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COMMENTS IN RESPONSE TO MITCHELL'S LETTER AND MARTELL'S APPENDIX

Enclosed are the comments I have prepared in response to Mitchell's letter and Martell's appendix. These were assembled from your input, as well as from other sources.

In reply to Geesaman's appendix I plan to use our response from the LMFBR PFEIS; similarly I plan to include WASH-1320, LA-5810 and the British MRC report in response to the NRDC comments.

Since DNA needs this material by Wednesday, April 2, and since it must be revised according to your comments, would it be possible for you to review this immediately and return your comments by COB Tuesday, April 1? My apologies for the short time frame, but my lead time, especially with lab replies, wasn't much longer.

Please review for accuracy, completeness, responsiveness, etc. If you feel the response is unnecessarily thorough, please discuss.

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Enclosure:
As stated

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Page 1, lines 12-23 - "It is difficult enough for the layman to comprehend what the experts in the various radiological science fields are saying about the effects of radioactivity, but that difficulty is compounded many times over the differences of opinion found among the experts, by the realization that even the experts agree that the long term effects of some of the more dangerous radionuclides are not known by anyone at this time and may not become known for many years to come, and it is unsettling to learn that the standards used for the kinds and amounts of radionuclides to be tolerated in the environment and in man are criticized by reputable experts as unreliable and inadequately conservative."

Comments: This sentence emphasizes the "difference of opinion found among the experts" regarding the "effects of radioactivity" and the adequacy of existing standards. It must be pointed out that the "reputable experts" to whom this author refers (i.e., Martell, Geesaman, Tamplin and Cochran) number four, and that, while they have expressed strong opinions regarding plutonium, they are not recognized as experts in plutonium by the national and international scientific community. Assessment of the hazards of radioactivity and the recommendation of standards are methodically, continually and properly reviewed by those persons most knowledgeable and experienced in evaluating the available relevant data. This is true both for national bodies such as the National Council on Radiation Protection and Measurements (NCRP) and the National Academy of Sciences (NAS), and for international bodies such as the International Commission on Radiological Protection (ICRP) and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). If newly published data dictates a change in the established standards, it is usually evaluated through the normal scientific process (e.g., publication) and is considered by one or more of the above bodies. None of the "experts" referred to have been or are members of any of these bodies, nor have their theories been presented through the normal

channels of scientific communication. In contrast, those individuals in this country and abroad who are recognized as experts because of their published research on plutonium and their analyses of the hazards of plutonium have regularly been members of or advisors to such bodies. These bodies have consistently adopted a conservative philosophy regarding recommendations; this is evidenced by the fact that no member of the public has been harmed by radioactivity released from or caused by non-military and non-medical activities, and that the radiological safety record of the nuclear industry over the past 30 years is an enviable one. Reputable scientists consistently have taken a conservative, scientific and unemotional approach to matters of radiation protection. Since these recommendations and resulting standards are inherently conservative, the term "inadequately conservative" becomes a relative one which essentially questions "How conservative is conservative?". One can be conservative to the point where standards are meaningless.

Page 2, lines 12-15 - "And it is in the assessment and, if possible, elimination of the radiobiological health risk that they are the most dependent upon the United States government."

Comments: The total elimination of any radiological health risk at Enewetak is an unrealistic objective which can never be attained. Some radioactivity and therefore some finite risk must be accepted if the people are to return under any of the clean-up alternatives.

Page 2, lines 15-19 - "The Defense Nuclear Agency and the Atomic Energy Commission have already devoted great amounts of time and money to assessment and remedy of radiological problems presented by this program, but more will have to be done and it will have to be done over a long period of time."

Comments: The author is aware that the Energy Research and Development Administration (ERDA) anticipates continued radiological monitoring of both the Enewetak environment and the people; these programs will be funded

in excess of $2 million during fiscal year 1976.

Page 3, lines 11-14 - "It is an absolute kind of responsibility to both return the people to their home and eliminate the likelihood of so much as a single radiation induced illness or anomaly."

Comments: This objective, while desirable, can never be achieved. The calculated dose levels following clean-up, however, are such that no radiation induced illness or anomaly is expected. As the author indicated, the effects of extremely low level radiation exposures over a long period of time are not known, nor will they be known with any degree of accuracy for a number of years. Decisions must be based upon the pertinent data available and the recommendations of those best qualified to evaluate the data; this approach has been necessary ever since the discovery of radiation. If the return of the people is deferred until the data base is complete, it may be some time before relocation can be effected. While no absolute assurance can be given, the predicted exposure levels are comparable to those in which populations have lived for centuries.

Page 6, line 14 to page 7, line 11 - "The survey of radiological conditions at Enewetak Atoll in 1972 under the auspices of the Atomic Energy Commission is, we believe exceptionally good as far as it goes, but we have been advised by capable experts in the field that more work remains to be done and that the qualifications of the four-member Task Group which supervised the conduct of the survey, the assessment of its data and developed final recommendations are open to question. It is also apparent that as detailed and elaborate as that survey was, follow-up gathering of data and careful assessment of that data is absolutely essential, particularly with respect to the risk to health from all low-level, long-life radionuclides and especially the danged posed by those alpha-emitting radionuclides known as hot particles, such as Plutonium-239 and Americium-241.

"We do not wish to detract from the qualification of the members of the Task Group, but in a field involving so many specialties and where equally expert opinions differ markedly, it is imperative that the Task Group for follow-up studies be enlarged to include scientists known to take the most conservative approach to radiation protection, such as Drs. E. A. Martell at the National Center for Atmospheric Research, Arthur R. Tamplin at Lawrence Livermore Laboratory, and Donald P. Geesaman, at the University of Minnesota. Their presence in the Task Group, or their participation in some other direct way in designing methods to

be used for the gathering of information and its evaluation is strongly recommended."

Comments: The author is confused regarding boththe composition of the Task Group, its resources and its responsibilities. The "AEC Task Group on Recommendations for Clean-up and Rehabilitation of Enewetak Atoll" was established by the AEC General Manager and requested to prepare recommended criteria and guidance for clean-up and rehabilitation of Enewetak Atoll for consideration by the Commission. The thirteen-man Task Group consisted of four units: Drafting Group (4), Headquarters Liaison (2), Interagency Liaison (3), and Advisors (4). Comments and recommendations on draft reports were obtained from the Environmental Protection Agency (EPA), Health, Education and Welfare (HEW), and many of the National Laboratories and University contractors. Thus, the ERDA recommendations rest not only on the qualifications of the four-man Drafting Group, but on a broader base of expertise available to ERDA from numerous sources. In addition, the "four-member Task Group" did not supervise the conduct of the environmental survey at Enewetak Atoll. While the Task Group made recommendations regarding clean-up and rehabilitation of the Atoll and for later follow-up studies and surveys that were approved by the Commission, the Task Group's responsibilities ended with the approval of their recommendations. Therefore, it is irrelevant to consider enlarging the Task Group. In addition, any new task group would have no additional results to evaluate at this time, or for perhaps years to come. It should also be noted that the recommendations of the Task Group are lower, and therefore more conservative, than the exposure limits recommended by the national and international bodies.

Whether or not the author's advisors are "capable experts in the field," "equally expert," etc. is the author's opinion. Thus far they have not

received support from the scientific community at large. Furthermore, the participation of those with extremely conservative philosophies may lead to decisions based on no effective standards at all, a situation which might result in demands in excess of current technology and potentially available funds, thereby preventing return of the people. Resolution of scientific issues should be conducted through normal scientific channels rather than on a day-to-day basis at Enewetak.

Page 7, lines 12-16 - "The 1972 radiological survey (NVO-140) must be regarded as an impressive beginning of long-range radiological assessment and monitoring of the Enewetak environment with appropriate emphasis placed upon not only the marine and terrestrial environments but upon the radionuclide pathways to man."

Comments: NVO-140 is the most comprehensive radiological assessment and monitoring ever carried out on a marine and terrestrial environment. The results of the Enewetak Radiological Survey, except for minor areas identified in the ERDA Task Group Report (sampling of water lenses and air over longer periods of time), were found to be an exceptionally complete data base for the purpose of evaluating conditions and for making decisions on clean-up and rehabilitation. Twelve months of lens water and air sampling will be conducted by the radiological support group within the clean-up organization during clean-up operations. No additional radiological survey is warranted or planned prior to the start of clean-up operations.

It should also be noted that the Survey not only considered but focused on radionuclide pathways to man. Various diets and quantities of ingested foods were considered in deriving potential radiation exposure via these routes.

Page 6, lines 16-18 - "As we shall discuss more fully below, more information is needed about the presence of hot particles."

Comments: We agree that more information is needed about the potential presence of hot particles. However, it will be some time before all of the

issues are resolved, and today's decisions must be based upon today's knowledge, as was discussed above. Most experts in the field feel that the hot particle problem has been grossly exaggerated, as is evidenced by the British Medical Research Council report.

Page 7, lines 18-20 - "The long range effects of Strontium-90 and Cesium-137 and other nuclides in the food web cannot be known without experimental planting."

Comments: We agree that the significance of strontium-90, cesium-137 and other nuclides in the food web cannot be known without experimental planting. For that reason ERDA has already funded and initiated a research effort to study the uptake of various radionuclides in seedlings and plantings. Although applicable to general considerations, this effort is directed at the question of if and when the Enjebi people might be permitted to return to their island.

Page 7, lines 21-23 - "And as time goes on, scientific knowledge of the nature and effect of radioactivity is bound to improve and new techniques for remedial measures will be found."

Comments: Anticipation of the development for startlingly new remedial measures to counteract the effects of radiation does not appear realistic in this situation for several reasons: new findings are not anticipated in the foreseeable future; those techniques which are available have limited usefulness for specific situations; at the extremely low levels of exposure discussed in this situation there is no way of determing the effectiveness of any such technique, nor is any need for same anticipated for this population.

Page 7, lines 23-26 - "These scientific advancements will be lost to the Enewetak people unless the United States government assumes a long-range commitment of the kind we suggest here."

Comments: The "long-range" commitment is not defined. Governments are not known to commit themselves to the application of scientific advancements prior to the discovery of such advancements even to their own populations,

especially in the absence of a proven need for such application.

Pages 8-11 - The Hot Particle Problem - The comments of NRDC, Geesaman and Martell are addressed separately following their respective comments.

Page 8, lines 12-17 - "For a discussion of the seriousness the hot particles problem we attach as Appendix II, E. A. Martell, "Basic Considerations in the Assessment of the Cancer Risks and Standards for Internal Alpha Emitters," (Statement presented at the public hearings on plutonium standards sponsored by the United States Environmental Protection Agency, Denver, Colorado, January 10, 1975)."

Comments: Martell's conclusions are reached quite independent of those who advocate the "hot particle" hypothesis. Although he reaches similar conclusions the logic by which he justifies his position cannot be considered suportive of the "hot particle" argument (see Martell's comments, page 8, lines 11-22).

Page 8, lines 25-27 - "It is highly likely that inhalation of very small amounts of plutonium gives rise to a high risk of lung cancer."

Comments: There is no question that inhalation of sufficient quantities of plutonium will lead to lung cancer. It is not clear what is meant by "very small amounts." Research to determine the levels below which lung cancer is not seen is currently in progress.

Page 8, line 27 to Page 9, line 2 - "And the DEIS completely fails to address the recent findings of Martell and others that hot particles may very well be a causative factor in a number of other disorders."

Comments: The "...recent findings of Martell..." were presented at public hearings in Denver, Colorado, on January 10, 1975, as stated on page 8, lines 15-17, and were appended as Appendix II. These "...findings of Martell..." are reviewed separately (see "Comments on Appendix II: 'Basic Considerations in the Assessment of the Cancer Risks and Standards for Internal Alpha Emitters'").

Page 9, lines 12-16 - "In addition, the ^{241}Am concentrations range up to 8.2 pCi/g averaged over the tope 15 cm depth of soils, with $^{241}Am/^{239}Pu$ ratios varying widely and ranging up to 3.5 (NVO-140, Vol. 1, p. 507)."

Comments: The Survey does claim (page 507) that activities of Am-241-to-Pu-239, 240 in Enewetak soil range up to 3.5. However, it goes on to indicate that the highest ratios are for those islands which have the lowest absolute amounts of Am-241 and Pu-239. In a communication with D. W. Wilson, author of the cited information, he explained that the high ratios result from comparing two numbers which are themselves small and near the limit-of-detection; consequently, the 3.5 value is not significant. (All ratios greater than 1.0, moreover, were calculated for samples taken from the southern islands of the Atoll.) The more meaningful ratios are listed in Table 14, page 98, of the Survey. Those ratios are for the northern islands and they average about 0.40 with the largest being 0.51. Plutonium continues to be the more abundant of the two elements.

Page 9, lines 16-18 - "Due to further radioactive decay of ^{241}Pu, the ^{241}Am activity concentrations can be expected to double over the next 50 years."

Comments: The source of ^{241}Am at Enewetak Atoll is the decay of the parent nuclide ^{241}Pu. The maximum ^{241}Am activity which can result from the decay of ^{241}Pu is 2.6% of the initial ^{241}Pu activity. This maximum ^{241}Am level is reached in 69.6 years. However, at 17 years 65% of the maximum ^{241}Am activity is already present and at 20 years 71% is present. The average time since most of the tests at Enewetak is about 20 years. On this basis alone the ^{241}Am levels at Enewetak will not increase by more than 30-40% above present levels.

Page 9, lines 21-22 - "The DEIS limits consideration of $^{239+240}Pu$ to inhalation risks."

