Testimony for the GESMO Hearings

Submitted by

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February 21, 1977

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This is CNR Report 1977-1.

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Testimony by John W. Gofman for the NRC GESMO Hearings

The testimony presented here addresses the considerations of the toxicity of plutonium. It addresses GESMO, Volume 3, Section lV-J, Radiological Health Assessment. There are other sections of GESMO which also address or mention the toxicity of plutonium. To the extent that this is so, this testimony addresses those sections as well.

Contents of Testimony

Note: All five items listed above are integral parts of this testimony.

General Response to All Critiques

of

John W. Gofman's Estimates of the Lung Cancer Hazard of Plutonium

Introduction:

The estimates of the lung cancer hazard of inhaled plutonium are set forth in three papers by John W. Gofman. These are

(1) The Cancer Hazard From Inhaled Plutonium, CNR Report 1975-1R, dated May 14, 1975

(2) Estimated Production of Human Lung Cancers By Plutonium from Worldwide Fallout, CNR Report 1975-2, dated July 10, 1975

(3) The Plutonium Controversy, JAMA, 236, No.3, pp284-286, July 19,1976.

For simplicity in the ensuing discussion , reference to these reports of a general character will be by the term "Gofman-Pu papers". References of a specific character will be to "CNR-1R", or "CNR-2", or "JAMA".

Before the appearance of the Gofman-Pu papers, the general "wisdom" was that the lung cancer potential of plutonium could be adequately described by consideration of the deposition and retention of PuO_2 aerosols (or other insoluble Pu compounds as aerosols) in the lung tissue of the bronchiolo-alveolar regions, with neglect of any potential effect on more proximal bronchial tissues, that is tissues proximal to the terminal bronchioles. The models used in achieving that "wisdom" predicted no significant long-term retention of plutonium would occur in the bronchial tissues proximal to the terminal bronchioles. Hence it was predicted that there would be no significant dose to such more proximal bronchi . Gofman raised the question as to whether the conventional "wisdom" might be in error, which it certainly seems to be. Since even a small percentage retention in the proximal bronchial tissues could drastically increase the lung cancer hazard of plutonium aerosols, it becomes of extreme importance to know just how much plutonium is subject to long-term retention in the bronchial tissues proximal to the bronchiolo-alveolar region. The plain fact is that no studies have been accomplished to describe such retention of plutonium in either of two major classes of humans, cigarette smokers and non-smokers. It is regrettable that such studies have not been accomplished .It is further an indication of the lack of competence of AEC and ERDA that in all their highly-funded studies of plutonium, these crucial questions have not been addressed, particularly when one considers the central importance of the issues in-

volved.

In the Gofman-Pu papers it was pointed out that the major part of the mechanism counted upon to remove Pu rapidly from the bronchi was the set of cilia lining the bronchial cells. These cilia beat in a direction such that material is propelled in the direction of the nasopharynx. The actual material being propelled is, at least in part, mucus secreted by goblet cells in the bronchial lining, such mucus probably incorporating part of the particulate material being cleared. Part of the clearance mechanism may also be the propulsion of cells of the macrophage class by cilia. The cilia are severely damaged in humans who smoke cigarettes. Such damage is two-fold in character, namely actual denuding of the cilia of cells and functional impairment of ciliary action in those retaining cilia. Additionally the smokers have replacement of the normal bronchial epithelium by metaplastic epithelium in numerous regions. Similar alterations occur in non-smokers but to a far lesser degree. JAMA presents the references both to the studies of Ide and of Auerbach on these issues.

No one really knows why PuO_2 aerosols are retained so tenaciously in the bronchiolo-alveolar region, tenaciously enough to give $T_{\frac{1}{2}}$ clearance times of the order of 500 days from this region. But there is no doubt that the clearance time is long. Many who have commented on the long-term retention in this region have ascribed it to the absence of ciliated cells, so that the so-called "muco-ciliary escalator" doesn't work there as it does in the more proximal bronchi.

If the cilia are absent in 25-30 % of the bronchial cells and if the cells are altered to metaplastic epithelium, what has happened in such regions to the ability to clear PuO_2 aerosols? As Gofman pointed out <u>carefully</u> in the Pu papers, there simply are no data on humans (nor any valid experimental animal model) to answer this crucial question. <u>No data at all.</u> Gofman therefore suggested that, as a first approximation, the best estimate is that clearance from such regions of ciliary absence and metaplasia will become equivalent to that for the non-ciliated bronchiolo-alveolar region , yielding $T_{\frac{1}{2}}$ values of the order of 500 days. To be sure, altered bronchial epithelium is <u>not</u> identical with bronchioloalveolar tissue. It may be, as Gofman pointed out, that altered bronchial epithelium is more capable, equally capable , or less capable in PuO_2 clearance compared with bronchiolo-alveolar epithelium. It is the height of public health irresponsibility to <u>assume</u> that clearance is unaltered in the damaged

bronchial epithelium. Such an assumption emanating from laboratories supported by ERDA or NRC should be totally suspect, in view of the vested interest of those laboratories in a low order of plutonium toxicity.

The Criticisms of the Gofman Hypothesis

In five separate reports, the ERDA laboratories have produced critiques of the Gofman-Pu papers. While I shall deal with each of those reports separately below, the substance of the criticism has to do with two points:

(a) The $T_{\frac{1}{2}}$ clearance time utilized by Gofman for the injured bronchial epitheium just "must be too high", and

(b) Gofman has used a much higher figure for the relative risk of lung cancer per rem of ionizing radiation than the BEIR committee and this accounts in part for his high toxicity of plutonium.

I shall deal with these two points in this general response. As for the remainder of the criticisms, they are so far over on the side of absurdity that they hardly deserve any comment whatever. This will be demonstrated in the responses to the specific critiques.

