DEFENSE NUCLEAR AGENCY
WASHINGTON, D.C. 20305

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12 February 1975

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Mr. Theodore R. Mitchell
Attorney at Law
Executive Director, Micronesian Legal
Services Corporation
Post Office Box 826
Saipan, Mariana Islands 96950

Dear Ted:

I started to write a longer and more formal response to your letter forwarding the reply to the DEIS. I threw it away, and decided to forward the attached copy of the memo I wrote to my staff after reading your reply. This is rather informal, indeed, but I wanted you to know how seriously I view the problems we face, and I didn't see any excuse in "beating around the bush."

I do not fault you. I simply am frustrated and concerned. I'm still going to try my best, but we now are confronted with new decisions and with a time schedule that may be impossible.

Sincerely,

WARREN D. JOHNSON
Lieutenant General, USAF
Director

1 Incl
as

DIR 12 February 1975

MEMORANDUM FOR: DDOA

SUBJECT: Draft Environmental Impact Statement (DEIS)

1. We need to have AEC (ERDA) representatives and DOI representatives read this. Then we need a conference at the earliest possible date to discuss and determine steps to be taken. I don't want a big meeting, but we can't wait for letters!

2. If it is decided that we should follow all or a substantial part of Mitchell's recommendations, I believe the project faces a minimum of a one year delay. This needs to be assessed ASAP.

a. I cannot go to Congress for the funds we have now requested anticipating such a delay, unless I frankly tell them we face such a delay and the almost certain cost escalation. (This applies even if we reject the more extensive soil removal and the disposal of radioactive debris away from the atoll. If we accept these more stringent measures, the 100 million dollars cited by Ted Mitchell is probably much lower than the ultimate cost.) In today's fiscal environment, I do not believe we would have a prayer of getting any such funds. Possibly we could still convince Congress with the uncertain cost figures, but I seriously doubt we could obtain even the first increment (for the base camp) until we can nail down the probable total. We need to assess this ASAP.

b. If we reject all or part of Ted Mitchell's recommendations, what would his reaction be?

(1) Would we face likely litigation?

(2) If so, again I need to tell Congress and again I do not believe we'd get any money this year!

(3) Once we decide on a course of action, we need a meeting with Ted Mitchell and this has to be laid out to him - honestly and frankly. If we accept his recommendations and face delays and likely failure to obtain funds, then what? If we reject his recommendations, then what? I want to ask him point blank so that I can be accurate and complete in my statements to Congress. Because he was so late with his reply to the DEIS, there just may not be time to do all of this before we testify.

3. There may be an alternative course for dealing with Congress: tell them of the problems and ask for a reduced amount sufficient only to assure present facilities do not further decay. This should be the bare minimum to assure a smooth transition to later preparation of the base camp. (In addition to present O&M, I would "guess" this would be somewhere around four million dollars.)

a. How can this be done since the President's budget has gone in? (What procedures?) (I realize this will anger OMB and Congress but it may be the lesser of evils.) We have been honest with them believing (as Mitchell said to me in his telephone call) that his response to the DEIS wouldn't contain any surprises. It did!

4. Having read the various replies to the DEIS, it seems to me we have to either reject some outright, or the return of the people to Enewetak can never take place. This just doesn't seem logical, since there are places in the world where people have lived for centuries with radiation levels equal to or in excess of those which would remain at Enewetak. It seems to me the statistical risk should be considered. If the Dri-Enewetak want to return to the atoll, are they unwilling to accept any risk? Don't they face a possibly greater statistical health risk from other sources (like the suspected ruptured tubular pregnancy while we were there)? I believe if that question were put to them in language they could clearly understand, they would elect to accept the "prudent risks" we (ERDA and DNA) have assessed.

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a. However, if Ted Mitchell is correct in assuming we don't have enough facts to assure "prudent risks" we should acknowledge that lack, accept the delay, and attempt to find the facts.

b. If Ted Mitchell is wrong, can we convince him he is wrong? I sure am not the expert. We must rely on ERDA for this. It is not only their assigned role, - but they are the "experts."

c. All of us, including Ted Mitchell are playing "God" and we are damn poorly equipped to do so!! (Despite our best efforts.)