Comments: The statement is in error. The DEIS includes also doses to various organs resulting from the ingestion of $^{239,240}Pu$ via food chains. See, for example, Table 235, p. II-48, Vol. II for integral doses from the various food chain; Table 239, pp. II-53 to 55, Vol. II for doages from

ingestion of terrestrial foods assuming diet at the time of return for each of the island groups; Table 240, pp. II-56 to 58, Vol. II for dosage from ingestion of terrestrial foods assuming a 10-year post return diet for each of the island groups. The critical organ (bone) dose resulting from the ingestion of $^{239,240}Pu$ is insignificant when compared to the dose from all other radionuclides.

Estimates of doses for residents of Enewetak Atoll for various living and dietary patterns presented in the Enewetak Radiological Survey Report, NVO-140 and in the Task Group Report considered the critical organ for the most sensitive segment of the population and all pathways and all significant contributors (radionuclides) to this exposure.* The judgments and recommendations of the AEC Task Group were made using current guidance on radiation protection practices. Guidance from the Federal Radiation Council (FRC), International Commission on Radiological Protection (ICRP), and the National Council on Radiation Protection and Measurements (NCRP) was used.

Page 9 - lines 22-25 - "However significant uptake of Pu from the gastrointestinal tract has been observed in young mammals and similar uptake may occur in young children."

Comments: This statement is partially correct. Absorption of plutonium from the gastrointestinal tract is as much as 100 times greater in the newborn rat than in the adult. A similar effect of enhanced absorption of normally non-absorbed substances is observed in the newborn of other animal species. If such an effect occurs in the human infant, it will probably persist for only a few days following birth. However, the predominantly milk diet consumed during this period is a very poor source of transuranic elements.

*Dose assessments were made for the fetus, the newborn, children and adults, using exposure levels in the highest year predicted. Because of the extremely small (<0.001%) contribution to the bone dose due to plutonium, the transuranic elements were not included in this age-related assessment.

There is no indication that a similar increased uptake would occur in young children.

Special calculations of dose resulting from ingested plutonium have not been made for the infant or child. The available data on metabolic behavior of the transuranic elements in the infant and child indicate that no significant underestimate of hazard will result from considering the total population as adults.

Because of the very long retention of transuranic elements in man, most of the radiation dose deposited in infants or children will be delivered when the child has grown to a much larger size. The radionuclide is not only diluted by this growth process, but, in the important instance of bone, is buried under new bone growth and its alpha particles largely shielded from the radiosensitive cells on the bone surface. Thus, the smaller intake of radionuclides by the infant or child results, over the life span, in a very much smaller dose than the metabolic models predict using adult intake parameters.

Page 9, line 25 to Page 10, line 10 - "In addition the uptake of americium in soils by vegetation is substantially higher than plutonium uptake. Similarly americium is readily taken up from the gastrointestinal tract and accumulated in the liver, spleen and bone of mammals, and thus undoubtedly in man.

"Based on these considerations it is possible that uptake of americium in the food chain and its accumulation in the liver and skeletal tissue of man may be the critical path for exposure to internal alpha emitters in the Enewetak Atoll area.

Comments: The use of ^{241}Am data in the dose evaluations for Enewetak Atoll in "Enewetak Radiological Survey," NVO-140, 1973, are as follows:

Marine Food Chain - ^{241}Am data were included in the dose assessment. ^{241}Am was non-detected in most of the fish samples analyzed. In such cases the value used for assessment was the detection limit.

Terrestrial Food Chain - ^{241}Am data were not included in the dose assessment because it was not detected in any of the edible food chain samples. $^{239,240}Pu$ was detected in terrestrial products and the relative significance of Pu was evaluated for this pathway. $^{239,240}Pu$ contributes less than 0.001% of the bone (the critical organ) dose via this pathway.*

Inhalation Pathway - ^{241}Am was not evaluated via this pathway.

Page 10, lines 10-14 - "The radiological survey is seriously inadequate with respect to americium distribution in both vegetation and in edible marine life to assess the consequent body burdens and health consequences to future atoll inhabitants."

Comments: A more precise estimate of the relative increase in ^{241}Am levels and the potential concentrations of ^{241}Am relative to $^{239,240}Pu$ can be made from data available in "Enewetak Radiological Survey", NVO-140, 1973, (Ref. 1), from a report by V. E. Noshkin, et al., "Transuranics at Pacific Atolls, 1. Concentrations in the Waters at Enewetak and Bikini," UCRL-51612, 1974, (Ref. 2), and from more recent data of Noshkin at Enewetak Atoll (unpublished) (Ref. 3). These data are considered for several different compartments at Enewetak Atoll.

Water Concentrations

a. One crater and on lagoon sample have been analyzed for both ^{241}Pu and $^{239,240}Pu$. The $^{241}Pu/^{239,240}Pu$ ratios were 1.14 and 2.56 with a mean of 1.85 and a standard deviation of 1.0. This average ratio

*After maximum ingrowth of ^{241}Am, the ^{241}Am concentration will be approximately 40% of the $^{239+240}Pu$ present. Assuming that the Am uptake of americium in soils by food plants is an order of magnitude greater than for plutonium and that the absorption of Am across the gastrointestinal tract is two orders of magnitude greater than for Pu, the Am contribution to the bone dose is still less than 0.5% of the total dose. With time the proportion of dose resulting from the transuranic elements will increase due to a decrease in the total dose resulting from the radioactive decay of many of the fission products.

has been used to estimate the ^{241}Pu activity in other compartments. The calculations have also been made using a ratio of $^{241}Pu/^{239,240}Pu$ of 3.0 which is slightly more than X + 1σ(i.e., 1.85 + 1.0 = 2.85).

b. Lagoon surface water has an average $^{239,240}Pu$ concentration of 39 fCi/1 (Ref. 2, Ref. 1). Using a ratio of 1.85 $^{241}Pu/^{239,240}Pu$ the ^{241}Pu activity is 72 fCi/1. The ratio of ^{241}Am to $^{239,240}Pu$ is 0.11 (Ref. 3); therefore, the ^{241}Am activity is 4.3 fCi/1. The maximum ^{241}Am activity which will result from decay of present levels of ^{241}Pu is 72 (0.026) = 1.87 fCi/1. This is a 43% increase over present ^{241}Am activity. The total ^{241}Am which will result is 6.2 fCi/1 which is 16% of the $^{239,240}Pu$ activity.

Plankton

Concentrations in pCi per gram wet weight of ^{241}Am and $^{239,240}Pu$ in plankton samples are 0.23 and 0.39 respectively (Ref. 1). Using the factor 1.85 for $^{241}Pu/^{239,240}Pu$, the ^{241}Pu concentration would be 0.72 pCi/g. Maximum growth from this level of ^{241}Pu will be 0.72 (0.026) = 0.019 pCi/g. Relative to the present ^{241}Am level of 0.23 pCi/g this represents an 8% increase. The total ^{241}Am is 0.23 + 0.019 = 0.249 pCi/g. Relative to the $^{239,240}Pu$ levels this is 0.249/0.39 = 0.64 or 64%.

However, a more realistic situation is that the plankton will always reflect the same ratio of ^{241}Am to $^{239,240}Pu$ as they do to the present water concentrations. Therefore, the increased ^{241}Am water concentrations resulting from the "grow in" from ^{241}Pu will lead to ^{241}Am concentrations in plankton which are 85% of the $^{239,240}Pu$ concentrations.

Fish

The average concentration of ^{241}Am and $^{239,240}Pu$ is 0.11 and 0.25 pCi/g respectively (Ref. 1). ^{241}Am was non-detected in most fish samples and the value 0.11 pCi/g represents the value obtained when the detection limit is assumed to be a real concentration. Again using 1.85 as the ^{241}Pu to $^{239,240}Pu$ ratio the ^{241}Pu concentration is 1.85 (0.25) = 0.46 pCi/g ^{241}Pu.

The maximum ^{241}Am which can grow in from this level of ^{241}Pu is 0.46 (0.026) = 0.012 pCi/g. Relative to the present ^{241}Am concentrations of 0.11 pCi/g this represents an 11% increase. The total ^{241}Am will be 0.11 + 0.012 = 0.122 pCi/g; compared with the $^{239,240}Pu$ of 0.25 pCi/g this is 0.122/0.25 = 0.49 or 49% of the $^{239,240}Pu$ levels.

However, a more realistic situation is that the fish will always reflect the same ratio of ^{241}Am to $^{239,240}Pu$ as they do to the present water concentrations. Therefore, the increased ^{241}Am water concentrations resulting from "grow in" from ^{241}Pu will lead to ^{241}Am concentrations in fish which are 64% of the $^{239,240}Pu$ concentrations.

Sediment Concentrations and Ratios

The ratio of ^{241}Am to $^{239,240}Pu$ in lagoon sediments is 0.37 and for the craters is 0.29 (Ref. 1). These data will be shown to correspond very well with ratios observed in the soil. The ^{241}Am and $^{239,240}Pu$ concentrations are 172 and 463 mCi/Km^2 respectively. The ^{241}Pu concentration is therefore 1.85 (463) = 857 mCi/Km^2. The maximum ^{241}Am activity from ^{241}Pu is therefore 0.026 (857) - 22 mCi/Km^2. This will be a 13% increase above present levels of 172 mCi/Km^2. The total ^{241}Am activity will be 172 + 22 = 194 mCi/Km^2 which 194/463 = 0.42 or 42% of the $^{239,240}Pu$ activity.

Soil Concentrations

The average soil concentration of $^{239,240}Pu$ for the northern half of Enewetak Atoll is 12 pCi/g (Ref. 1). The $^{241}Am/^{239,240}Pu$ ratio in the soil at Enewetak is 0.36 (Ref. 1). Therefore, the present ^{241}Am concentration is $^{239,240}Pu$ 12 pCi/g (0.36 $^{241}Am/^{239,240}Pu$) = 4.3 pCi/g ^{241}Am.

Assuming the same ratio for $^{241}Pu/^{239,240}Pu$ of 1.85, the ^{241}Pu activity is 1.85 (12) = 22 pCi/g ^{241}Pu. The maximum grow in of ^{241}Am from this level of ^{241}Pu is 22 (0.026) = 0.57 pCi/g ^{241}Am.

When the calculated increase from ^{241}Pu of 0.57 pCi/g ^{241}Am is compared to 4.3 pCi/g of ^{241}Am now present it represents a 13% increase. The total ^{241}Am will be 4.3 + 0.57 = 4.87 pCi/g. When compared with the 12 pCi/g of $^{239,240}Pu$ present, the ^{241}Am level will be 41% of the $^{239,240}Pu$.

Ground Water

The average $^{239,240}Pu$ concentration in the ground water on Enjebi is 5.4 fCi/l (Ref. 3). Using the 1.85 factor the ^{241}Pu concentration is 10 fCi/l. ^{241}Am concentrations have not yet been measured but assuming a similar ratio for ^{241}Am to $^{239,240}Pu$ in both the lagoon water and ground water an estimate of the ^{241}Am in ground water can be made. 5.4 fCi/l $^{239,240}Pu$ in ground water (4.3 fCi/l ^{241}Am in lagoon water)/39 fCi/l $^{239,240}Pu$ in lagoon water = 0.6 fCi/l ^{241}Am in the ground water. Decay of ^{241}Pu leads to a 43% increase in ^{241}Am concentration and the total ^{241}Am activity is 16% of the $^{239,240}Pu$ activity.

Vegetation

Over 400 vegetation samples were analyzed during the Enewetak survey. There were only 3 samples where both ^{241}Am and $^{239,240}Pu$ were detected. The average values for these 3 samples were 0.44 pCi/g $^{239,240}Pu$ and 0.053 pCi/g ^{241}Am. The ^{241}Pu concentration is 1.85 (0.44) = 0.82 pCi/g. The maximum ^{241}Am activity resulting from this level of ^{241}Pu is 0.026 (0.82) = 0.021 pCi/g ^{241}Am which is a 40% increase above present ^{241}Am concentrations. The total ^{241}Am activity is 0.053 + 0.021 = 0.074 pCi/g which is 0.074 pCi/g/0.44 pCi/g = 0.168 or 17% of the $^{239,240}Pu$ concentration.

Page 10, line 19 to Page 11, line 10 - "The resuspension measurements and calculations which relate the air contamination to the soil contamination are not immediately compelling, and deserve a much more careful analysis than I have given them. I would be surprised if the analysis is meaningful to factor of 100, when used to determine public health guidelines. Resuspension is poorly understood, it is sensitive to windspeed, soil characteristics, vegetation, humidity, rainfall, mechanical disturbance, physical and chemical history of plutonium particles in soil. How then does one consider the exposure of children throwing dry sand on a windy day at the beach? I would anticipate large fluctuations about the implicity exposure levels, which, even for the limiting soil contamination guidelines and predicted air concentrations associated with these guidelines, will be approximately a maximum permissible lung burden."

Comments: The issues raised by Dr. Geesaman are not new. We are well aware that all of the variables which are identified have not been analyzed with respect to their individual or combined influence upon resuspension factors. For that reason additional air sampling studies will be carried out for a period of twelve months, as described in the DEIS.

Even though measurements made primarily reflect airborne plutonium from worldwide fallout levels and cosmic ray activity, because of the uncertainties identified the assumptions made in deriving the various organ doses due to the inhalation of plutonium are quite conservative. A constant air concentration of plutonium is assumed, consisting of low

solubility, optimal size particles for deep lung deposition; furthermore, cases for both surface soil concentrations of plutonium and average soil plutonium concentrations are calculated. The conservatism of these factors is apparent in that: the average person is not likely to be constantly exposed to an air dust loading of 100 $\mu g/m^3$; the plutonium content of the air is not likely to continually be the same as that in the soil; all resuspending particles of plutonium will never be of an identical and optimal respirable size; particles of low solubility are considered to be the more hazardous of the chemical forms of plutonium.