Criticism (a)

Other than wishful thinking, the five ERDA lab critiques have provided nothing at all to challenge the Gofman value of $T_{\frac{1}{2}}$ of clearance from injured bronchial epithelium. They have, therefore, assumed (not presenting any basis whatever) that even after cilia have been lost and damaged, after normal cell epithelium is replaced by metaplastic epithelium, this bronchial epithelium will still be far more efficient in clearing PuO_2 particulates than the non-ciliated epithelium of the bronchioalveolar region. In the absence of any evidence to support their position, they simply choose to be optimistic about the lung cancer potential of plutonium . This is public health irresponsibility at its worst! To the extent that ERDA and/or NRC accept(s) such nonsense as science , they are being totally derelict in their legal responsibilities concerning the health and safety of the public. All estimates of the hazard of the nuclear fuel cycle, of reprocessing, of mixed oxide fuel fabrication, of appropriate standards for so-called " permissible" exposure either to the public or to occupational workers are simply worthless until the question of plutonium retention in the bronchial epithelium is resolved. Gofman

has made the very reasonable (and modestly conservative) suggestion that conversion of ciliated bronchial epithelium to non-ciliated epithelium plus some metaplastic epithelium makes them act , with respect to PuO_2 retention, like non-ciliated bronchiolo-alveolar epithelium. Moreover, he suggested that metaplastic epithelium might be more active, by phagocytosis or endocytosis, than is the case for the bronchiolo-alveolar region, and that this could indeed make his estimate inadequately conservative. Gofman emphasized that the burden of proof that his proposed behavior does not represent reality rests upon those who choose to believe a more "optimistic" behavior will occur. This is clearly and unequivocally as it should be in such matters of public health importance, a point not apparently appreciated in any of the five ERDA lab critiques. While such lack of appreciation cannot be condoned, it can be understood in the light of the desire not to bite the hand that feeds them.

Meanwhile, in striking contrast to the opportunistic gobble-dygook presented in the five ERDA lab critiques, there has appeared a most valuable study of human material by Radford and Martell. This paper bears directly upon the clearance of insoluble particulates from bronchial tissue. The paper is " Polonium-210: Lead-210 ratios As An Index of Residence Times of Insoluble Particles From Cigarette Smoke in Bronchial Epithelium" by Edward P. Radford and Edward A. Martell, and was presented at The Fourth International Symposium on Inhaled Particles and Vapors , Edinburgh, 22-26 September 1975. (in press, Pergamon Press Ltd.). The introduction to this paper, appearing just a few months after the CNR-1 and CNR-2 papers, re-iterates and confirms the points made by Gofman, and it does that so succinctly, that I think it important to reproduce that introduction here as follows: (I now quote Radford and Martell)

> " Up to the present, models of the deposition, retention, and clearance of insoluble particles from pulmonary tissues have emphasized processes within the lung parenchyma, with the bronchi being considered primarily conduits through which mucociliary clearance occurs. There is recognition that preferential deposition of some inhaled particles occurs by impaction at special locations in the bronchial tree, such as at bifurcations of the trachea and major bronchi, and there is also evidence that localized regions of the epithelium may have inefficient ciliary clearance because of splitting of the mucociliary stream at bifurcations, development of squamous metaplasia and loss of ciliary function in small areas, or other mechanisms affecting mucociliary competence. For these reasons, one would conclude that in some regions

(Radford-Martell quote continued)

> of the bronchial epithelium, especially at bifurcations, localized concentration of insoluble inhaled particles might accumulate, both by direct deposition or after entrainment in the mucociliary stream and subsequently becoming trapped in regions of of inefficient clearance. Especially in relation to development of bronchial cancer, certainly one of the most serious consequences of inhalation of particles, the failure of current lung clearance models to take account of the retention of materials in the bronchial epithelial structures is one of the most serious limitations of these models."

This quotation expresses precisely the same type of thinking presented in the Gofman-Pu papers.. The experimental data of Radford and Martell are used to try to estimate the residence time (mean value) for the Lead-210-containing insoluble particles in mainstream cigarette smoke. These authors point out that the presence of some soluble Lead-210 in bronchial tissues will have the effect of giving too low a mean residence time for the insoluble particles by their method of analysis. Yet, in spite of this, they estimate a mean residence time of 3 to 5 months for the insoluble particles. This , in days , is between 90 and 150 days. Taking a middle value of 120 days this would be some six times lower than the mean residence time Gofman estimated for PuO_2 aerosols in the injured part of the bronchial epithelium (500/0.693 is approximately 700 days). It must be noted that the Radford-Martell values are for overall bronchial tissue samples, not the injured regions alone. Thus over and above the Lead-210 (soluble) error which tends to make their residence times too low they are including some normal bronchial tissue which may have a very much shorter residence time than the injured tissue. When these two factors are taken into account, their experimental evidence on human material may finally suggest an even longer $T_{\frac{1}{2}}$ for injured bronchial tissue than that suggested by Gofman. These data ,preliminary as they are, are enormously closer to the estimates of Gofman than to the optimistic suggestions of a fraction of a day or a day for $T_{\frac{1}{2}}$ in injured bronchial epithelium to be found in the ERDA lab critiques.

Dr. J. Martin Brown (Health, Safety , and Social Issues of Nuclear Power and the Nuclear Initiative, Chapter 4 in "The California Nuclear Initiative" , Stanford University Institute for Energy Studies, April 1976) stated, concerning the ERDA critiques, the following;

" However, none of the critics seems to be aware of recent data

showing the extensive holdup of insoluble particles of polonium (an alpha-emitter, like plutonium , that is concentrated on tobacco leaf hairs) in the bronchial tree of smokers. This, at least qualitatively, supports Gofman's hypothesis and certainly suggests further study is urgently needed."

Dr. Brown is referring to the Radford-Martell studies cited above.

Of all the five ERDA lab critiques, only one, that by Healy and co-workers,shows even a glimmer of appreciation of the problem of plutonium retention in the bronchi. In their conclusion they state

" In our review of his papers we have concluded that the speculations of Gofman require the arbitrary acceptance of too many numerical parameters and unconfirmed mechanisms to be acceptable as even an approximate numerical estimate of potential lung carcinogenesis by plutonium . There is, indeed, a paucity of direct measurements of clearance rates for intact and damaged bronchial ciliated epithelium but current information would indicate that the problem is not as serious as postulated by Gofman. We would recommend that measurements continue with more emphasis on the absolute bronchial retention, and that until such evidence is available , the Gofman predictions be regarded as interesting and imaginative speculations which should serve to stimulate increased interest in certain phases of current studies. However, we cannot concur with his often stated position that speculation, no matter how poorly founded, is a proper basis for public health decisions."

It is of some importance to dissect this amazing paragraph of admission of ignorance concerning the clearance of plutonium by intact and damaged epithelium. Starting with the gratuitous insult at the end of their quote, I might point out that it will rain 40 inches per day in the Sahara Desert before the Healy group will be able to show a <u>single</u> instance where Gofman suggested that speculation , no matter how poorly founded , is a proper basis for public health decisions , let alone " his often stated position" . Nevertheless, one must be tolerant in such matters. Here is one of ERDA's set of experts on plutonium toxicity publicly admitting that there is absolutely no way that they can help refute Gofman's estimates, admitting that Gofman is correct concerning the absence of the crucial measurements, advising that the measurements Gofman says are essential must be given high priority. It is easy to understand their frustrations about their admissions which lead them to use insults when they have no science to offer in refutation.