5. Please lay out a time table and keep me informed.

Warren D. Johnson

WARREN D. JOHNSON
Lieutenant General, USAF
Director

Copy furnished:
Comp

Comments

of the

People of Enewetak

Concerning

The Draft Environmental Impact Statement

CLEAN UP, REHABILITATION, RESETTLEMENT

OF

ENEWETAK ATOLL-MARSHALL ISLANDS

(DATED September 1974,
issued by the Defense
Nuclear Agency, Washington,
D. C. 20305)

Prepared by

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February 1, 1975

1. Introduction

The impetus for development of this program comes from the long-expressed desire of the People of Enewetak to return to their homeland. Although resigned to their nearly thirty year exile at Ujelang Atoll, they have never given up hope of returning to Enewetak, if but only if, it is radiologically safe for them to do so. They are aware of the substantial social and economic problems which necessarily attend the relocation and resettlement of their more than 400 persons, but the difficulty of assessing the risk from the extensive radioactivity present at the Atoll as a result of the nuclear weapons testing program there is by far the most troublesome. 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.

Their individual and collective desire to return to their ancestral homeland is difficult for Americans to fully appreciate. To them land is not a commodity, a thing apart, to be

brought or sold. In their culture the land and marine environment of the atoll are fully integrated with the human members of the society. It is an economic resource and more. Ownership and use of the land reflects and is inextricably linked to the social organization and to the culture as a whole. To be sure, their society has undergone and continuously is undergoing change as a result of forces both within and without, but the extraordinary significance of their being able to resettle to the atoll discovered by their ancestors remains constant.

Thus, the People of Enewetak are both the prime beneficiaries and the prime risk-takers in this resettlement program. And it is in the assessment and, if possible, elimination of the radiological health risk that they are the most dependent upon the United States government. 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. And throughout, the People of Enewetak will rely upon the responsible agencies of the United States government to do everything possible to assess and minimize the risk due to the residual radioactivity in the Enewetak biosphere. Nothing said in these comments, for example, should ever be taken as an assumption of risk by the people of Enewetak. When they left the Atoll in 1947 at the insistence of the United States government it was radiologically safe. That is the state in which it should be for their return.

Of course, it cannot ever be restored to that condition, but that must be the assumed objective in order that remedial measures can more likely fall within the safest possible limits, and so that on-going efforts will be made to continually add to the knowledge of radiological conditions at Enewetak and refine and improve both risk assessment and remedial measures as the various relevant sciences develop over the years.

Not only is the United States trustee for these people, but it has an especial humanitarian obligation to them because of the uniquely dangerous potential effects due to the use to which the trustee put the Atoll. 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.

A full measure of gratitude is due and hereby given, however, to the considerable efforts which the United States has made thus far. The planning for resettlement, the radiological survey, the planning for the clean-up, all represent a very large contribution to the ultimate success of the program. And we do not wish to dampen the enthusiasm and interest of the many persons in and out of the government who have given devoted effort thus far. The comments made here are offered in the spirit of cooperation, with the realization that they will be received in that same spirit.

2. Social and Economic Problems Associated with Resettlement

Further consideration of the social and economic problems associated with the resettlement must be given. This is perhaps

one of the weakest aspects of the DEIS as it now stands.

Attention is given to both short and long range economic planning (Vol. I, § 7, Vol. II, Tab D), but in consultation with the people themselves specific objectives and specific economic development possibilities must be found so that the shared aim of economic self-sufficiency can be achieved. We realize that with all the other aspects of this complex project demanding attention up to now, this was not intentionally underemphasized. But as the program moves into its clean-up phase more attention must be given to meeting the future economic needs of the people. This is especially true because since the writing of the DEIS it has become known that adverse radiological conditions in the northern part of the Atoll do not permit the rehabitation of Engebi islet and severely if not completely restrict the use of the northern islets for the foreseeable future.

The Enewetak Planning Council must continue to be relied upon to make the final value judgments upon one proposal or another and upon the development of the economy as a whole so that it will be consonant with their own capabilities and values, but one or more specialists should be engaged by the government and made available in an advisory capacity. They must be carefully selected both in terms of expertise in the field and suitability to this kind of cross-cultural task and to the maximum feasible extent the Planning Council should participate in the selection.

Resettlement to Enewetak Atoll from Ujelang will involve an unusual amount of stress for individual members of the group and for the group as a whole. Physical stress will, if all goes as

planned, be at a minimum, but we have in mind here the emotional stress upon the individual and the stress upon group processes. This matter is not addressed at all by the DEIS.

Ultimately, of course, it is for the people to manage the transition well and to adapt with their society intact, but experience with similar resettlement schemes is available and should be used to increase the likelihood of successful resettlement. The people themselves can benefit from greater awareness of the stresses they will experience and those outsiders involved in planning and working with them must have the same understanding.