In view of the author's stated uncertainties it is not clear what the basis is for the conclusions stated, or their derivation or justification. In the absence of any of these it must be regarded as opinion.

Page 11, lines 15-20 - "Concerning the standard employed by the DEIS for maximum permissible plutonium contamination of soils at Enewetak, Dr. Martell points out that 'There are no ICRP standards for soil levels of Pu and the actinides or for lifetime exposures to internal alpha emitters.' (Personal Communication.) And he provides the following critique of the standards adopted by the AEC Task Group for Enewetak:"

Comments: Numerical values of radiation exposure and concentrations of plutonium in soil were recommended by the Task Group as guides for use in evaluating radiological conditions at Enewetak Atoll only. Such guides were not intended as and are not to be considered as standards. These guides were used as limits in evaluating remedial action options in order to recommend actions and restrictions that will ensure that exposures of people when they return will not exceed the basic FRC, ICRP, and NCRP standards. These considerations are the basis for actions and restrictions recommended in the DEIS. While there is no national or international standard for plutonium expressed as a concentration in soil, the guides recommended, 40 and 400 pCi/g, were derived using the best current information relating such soil concentrations to possible exposure to man. The guidance for cleanup of contaminated soil

was selected such that exposures of people are expected to be well within the basic standard. This guidance has been approved by EPA for use at Enewetak.

The statement that, "There are no ICRP standards...for lifetime exposures to internal alpha emitters," is in error. All ICRP standards for internal emitters are based upon the assumption of lifetime exposure.

Page 11, line 21 to Page 12, line 4 - "The recommendation that plutonium contaminated soils, with levels not exceeding 40 pCi $^{239+240}Pu$/g of soil averaged over 15 cm depth, is suitable for human habitation, can be very seriously questioned.

"The State of Colorado Board of Health has adopted interim standards for Pu contamination limits in soils in land areas for residential use, specifying that ^{238}Pu levels shall not exceed 2 dpm (0.91 pCi) per gram of surface soil (i.e., averaged over the top 1 cm depth of soil)."

Comments: The information quoted from Dr. Martell's "personal communication" relative to an interim standard for plutonium in soil adopted by the State of Colorado Board of Health is grossly misleading. The guidance referred to does not apply to cleanup or removal of soil containing plutonium or to restrictions on use of plutonium contaminated land as Dr. Martell's communication implies. After conduct of an appropriate hearing, the Colorado Board accepted 2 dpm/g or 1 pCi/g of plutonium in soil as requiring special techniques of construction upon such property. These special techniques are intended to minimize plutonium resuspension by construction activities. This guidance is irrelevant to development of plutonium cleanup guidance for Enewetak Atoll.

in cases with long-standing pulmonary hypertension. In costal cartilage there was no increase in ash with age and the alpha activity declined with age. Turner et al. and Mayneord were cited as having reported that the radioactivity of bone ash does not increase with age.

In short, Elkeles reported that in those elastic arteries which are the most common sites of atherosclerosis, there is increase of both ash and alpha activity with age, and advanced the concept that progressive deposition of calcium together with small amounts of alpha emitters lead to subtle injury and reactive changes of connective tissue in arterial walls leading to atherosclerosis.

However, as mentioned above, there are changes in some parts of some vessels which precede and provide a receptive environment for deposition of calcium and the alpha emitters that behave like calcium metabolically and go with calcium, e.g., from bone to vessel walls. Much larger doses of alpha radiation than the amounts measured by Elkeles are required to damage arteries to the point of causing substantial increase in calcium deposition. Increasing blood pressure with age should be highly suspect as one condition which may contribute to subtle but progressive changes in aorta, coronary arteries, and perhaps even renal and other arterioles to some extent, which may provide the conditions favoring calcium deposition. Just as atherosclerosis occurs in pulmonary arteries under the conditions of pulmonary hypertension, atherosclerosis in aorta or coronary arteries and damage of renal arterioles are associated with general hypertension or increases in blood pressure with age.

Page 11, lines 4-7 - "In addition atherosclerosis plaques normally occur in the main and abdominal aortas and the coronary arteries, but rarely in the pulmonary arteries[42,44]. This distribution suggests a respiratory origin for the mutagenic agent."

FRC, ICRP, and NCRP have all taken the position that there can be different guides with different numerical values for different circumstances. This is one reason why it is difficult for standards agencies to develop standards applicable to a broad range of circumstances. This is also why such guidance is often developed on an Ad Hoc basis. The Task Group 40-400 pCi/g and the 1 pCi/g cited by Dr. Martell are both examples of Ad Hoc guides for plutonium in soil developed for a completely different purpose and for very different conditions. Inherent in both guides are considerations of what may be feasible. Inherent in both is the assumption that neither is absolutely safe. Neither of the guides should be considered as standards.

It is not feasible to plan operations such as Enewetak cleanup on the basis of what may be learned in the future that would warrant changing standards. Likewise, application of current Federal Regulations containing basic radiation protection philosophy, practice, and standards cannot be too much influenced by a contrary State Health Department "interim standard" devised for a unique set of circumstances. Assuming that basic standards can be met, it is reasonable to assume that the guide selected for each set of circumstances involving protection of people from radiation exposure would be the lowest level within the standard that is feasible, a level that is attainable without inordinate difficulties. This is the idea behind the "lowest practicable" concept.

From a radiation exposure consideration, there is in fact little choice in the level of protection that can be provided the Enewetak people. The choices for cleanup degree at Enewetak are limited in one

direction by the basic FRC standards considered as an upper limit to what might be acceptable (this is a health consideration), and in the other direction by a rapidly increasing engineering effort that is required for even small increments of exposure reduction below the standards (this is a cost consideration). It would not take much excess conservatism in cleanup "monitoring" or changes in the Task Group numerical guides to upset the agreement on standards and/or delicately balanced position on cleanup guidance that has been achieved among the Federal agencies. The health risk associated with exposures at the level of the FRC standards is known to be very low and considered acceptable for the general public, but this risk may not be zero. No guarantee can be given that those who return to Enewetak will experience zero ill effects from radiation received. However, we do not expect to see any such effects.

Nevertheless, if the wording is examined carefully, the comparison is made between the recommended cleanup criteria and the Colorado "interim standards" in land areas for residential use. It should be noted that Case 3, the recommended cleanup plan, would limit the residence locations of the Enewetakese to the southern islands of the Atoll, at least initially. According to Table 3-8, p. 3-70, Vol. I of the DEIS, the mean plutonium concentration in soil on most of these southern islands varies from 0.04 to 0.07 pCi/gm (ranging from 0.004 to 1.1 pCi/gm), with one island showing a mean concentration of 0.63 pCi/gm (range 0.2-2.0), all of which are below the interim guideline established by the State of Colorado and referred to by Dr. Martell. While these are mean values over 15 cm of soil depth, the islands consisting of the initial islands of habitation show a mean value of 0.04 with a range of

0.004 to 0.31. Even if the total quantity of plutonium in this sample were concentrated in the top 1 cm of soil (a most unlikely situation), the concentration would be 1.5 pCi/gm, quite comparable to the Colorado guidelines.

The recommendations for Enewetak are based upon reasonable constraints to the living patterns and the diet of the people after their return. Colorado criteria did not consider such factors. Furthermore, the Colorado values are not based upon any demonstrated health hazard to man, but rather are based upon an arbitrary factor times the plutonium concentration in Colorado soils resulting from world-wide fallout.

Page 12, lines 4-6 - "It is noteworthy that the AEC has not established that this standard is unduly conservative..."

Comment: ERDA (AEC) has never recognized the interim plutonium concentration in soil guidelines of the State of Colorado, nor are we aware that any other Federal agency has recognized this guideline; such guidance is not applicable to Federal property. However, we do not dispute the right of the sovreign State of Colorado to establish whatever guidelines it wishes for applicability elsewhere within the state. The lack of any challenge on this issue should not be interpreted as tacit acceptance of the guidelines, however.

Page 12, lines 6-10 - "...it is not apparent that the AEC has requested the ICRP or NCRP to make specific recommendations with respect to standards for Pu in soils applicable to chronic exposure to the general public, including children."

Comments: Contrary to the impressions of Dr. Martell, the ICRP and the NCRP consistently have avoided giving guidance with respect to plutonium contaminated soil. Whether or not this response may change in the future is a matter for speculation. To date, however, no guidance has been forthcoming.

As stated above, FRC, ICRP and NCRP have all taken the position that there can be different guides with different numerical values for different circumstances; in other words, each situation is unique and must be considered in relation to its specific characteristics.

Page 12, lines 15-20 - "...for most Enewetak soils the top cm contains substantially higher levels of Pu per gram than the 15 cm depth average. Thus, for example, at location 101 on Pearl, the top 1 cm depth shows 400 pCi ^{239}Pu/g, whereas the average over 15 cm depth is about 60."

Comments: While Dr. Martell is correct that "for most Enewetak soils the top cover contains substantially higher levels of Pu per gram than the 15 cm depth average," there are also locations where higher plutonium concentrations are found below the top cover of soil (Janet, locations 135, 142, 143, 144, 901; Irene, 24, 27, 51, 100; Alice 24; Belle, 35, etc.). None of these islands are expected to be inhabited islands. Comparison with the Colorado guidelines, therefore, are grossly misleading. Furthermore, the recommendation of the Task Group clearly states that there should be, "Recovery of plutonium in soil at concentrations greater than 400 pCi/g ^{239,240}Pu at any depth these levels are found. Also, recovery of contaminated soil sufficient to reduce surface levels to a value well below 40 pCi/g ^{239,240}Pu" (p. 5-80, Vol. I; emphasis added).

Page 12, lines 23-26 - "There are recent research developments which are expected to lead to reductions in acceptable organ burdens of Pu in man by a factor of 100 to 1000 or more."

Comments: There are no recent research developments of which we are aware that are expected by knowledgeable experts "to lead to reductions in acceptable organ burdens of Pu in man by a factor of 100 to 1000 or more." If Dr. Martell is aware of research data which would justify such changes,

it would be expected to be distributed to the scientific community so that the ICRP and NCRP might consider the implications.

Page 12, line 26 to Page 13, line 3 - "In my opinion it is likely that a 10 pCi lung burden of insoluble alpha emitting particles will give rise to significant adverse health effects for lifetime exposures."

Comments: Similarly, if Dr. Martell has evidence that "a 10 pCi lung burden of insoluble alpha emitting particles will give rise to significant adverse health effects for lifetime exposures," we would expect to have such data presented scientifically. Until such time as evidence is available, these conclusions remain as stated by Dr. Martell as "my opinion."

Page 13, lines 14 to Page 14, line 6 - "Drs. Cochran, Tamplin and Geesaman all raise the same or similar objections to the DEIS plutonium standards.

"Further explanation of the plutonium cleanup criteria developed by the AEC Task Group is necessary. (DEIS, Vol. II, Tab B, pp. III-8 to III-11.) We have already mentioned the questionable wisdom of the 40 pCi/g standard. For any concentrations exceeding 400 pCi/g the Task Group recommendations require removal of the soil. But in the range between 40 and 400 pCi/g, the DEIS standards call for 'corrective action ..... on a case-by-case basis.' (Vol. II, Tab B, p. III-9.) Certain criteria are offered for guidance in the exercise of this judgment, but they appear to be entirely too unspecific and subjective. Once a decision is made to take corrective action, the objective is to achieve a substantial reduction in plutonium soil concentrations, and further, to reduce concentrations to the lowest practicable level, not to reduce them to some prescribed numerical value. (Ibid. Emphasis added.)"

Comments: As stated in the DEIS, decisions regarding corrective action for plutonium concentrations in soil between 40 and 400 pCi/gm $^{239,240}Pu$ will be on a case-by-case basis. Many specific factors enter into such a decision for which definite statements and numbers are inappropriate: location, environmental factors (e.g., wind and wave action); soil matrix; soil use; frequency and duration of human, animal or crop contact; risk/benefit balance; significance of removal, etc. To establish predetermined criteria for those and other variables is unrealistic. Judgment must be used to determine what can be done without doing more harm than good.

Page 14, lines 7-10 - "Nor is it entirely clear who will be making these "case-by-case" decisions. Presumably it is the "team of experts" referred to in the recommendations of the Task Group (Vol. II, Tab B, p. 27), but we are not told who they are or how they will be selected."

Comments: Defense Nuclear Agency is responsible for cleanup of Enewetak Atoll. Staff for radiological support of cleanup operations will be selected by that Agency. The AEC Task Group recommended inclusion of Public Health Service (EPA) experts in the team that will interpret radiation and radioactivity measurements and provide advice and guidance in the field on cleanup actions, as was done for Bikini Atoll cleanup.

Page 14, lines 11-15 - "This whole approach must be explained and justified, especially at a time when the EPA is conducting hearings around the country on plutonium soil standards for precisely the purpose of developing 'numerical values' for the maximum concentrations permissible."

Comments: EPA has conducted public hearings in Washington, D.C., and in Denver, Colorado, to "evaluate the environmental impact of plutonium and the other transuranium elements and to consider whether new guidelines or standards are needed to assure adequate protection of the general ambient environment and of the public health from potential contamination of the environment by radionuclides of these elements." It can be seen from the above that the purpose of the hearings was to determine whether or not additional or new guidelines or standards are required; it was not the purpose of the hearings to set new standards or to specifically develop plutonium soil standards, much less "for precisely the purpose of developing 'numerical values.". These activities may or may not subsequently take place, but the hearings were held to obtain information relevant to the above stated objectives which appeared in the Federal Register. It is expected that in time EPA may provide additional guidance pertaining to plutonium soil standards, but it is not at all clear at this time that this guidance will consist of numerical values, even as FRC guidance in the past has not referred to

numerical values.