There is one additional point in the Healy et al critique that deserves translation. That is in the quote above where they say "current

information would indicate the problem is not as serious as postulated by Gofman". On a matter of such transcendent importance one would expect scientists, in possession of such "current information", would tell us how serious the problem is, if it is not as serious as postulated by Gofman. On this, they are silent. One cannot help thinking, since they do not provide the " current information", that what they mean is current wishful thinking that might please ERDA.

Criticism (b):

The essence of this second criticism is that Gofman used too high a value for the relative risk of lung cancer development per rem of exposure . Cited as evidence that Gofman used too high a value are a value of 0.20 for the relative risk and a value of 0.29 for the relative risk in both the BEIR Report. I shall return to the excellent reasons why Gofman rejected these values. At this point I should like to take up the reason why Gofman quoted the BEIR statement that the relative risk might be 0.5% or even higher--- and even so Gofman rejected that value as four times too low. Much of this discussion is in a sense moot since new evidence is now available from the Hiroshima-Nagasaki studies (1975) and they not only show that Gofman's choice of 2% per rem was the proper choice at the time, but also the new evidence indicates that even the Gofman estimate of relative risk per rem might be too low, NOT too high.

The relative risk method suggests that radiation acts as a multiplier on the risk of carcinogenesis from so-called "spontaneous" or "natural" causes. Now, virtually everyone in the cancer field considers that the so-called "spontaneous" causes of cancer are ,at least to the extent of some 90% of all causes, environmental factors of a variety of sorts. In the case of lung cancer in man, it is widely accepted that the environmental factor is the smoking of cigarettes, and it is widely considered that cigarette smoking is responsible for 90 % of all human lung cancers. To my knowledge the only contrary opinion is that of the tobacco industry, which is hardly the surprise of the century. If radiation acts as a multiplier of "spontaneous" cancer, the corollary is that it will, in general, act as a multiplier of the cancers caused by the environmental factors that are the basis for so-called "spontaneous" cancers. Is this reasonable? Particularly in the case of lung cancer induced by alpha particle radiation which

is at issue here, is this reasonable? The answer is overwhelmingly in the affirmative. An excellent study was reported by Lundin et al

(Lundin, F.E., Jr.,Wagoner, J.K.,Archer, V.E. "Radon Daughter Exposure and Respiratory Cancer:Quantitative and Temporal Aspects" NIOSH-NIEHS Joint Monograph No.1 US DHEW,1971.)

contrasting the effect of alpha particle irradiation of uranium miners in the production of lung cancer in cigarette smokers versus non-smokers . They demonstrated that the effect of radon daughter alpha particle exposure was approximately ten times higher in the cigarette smokers than in the non-smokers for production of human lung cancer. It would take the utmost mayhem with these results to interpret the radiation as anything but a multiplier. These results are very directly relevant since they arise from high LET radiation (alpha particles) which is at issue with respect to plutonium radiation. But there have long existed data concerning radiation as a multiplier for low LET radiation as well. The studies of Stewart and her colleagues on the effect of pre-natal radiation (X-rays) in induction of cancer and leukemia in childhood are by now classic. A prime result of those studies was the demonstration that cancers and leukemias were induced by radiation in proportion to the "spontaneous" occurrence of those diseases.

Among those who fail to understand this problem there seems to exist the mystical idea that the relative risk method and the absolute risk method give rise to different answers concerning the number of cancers per rem of ionizing radiation. Nothing could be further from the truth. Both methods must give the same answer or one of the methods is simply not correct! The virtue of the relative risk method is that it enables predictions in situations where actual experimental data are unavailable. Should the results of the relative risk method ever predict any result at variance with absolute risks as measured, one would have to give up the relative risk method. That has not yet happened in any study with reliable,meaningful data.

A crucial feature of the relative risk method is its prediction that the multiplier for radiation is very sensitive to the age at irradiation. In CNR-2, Gofman reproduced a table (Table lV from Reference 10 cited there) showing how the percent increase in cancer mortality per rad varies with age. For the 21-30 year age group (and it is this age group that CNR-1, CNR-2 and JAMA explicitly address) the value is 2 % per rad. For the 41-50 year age group the value drops to

0.5 % per rad. Gofman and Tamplin published a report in 1970

Gofman, J.W. and Tamplin, A.R. " Radiation-Induction of Human Lung Cancer", Hearings of the Joint Committee on Atomic Energy, January 28, 1970) pp. 389-399.

which presented the doubling dose for radiation induction of human lung cancer based upon the Japanese studies of Wanebo et al . That doubling dose was estimated to be 79.7 rads and applied to a population of Hiroshima-Nagasaki subjects over 35 years of age. A value of 79.7 rads as a doubling dose corresponds to 1.25 % per rad. Just recently the extended data from Japan have appeared in a paper by Beebe and Kato from which the per cent increase in cancer per rad

Beebe, G.W. and Kato, H. "Review of Thirty Years Study of Hiroshima and Nagasaki Atomic Bomb Survivors II. Biological Effects,E. Cancers Other Than Leukemia ",Journal of Radiation Research , Supplement, pp. 97-107, 1975.

for lung cancer can be estimated. The data of Table 1(reference just cited) give a relative risk of 140% for the group having received between 10 rads and 100 rads and a relative risk of of 200% for the group having received over 100 rads. The mean dose for the 10-100 rad group cannot be far different from 50 rads. It would be a conservative estimate to assign a value of 200 rads for the group over 100 rads . These data then translate into 0.8% per rad for the 50 rad mean, and into 0.5% per rad for the 200 rad mean. Since there is no overt reason to weight one group more than the other, the final best estimate is 0.65 % per rad. It is virtually certain that this value is too low, though we shall not alter it. The reason it is too low is that all cancers developing between 1950 and 1972 are included. This means that part of the observation period was during the latent period for lung cancer development. Thus spontaneous cancers occur during the latent period, but radiation-induced ones do not, giving a falsely low value for the radiation-induced cancers relative to the spontaneous ones. The earlier estimate of Gofman-Tamplin of 1.25 % per rad is probably closer to the true value, but since it is an estimate of the effect of including part of the latent period , we shall not insist on it here, and shall accept the 0.65% per rad figure that comes out of the extensive recent Japanese data. Beebe and Kato are quite explicit in stating that these data refer to those who were 35 years of age or older at the time of bombing. They do not provide

the mean age at time of bombing for this group , but that mean age cannot possibly have been far away from 45 years. If we use 45 years we shall be conservative. So, for a group of mean age of 45 years we have a mean value of 0.65% increase in cancer per rad, versus a value of 0.5% predicted by Gofman and Tamplin through the relative risk method. Calculating back to persons 21-30 years of age, the new Japanese data would suggest 2.6% per rad rather than the 2 % per rad utilized by Gofman in the Gofman-Pu papers. If anything ,therefore, Gofman underestimated the cancer hazard of radiation .

