of the USA can be obtained from Bennett's data for New York.[3] His estimate is that the cumulative deposition through 1972 is 2.65 millicuries per km^2 for the New York area. Assuming the average

deposition for the USA is not far different from that for New York, this means that for the USA, with an area (including Alaska + Hawaii) of 3.62×10^6 mi^2, or 9.27×10^6 km^2, the total deposition was $(9.27 \times 10^6)\ (2.65) = 2.46 \times 10^7$ millicuries, or 2.46×10^4 curies Pu^{239} equivalent. Conversion to grams yield $(2.46 \times 10^4)\ (16) = 3.94 \times 10^5$ gms. Conversion to pounds yields $\frac{3.94 \times 10^5}{454}$, or $0.87 \times 10^3 =$ 870 pounds. So, approximately 900 lbs. of plutonium were deposited in the USA through 1972 from weapons testing.

The Tamplin-Cochran estimate (see Reference 1) is that the developing nuclear power industry, from AEC projections, will involve the handling of 400 million pounds for plants installed through the year 2020. Since this will be reactor-grade plutonium, it will be approximately 5 times as α-active as the weapons grade plutonium. Therefore all cancer estimates must be multiplied by five-fold to correct for reactor-Pu versus Pu^{239}.

In the calculations presented here, the deposition of 900 pounds of weapons plutonium has committed some 116,000 lung cancers for the USA. It is instructive to ask what various levels of containment in the nuclear power industry imply for the future production of lung cancers. For such an estimate, it will be <u>assumed</u> that the inhaled plutonium per pound of Pu dispersed will be comparable to that for weapons fallout. In fact, it may turn out to be equal to, greater or less than the case for weapons fallout.

Containment Perfection	Pounds Pu Dispersed	Lung Cancers Produced (corrected for reactor grade Pu)
99%	4,000,000	2,575,000,000
99.9%	400,000	257,500,000
99.99%	40,000	25,750,000
99.999%	4,000	2,575,000
99.9999%	400	257,500
99.99999%	40	25,750

Considering the fallibility of men and equipment plus circumstances of accidents, it would hardly be surprising that containment will not be better than 99.99%, and that represents excellent containment under industrial circumstances. The lung cancer production would be, for such excellent containment, a total of some 25,750,000 cases. Since these cases would be spread over about 50 years, it would represent 500,000 additional lung cancer fatalities per year. Since the current death rate from all causes combined in the USA is about 2,000,000 per year, a nuclear-based energy economy with 99.99% perfection in plutonium containment could mean a 25% annual increase in total death rate from this one source alone. The prospects seem hardly less gloomy even for 99.999% perfection in containment, a containment level that falls squarely in the miracle realm.

It is to be noted that the assumption being made here is that under the circumstances of plutonium release from the nuclear power industry, the plutonium dispersal would be limited to USA, rather than worldwide.

Supplemental Notes

Note 1: Sensitivity to induction of cancer by ionizing radiation is age-dependent. The following table (excerpted from Reference 10) describes the sensitivity variation quantitatively.

Table IV (from Reference 10)

VARIATION IN CANCER INDUCTION PER RAD WITH AGE

These estimates represent a step function approximation in reasonable accord with the data points available in the text.

Age at irradiation (years)	Increase in cancer mortality rate per rad (in Plateau Region) (per cent)
In utero	50
0-5	10
6-10	8
11-15	6
16-20	4
21-30	2
31-40	1
41-50	0.5
51-60	0.25
61 and beyond	Assumed negligible

Note 2: It has been stated here and in Reference 1 that the period on the plateau of radiation effects may be 30 years or it may be the entire lifespan of the exposed population. It must be pointed out that if the plateau truly lasts only 30 years, then the estimated number of lung cancer deaths from inhalation of weapons-test plutonium fallout would require revision, most probably in a downward direction. Crudely, this would be so because for those individuals exposed early in life, e.g. below 20 years of age, the 30-year plateau period (after the latent period) could be over before these individuals have reached the ages characterized by high absolute lung cancer fatality rates.

A more refined treatment would also require consideration of the additional fact that for those exposed while very young, the cigarette smoking factor is almost certainly absent, so that there would be a revision required in the lung cancer dose for such individuals. Such a refined treatment, similar to that of Reference 10, would divide the population exposed by age decade at time of exposure, would calculate an appropriate lung cancer dose for each age decade, and would calculate the absolute numbers of expected fatalities for various plateau durations, particularly for 30 years and for the remaining lifespan of the exposed populations.

The currently-presented calculations really represent a hybrid calculation. They tend to underestimate the overall effect by crediting only 30 years as the period at risk. On the other hand, for the reasons stated above relating to expiration of the plateau period, they tend to overestimate the overall number of cancers. The refined calculations will be presented in a later report of this series. It must be emphasized, however, that ultimately the real resolution to the problem must come from determination of plateau duration in humans through continued followup of exposed population groups, e.g., the Hiroshima-Nagasaki and spondylitis groups.

Note 3: The individual exposure data for the 25 Rocky Flats workers are not recorded in the published literature, nor are their ages. Since the Rocky Flats Management was exceedingly cooperative in providing these data, they are reproduced as Table V below.

The immediate lung deposition in these workers versus time-weighted average (as in Table V) would depend critically upon the

exact time after exposure for each initial measurement and on the very early clearance fraction of deposited PuO_2 in man. Since these are not available, there is no way to correct the data here for these effects. At most, the lung cancer expectation would not be increased by a factor of two, so that no change in conclusions reached would be indicated. And since the expectation has been maximized by assumption of full plateau by 10 years, the argument presented is further strengthened.