Page 14, lines 18-29 - "Before any final standards are set for the radiological cleanup of Enewetak, the International Commission on Radiological Protection should be called upon for plutonium and actinide standards applicable to air, water, soils and food concentrations for both soluble and insoluble activities, applicable to long-range exposure to the general public. Application should also be made to the U. S. Environmental Protection Agency for special hearings for the same purpose. Consideration should also be given to the desirability of requesting the United Nations Scientific Committee on the Effects of Atomic Radiation to conduct hearings and set these standards. (We are indebted to Dr. Martell for these suggestions.)"

Comments: It is doubtful if UN hearings or additional EPA hearings would bring to light any information not already known and considered, or that ICRP would address the specific question of what would be acceptable for Enewetak cleanup. ICRP policy leaves to each nation a degree of flexibility in applying the basic standards. Theonly experience at clean-up and rehabilitation of an atoll lies within ERDA, EPA, HEW, DOD, and DOI.

The request for EPA public hearings on plutonium soil contamination standards appears inconsistent with the preceding paragraph on page 14 which states that such hearings are now being held around the country.

It should also be noted that there are in existence applicable ICRP standards for air and water (and by extension, food) for both soluble and insoluble forms of these isotopes for long-range exposure to the general public. Thus the only issue is the one of soil standards, which has been discussed at length previously.

Page 15, line 5 to page 16, line 7 - "Removal and Disposal of Radiocontaminated Materials These comments relate to the proposed removal and disposal of contaminated scrap metal and soil treated in the DEIS at Vol. 1, SS5.3.3.3 and 5.5.

"All radiocontaminated scrap metal on the Atoll has been identified and will be removed, as of course it must be, but the precise method of disposal has not been determined. Four alternative methods are discussed: ocean dumping of the loose scrap, concrete encapsulation in the Cactus and Lacrosse craters at the north end of Runit islet, or removal to the United

States mainland for storage. We appreciate the practical and political difficulties presented by the various disposal methods which would remove the scrap from the Atoll entirely, but the People of Enewetak are adamantly opposed to any disposal upon or within theenvirons of the Atoll. Ocean cumping, according the DEIS (Vol. I, S 5.5.2.1), was rejected 'in view of the difficulty in obtaining a permit and certainty of international complications.' Disposal to the United States mainland was disfavored for similar reasons. (Vol. I. S 5.5.2.4) Disposal on the Atoll must be rejected and the other methods should be explored, the necessary permits and authority obtained and disposal off the Atoll selected as the preferred method.

"Removal and disposal of contaminated soil presents more serious cost and practical difficulties, but here again the complete removal and off-Atoll disposal of all contaminated soil must be the stated objective of the program.

"Even using the high plutonium contamination standard set by the Task Group (40 pCi/g, etc.), the total amount of Atoll soil which would have to be removed and disposed is 779,000 cubic yeards. (Vol. I S 5.5.2). If the soil standards are lowered as they should be, that volume will increase."

Comments: The comments pertaining to disposal of contaminated material are most appropriately dealt with by agencies other than ERDA because of the legal, political and fiscal implications.

Page 16, lines 15-18 - "... but a clear decision must be taken to study and fully assess the relation of soil removal to dose reduction (including the risk from airborne hot particles) and the likely ecological effects of soil removal and replacement."

Comments: From a radiation exposure consideration, there is in fact little choice in the level of protection that can be provided the Enewetak people. The choices for cleanup degree at Enewetak are limited in one direction by the basic FRC standards considered as an upper limit to what might be acceptable (this is a health consideration), and in the other direction by a rapidly increasing engineering effort that is required for even small increments of exposure reduction below the recommended guidelines (this is a cost consideration). It would not take much excess conservatism in cleanup "monitoring" or changes in the Task Group numerical guides to upset the delicately balanced position on cleanup guidance that has been

achieved among the Federal agencies.* The health risk associated with exposure at the level of the FRC standards is known to be very low and considered acceptable for the general public, but this risk may not be zero. No guarantee can be given that those who return to Enewetak will experience zero ill effects from radiation received; however, we do not expect to see any such effects.

An assessment of the relation of soil removal to dose reduction is discussed in the DEIS, Vol. II, Section 13, pp. 8-14.

Page 16, line 25 to page 17, line 11 - "Radiological Monitoring of Cleanup The AEC Task Group has wisely recommended the establishment of 'team of experts' to monitor the execution of the radiological cleanup phase of the program. (DEIS, Vol. I, pp. 5-79, 6-5) Even if the Task Group is enlarged as we have suggested and specific soil standards are developed and implemented, this monitoring group will perform a crucial function. Thus, it is important that its membership be carefully selected. It is imperative that radioscientists of the most conservative cast be included in the monitoring group. Here again, we suggest that the names of Drs. Martell, Geesaman, Tamplin and Cochran.

"And the on-site authority of the monitoring group should be clearly defined, with all important or unexpected problems to be referred to the enlarged Task Group."

Comments: The scientific approach used in development of radiation protection standards and practices is inherently conservative. The basic standards of the FRC, which according to law must be implemented by Federal agencies, are in themselves conservative. Recommendations by the AEC Task Group contain additional safety factors and provisions for checking the effectiveness of remedial actions and restructions. Application of these recommendations by those of the most conservative cast might well lead to actions based on no standards at all. In our opinion, the Enewetak cleanup

*A situation can easily be envisioned in which in order to obtain the level of cleanup desired, an island might essentially have to be scraped away. Whether or not such requirements benefit anyone is debatable.

field operation is not theproper forum to pursue debates and discuss issues between individual scientists where there can be no early resolution. This would be disruptive and devisive for those conducting cleanup. The hypotheses of Drs. Martell, Geesaman, Tamplin and Cochran are subjects for proper scientific debate. This is best conducted through the medium of technical journals and scientific meetings, not in day-to-day deliberations at Enewetak Atoll.

Page 17, lines 12-19 - "Test Plantings, Groundwater and Air Sampling
We are in full agreement with the AEC Task Group recommendations for test plantings, lens water and air sampling. (Vol. I., pp. 5-80 to 5-81). But it is not clear whether these recommendations have been implemented. They must be and the studies should be commissioned to the best scientists and technicians available, under the over-all guidance of the enlarged Task Group. All of these studies must deal explicitly with the hot particle problem."

Comments: All of the recommendations referred to here have been implemented and either are or soon will be underway. Additional information per these specific projects are appended.

It is not clear how the lens water and test plantings projects are "to deal explicitly with the hot particle problem." Plutonium analyses will be performed routinely, but "hot particle" analyses is not contemplated in these studies. Characterization of resuspended particles will be conducted as a part of the air sampling project, however.

Page 17, line 20 to Page 18, line 8 - "Radiobiological Health Followup
AEC Task Group recommendation 12 (Vol. I, p. 5-81) calls for 'Baseline surveys of body burdens and urine content of Cs-137 and Sr-90...for the Enewetak people prior to return to Enewetak Atoll, and periodically thereafter.' But here, too, it is not clear whether a firm commitment to long-range radiological health monitoring of the Enwetak population has been made, and, if so, precisely how it will be implemented.

"A fully adequate radiological health program must be designed, funded and implemented. It can and should include the people of Bikini, who will one day soon be resettled, the exposure victims at Rongelap and Utirik Atolls and the Enewetak people.

"The final impact statement should address this question and state clearly whether such a program is planned and what it will include."

Comments: Provision for periodic radiological health monitoring of the Enewetak population, including analyses for strontium, cesium and plutonium, has already been arranged.

Page 18, lines 8-10 - "It too must deal with the health effects of hot particles and all forms of low level radiation, with emphasis on internal emitters."

Comments: It is not clear whether "It" refers to the Final Statement or to the radiological health monitoring program to be established. If the former, the issue is discussed in our response to the comments of Drs. Martell, Tamplin and Geesaman. If the latter, it is a very unrealistic request. To identify any possible health effect in a very small population resulting from extremely low levels of respirable particles, which may or may not be present, of questionable significance even in controlled laboratory environments would be nearly impossible. Criteria justifying the initiation of any controlled epidemiologically and radiologically valid study are not present. Studies of "the health effects of hot particles and all forms of low level radiation" have not yet been completed in laboratories; it is not realistic to expect to study them in the population under discussion. To study any relationship between morbidity or mortality and the anticipated levels of exposure to radiation is a considerably different situation from monitoring the people to ascertain thelevels of internal emitters to which they are exposed. The latter will be done, as stated above, but there is no intention of the former.

Page 22, lines 19-23 - "But at the same time all of the radiological investigations recommended here should be undertaken and high confidence results obtained as soon as possible so that they can be used to revise and improve the radiological cleanup phase before moving forward with it."

Comments: The additional radiological investigations recommended by the Task Group have already been funded and initiated. If all of the recommendations suggested by the author had to be undertaken and high confidence results obtained to revise and improve the radiological cleanup phase before proceeding, the return of the Enewetakese might have to be abandoned or delayed a good many years.

Not proofed or edited

Comments on Appendix II: "Basic Considerations in the Assessment of the Cancer Risks and Standards for Internal Emitters" by Edward A. Martell*

Page 2, lines 9-13 -"Bair et al. [10] fail to take into account the full implications of some of the recent published results: in particular, the observed higher tumor risks for $^{238}PuO_2$ than for $^{239}PuO_2$ [11], the apparently limited biological response of mammal lung cells from ^{238}Pu and ^{239}Pu incorporated into ceramic microspheres [12,13]..."

Comments: It is highly unlikely that Dr. Bair would fail to take into account the implications of data emanating from his own laboratory (11)**; similarly, it is not realistic to assume that Dr. Richmond would fail to appreciate data from his own experimental work.

Page 2, lines 13-17 - "...and the tobacco smoke radioactivity results[14]. The latter results imply that as little as a few picocuries of insoluble alpha emitting particles in the lung may give rise to a significant risk of lung cancer and other serious health effects in the chronic exposure case."

Comments: The Public Health statistics correlating tobacco smoking and incidence of lung cancer and other diseases do not distinguish selectively between the alpha emitters and the rest of the tobacco smoke as causative agents. Extensive epidemiological observations suggest that the etiology of lung cancer in smokers is different from that in irradiated populations. The excess risk of lung cancer produced by radiation persists for at least three decades after single or briefly fractionated exposures; it does not show any appreciable return toward normal levels for twenty years following an initial 8-12 year latency. On the other hand, when smoking is continued for a prolonged period and then terminated, excess risk remains constant for only 1-3 years after which a steady decline to normal occurs in 10-14 years. The striking difference in the temporal pattern of excess mortality

* The author referred to herein is Dr. Martell.
**Numbers throughout are to Dr. Martell's references.

in former smokers and in irradiated humans indicates, contrary to Dr. Martell's suggestion, that the etiology is different in the groups. Uranium miners who have stopped smoking do not show this pattern of declining risk typical of unirradiated populations; about 30% of the cancer deaths in uranium miners have occurred in former smokers even though more than half of them had not smoked for five years or more. This suggests that the effects of the smoke as a whole, or apart from the contained alpha emitters, are probably much more important than the permanent effects of the small amounts of alpha emitters in the smoke, in regard to the mechanism of induction of the associated lung cancer.

While Dr. Martell provides evidence regarding the presence of very small quantities of alpha emitting radionuclides in definitive structures of the tobacco leaf, his conclusions go considerably beyond the data provided.

Page 2, lines 28-29 - "And for long term exposures, unacceptably high tumor risks appear to be associated with picocurie burdens of internal alpha emitters."

Comments: If this conclusion is based upon the tobacco smoking statistics, the previous response is applicable here also.

also

Page 3, lines 1-5 - "It/is possible that the critical health effects for alpha emitting particles are the incidence of atherosclerosis and other degenerative diseases of the cardiovascular system. The published evidence supporting these conclusions is briefly reviewed below."

Comments: The evidence which the author presents in support of his conclusions is reviewed below.

Page 4, lines 13-16 - "The alpha radiation-induced bone tumor incidence in dogs is observed to be proportional to the square of the alpha dose[19] implying that a sequence of two or more low probability events must be involved."

Comments: This is true and should be noted for future reference. It should also be noted that this dose-incidence relationship indicates not only an increase in tumor incidence with increasing dose size and dose rate (associated with dose size), but also an increase in tumorigenic effectiveness per unit dose with increasing dose size and dose rate. This kind of dose-incidence relationship shows decreasing effectiveness of doses in the rising portion of the dose-incidence curve with the decreasing dose rate that is associated with decreasing dose.

Page 4, lines 16-18 - "This is consistent with the two-mutation and multiple-mutation theories of cancer[20,21] based on the age distribution of cancer in man."

Comments: This is also consistent with the multistage theories of mechanisms of carcinogenesis requiring cellular initisting events (malignant cell transformation) plus promotional events such as local tissue damage or damage of structure and function of more remote but relevant organs or systems by one or a combination of agents or conditions.

Page 4, lines 18-27 - "On the basis of these considerations the production of a malignant cell involves a sequence of events, as follows: (1) production of a viable mutated cell; (2) clone growth from the mutated cell; (3) production of a second viable mutation in one or more of the clone; (4) growth of a clone of doubly-mutated cells; etc. Thus, for a two-mutation sequence, the tumor risk would be proportional to the $R^2t^2(t/T_c)$, where R is the alpha dose rate, t is the time of exposure, and T_c is the mean life of the normal cell and singly mutated cell. The term (t/T_c) represents the influence of the growth of the clone of the singly-mutated cell on the long-term risk."