The ERDA lab critiques suggest that Gofman should have used a value of 0.2 % per rad (actually 0.19%) to be found in table f-2 of the BEIR report. This suggestion is so absurd on the face of it that it shouldn't require explanation, but apparently explain we must. First of all the BEIR value is for people over 35 years of age* Actually the BEIR committee erred in that table because they label the group as having a mean age of 35 years* Beebe and Kato are explicit in pointing out that even now they are unable to address the issue of persons under 35 years* The most probable estimate of true mean age for the group was 45 years or more. Since the Gofman-Pu papers address the risk for persons 21-30 years(and most explicitly so), it would be appropriate to multiply that 0.2% value by four, giving 0.8%. But this is only the beginning of the error that would occur using the crude tabulated value from BEIR. The BEIR Committee recognized this in a very important paragraph which is here reproduced from p.156 of the BEIR report;

> " All four of these groups are still under investigation, and it is probable that because of the long latent period for lung cancer, the rates calculated will rise as further cases develop. This is particularly true for the spondylitis patients. It is possible , therefore, that in the final analysis the absolute risk in these groups will approach $2/10^6$/year/rem and the relative risk reach 0.5% or higher. For the three groups (miners and Japanese survivors in which up-to-date information is available,)it is significant that many new cases have been added in the past few years."

Note that even the 0.5% figure suggested would have to be corrected to convert the value to that appropriate for 21-30 years of age. So, the BEIR Committee very explicitly tells us that the value in the table is outdated, is contaminated by cases in the latent period, and that hence that value may be seriously low. The BEIR Committee provides some guidance as to how high the value might go. All Gofman

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Values with a star (*) refer to age at time of bombing.

did was to read what the BEIR Committee said about its own values. The ERDA lab critiques would suggest that reading is out of style.

There is no rational way that the criticism that Gofman used too high a value for relative risk per rad will stand up. If we add to this the fact that the most recent extensive Japanese data suggest Gofman may have underestimated the value for relative risk per rad, it would appear that this ERDA Lab criticism falls of its own weight unequivocally.

Lastly on the issue of estimation of risk per rad, some of the ERDA lab critiques have exhumed the Neanderthal argument that the linear hypothesis should not have been used by Gofman in the Gofman-Pu papers. There is every reason to have used the linear hypothesis in estimating the lung cancer hazard of plutonium. First of all, it represents public health responsibility, instead of irresponsibility as has been pointed out by virtually every serious group concerned with radiation protection. That the National Council on Radiation Protection and Measurements might not concur is hardly surprising for this is the same nuclear industry hack organization that in 1954 stated that 36000 millirems would be without physical effect on man. The Environmental Protection Agency , in its 1976 rule-making on radiation standards , has just rejected the NCRP suggestions. But public health responsibility is only one reason why the linear hypothesis should be used . Let us consider the evidence abundantly available to us.

1. Referring to the Spiess- Mays studies(Spiess,H, Mays, C.W., "Bone Cancers Induced by ^{224}Ra(ThX) in Children and Adults", Health Physics, Vol. 19, Dec. 1970, pp 713-729) the BEIR Committee stated the following;

> " The data for the ^{224}Ra-injected patients are consistent with the linear nonthreshold dose-response curve within the limits of the dose range available and when the dose is expressed as mean dose to bone."

It is worth noting that these studies are for high LET alpha radiation in humans, the same type of radiation as for plutonium.

2. The data on Ra^{226} induction of bone cancer in man have been re-analyzed by several workers after Evans claimed that those data were inconsistent with the linear hypothesis. One such refutation is by Gofman,J.W. and Tamplin, A.R. " The Question of Safe Radiation

Thresholds for Alpha Emitting Bone Seekers in Man ", Health Physics, Vol. 21, July, 1971, pp 47-51. Again, these are data for high LET alpha radiation in humans and they are consistent with the linear non-threshold hypothesis.

3. In an analysis of the induction of lung cancer in the uranium miners, Gofman and Tamplin showed that the doubling dose in working-level-months for radon-daughter induction of lung cancer showed no evidence of increasing at the low dose ranges, a fact supportive of the linear, non-threshold hypothesis. Indeed, in that analysis what variation in doubling dose occurred suggested, if anything, that the radiation effect might be even higher than predicted by linear thesis for the low-dose region. (Gofman, J.W. and Tamplin,A.R. , "The Colorado Plateau: Joachimstahl Revisited : An Analysis of the Lung Cancer Problem in Uranium and Hardrock Miners" Testimony presented at the Hearing of the Joint Committee on Atomic Energy, 91st Congress of the United States, Jan. 28, 1970, and GT-106-70.)

4. E.E. Pochin, in a very recent paper (Pochin, E.E, "Radiology Now: Malignancies Following Low Radiation Exposures in Man" British Journal of Radiology, Vol.49, No. 583, 577-579, July 1976) has commented that for the low LET radiation (X-rays) exposure of infants-in-utero, the risk of malignancy is linearly related to dose. The direct quote of Pochin follows;

> " The inference that the radiation caused malignancies was, however, questioned at first. For example, a sub-group of mothers might have been more frequently X-rayed than mothers in the control group because of hereditary abnormalities, e.g. of the pelvis, and this sub-group might have a preponderance of children who developed malignant disease as a result of some associated congenital abnormality.
>
> Such a source of bias now appears to be excluded, however, by two considerations. Firstly, Stewart and Kneale(1970) showed that the likelihood of subsequent malignancy per examination increased about linearly with the number of films used in the examination, where this number was known. ....."

Linearity , for these studies, covers the range of about 0 to a few rads, and for low LET radiation at that.

It appears that the public health problem of radiation protection will not indefinitely be burdened by the old crowd of AEC diehards, now wearing ERDA gowns. There are, after all, retirement rules and time has a way of marching on.

Conclusions: The critiques of the Gofman-Pu papers have been considered here in detail, with respect to the points deserving serious comment and those critiques are rejected in toto. Some very trivial criticisms from the five ERDA Lab critiques will be dealt with in the Appendix to this testimony. The only reason to deal with them at all is to re-assure the reader that major gems do not exist there which cannot be answered.

I therefore suggest that the NRC in its GESMO considerations accept the estimates of plutonium lung cancer hazard precisely as they appear in the three Gofman papers which are part of this testimony, CNR-1, CNR-2, and JAMA. It is my considered opinion the GESMO must deal with the consequences of those estimates if the NRC chooses to be responsible in its mission.