Note 4: It is highly probable that the bulk of the exposure reflected in Bennett's inhalation estimates are from direct fallout of plutonium rather than from resuspension of already deposited plutonium. Estimation of contributions from resuspension is difficult, during a period when direct fallout is still occurring. To the extent that resuspension occurs in the future, the estimated numbers of lung cancers will increase beyond the estimates presented here.

Note 5: In the discussion of the Manhattan Project and Rocky Flats workers, the possibility of having more lung cancer doses than the number of workers was included. It is self-evident that it only takes one cancer to kill a person. However, it is essential to allow for multiple lung cancer doses per person for correct analysis. In actual observation, effects arising from this are manifested as an earlier appearance of the lung cancers that would be otherwise expected.

TABLE V

THE DOW CHEMICAL COMPANY

ROCKY FLATS DIVISION
P. O. BOX 888
GOLDEN, COLORADO 80401

June 23, 1975

John W. Gofman, M.D.

RESPONSE TO REQUEST FOR INFORMATION ON 25 EMPLOYEES EXPOSED TO PLUTONIUM IN OCTOBER 1965

The following is a list of employees by age and their respective plutonium exposures. The amount in the lungs (chests) of the 25 employees is a time-weighted-average* over the 12 months following the exposure.

age	plutonium (nCi)	age	plutonium (nCi)
24	13	44	7
24	16	45	12
24	19	46	11
29	15	49	20
33	56	52	100
33	12	53	140
38	8	56	130
39	14	56	12
39	23	59	34
39	18	59	59
40	18	60	10
42	9	64	24
42	11		

* Time-weighted-average of 16 nCi in the lung produces 15 REM per year.

We have no records of individual's smoking habits.

H. E. Bowman
General Manager

CRL:mk

cc: W. M. Lamb, RFAO
C. R. Lagerquist

LIFE IS FRAGILE - HANDLE WITH CARE

References

1. Gofman, J. W. "The Cancer Hazard From Inhaled Plutonium". CNR Report 1975-1. The Committee For Nuclear Responsibility, P.O. Box 2329, Dublin, California, California 94566. May 14, 1975.

2. Hardy, E. P., Krey, P. W., and Volchock, H. L. "Global Inventory and Distribution of Fallout Plutonium", Nature, 241, 444, 1973.

3. Bennett, B. G. "Fallout ^{239}Pu Dose to Man". pp I:41-61, in: Fallout Program Quarterly Summary Report, Health and Safety Laboratory, U.S. Atomic Energy Commission Report HASL-278, January 1, 1974.

4. American Cancer Society "Cancer Statistics, 1975", in Ca - A Cancer Journal for Clinicians, p. 10-11, 25, No. 1 (Jan-Feb), 1975.

5. Advisory Committee to the Surgeon-General of the U.S. Public Health Service. "Smoking and Health", Chapter 9, Cancer, p. 138, on Age-Adjusted Mortality Rates for Cancer of Lung and Bronchus. Public Health Bulletin 1103, 1964. Superintendent of Documents, U. S. Government Printing Office, Washington, D.C. 20402.

6. Hempelmann, L. H., Richmond, C. R., and Voelz, G. L. "A Twenty-Seven Year Study of Selected Los Alamos Plutonium Workers". LA-5148-MS, Informal Report, Los Alamos Scientific Laboratory, Los Alamos, New Mexico 87544, January, 1973. Available, National Technical Information Service, 5285 Port Royal Road, Springfield, Virginia 22151.

7. Bair, W. J., Richmond, C. R., and Wachholz, B. W. "A Radiobiological Assessment of the Spatial Distribution of Radiation Dose From Inhaled Plutonium". WASH-1320, US AEC, September, 1974. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

8. International Commission on Radiological Protection, Publication 19, "The Metabolism of Compounds of Plutonium and Other Actinides", Adopted by the Commission, May 1972, Pergamon Press, Oxford, England, 1972.

9. Mann, J. R. and Kirchner, R. A. "Evaluation of Lung Burden Following Acute Inhalation Exposure to Highly Insoluble PuO_2". Health Physics 13, 877, 1967.

10. Gofman, J. W. and A. R. Tamplin, "Epidemiologic Studies of Carcinogenesis by Ionizing Radiation", in Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability", Statistical Laboratory, University of California, U.C. Press, Berkeley, California 94720. 1972.

11. Johnson, D. M. (Vice-Chairman of the Atomic Industrial Forum). "A Voice from the Barricades", p. 9 in Harper's Weekly for May 2, 1975. Harper's Magazine Co. Two Park Avenue, New York, N.Y. 10016.

12. Tompkins, P., quoting directly from 1954 N.C.R.P. Statement. in "Environmental Effects of Producing Electric Power" Hearings before the Joint Committee on Atomic Energy", 91st Congress, 1st Session, October-November, 1969, Part I.

13. Advisory Committee on the Biological Effects of Ionizing Radiation. "The Effects on Population of Exposure to Low Levels of Ionizing Radiation", Division of Medical Sciences, National Academy of Sciences, National Research Council, Washington, D.C. 20006, November, 1972. (Popularly known as the BEIR Report).

14. Cohen, B. L. "The Hazards in Plutonium Dispersal". Report of the Institute for Energy Analysis, Oak Ridge, Tennessee 37830, March, 1975.

Committee for Nuclear Responsibility, Inc.

P.O.B. 332, Yachats, Oregon 97498

Correspondence address: **P.O.B. 11207, San Francisco, California 94101**