Comments:

The author states that, for a two-mutation sequence, the tumor risk would be proportional to $R^2t^2\ (t/T_c)$, where R is alpha dose rate, t is time of exposure, T_c is mean life of normal cell and singly mutated cell, and the term (t/T_c) represents the influence of the growth of the clone of the singly-mutated cell on the long-term risk.

Assuming that this formula is appropriate for the continuous alpha particle irradiation from internal alpha emitters, with very high LET, short track in tissue, and high cell sterilizing and killing efficiency and effect within short distances of the sources, and applying to it various dose rates (R), a given constant time of exposure (t) and a given constant mean life of normal cell and singly mutated cell (T_c), the formula seems to indicate that for varying internally administered amounts of alpha emitter (continuous alpha irradiation), i.e., different doses and associated dose rates, the tumor incidence would be proportional to the square of the dose or the square of the dose rate, and the incidence per unit dose would increase in proportion to increasing dose or increasing dose rate. This is compatible with the dose-incidence relationship for alpha radiation-induced bone tumor incidence in dogs cited by the author in lines 13-16, page 4 (*see comments on that sentence above*). This kind of dose-incidence relationship for alpha emitters (involving continuous irradiation) indicates decreasing effectiveness of doses in the rising portion of the dose-incidence curve with the decreasing dose rate that is associated with decreasing dose.

It is difficult to reconcile these findings with the author's next statement, as follows:

Page 4, line 28 to Page 5, line 3 - "This tumor risk relationship makes it abundantly clear that a linear extrapolation to low dose rates is not only conservative for alpha radiation induced tumors, but rather that there is a marked increase dose-rate vs. risk relationship."

Comments: Reference is made to the immediately preceding set of comments. For varying amounts of internally administered alpha emitter

of a given type, the continuous irradiation occurs over a constant time of exposure, but the dose rate (and the total accumulated dose in a given time) depend directly on the amoung of alpha emitter administered. Both dose and dose rate are relevant and important in internal alpha radiation induction of cancer. In the author's formula, if the only parameter that is varied is the dose rate (R), this would also vary the dose proportionally for a constant time of exposure. On this basis there is a direct relationship between incidence (or risk) and dose rate or dose size.

Perhaps what the author intended, but did not make clear and explicit, was that the total dose should be kept constant by varying both the dose rate (R) and the time of exposure (t) such that the product of the two (the total dose) would always be the same. Under these circumstances, as one increases the dose rate (R) one decreases the exposure time (t) proportionally, and the consequent reduction of the function t/T_c reduces the value of $R^2t^2(t/T_c)$ which is related to incidence or risk. However appropriate the use of the formula may be for estimating a two-mutation sequence from some kinds of radiation from external sources, it is artificial and neither appropriate nor realistic for tumor incidence or risk for continuous irradiation from internal alpha emitters which cannot be limited to varying times of exposure in relation to dose rate, and it requires presumptions on the interrelationships among dose rate, time of exposure, total dose, relevant induction dose, and latent period in internal alpha radiation induction of tumors, as well as the assumption that a two-mutation sequence is, or is equivalent to, the mechanism of cancer induction.

Page 4, lines 3-24 - "There is an increasing body of published experimental evidence that reflects this trend.

"Speiss and Mays[22] observed that for ^{224}Ra alpha radiation induced bone sarcoma in man, the tumor incidence per rad approximately doubled for a four-fold increase in the spacing of ^{224}Ra injections and that the observed incidence of bone tumors per rad in children was nearly twice that for adults. Upton et al.[23] show a significantly higher incidence of tumors in mice for a given neutron dose at more protracted periods of exposure. Moskalev and Buldakov[24] showed that fractionation of the administered ^{239}Pu dose over larger periods of time increased bone tumor induction. The higher tumor incidence per rad for the smaller lung burdens of crushed $^{239}PuO_2$ microspheres observed by Sanders[11] seems best explained by the limited alpha irradiation of large numbers of cells by numerous very small, mobile particles of low acticity per particle (see below). Hamsters subjected to low alpha doses from ^{210}Po distributed quite homogeneously in the bronchiolar-alveolar region show a marked increase in the lung tumor incidence per rad at very low doses and dose rates[25]. And the incidence of bronchial cancer in uranium miners reflects a higher tumor risk per rad at the lower doses[26] for this low dose rate exposure group. The tobacco radioactivity results[14] indicate a significant tumor risk for the cumulative alpha radiation dose from ^{210}Po in insoluble particles in the bronchi of smokers, involving much lower dose rates."

Comments:

Here the author indicates that "there is an increasing body of published experimental evidence that reflects this trend", i.e., referring to "marked inverse dose-rate vs. risk relationship." Then the author cites various reports to support this.

It is well known that fractionation or protraction (reduction of dose rate) of doses that as intensive doses are in the range of high doses that are relatively inefficient (per rad) for carcinogenesis (i.e., in the declining part of the dose-incidence curve following the peak at the most efficient dose level), owing to excessive cell sterilization or destruction, will increase the efficiency of such doses. It is also well known that fractionation or protraction of a dose that as an intensive dose is in the rapidly rising portion of a dose-squared dose-incidence relationship (an efficient dose) will reduce the

effectiveness and efficiency of the dose. It is the dose rate influence on these efficient doses that is important in considering the possible influence of dose and dose rate reduction to levels of interest in radiation protection. The author neglects this distinction in his selection of reports for presentation in this paragraph.

While the material Sanders (11) administered to rats was derived from crushed $^{238}PuO_2$ microspheres, the animals did not receive crushed $^{238}PuO_2$ microspheres. The material had been stored in a saline solution for a long period of time and had been altered to a non-crystalline form of plutonium (i.e., it had no detectable x-ray diffraction pattern) throught to be solubilized in the saline and "monomeric" ^{238}Pu in form.

Page 5, line 25 to Page 6, line 20 - "Based on the above considerations it is evident that the tumor risk is optimized when a very large number of cells and their descendants are subjected to only a few widely spaced alpha interactions with the small target afforded by the cell chromosomes. This follows necessarily from the fact that most alpha interactions with cell chromosomes lead to the subsequent mitotic death of the cell, as Barendsen has shown[17,18]. The production of a malignant cell calls for a sequence of two or more low probability events and thus cannot be speeded up by the application of massive alpha doses, but rather only by subjecting a much larger number of cells to a limited number of interactions. Additionally, assuming that the tumor risk to the tissue subjected to alpha irradiation is proportional to $R^2t^2(t/T_c)$, explained above, it is apparent that the alpha activity concentration or the activity per particle which is equated to a given tumor risk decreases with increasing time of exposure and also that a given risk can be attributed to smaller cumulative doses when the time of exposure t is appreciably longer than the mean life of the cell, T_c. Brues[27] and Burch[28] both pointed out that the two-mutation theories of carcinogenesis[20,21] would imply an exceptionally high effectiveness of widely spaced radiation for tumor production. It is proposed that just such a dose rate relationship serves to reconcile the observed significant tumor risk in cigarette smokers with the presence of a persistent lung burden of insoluble smoke particles involving a total of only a few picocuries of ^{210}Po[14]."

Comments:

Here the author does recognize the high cell sterilizing or killing efficiency of alpha radiation in attempting to further his argument that tumor risk is optimized at very low dose. This argument and his additional argument, on the basis of the assumption that the tumor risk to the tissue subjected to alpha irradiation is proportional to $R^2t^2(t/T_c)$, that "the alpha activity concentration or the activity per particle which is equated to a given tumor risk decreases with increasing time of exposure and also that a given risk can be attributed to smaller cumulative doses when the time of exposure t is appreciably longer than the mean life of the cell, T_c", are rather enigmatic with respect to dose and dose-rate relationships with effect, but are subject to the previous (above) comments on the author's pages 4 and 5.

The dose-squared relationship between alpha radiation induced cancer incidence and dose (as in the dog experiments referred to) indicates increasing effectiveness and efficiency of dose in the rising portion of the relationship curve until the curve changes to a plateau before declining at still higher doses. According to this, the rising portion of the downwardly convex curve shows decreasing efficiency with decreasing dose. Under actual conditions, different amounts of a particular alpha emitter in a particular form and distribution within an organ irradiates cells and tissues for the same time period (t) and with the same decay kinetics, and therefore the dose rate (R) and the dose are determined by the amount of the alpha emitter taken in or present. The use of the formula $R^2t^2(t/T_c)$ with

proportional va tion of R and t to achieve con nt dose for examination of the influence of variation in dose rate and time of exposure may be useful for external sources of radiation which can be controlled with respect to R and t, but this manipulation is artificial and unrealistic for internal alpha emitters which are not subject to such control of the variation in time of exposure.

Page 6, "Hot" PuO_2 Particle Risks - "If the above tentative conclusions are correct, then the same considerations must apply in the assessment of tumor risks for hot particles."

Comments:

The correctness of "the above tentative conclusions" are subject in part, at least, to previous (above) comments on the author's pages 4, 5 and 6.

Page 6, lines 26-28 - "Raabe et al.[29] report an apparent rate of dissolution of $^{238}PuO_2$ in lung fluid which is two orders of magnitude higher than that observed for $^{239}PuO_2$ particles."

Comments: The $^{238}PuO_2$ dissolution experiments referred to were not carried out using "lung fluid", but rather a synthetic serum simulant. In addition, these experiments were conducted in vitro, not in vivo as is implied.

Page 7, lines 5-8 - "In addition the $^{238}PuO_2$ particles exhibited a very significantly lower density than the $^{239}PuO_2$ particles[30], indicating a highly faulted structure and weakened intermolecular bonding for the $^{238}PuO_2$ particles."

Comments:

Early measurements of density of PuO_2 with the Lovelace Aerosol Particle Separator system were highly variable due to experimental errors, with values averaging about 10 g/m^3; this value was reported for $^{239}PuO_2$. Improved techniques were developed by the time the $^{238}PuO_2$ experiments were conducted and the particle densities measurements were more constant and probably more accurate with average values around 8 g/cm^3. That this difference in reported density was caused by "...a highly faulted structure

and weakened in molecular binding for $^{238}PuO_2$. ' is speculation by the author and appears somewhat oversimplified.

Page 7, lines 8-12 - "Fleischer[31] proposes that the apparently higher dissolution rate for $^{238}PuO_2$ may be explained by the alpha recoil nucleus ablation of the surface layers of the particles, with a fragmentation rate proportional to the specific alpha disintegration rate and with variable sizes of fragments ranging up to $\sim 10^4$ atoms."

Comments:

Fleisher's suggestion that aggregate recoil explains the increased dissolution rate of $^{238}PuO_2$ over $^{239}PuO_2$. Clearly, this is a radiolytic effect, but the exact mechanism has not been unequivocably demonstrated.

Page 7, lines 12-14 - "The poorer structural integrity of the $^{238}PuO_2$ particles may give rise to an increase in the size range of the ejected fragments."

Comments:

The reference to "...poorer structural integrity of the $^{238}PuO_2$..." gives the impression of being a factoral statement; in point of fact it is the author's speculation, and possibly an erroneous one. When $^{238}PuO_2$ is prepared in a manner identical to the preparation of ^{239}Pu, investigators do not feel that the $^{238}PuO_2$ has "poorer structural integrity" or lower density than $^{239}PuO_2$, although it does have a lower median particle size. This alone could account for a higher solubility rate, in so far as the $^{238}PuO_2$ particles would have a larger surface area per unit mass (or activity) than $^{239}PuO_2$ particles.

Page 7, lines 14-17 - "Such small fragments, ranging up to tens of angstroms in diameter or more, would pass readily through the 0.1 μm diameter pores of the membrane filters used in the dissolution experiments[29]."

Comments:

Whether small ablation fragments, if they are formed, can pass readily through a membrane filter rated at 0.1 μm pore has not been demonstrated. This assumption and those following, while perhaps reasonable, are assumptions of the author.

Page 7. lines 23-27 - "Another explanation for the apparently higher solubility of $^{239}PuO_2$ than $^{238}PuO_2$ is the possibility that the intense alpha radiolysis of the lung fluid at the surface of the particles leads to the production of chemically active free radicals which in turn react with PuO_2 molecules on the particle surface."

Comments:

The author is incorrect; presumably a typographical error occurred. We expect that "...higher solubility of $^{239}PuO_2$ than $^{238}PuO_2$ is..." should read "...higher solubility of $^{238}PuO_2$ than $^{239}PuO_2$ is...".

In the experiments referred to (29), "intense alpha radiolysis" seems rather inappropriate to describe the irradiation of a solvent surrounding a submicrometer particle of $^{239}PuO_2$ or $^{238}PuO_2$ which are probably widely separated in the lung. A 0.44 μm $^{238}PuO_2$ particle of the same size emits only 3 alpha particles per hour. In well buffered solvents such as were used in the dissolution experiments, radiolysis products are probably quickly inactivated at the slow rate and in the small quantities that they are formed.

Page 8, lines 2-6 - "However this dissolved plutonium undoubtedly would be slowly redistributed in the lung in the same fashion as that reported by Moskalev[34] for inhaled soluble compounds of plutonium, resulting in a highly non-uniform distribution, with hot spots located predominantly in the sub-pleural region of the lungs."

Comments:

That "...this dissolved plutonium undoubtedly would be slowly redistributed in the lung..." (emphasis added) is a source of confusion. The material that is redistributed in the lung is the material that does not become solubilized, e.g., PuO_2 particles, or polymerized $Pu(NO3)4$. The solubilized plutonium enters the bloodstreams and is translocated to the liver or the skeleton; this has been shown quite clearly in both the rat and the dog in studies at the Pacific Northwest Laboratories.

Page 8, lines 6-8 - "This gradual conversion of the soluble plutonium compounds to small colloidal size particles at focal points of activity may be the result of the self-chelating properties of tetravalent plutonium in solution."