Future evidence may show that the Gofman estimates are correct as they stand, are too high, or are too low, as Gofman has clearly and explicitly stated in those reports. And if and when such evidence(not wishful thinking) appears, the estimates can be appropriately altered, if necessary. There does exist one possible source, aside from the issue of $T_{\frac{1}{2}}$ of clearance,that could force some downward revision of the lung cancer hazard of plutonium from that estimated by Gofman. If some of the energy of alpha particle decay is expended in cells other than those at risk for carcinogenesis or is expended in mucus , the true dose to the cells at risk would have been overestimated. But we simply do not know the physical distribution of the plutonium that is retained long-term, so there is no way now to estimate whether a correction is needed or how large it might be. A correction of the order of two-fold is by no means inconceivable. On the other hand there is also the possibility that the RBE factor should have been chosen as 20 instead of 10, which would introduce a factor of two in the opposite direction. Probably other minor factors will occur to alter the estimates. This is why Gofman suggested that the final result might be a factor of two or three too high or too low (JAMA, p285). The key point is to realize that any correction required could go either way, and hence from the public health point of view there is no basis for optimism that the result predicts too high a carcinogenicity. Until such time that evidence dictates a change in estimate, any action based upon hopes the the risk estimates are

too conservative is clearly irresponsible public health practice. GESMO is necessarily a public health document in addition to its functions in describing other aspects of the use of mixed oxide fuel.

In the immediately preceding discussion , possible sources of correction of the Gofman estimates of the lung cancer hazard of inhaled plutonium by a factor of 2 or 3 one way or the other are considered. However any such possible corrections turn out will not alter the fact of the enormously greater carcinogenicity of plutonium as demonstrated by the Gofman analysis versus prior analyses of this problem. The plutonium carcinogenesis as estimated by Gofman is of the order of 1000 times greater than prior estimates, (or of some current "we hope and pray" estimates) So corrections of a factor of 2 or 3 will not alter the major revisions in all its thinking that NRC will have to do to bring GESMO anywhere near the realm of the real world .

APPENDIX

Responses by J.W. Gofman to the Minor Issues Raised in the ERDA Lab Critiques

1. Document: "Review of John W. Gofman's Reports on Health Hazards from Inhaled Plutonium" Chester R. Richmond, ORNL/TM- 5257, February 1976.

Point No. 1 Richmond states, p.2, " It would appear that Gofman is completely dismissing the hot particle arguments, yet it is not clear until one reads the paper that he leans heavily as he derives his risk estimates upon the argument of a large reduction in the mass of the presumed critical target tissues within the lung."

Answer Gofman had no need whatever to address the hot particle arguments in this work, so he had no occasion either to accept or dismiss hot particle arguments. A bit of reading would help Dr. Richmond on this mis-statement of his. Dr. Richmond also complains that one must read Gofman's papers in order for it to become clear what Gofman has done. I hardly think it is unusual to have to read a paper to understand it.

Dr. Richmond is indeed correct that Gofman leaned heavily upon the argument of a large reduction in mass of the presumed target tissues within the lung. In fact a reduction factor of 570, thoroughly justified by the fact that it is the bronchial cells one must consider if bronchogenic carcinoma is at issue! I doubt very seriously that Dr. Richmond can find any scientific support for inclusion of irradiation of blood vessels, nerves, alveolar tissue, terminal bronchiolar tissue , or other tissue in determination of the radiation effect upon bronchial cells in the production of bronchogenic cancer. I believe Dr. Richmond would make as much sense to include those extraneous tissues in the critical mass as to include the mass of the ass. There are some rare instances in carcinogenesis where indirect effects of radiation can have effects upon carcinogenesis.Thus radiation injury to a hormone-producing target may increase tropic hormone output of the pituitary and thus have influence. I doubt very much that we are dealing with anything like this in eliminating the extraneous lung tissues for the critical target assessment here. Second there is the relation of immune suppression that may operate to accelerate carcinogenesis. It is

inordinately doubtful that elimination of extraneous lung tissue from the mass of critical bronchial cells could possibly lead to overlooking a significant immune response in the carcinogenesis process in the bronchial cells.

Point No.2 Dr. Richmond states that if Gofman's $T_{\frac{1}{2}}$ of 500 days in the damaged bronchial areas were true,the"lungs of many heavy smokers would obviously become rapidly filling reservoirs for all sorts of atmospheric contaminants and particulates and perhaps, more important, if large regions (whatever anatomical reference this might have) were severely damaged by loss of cilia resulting in extremely long clearance half-times, the affected individuals would most probably drown in their own fluids." (Direct quote of Richmond)

Answer: Dr. Richmond has simply missed the entire point about what is being discussed. The subject, Dr. Richmond, is highly insoluble particulates of PuO_2 and possibly other highly insoluble particulates. So Dr. Richmond's concern about fluids is simply absurd. Equally foolish is concern about "all sorts of atmospheric contaminants", a large proportion of which are soluble and hence not even at issue. Moreover, it is still a highly open question as to why PuO_2 particulates are retained so tenaciously when they are. The alpha activity has not been ruled out as a factor. The density of the particulates, very high for PuO_2 compared with most atmospheric insoluble particles is undoubtedly important. On a quantitative basis Dr. Richmond would be extremely hard-pressed to show that the insoluble, high-density, alpha-particle emitting particulates of atmospheric contaminants would, according to the Gofman thesis, lead to any blockage of the airway, to say nothing of such nonsense as "drowning in their fluids." Lastly I would advise Dr. Richmond to have a hard look at the Radford-Martell data discussed in the body of this testimony, which data suggest order-of-magnitude(at least) agreement with Gofman's $T_{\frac{1}{2}}$ values for the highly insoluble Lead-210-containing particulates from mainstream smoke of cigarettes.

Point 2a: Here I shall take up an expansion of Point 2 concerning possible airway blockage since in one form or another it occurs in all five of the ERDA Lab critiques. The Los Alamos critique comments on the amount of dust that might accumulate although there is not a claim of airway blockage. The other four critiques claim , outrightly,

that airway blockage would occur if Gofman's $T_{\frac{1}{2}}$ of 500 days for highly insoluble PuO_2 particles were correct for injured regions of bronchial epithelium. This assertion is simply absurd,as will now be demonstrated in extenso.

In the Los Alamos critique, Healy et al state the following;

"Secondly, using his values for the quantity in the bronchial area (2.7%) retained with a long half-life (500 days) a mechanism of entry into the tissue would predict that, for a normal dust concentration of 100$\mu g/m^3$, the one gram of bronchial epithelium would accumulate at equilibrium some 39 mg of dust."

Let us accept the correctness of the Healy et al calculation that the accumulation would be 39 milligrams. Healy et al then go on to state the following;

"Of course, in industrialized communities, the actual concentration in the air may be several times the 100 $\mu g/m^3$ assumed above."

Let us grossly exaggerate the problem by not only crediting the air concentration as "several times the 100 $\mu g/m^3$", but by assuming an air concentration of <u>one hundred times</u> the 100 ug/m^3, a rash assumption indeed. We would,therefore, have to multiply Healy's value of 39 mg of dust by 100, giving an accumulation of <u>3.9 grams of dust</u>.

I'll accept, for rashness' sake, that Healy's calculation that all atmospheric dust will behave like insoluble PuO_2 aerosol is correct (rash, indeed, but let us assume it nevertheless). Our objective now is to estimate <u>how much airway reduction occurs</u> if the accumulation in the crucial bronchial areas is 2.7% as assumed by Gofman.

In CNR-1R, p.16, Gofman presented a value of 0.115 cm. as the radius of the relevant intrapulmonary bronchi. On p.17 of that same reference he calculated the surface area of such bronchi to total up to 361 cm^2.

The cross-sectional area of the lumen of such bronchi is ~~π~~ r^2, which for a radius of 0.115 cm is 3.1416 x $(0.115)^2$, or 0.0415 cm^2. Now let us calculate how much cross-sectional area is sacrificed to accomodate 3.9 grams of dust. Let us start by assuming a uniform distribution of the dust over the surface area of all the relevant bronchi, which is 361 cm^2. Let h = the height of the layer of dust. For mass equality, we have the following relation:

(Height) x (Area) x (Density) = Mass of dust.

As a reasonable value for the dust we shall use a density of 2 grams/ cm^3 (noting that PuO_2 is much more dense than this). Substituting,

$$(h) \times 361 \times 2 = 3.9$$

$$h = 3.9/722 = 0.0054 \text{ cm.}$$

This value of h is, to a close approximation, equal to the radius reduction for these bronchi after dust accumulation. The new radius (post-dust collection) is 0.115-0.0054 = 0.1096 cm. The cross-sectional area of the reduced lumen is πr^2, or $3.1416 \times (0.1096)^2$, or 0.0377 cm^2. This means that the reduced lumen area is (0.0377/0.0415) times 100, or 90.8 % of the original lumen area. Expressed otherwise the loss in lumen area is only 9.2 %. This is so little reduction, even for the extremely unrealistic, rash assumptions used, that it is absurd to speak of airway blockage. The variations in lumen diameter from smooth muscle contractions in the bronchial wall will be far greater. Now the issue can be raised that the dust will only accumulate over the injured bronchial regions, so that the layer would be raised there. That is true, but irrelevant. Even if the "dust" is irregularly distributed, tne approximate cross-sectional area lost is still the same.

The final conclusion that must be drawn is that the concerns of all the five ERDA Lab critiques concerning airway blockage is ridiculous indeed, since even making rashly worse assumptions than they did, the blockage is insignificant. Snipes et al, in the Lovelace Foundation critique , made a calculation that if all the smoke inhaled with the smoking of 20 cigarettes per day were to behave like insoluble PuO_2 aerosols (an assumption that is not in the rash class, but rather in the wild blue yonder) then there would be about 6.4 grams accumulated in the relevant bronchi. This is followed by the completely guessstimated opinion that " Thus , ventilation would cease or be seriously impaired in heavy smokers." Had Snipes et al taken the trouble to calculate the airway reduction by the methods above , he would have found that the radius reduction is (6.4/3.9) x 0.0054, or 0.0089 cm, the reduced lumen area is 0.0354 cm^2, and the final area remaining is 85.2% of the original area (0.0354/0.0415) x 100), and this means a loss of 14.8 % of the airway cross-section----far, far from"ventilation would cease or be seriously impaired". And all this assumes some reasonableness for the assumption that all cigarette smoke behaves like insoluble PuO_2 aerosol.

Point 3: Richmond goes through a long discussion of Gofman's demonstration that Hempelmann overestimated the body burden of the Manhattan Project workers by a factor of 14. I thought he was going to show that it was an incorrect criticism; instead he finally ended up admitting that many sources of new data would suggest that Hempelmann was wrong by a factor of ten in using urinary excretion data to estimate body burden. A factor of ten from experimental data is an excellent confirmation of Gofman's factor of 14. And as for the use Gofman made of the factor of 14, the same general conclusion would have been arrived at with a factor of 10. So this issue needs no further elaboration.

Point 4: In a wild, blatantly false statement on p. 12, Richmond states the following;

" The summary and conclusion section of Gofman's paper (CNR-1) states that there are 7.83×10^9 " lung cancer doses" per pound of plutonium . He neglects to point out in this section , however, that this estimate-- if true -- is per pound of plutonium deposited in the lung".

To show how ridiculously false Richmond's assertion is, I shall quote the summary and conclusions section on this subject from CNR-1 of Gofman:

" For cigarette-smokers:

Pu^{239}

(a) 0.058 micrograms deposited Pu^{239} represents one "lung cancer dose".
(b) 7,830,000,000 "lung cancer doses" per pound of Pu^{239}. "

It is hard to see how Gofman could have more explicitly indicated he was referring to DEPOSITED plutonium .He defined the " lung cancer dose" in terms of deposited plutonium , and that definition is used to give "lung cancer doses" per pound. What could be more straightforward? Perhaps an opthalmological examination is in order.

The follow-on to this nonsense is even worse in Richmond's critique. He says that the unsuspecting reader might incorrectly calculate 10^{13} to 10^{14} lung cancer deaths for the five to seven tons of plutonium produced as weapons fallout. On the very next page of Richmond's own report he then quotes Gofman's own calculation of 1,000,000 deaths. Any reader who calculates 10^{13} deaths, with a number of 10^6 deaths explicitly stated , would simply be totally incompetent.

Summary concerning Richmond's critique; There is absolutely nothing

that can be taken seriously in the Richmond critique. It should be totally and unequivocally rejected in its entirety.

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2. Document: "Comments Prepared by Dr. D. Grahn , Division of Biological and Medical Research, Argonne National Laboratory , Argonne, Illinois 60439, October 8, 1975.

Point 1: Grahn states the following on page 1 of his critique;

"Gofman then rederives risk in terms of " lung cancers per pound of plutonium " . This seems algebraically acceptable and technically it is a kind of extension of the "man-rem" concept. However, scientifically it is inappropriate because it does not allow for the all-important factors of atmospheric dispersion and dilution. "

Answer: This is sheer nonsense. Gofman derived all estimates of risk in terms of deposited plutonium, clearly , explicitly , and unequivocally. In no way can this be mis-interpreted as risk related to released plutonium. It should be totally obvious to Grahn that the factors of dispersion and dilution were clearly taken into account in all uses made by Gofman of the risk of lung cancer. All that can be said further to Dr. Grahn is ,that when all else fails when one is criticizing a manuscript ,it pays to read the manuscript.