Comments:

It is not clear what is either meant here or what assumptions have been made to reach this conclusion.

Page 8, lines 9-11 and line 17 - "In recent studies of rat inhalation of $^{238}PuO_2$, Sanders(11) has demonstrated a substantially increased risk per rad for small lung burdens of aged, 'crushed' $^{238}PuO_2$ microspheres."
"...the greater mobility and wider redistribution of the $^{238}PuO_2$ microspheres..."

Comments:

The material to which Sanders (11) exposed rats was not "'crushed' $^{238}PuO_2$ microspheres." It was material derived from crushed $^{238}PuO_2$ microspheres. It had been stored in a saline solution for a long period of time and had been altered to a non-crystalline form of plutonium (i.e., it had no detectable x-ray diffraction pattern) thought to be solubilized in the saline and "monomeric" in form.

In addition, it should be pointed out that the smallest initial deposition in this study was 5 nCi, or about 300 times the current maximum permissible occupational lung burden for humans of 0.016 nCi/gm (assuming a 1000 gm lung). The low rad dose, in contrast to this deposition, was due to the rapid alveolar clearance of the inhaled ^{238}Pu.

Page 9, lines 20-22 - "It is proposed that these two tumors may be attributed to secondary protons ejected by alpha interactions with hydrogen atoms. The expected yield is one proton per 10^4 alpha interactions."

Comments:

It is not clear exactly who is making the proposed mechanism of induction of the two tumors referred to, what the basis is for the proposed conclusion, or what evidence is available to support it.

Page 10, lines 12-18 - "It has long been known that those tissues in which there is more active cell division suffer the earliest and most seve severe radiation damage effects, and that this includes the blood forming cells in lymphatic glands and in bone marrow.[16,37]. Such effects include the destruction of rapidly multiplying cells that produce the blood platelets which assist in the control of blood clotting. Similarly the population of leucocytes is reduced with a corresponding reduction in resistance to disease."

Comments:

These sentences apply primarily to the acute radiation effects seen after high exposure levels. Their relevance to the effects of alpha emitting radionuclides is not clear.

Page 10, lines 18-21 - "These effects plus the accompanying chromosome structural changes can give rise to the earlier incidence not only of cancers, but the whole pattern of diseases of the cardiovascular and renal systems[37,38]."

Comments:

The author, referring to the fact that radiation can damage or destroy cells that produce blood platelets and leukocytes, states that, "These effects plus the accompanying chromosome structural changes can give rise to the earlier incidence not only of cancers, but the whole pattern of diseases of the cardiovascular and renal systems[37,38]." This statement, the manner in which it is made, and the sweeping implications of it are misleading, non-sequitus and unaccompanied by adequate meaningful explanation of foundation. It attempts to faction-alize mechanistic connections which are so remote and speculative, and neglectful of known aspects of the mechanisms, as to practically meaningless. The references [37 (a publication appearing in 1938) and 38] do not provide substantive support for the sweeping mechanistic aspects of the statement.

Page 10, lines 22-26 - "Let us review the mounting evidence which suggests that inhaled insoluble alpha emitting particles may be the agent of atherosclerosis and thus give rise to an increased risk of death by early coronaries and strokes. Atherosclerosis is reported to be present in every instance of partial or complete arterial occlusion and every case of coronary thrombosis(39)."

Comments:

Reference 39 is identified as a 1940 paper in The American Heart Journal pertaining to arteriosclerosis of the coronary arteries and the mechanism of their occlusion. The journal volume number was not provided in the reference list, and we were unable to find the paper by means of the year (1940) indicated in the reference listing given. To date we have not yet located this paper.

Page 11, lines 1-3 - "Recently Benditt has shown(40) that the human atherosclerotic plaque is a monoclonal proliferation of a mutated cell of the artery wall, and thus an arterial tumor."

Comments:

Clarification of this matter requires explanation of what the paper of reference (40) by Benditt and Benditt (1973) actually reports and shows.

First, it should be pointed out that early in embryonic development of mammalian females there is random inactivation of one or the other of the two x-chromosomes in various cells. Thus, the female becomes a mosaic of two cell types, each type having one or the other of the pair of x-chromosomes active with respect to x-linked glucose-6-phosphate dehydrogenase isoenzymes. The two cell populations reproduce true to type in this respect throughout somatic growth, it is thought. Benditt and Benditt (1973) referred to Linder and Fartler as having examined the nature of the cell population in benign uterine smooth muscle

tumors by investigating the pair of x-linked isoenzymes.

Benditt and Benditt (1973) (author's reference 40) investigated by this means individual atherosclerotic plaques from various regions of the aorta and common iliac arteries of 4 human females. The data were reported to show that the fibrous caps of the atheromatous plaques were composed of cells that produce solely or predominantly one of the two isoenzymes, whereas samples of artery wall media and intima were regularly composed of a mixture of the two isoenzyme cell types.

These investigators considered an alternative to the injury-repair hypothesis of spontaneous atherosclerosis on the basis of the following considerations: cells of spontaneous atherosclerotic lesions differ from cells of normal artery wall and cells populating a repair site in size, composition of associated extracellular material (e.g. preponderance of collagen rather than elastin), and in the absence of intercellular junctions. These investigators stated that these differences and the results of their enzyme analysis of plaques and normal vessel components imply that atherosclerotic plaques in human beings arise by another mechanism. They stated that these features suggest two possibilities: either the cells of atherosclerotic plaques derive from a population of cells different from those of the normal arterial media or they are transformed cells, and if the latter is so, cells of atherosclerotic plaques, like those of the benign smooth muscle tumors of the uterine, could be expected to be monoclonal. It is at this point that Benditt and Benditt seem to have used the term, monoclonal, to suggest origin not only from one of the two

isoenzyme cell types of the female but from a single transformed cell. In this latter context they stated that the mechanism compatible with the monoclonal nature of atherosclerotic plaques is mutation, and that the likely causes are chemical mutagens or viruses.

All that the actual data (isoenzyme data) in this paper (Benditt and Benditt) really show is that the plaques arise solely or predominantly from one or the other of the two embryonically determined isoenzyme cell types, with differentiation or metaplasia of the cells of either type in certain characteristic ways under the atherosclerotic circumstances, and not that plaques necessarily had origin from single (versus multiple) cells of the isoenzyme cell type predominating. The actual data did not show that cell mutation was involved, as is stated by the author of the present document under review. Nor does proliferation of a mutated cell necessarily result in a tumor.

Benditt and Benditt acknowledged the possibility that the reason for the sole or predominant presence of one or the other of the two isoenzyme cell types in the plaques is not a monoclonal origin but rather some process selecting from one or the other of the two cell types.

Benditt and Benditt did not actually define the atherosclerotic plaque as an arterial tumor as the author of the document under review seems to imply in relation to his reference to the paper by Benditt and Benditt.

Page 11, lines 3-4 - "Elkeles[41,43] has observed anomalously high concentrations of alpha activity at the calcified plaque sites."

Comments:

Elkeles (1966) (author's reference 42) pointed out the well known fact that calcium deposition in various soft tissues is a manifestation of aging. Elkeles referred to a paper by Blumenthal et al. who micro-incinerated human aortas and showed that calcium was deposited in the

media after 20 years of age, and emphasized that such deposits increase with age and precede the formation of intimal plaques. Blumenthal et 1. also observed that the ratio of calcium phosphate to calcium carbonate in the aortas is similar to that in bone.

It should be pointed out here that whether or not the calcium deposits in blood vessels may be regarded as an irritant leading to damage and repair in the formation of plaques, there are subtle to obvious changes in parts of some blood vessels (e.g., aorta, coronary arteries, renal arterioles) that somehow provide a receptive environment for deposition of calcium (dystrophic calcification). Calcium deposition may be especially marked in conditions involving excessive demineralization of bone, as in advanced osteoporosis of aging, osteitis fibrosa, and parathyroid disorders, and with elevation of blood levels of calcium, from whatever cause. The deposition of calcium in blood vessels as a consequence of damage of bone of experimental animals after internal administration of substantial doses of bone-seeking alpha emitters has been observed.

Elkeles (to whom the author referred) pointed out that certain radioactive substances are deposited with the calcium in the skeletal system. He reported that in elderly patients, the alpha particle activity per unit net weight of aorta, although variable, tended to follow the degree of calcification. He studied the abdominal aorta, coronary arteries, pulmonary artery, and ribs and costal cartilage. Ash % and alpha activity rose with age only in the aorta and coronary arteries. In pulmonary arteries, ash % did not rise and alpha activity declined with age. Pulmonary arteries were chosen as the control arteries because they are histologically similar to aorta but not subject to atherosclerosis except

in cases with long-standing pulmonary hypertension. In costal cartilage there was no increase in ash with age and the alpha activity declined with age. Turner et al. and Mayneord were cited as having reported that the radioactivity of bone ash does not increase with age.

In short, Elkeles reported that in those elastic arteries which are the most common sites of atherosclerosis, there is increase of both ash and alpha activity with age, and advanced the concept that progressive deposition of calcium together with small amounts of alpha emitters lead to subtle injury and reactive changes of connective tissue in arterial walls leading to atherosclerosis.

However, as mentioned above, there are changes in some parts of some vessels which precede and provide a receptive environment for deposition of calcium and the alpha emitters that behave like calcium metabolically and go with calcium, e.g., from bone to vessel walls. Much larger doses of alpha radiation than the amounts measured by Elkeles are required to damage arteries to the point of causing substantial increase in calcium deposition. Increasing blood pressure with age should be highly suspect as one condition which may contribute to subtle but progressive changes in aorta, coronary arteries, and perhaps even renal and other arterioles to some extent, which may provide the conditions favoring calcium deposition. Just as atherosclerosis occurs in pulmonary arteries under the conditions of pulmonary hypertension, atherosclerosis in aorta or coronary arteries and damage of renal arterioles are associated with general hypertension or increases in blood pressure with age.

Page 11, lines 4-7 - "In addition atherosclerosis plaques normally occur in the main and abdominal aortas and the coronary arteries, but rarely in the pulmonary arteries[42,44]. This distribution suggests a respiratory origin for the mutagenic agent."

Comments:

Here the author assumes that atherosclerotic plaques are the result of alpha radiation induced cell mutations and suggests that the alpha emitters responsible originate from the lung (presumably inhaled) because the pulmonary arteries rarely develop atherosclerotic plaques. This would imply that inhaled alpha emitters that get into the blood are trapped efficiently in their first passage in the blood stream through the pulmonary veins, heart, coronary arteries, and perhaps the rest of the vascular tree except that virtually none is left in the blood by the time the blood reaches and services the pulmonary artery. The author does not discuss this matter, or the fate of alpha emitters absorbed from the gastrointestinal tract, or the mechanisms by which alpha emitters may be taken up so specially in aorta, coronary arteries, etc, on the first passage of the blood containing them.

The sentences which are the subject of these comments represent a very poor argument for the respiratory origin of the causative agent, for the nature of the causative agent, or for the reason for the rarity of atherosclerosis in the pulmonary artery. It is highly unlikely that there would be no alpha emitters passing through the blood of the pulmonary artery or of its vasa vasorum after inhalation and ingestion of alpha emitters that were in a state allowing them to pass into the blood.

Page 11, lines 8-10 - "Attempts to reproduce arterial lesions in animals by chemical, mechanical and nutritional means have not produced plaques similar to those of atherosclerosis in man[40]."

Comments:

The author's reference (40) is to the paper by Benditt and Benditt (1973), whose statement in this matter is as follows: "Chemical, mechanical, and nutritional manipulations have been used in animals in an effort to reproduce lesions like those of atherosclerosis in man: none of these experimental lesions yields wholly satisfactory copies of lesions of the human disease."

The next sentence in the Benditt and Benditt paper is as follows: "Spontaneous atherosclerosis occurs in chickens and, as we have found, produces lesions that strikingly resemble those of man."

The fact that certain types of experimental manipulation may have failed so far to reproduce wholly satisfactory copies of lesions of the human disease does not mean that some of those lesions which have been produced are wholly irrelevant or that radiation is the only agent that would be perfectly successful. Experimental manipulation with radiation has not succeeded in meeting this requirement either. Since other animals are not wholly like humans it has been difficult, but it is not necessarily impossible for the future, to produce good copies of the human disease by experimental manipulation of factors other than radiation. Perhaps investigation of the spontaneous lesions in chickens would provide valuable clues.

Page 11, lines 10-12 - "However atherosclerotic plaques have been directly induced in human arteries by intensive irradiation with x-rays and radium(45)."

Comments:

The reference (45) is to a paper by Sheehan (1944) on what Sheehan calls an uncommon or at least rarely described lesion, i.e., foam cell plaque, observed in the intima of irradiated small arteries (100 to 500

microns external diameter) in several irradiated organs. The lesion was described as a plaque-like thickening of intima due to collection of foam cells alone or foam cells mixed with various other cells, fluid, fibrin or hyaline material, between endothelium and internal elastic membrane. Although pathological changes were found sometimes also in adjacent internal elastic membrane, media and adventitia, these structures were often normal. The plaque may cause marked narrowing or even occlusion of the lumen. Thrombosis, fibroblastic proliferation of deposition of elastic tissue in the thickened intima seldom result. The foam cell plaques were found in small arteries in organs subjected to radiation therapy (large doses) by roentgen rays and/or gamma rays from radium sources. The paper states that the plaques probably result from migration of lymphocytes and monocytes into the intima from the blood stream and subsequent transformation (meaning differentiation or metamorphosis) of these cells into foam cells by their ingestion of lipids which have been freed by the dissolution of red cells in the intima or which have accumulated in the intima after passage across portions of the endothelium that was rendered more permeable than normal by irradiation. The paper contained a casual or incidental statement to the effect that the foam cell plaques in irradiated small arteries closely resemble the early lesion of atherosclerosis. If this were true, it would be indicative of some of the kinds of changes which may occur in vessels before, and presumably responsible for, subsequent deposition of calcium. It is interesting to point out, however, that earlier in this paper, Sheehan indicated that foam cell plaque was an uncommon or at least rarely described lesion.