Point 2: Grahn states the following on page 2 of his critique;

" A rather serious misconception is entrained in Gofman's discussion of the residence time of particulate matter on the bronchiolar epithelium in regions where cilia are absent. The assumption that epithelial residence half-time would be 500 days in the bronchioles , as employed for the alveolar regions , is not supported by evidence."

Answer: Gofman made abundantly clear that, for plutonium aerosols in the bronchi of cigarette smokers, there simply existed no evidence, and that, as a result, reasonable assumptions were being made. Indeed, since that time, the Radford-Martell evidence did appear concerning long residence half-times (see main body of this testimony) for insoluble particles in the bronchi of cigarette smokers. Of one thing we can be certain--- Dr. Grahn certainly presented no evidence to contradict the Gofman $T_{\frac{1}{2}}$ estimates. As he goes on in his discussion he cites the work of Albert et al in donkeys.

Several of the critiques cite the work of Albert, either for donkeys or humans, as evidence showing that a long clearance half-time cannot obtain for PuO_2 aerosols in the injured bronchial epithelium. The Los Alamos critique presents a detailed listing of the papers by Albert and his colleagues. It can be stated flatly in response to Grahn and all the others who cite Albert et al on this subject that the Albert work simply cannot address the question of a few percent long-term retention of insoluble aerosols in the bronchi. The very definition of bronchial clearance by Albert by his methodology would exclude long-term retention from the bronchi. It is to the credit of the Los Alamos group's critique that they recognized clearly that the Albert work cannot bear upon the Gofman thesis at all. They stated on page 7 of their critique the following:

" However, on careful examination these experiments would not detect the increased retention in the bronchial region postulated by Gofman since this fraction would be considered as pulmonary deposition and the normal fluctuation among individuals is too great to detect the Gofman assumption of 2.7% deposited in the bronchiolar region."

To this, I can only say "Amen, it is nice to see that somebody understands the literature." Richmond, Bair, Grahn, and Snipes, representing 4/5 of the ERDA Lab critiques , all grossly misuse the Albert data in their efforts to refute the Gofman thesis. It is regrettable that they indicated such a total lack of understanding of the problem at hand. Essentially the same can be said for the misuse by some of the critiques of related data of Lourenco et al.

Summary Concerning Dr. Grahn's Criticisms:

Aside from the items mentioned above,Dr. Grahn's comments and erroneous calculations are amply covered in the issues discussed in the main body of this testimony. I conclude that none of Dr. Grahn's points are valid in any way. I reject each and every one of his criticisms as erroneous.

Document 3: Bair, W.J. " Review of Reports by J.W. Gofman on Inhaled Plutonium" BNWL-2067/UC -41 , October 10,1975, Battelle Northwest Laboratories.

The major points of Bair's critique are thoroughly refuted in the main body of this testimony . Some of the minor points are those where Bair erred as did other ERDA laboratories,and they are covered with mention of Bair in the discussion of the specific points for the other ERDA critiques.

Point 1: Bair makes the point that it is possible that some of the plutonium alpha particle energy is lost in mucus or in metaplastic cells other than those which are subject to carcinogenesis.
Answer: As conceded in the main body of this testimony, Gofman agrees with Bair that it is entirely possible that some correction may have to be made for such effects. But neither Bair nor anyone else has presented any evidence that such a correction is of consequence or even an order of magnitude of possible size. Only some real data can help us here.

Point 2: Bair states(on page 9 of his critique)the following:

" If Gofman's premise that clearance of plutonium from lungs of smokers is greatly impaired is true , human autopsy data should be showing much higher lung burdens of plutonium in smokers than in non-smokers . This has not been revealed in the results published to date."

Answer: Patent nonsense! It would be a revelation indeed if Dr. Bair could point out a single published study of human autopsy material that has been studied in a manner to test whether insoluble PuO_2 aerosols are retained in cigarette smokers to the extent of 2.7% versus 0.2 % in non-smokers. This would have required meticulous analysis of relevant bronchi versus lung parenchyma. While such data would help resolve the problem, Dr. Bair seems unable to point such data out to the world. Moreover, the analysis of autopsy material obtained a long time after the exposure(more than a couple of clearance half-times) would be unsatisfactory even if a careful dissection of bronchial epithelium were made to compare with lung parenchyma.

Summary Concerning Dr. Bair's Criticisms:

The major points of Bair's criticisms are adequately dismissed as erroneous in the main body of this testimony since they deal with such issues as the $T_{\frac{1}{2}}$ of 500 days, the use of the linear hypothesis, and the BEIR Report estimate of the relative risk per rad --- all of which is disposed of there. The one point concerning a possible correction of alpha energy absorbed in mucus or non-relevant cells is a valid point and has been discussed above. There is nothing else in the Bair critique that is valid, and hence aside from this one point, all else in the Bair critique is rejected in toto.

Document 4: Snipes, M.B., Brooks, A.L., Cuddihy, R.G., and McClellan, R. O. "Review of John Gofman's Papers on Lung Cancer Hazard From Inhaled Plutonium" , L F-51/ UC-48 , Lovelace Foundation for Medical Education and Research, P.O.Box 5890, Albuquerque, NM 87115.

The major points of this critique have already been dismissed either in the main body of this testimony or in the discussion of other critiques which share the erroneous analysis of this critique. There is one point worth mentioning , ridiculous as it is. Incredibly, the Snipes critique states the following;

" Gofman states that his approach does not involve the "hot particle" hypothesis which precipitated considerable controversy in 1974. However, his approach to this problem is in fact an extension of the hot particle hypothesis."

Answer: "Incredible" is far too weak a word to describe this absurd assertion by the Lovelace workers. There are only two possibilities . First, the Lovelace workers don't have the foggiest notion of what the "hot particle" hypothesis is all about. Second, they don't understand anything they ostensibly read in the Gofman-Pu papers. On second thought, a third possibility must be entertained, namely, the Lovelace workers understand neither the "hot particle" hypothesis nor what they ostensibly read in the Gofman-Pu papers. (See footnote, p.26)

Summary Concerning the Lovelace Foundation Critique: It is impossible to find a single item in this critique that has any validity at all. Everything in this critique is therefore rejected unequivocally and totally.

Document 5 : Healy, J.W., Anderson, E.C. , McInroy, J.F., Thomas, R.G., and Thomas, R.L. " A Brief Review of the Plutonium Lung Cancer Estimates by John W. Gofman" , LA-UR-75-1779, Los Alamos Scientific Laboratory, Los Alamos, New Mexico , October 8, 1975.

There are a few minor points to address specifically in the Los Alamos critique. A general commentary follows in the Summary concerning this critique.

Point 1: The Los Alamos workers raise the question as to whether the Auerbach data can be directly described in terms of percent of cilia lost in cigarette smokers. The Auerbach data give a composite measure, namely percent of slides showing two features , loss of cilia plus an average of 4 or more cell rows in epithelial depth. According to this criterion of scoring slides, a slide with total absence of cilia but with fewer than four cell rows in depth would be scored as negative. Inasmuch as cilia loss is , in all likelihood , a lesser degree of damage than metaplasia or hyperplasia, cilia loss might be expected to be even more frequent a finding than is indicated in the Auerbach tables. Because of this uncertainty in the appropriate use of the Auerbach data, Gofman sought data that are unequivocal on the issue of per cent of cilia lost in smokers. Such data are published by Ide and co-workers, and are referenced in JAMA by Gofman. The Ide publication shows 30% ciliary loss in cigarette smokers, whereas Gofman used a value of 25 % as his interpretation of the Auerbach tables. Thus it turns out that any revision of the estimation of the lung cancer risk in smokers , using the Ide data,would make the plutonium risk somewhat worse than estimated by Gofman.

Point 2: The Los Alamos workers point out that some data indicate very long -term retention of part of the plutonium deposited in the lung--- much longer than would be suggested by a $T_{\frac{1}{2}}$ value of 500 days for clearance . They suggest, therefore, that Gofman's criticism of the Hempelmann estimates of body burdens in the Manhattan Project Workers may be somewhat too severe. Unfortunately , this finding of some very long-retained burden in the lung does not allow for such a simple interpretation. First of all , there are small lymph nodes in the lung, other than the large masses of nodes in the tracheobronchial region. It is entirely possible that some, or even most, of the residual burden is in lymphatic tissue and not in true lung

tissue. Second, even if the residual burden is in true lung tissue, there would be a world of difference if the burden is in the bronchiolo-alveolar region versus being in the bronchial tissue(that is , the bronchial tissue relevant for bronchogenic cancer.) If the burden is in the bronchiolo-alveolar tissue, the concentration of plutonium would be estimated to be 570 times lower there than in the bronchial tissue , with its corresponding marked reduction in radiation dose due to the factor of mass of tissue. Additionally , the intrinsic risk of bronchiolo-alveolar cancer, "spontaneously " is very much lower than that for bronchogenic cancer. Bair , in his critique, references DeLaRue et al,(Cancer, 29, 90-97-1972) as giving a value for bronchiolo-alveolar cancer representing only 3-6% of the total number of lung cancers both in smokers and non-smokers. Thus , if any especially long-term retention does exist , it is of the utmost importance to know precisely where in the lung it is. If it is in the parenchyma, the estimated risk by Gofman would hardly be much increased, contrasted with an estimated risk elevation that would be required if the long-term retained material is in the bronchi.

Summary Concerning the Los Alamos Lab Critique: There are a number of minor points that could be addressed additionally concerning the Los Alamos critique. However, the following quotation from the Los Alamos paper indicates it is not even worth going into these minor points in detail. The final section of the Los Alamos paper has the quotation on pages 17-18. I quote,

"We would recommend that measurements continue with more emphasis on the absolute bronchial retention, and that until such evidence is available, the Gofman predictions be regarded as interesting and imaginative speculations which should serve to stimulate increased interest in certain phases of current studies. "

Let us translate what the Los Alamos group is saying to ERDA. They are saying, in plain English, we don't have the ability to say whether Gofman's estimates are correct or not correct, and we aren't going to know until there are more data.

The message should be pretty clear for GESMO. The NRC draft of GESMO makes all kinds of comments about the radiological hazards of mixed oxide fuel use utilizing a hazard estimate for plutonium induction of lung cancer that may 1000-fold or so too low. In view of the Los Alamos admission on this point, there is no conclusion to

be drawn other than that which follows. The NRC draft of GESMO is simply incompetent on the issue of plutonium toxicity . If the NRC persists in using such an incompetent estimate of the lung cancer hazard of plutonium,plus fairy-tale dreams of what the release fraction of plutonium will be at reprocessing and fuel fabrication facilities, then the NRC will be declaring it has no respect whatever for the health and safety of the public.

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Footnote: In several of the ERDA Lab critiques ,reference has been made to the "hot particle" hypothesis of Geesaman-Tamplin-Cochran. Indeed some of the ERDA Lab critiques suggest that Gofman's thesis makes use of, or is a variant of,the "hot particle" thesis. Such an assertion is blatant nonsense of the very worst kind. Those who make such an assertion show a total lack of understanding of the "hot particle" thesis and the Gofman hypothesis.

The ERDA Lab critiques which infer that Gofman made use of the "hot particle" hypothesis do so because Gofman calculated the mass of the relevant bronchial tissue (for carcinogenesis)to be one gram rather than the 570 grams of the whole(bloodless) lung. In using one gram Gofman is stating a most simple scientific fact, namely, if the bronchial cells represent the target for carcinogenesis, one calculates the dose to that target , not to miscellaneous and sundry assorted garbage. It is pathetic to find this totally straightforward scientific operation misconstrued as use of the "hot particle" thesis to which it bears exactly zero relationship.

The "hot particle" thesis predicts that as the dose to a so-called "critical architectural unit" increases, the effectiveness of radiation increases far more than one would anticipate from linearity because a new mechanism of carcinogenesis has entered the picture. In striking contrast,the Gofman approach says the situation is precisely what linear theory would predict, namely if the mass of target is reduced for a given radiation source, the increase in carcinogenic effect of radiation is in the same proportion as is the decrease in mass of the target tissue. This is precise use of the linear hypothesis, and is in no way related to the "hot particle" hypothesis.

In the Los Alamos critique it is stated that,since only 25% of the one gram of bronchial epithelium is injured, and hence the plutonium concentrates its radiation there, this constitutes use of the "hot particle" thesis. Sheer, unadulterated nonsense! If the plutonium concentrates in ¼ of the bronchial epithelium and delivers its dose there, the carcinogenic effect is 4 times as high there as it would be for the plutonium distributed over the whole gram of bronchial tissue. Thus, in terms of effect, ¼x4 = 1, and 1x1 =1. This shows that the plutonium concentrating in ¼ of the cells gives exactly as much carcinogenic effect as that same amount of plutonium distributed in all the cells. This is exactly what linear theory predicts and it is linear theory that Gofman uses. None of these considerations even remotely resemble any aspect of the "hot particle" thesis.