Page 11, lines 15-17 - "For all of these reasons it is proposed that inhaled insoluble alpha emitting amoke particles are very likely to be the mutagenic agent which gives rise to atherosclerosis in cigarette smokers."

Comments:

This statement is a string of poorly founded presumptions covered by previous comments.

Page 11, lines 18-21 - "If this is the case, similar increased risk of early coronaries are to be expected for other groups of individuals who are occupationally or environmentally exposed to the inhalation of insoluble alpha emitting particles of respirable size."

Comments:

"If this is the case" is a poorly founded presumption, for reasons covered in previous comments.

Page 11, lines 26 to Page 12, line 2 - "Very significant increases in the incidence of early coronaries as well as lung cancers and cancers at other sites is observed among cigarette smokers[46] with insoluble alpha emitting particle burdens of only a few picocuries of ^{210}Po in the lung[14] and similar total alpha activity per 100 grams of arterial wall tissue[41-43]."

Comments:

This sentence is misleading in tying the alpha activity of arterial wall tissue to the statement about incidence of diseases among cigarette smokers. The references (41-43) refer to Elkeles' papers reporting alpha particle activity in calcified atherosclerosis and in coronary artery disease, based on measurements in plaques and vessels, where it is most likely that calcium, and alpha radioactivity with it, increase after alterations of the arterial tissue that lead to the rest of the atherosclerotic mechanisms have occurred (see comments on page 11, lines 3-7 and 10-12).

Page 12, line 3 to Page 13, line 2 - "By comparison, plutonium workers exhibit plutonium organ burdens ranging from a few picocuries to a few

nanocuries or more[47,48]. And although there has been no epidemiological study of the age-incidence of heart disease and cancer among plutonium workers, the limited published information bearing on this question is more disturbing than reassuring. Most often cited is the medical experience of 26 plutonium workers at Los Alamos[49,50], usually accompanied by a statement to the effect that none of the medical findings for this group can be attributed definitely to internally deposited plutonium. With equal justification one may state that mostof the serious medical findings in this group can be attributed to plutonium. One member of the original group died in the early 1950's. Cause of death is not reported. Another died of a coronary at age 38. A third suffered a coronary occlusion bur recovered and was well compensated. A fourth developed a hamartoma of the lung and his right lower lobe was surgically removed in May 1971. A fifth had a melanoma of the chest wall. A sixth had a partial gastrectomy for a bleeding ulcer. One subject suffered loss of teeth, apparently due to damage to the lamina dura of the jaws which show the earliest effects in beagles given toxic doses of plutonium. Another subject has gout. The full medical history of this group, now mostly in their fifties, has not yet completely unfolded. Only 12 of these 26 plutonium workers were exposed to plutonium inhalation. While of the observed effects were experienced by the inhalation exposure group? Regardless of the distribution, the medical experience of this small group thus far provides no basis for complacency about the health consequences of plutonium exposure.

"Hanford employees and others whose autopsy tissue samples exhibited plutonium levels in excess of 5 fCi/g died mainly of coronary heart disease and other cardiovascular effects and to a lesser extent of cancer and pulmonary emphysema[47]."

Comments:

The author's discussion here, of what he calls "the limited published information" on plutonium workers with plutonium organ burdens, cannot be categorized as a scientific analysis or discussion of the problem, but rather as his subjective reaction and opinion. In our opinion the Los Alamos workers with plutonium burdens and the autopsy cases in the Transuranium Registry do not constitute an adequate sample for the assessment of theincidence of any type of disease. We have not and do not feel that it would be purposeful to compare the incidence of various diseases with the national figures for such small samples. Adequate comparison data for the incidence of disease is not available for morbidity in theliving members of the Los Alamos group. The

autopsy data may show significant bias due to the selection process in obtaining permission for autopsies.

Page 13, lines 2-6 - "Based on evidence reviewed above it appears that atherosclerosis is a cancer of the artery wall and thus that coronary heart disease and other diseases of the cardiovascular and renal system are expected effects of inhaled plutonium and of other insoluble alpha emitting particles."

Comments:

This sweeping generalization and string of presumptions are poorly founded, for reasons given already in previous comments above.

Page 13, lines 6-8 - "An adequate assessment of the magnitude of these risks can only be obtained by a comprehensive medical follow-up of all past and present plutonium workers."

Comments:

We do, however, fully agree with the author that there is need for a proper epidemiologic study of workers with plutonium burdens, and ERDA is now developing concrete plans for such a study. The continuation of such studies as well as thepertinent experimental research are certainly worthy of support and encouragement.

Page 13, lines 8-12 - "Until the age distribution of these effects among plutonium workers is fully assessed, any claim by the proponents of nuclear energy that there is little risk associated with the MPLB (maximum permissible lung burden), 16 nCi of plutoniu, or fraction thereof, is totally unjustified."

Comments:

The use of the phrase "these effects among plutonium workers", without specification, suggests that the author has already concluded or presumed, on the basis of "the limited published information" (his term on his page 12), that all of the deaths, causes of death, and diseases among plutonium workers that he mentions on his pages 12 and 13 are "effects" of plutonium rather than natural or other specific causes, despite statements he attributes to the investigators of "the medical experience of 26 plutonium workers at Los Alamos[49,50]" to the effect that "none of

the medical findings for that group can be attributed definitely to internally deposited plutonium". On his page 12, the author states: "With equal justification one may state that most of the serious medical findings in this group can be attributed to plutonium", and this he states apparently on the basis of his examination of the limited published information and his speculations.

Where objective scientific assessments of risk are concerned, we do not think that the justification of the assessments or related claims should depend on whether or not one is a proponent or opponent of, or indifferent to, nuclear energy or its alternatives.

That any risk associated with the MPLB, or fractions thereof, is totally unjustified is an opinion of the author. The evidence presented by the author cannot be considered supportive of his conclusions in light of the above comments. The opinion that there is no fraction of the MPLB at which the risk becomes insigificant appear unrealistic.

Page 13, lines 12-14 - "The growing evidence suggests that as little as a few picocuries of alpha activity in the lung, in arterial tissue, and in other organs gives rise to a significant cancer risk."

Comments:

If the statistics relating cigarette smoking and lung cancer are the basis for the statement concerning lung, there is still a question concerning the relative importance of the few picocuries of alpha activity as compared with the influence of the rest of the smoke (see previous comments on the author's page 2, lines 13-17). The case for cancer risk in arterial tissue, if it refers to the author's postulate that atherosclerotic plaques are arterial tumors, is poorly founded (see previous comments on the author's pages 10 and 11). We are not clear what data has been provided to support the statement about "other organs", whichever

the author meant by this term, and "a few picocuries of alpha activity".

Page 13, lines 15-17 - "The published evidence, reviewed above, clearly indicates that a linear extrapolation to lower doses and dose rates is not conservative for internal alpha emitters."

Comments:

This statement is not clear about anything in it, not the "published evidence" or what is "reviewed above" that is pertinent to the statement, not the level of dose or dose rate (and associated efficiency for the effect) from which extrapolation linearly is supposed to be "not conservative", not the shape of the dose-effect curve that is regarded as nonconservative as compared with the linear one, not the meaning of "conservative", not the effect being considered in the statement, and not the kinds of alpha emitters referred to or their properties. Again, this is a sweeping and poorly founded generalization. See also previous comments on the author's pages 4 and 5.

Page 13, lines 17-19 - "The initial effects of alpha interactions with cell chromosomes are irreversible and thus will vary linearly with alpha dose rate."

Comments:

On his page 3, the author states: "When alphas interact with the chromosome or its gene in the nucleus of the cell, the dense ionization in the track of the alpha particles give rise to closely spaced breaks which bring about a wide variety of irreversible chromosome structural changes, or mutations. X-ray and γ-ray interactions give rise to a diffuse distribution of ions, resulting in widely spaced individual breaks, most of which can undergo repair by recombining without structural change. Thus permanent structural changes for x-rays and γ-rays are proportional to the square of the dose, with greatly reduced incidence

at low dose rates. By contrast, structural changes resulting from alpha interactions are directly proportional to the number of interactions and are independent of alpha interaction rates."

The proper interpretation or expression of the content of the quoted statement under comment and the quoted statements in the comments, is that for alpha radiation the incidence of induced chromosomal structural changes increases linearly (proportionally) with increasing dose, and also with dose rate under realistic conditions for internal alpha emitters where dose rate and dose are dependent upon amounts of alpha emitter present. On this linear relationship basis the effectiveness of the alpha radiation per unit dose (efficiency) is independent of dose rate, as contrasted with the dose-squared dose-effect relationship for x- or γ-rays and the dependence of effectiveness and efficiency on dose size and dose rate. However, it should be pointed out that either the linear or the dose-squared dose-effect relationship pertains only to the point of saturation of effect, with no further increase in the specified effect with further increase in dose. Furthermore, at very low doses of alpha radiation, if there were any reduction in efficiency of production of any particular type of chromosomal effect or other effect, which could be possible, this would indicate the possibility of an effect-reducing influence of reduced dose-rate.

Page 13, lines 19-22 - "However, the cumulative effects of internal alpha emitters gives rise to an increase in the populations of mutated cells (cells with viable structural changes in their chromosomes) and in the health consequences of such changes."

Comments:

This is true insofar as it means that the increasing dose with

continuous irradiation increases the incidence of mutated cells capable of reproduction, but only up to the point at which the continuing irradiation and increasing dose begins to sterilize or kill more of such reproductively capable mutated cells than it is producing. Compared with x- or γ-rays, alpha particles are highly efficient for killing or reproductively sterilizing cells, so that for equivalent dose parameters, the fraction of cells surviving and capable of reproducing themselves as well as carrying chromosomal aberrations and mutations, is very small for alpha radiation. But the amount of tissue damage and disorganization, which requires larger doses for production than does chromosomal aberration or mutation, and which may also be an important factor in mechanisms of carcinogenesis, is relatively greater for the high LET alpha radiation.

Page 13, lines 22-23 - "Therefore the tumor incidence per alpha disintegration must increase with decreasing dose rate."

Comments:

This sentence seems enigmatic, non-sequitus and perhaps incompatible with the author's previous sentence. Perhaps it depends upon what the author means by his newly injected dose parameter, "per alpha disintegration," as compared with his meaning of "dose" (rad?) and of "decreasing dose rate." If he intends to mean by "decreasing dose rate" the decreasing dose rate in an organ with increasing time after a given alpha emitter burden to that organ, and then intends to relate this lowered dose rate with the delayed appearance of cancer after a long latent period, it should be pointed out that the earlier high dose rate may be the rate more associated with the cancer induction. On the other hand, if he intends to mean a comparison of different dose rates,

initially or later on, based on different amounts of alpha emitter burden given to an organ initially, then the interpretation of his remark depends upon whether he is comparing high dose - high dose rate levels which are relatively inefficient for cancer production (by virtue of overkill of cells) (see my comments on the author's pages 4, 5 and 13) with lower dose and dose rate levels, or comparing different dose and dose rate levels in the region of ascending dose-incidence relationship.

It should be pointed out again that the author apparently regards chromosomal structural changes or mutations as constituting the mechanism of carcinogenesis and has indicated that the chromosomal effects of alpha radiation are directly proportional to the number of alpha interactions, independent of alpha interaction rates, and vary linearly with alpha dose rate. The linear relationship between effect and dose or dose rate indicates lack of dependence of effect per unit dose on dose size or dose rate. Therefore, it does not follow that tumor incidence per unit dose, on the basis of a mechanism that has a linear dose-effect relationship, will change with change in dose size or dose rate.

Page 13, lines 23-26 - "For this reason a given cancer risk is equated with smaller cumulative alpha doses and with much smaller internal alpha emitter burdens as the period of exposure decreases.

Comments:

This is an enigmatic statement, but taken literally appears to be unfounded for the same reasons given and referred to in my comments on the previous sentences on this page and on the author's pages 4 and 5.

Page 14, lines 1-9 - "By contrast, the cellular effects of X-rays and γ-rays are largely repairable at low dose rates. This stems from the fact that the diffuse distribution of ion pairs produced by such radiation results in widely spaced single chromosome breaks which repair themselves readily. For these reasons the relative biological effectiveness of alpha particles, compared to X-rays and γ-rays increases continuously with decreasing dose rate. Thus alpha radiation acquires a greatly increased biological significance relative to soft radiation in the production of tumors and other health consequences of chromosomal structural changes."

Comments:

The first two sentences of this paragraph are essentially correct. The third sentence is essentially correct, but it should be pointed out and emphasized that the relative biological effectiveness of alpha particle irradiation, compared to x-radiation and γ-radiation as standards, increases with decreasing dose rate not because the effectiveness of alpha radiation changes with change in dose rate but because the effectiveness of the standard radiation (x- or γ-radiation) decreases with decreasing dose rate. The effectiveness of the alpha radiation is independent of dose rate and proportional to dose.

The 4th sentence of this paragraph is also subject to this qualification and to others of my preceding comments relating to it.

Page 14, lines 10 to page 15, line 2 - There are several other lines of evidence which reinforce the possibility that alpha interactions with cells play a unique role in human cancer production. The distribution of cancer sites in the bronchi, in the lymphatic system, in arterial tissue, in the liver and bone, all involve sites at whichinsoluble alpha emitters are known to accumulate. Anomalously high concentrations of alpha activity have been observed at the bronchial cancer sites[51], at cancer sites adjoining lymph glands in other organs[52,53] in atherosclerosis plaques[41-43], at liver cancer sites in thorotrast patients[54], at bone tumor sites in the radium dial workers[55], etc. The difficulties of producing lung cancer by external radiation has been pointed out by Warren and Gates[35,36]. The absence of cancers in muscular tissue, except at sites of thorotrast injection or plutonium injection, also is relevant to this issue. All of these observations reinforce the possibility that one or more of

the chromosomal structural changes which characterize a malignant cell must be brought about by alpha interactions and not by low intensity X-rays or γ-rays. In this connection, the determination of the nature of the structural differences between the healthy and the malignant cells of each organ could shed some light on this important question."

Comments:

The first sentence of this paragraph indicates that the paragraph will present "several other lines of evidence which reinforce the possibility that alpha interactions with cells play a unique role in human cancer production."

If the author intends that the word "unique" be used in the strict sense of the word, e.g. single or sole, it should be pointed out that no type of radiation effect has been observed which cannot be caused by other agents or conditions, and that no type of effect of alpha radiation has been observed that cannot be caused by other kinds of ionizing radiations. The differences between effects of various types of ionizing radiations are quantitative rather than qualitative, and are owing to differences in relative biological effectiveness, LET, distribution, etc.

The next two sentences in the paragraph refer to observations of high concentration of alpha activity at sites of cancer. In regard to bronchial cancer sites, the author refers to a paper on distribution of polonium in pulmonary tissues of cigarette smokers. In regard to cancer sites in the lymphatic system, the author refers to a paper entitled, "Only vertebrates with a lymphatic system are subject to malignant disease," and to a note to the Health Physics Journal, entitled, "The lymphatic system--a storehouse of long-stay deposits of inhaled radioactive particles." The author's references to anomalously

high concentration of alpha activity in atherosclerotic plaques, which he has designated as arterial tumors, are subject to my detailed comments on earlier pages of his paper. The liver cancer sites in thorotrast patients agree well with the deposition of administered sizeable amounts of thorium oxide and the consequent tissue damage, as do the bone tumor sites in radium dial workers agree well with the deposition of sizeable amounts of ingested bone-seeking alpha emitters and the consequent bone damage.

The 4th sentence of the paragraph states: "The difficulties of producing lung cancer by external radiation has been pointed out by Warren and Gates[35,36]." Such experimental difficulties have more to do with species and strains of animals, getting high enough radiation doses from external sources into the lungs or into what may be appropriate lung structures for the species and strain of animal, without severely damaging too much lung tissue in other ways or other tissues between the radiation source and the lung and thereby causing competing earlier causes of death than the tumors of interest. Some laboratories have been producing lung tumors in experimental means by radiation from external sources. There have also been reports of epidemiological studies showing increased incidence of lung tumors in human beings following irradiation from external sources. The author's statement should not be taken to mean that radiation from external sources cannot cause lung cancer.

The 5th sentence of this paragraph states: "The absence of cancers in muscular tissue, except at sites of thorotrast injection or plutonium injection, also is relevant to this issue." The author does not develop further the relevance or the issue referred to, or

indicate the types of cancers to which he is referring. This statement should not be taken to mean that other radioactive isotopes injected into similar regions, or irradiation from external sources in one or another mode, cannot cause similar cancers.

In the 6th sentence of this paragraph, perhaps the author is giving the purpose for his previous two sentences. He states: "All of these observations reinforce the possibility that one or more of the chromosomal changes which characterize a malignant cell must be brought about by alpha interactions and not by low intensity x-rays or γ-rays." Although this sentence has enigmatic characteristics, a mixture of confusing qualifications, I will take it literally and state that it is not well founded and is neglectful of the evidence for x-ray and γ-ray induction of mutations, chromosomal aberrations, and cancer. Nor does it address the possibility that malignancy may result from completely different mechanisms than those postulated by the author, or through several different mechanisms, one of which may be chromosomal abnormalities resulting from radiation.

Page 15, lines 3-13 - "It is also observed that the relative significance of chemical agents, viruses and radiation in the incidence of human cancer is not known. Details of the mechanisms of cancer induction by chemical agents and viruses also are poorly understood. And the proposed chemical carcinogens in cigarette smoke and in polluted urban environments have not been demonstrated to be carcinogenic at the low concentrations involved. For all of these reasons it is deemed likely that radiation, and alpha radiation in particular, may be the principal agent of human cancer. In view of such a possibility, it is very disturbing to note that the U.S. National Cancer Institute, now spending about one-half billion dollars per year on cancer research, has completely neglected the field of radiation induced cancer research."

Comments:

It is obviously true that "the relative significance of chemical agents, viruses and radiation in the incidence of human cancer is not known."

It is true that the "details of the mechanisms of cancer induction by chemical agents and viruses also are poorly understood." However, I would point out that many experts in carcinogenesis would regard these mechanisms as better understood than the mechanisms of cancer induction by radiation.

The 3rd sentence of this paragraph states flatly and without elaboration that "the proposed chemical carcinogens in cigarette smoke and in polluted urban environments have not been demonstrated to be carcinogenic at the low concentrations involved." Even if this were true, it may be only a question of more time and investigation, as it has been with understanding the effects of radiation. Furthermore, the same might be said for the low alpha radiation activity by itself in regard to cigarette smoke, as compared with the effects of the rest of the smoke (see previous comments on the author's page 2).

The fourth sentence of this paragraph states: "For all of these reasons it is deemed likely that radiation, and alpha radiation in particular, may be the principal agent of human cancer." The "reasons" referred to in this paragraph, even if they were true, would hardly be reasons for anything but further research, and they certainly do not provide an acceptable scientific basis for this enormously sweeping generalization and summary dismissal of all other known and suspected agents and conditions which may cause or help to cause human cancer.

The fifth sentence implies that NCI has greater interest in etiological factors other than radiation but ascribes this interest

to a misdirection of the NCI program.

Page 15, lines 14-16 - "Published evidence[39-45] indicates that atherosclerosis is a tumor of the artery wall and that the alpha activity at the calcified plaque site is likely to be the mutagenic agent."

Comments:

This "evidence" has already been reviewed. Nowhere is evidence presented that atherosclerosis is a tumor of the artery wall or that alpha activity is likely to be the mutagenic agent. This statement is presented as fact, whereas it is the author's opinion.

Page 15, lines 16-19 - "If so the major causes of death in the general population - coronary disease, other cancers, and strokes - may in large part be attributable to internal alpha emitters from natural and pollutant sources."

Comments:

The phrase "If so" conditions the remainder of the sentence to be true only if the preceding sentence is factual, which has yet to be proven. The sweeping generalization is poorly founded, as has been previously discussed.

Page 15, lines 19-21 - "If so, fallout plutonium and alpha emitting contaminants must already be contributing to increased health risks and life shortening to ghe general public."

Comments:

Again the sentence is a conditional one based upon the preceding conditional sentence. By this time the author's conclusions have progressed considerably beyond the data presented in his references. The author has not presented any evidence to support this supposition.

Page 15, lines 21-26 - "Cigarette smoking causes increased risks of early coronaries, lung cancer, cancers at other sites, and other health effects[46], with about 15 years reduction in life expectancy for those who regularly smoke 2 packs of cigarettes per day or more (attributable to lung burdens of only about five picocuries of ^{210}Po in excess of that of nonsmokers).

Comments:

The author flatly states the parenthetical expression as if it were fact. There is complete disregard for any and all mechanisms of cancer induction other than that postulated by the author.

Page 15, line 28 to Page 16, line 3 - "Although these levels are only about 10 percent of the ^{210}Po organ burdens of heavy smokers, the effects may be correspondingly greater because the total population is exposed, and the inhalation exposures begin at birth."

Comments:

The author here states that the "effects (due to fallout) may be correspondingly greater) (than those due to cigarette smoke). The sentence implies as fact that health effects resulting from smoking are due to ^{210}Po organ burdens; this unverified assumption is the author's.

Page 16, lines 4-12 - "If the health risks attributable to fallout plutonium exceed 10 percent of the risks of heavy smoking, then inhalation exposure at 20 times fallout (the surface soil concentration of plutonium which corresponds to the interim soil standard adopted by the Colorado Board of Health in 1973) would give rise to organ burdens more than twice that of heavy smokers. Exposing children to such levels would be tantamount to their smoking four packs of cigarettes per day, beginning at birth. This estimate assumes, as I believe to be the case, that the inhaled, insoluble radioactive smoke particles give rise to the serious health effects of smoking."

Comments:

Such numbers relationships have little meaning when the basic assumptions are so poorly founded (see earlier comments). The author is building assumption upon assumption, which he acknowledged in lines 10-12.

Page 16, lines 13-21 - "For the estimate of organ burdens which may result from the inhalation of soil contaminants, it is common practice to attempt to determine the average surface soil concentrations, the applicable resuspension factors, inhalation exposure patterns, particle size distributions, lung retention, clearance and translocation patterns and rates, etc. The large cumulative errors and uncertainties in the prediction of theultimate organ burdens from long-term exposure to contaminated surface soils and urban dusts by such a long sequence

of complex processes serve to make this procedure an almost useless exercise."

Comments:

While we agree that there are uncertainties in the procedures which the author describes, we do not agree with the author's opinion that such calculations are useless exercises. Where uncertainties exist conservative factors are utilized, thereby overestimating any potential hazard. Because of this procedure, any errors are likely to be in the direction of conservatism.

Page 17, lines 5-8 - "For this reason, surface soils with one picocurie of plutonium per gram (the Colorado interim soil standard) should contain an estimated 10 to 100 pCi of plutonium per gram of insoluble soil particles of respirable size."

Comments:

The conclusion again is a supposition of the author for which no evidence is presented, as is suggested by the word "should." Furthermore, the evidence for and justification of the relationship between plutonium per gram of soil and plutonium per gram of insoluble soil particles of respirable size is not clear.

Page 17, lines 8-11 - "Such a soil level should lead to plutonium lung burdens of 5 to 50 picocuries by age 20, or 15 to 150 picocuries by age 60, with correspondingly higher concentrations in the lymph nodes, liver and bone."

Comments:

Again, the sentence is speculation ("should") by the author. It would be of interest to know how this conclusion was arrived at without using the surface soil concentration, applicable resuspension factors, inhalation exposure patterns, particle size distributions, etc., etc. which the author described on page 16, lines 14-21, as being "almost useless."

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Page 17, lines 11-13 - "Thus the Colorado/soil standard is hardly a safe or acceptable standard unless it can be shown that such levels of plutonium have no serious long term health effects."

Comments:

Assuming thepreceding assumptions to be fact, the author now states that, "Thus..." This conclusion is no more valid than the assumptions upon which it rests, for which no evidence is presented.

Page 17, line 20-25 - "Thus the high tumor risk for the hot $^{238}PuO_2$ particles(11) can be variously attributed to (a) the mobility of the smaller particles (b) the recoil ablation and/or dissolution rates which increase with specific activity and with surface area of hot particles and (c) the irradiation or larger numbers of cells with scattered protons (an effect that may be significant for very hot particles).

Comments:

The reference to "the hot $^{238}PuO_2$ particles(11)" is misleading. As was stated previously (see author's page 8, lines 9-11), the material used was neither $^{238}PuO_2$ nor in particulate form, but was considered to be "monomeric" ^{238}Pu. Furthermore, the author's definition of "hot...particles" here is not clear. It is the "monomeric" ^{238}Pu, with its consequent exposure of more "'target' epithelial cells" which reference 11 attributes as the cause of the higher tumor incidence. Recoil ablation, scattered protons, etc. are not discussed in reference 11.

Page 18, lines 1-4 - "For these reasons, the insoluble alpha emitting smoke particle, uranium oxide, thorium oxide and other alpha emitting particles of moderate to low specific activity may be expected to give rise to a higher tumor risk per alpha disintegration or for a given cumulative dose."

Comments:

It is not at all obvious what "these reasons" are, nor what they do or are supposed to support. If the "reasons" are what immediately precedes the statement, there is little upon which to base a judgment other than the assumptions upon which the author bases his speculations. If the "reasons" are the preceding 17 pages, these have been discussed in detail previously.

Page 18, lines 5-7 - "Similarly plutonium-239 in mixed fallout particles may be expected to produce more tumors per disintegration than is the case for pure $^{238}PuO_2$ and $^{239}PuO_2$."

Comments:

That this "may be expected to produce more tumors per disintegration" depends upon the validity of the assumptions of the author.

Page 18, lines 7-11 - "However although larger burdens of hot particles will be required for a given tumor risk, such risks can be expected to increase with both alpha specific activity and with particle surface area, and the effects should occur earlier for a given burden of smaller particles of higher specific activity."

Comments:

This sentence is very enigmatic, starting with "a given tumor risk" which is subsequently variable, ending with "a given burden" which earlier was larger (larger than what is not stated), and meanwhile varying specific activity, surface area and time. Nor is it clear that it is consistent with the two preceding sentences and earlier statements regarding the relative risk of high specific activity versus low specific activity particles.

Page 18, lines 12-15 - "The above considerations make it obvious that the present practice of averaging the alpha dose over the whole lung or some arbitrary fraction thereof[10-13] is a highly questionable and grossly misleading procedure at best."

Comments:

There is nothing at all "obvious" about the "above considerations," whether "above" refers to the same page or to the preceding 17 pages (see also comments on the author's page 13, lines 15-17). These "considerations" consist of a string of hypothetical assumptions which the author portrays as fact and upon which he bases his conclusions.