Dr. Thayer Scudder of the California Institute of Technology, a recognized authority on the subject and an experienced consultant, should be considered for this assignment and if the Planning Council agrees, he should be engaged in this capacity. Dr. Scudder has taken a quick look at the DEIS at our request. His comments attached hereto as Appendix I provide valuable insights and his contribution to planning and execution of the program would appear to be necessary. (The article which he enclosed is also useful. It is "The Impact of Human Activities on the Physical and Social Environments: New Directions in Anthropological Ecology," by E. Montgomery, J. W. Bennett and T. Scudder, 2 Annual Review of Anthropology 1973.)

Participation of another anthropologist versed in Marshallese culture is also in order, to assist both the Enewetak people and the outsiders involved in the program. Working in conjunction with someone like Dr. Scudder, the total contribution would be invaluable. Dr. Robert Kiste at the University of Minnesota has been

consulted by the governmental planners and meets these requirements exceptionally well.

Short of involving so many advisors and planners that decesions and action are unduly impeded, it is essential that those representing all the relevant disciplines work together as a group with the Enewetak Planning Council and the governmental decision-makers. To some extent this is what has been done during planning to date, but for the remainder of the program, the relevant disciplines should be identified as such, appropriate representatives engaged and organized into a more or less formal advisory council.

3. Radiological Considerations

3.1. The Radiological Survey

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 danger 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 qualifications 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.

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. As we shall discuss more fully below, more information is needed about the presence of hot particles. The long range effects of Strontium-90 and Cesium-137 and other nuclides in the food web cannot be known without experimental planting. (DEIS Vol. II, Tab B, p. 29.) These are only examples. 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. 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. And in so doing it is highly probable that important contributions to the development of greater understanding of radioactivity

and its effects will result, to the benefit of the United States and the world at large.

3.2. The Hot Particle Problem

It is with the kind assistance of Drs. E. A. Martell, Donald P. Geesaman, Arthur R. Tamplin and Thomas B. Cochran that we derive our comments here concerning this unique radiological hazard. Drs. Tamplin and Cochran submitted formal comments upon this DEIS to the Defense Nuclear Agency under date of September 24, 1974, and we fully accept and endorse what they have said there. Their observations and concerns are entirely consistent with those of Martell and Geesaman, expressed to us in personal communications.

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.) To further emphasize our grave concern about this problem, we attach comments and materials provided to us by Dr. Donald P. Geesaman as Appendix III. We subscribe fully to the views they express and we insist that they be dealt with fully in the final impact statement.

It is beyond question that the presence of Plutonium-239, Americium-241 and perhaps other alpha-emitting radionuclides at Enewetak Atoll constitutes one of the most serious health risks for the returning population. It is highly likely that inhalation of very small amounts of plutonium gives rise to a high risk of lung cancer. 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. See Appendices II and III. The DEIS deals only with inhalation risk, yet Americium is known to present a risk for the liver, spleen and bone of man through take-up from the gastrointestinal tract. (Martell, Personal Communication.)

Concerning the adequacy of the radiological survey with respect to internal alpha emitters, Dr. Martell had this to say:

> It is noted that the survey results for the Enewetak Lagoon sediments show an average of 463 mCi $^{239+240}Pu/km^2$, 172 mCi $^{241}Am/km^2$ and 586 mCi $^{90}Sr/km^2$ (Table 3-11, p 3-75, DEIS Volume I). In addition, the ^{241}Am concentrations range up to 8.2 pCi/g averaged over the top 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). Due to further radioactive decay of ^{241}Pu, the ^{241}Am activity concentrations can be expected to double over the next 50 years. In addition, densely vegetated soils on each island show the highest radioactivity concentrations.
>
> The DEIS limits consideration of $^{239+240}Pu$ to inhalation risks. However significant uptake of Pu from the gastrointestinal tract has been observed in young mammals and similar uptake may occur in young children. 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. 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 heald consequences to future atoll inhabitants. (Personal Communication.)

Dr. Geesaman independently identifies the same inadequacy in the DEIS and also finds a need for further study of the mechanisms by which plutonium contamination in the soil may find its way into the body.

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 distrubance, 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 implicit 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. (Personal Communication.)

Each of the questions raised here and in the related appendices must be addressed fully and carefully prior to resettlement of the people of Enewetak Atoll.

3.3 Plutonium Soil Standards

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 follcwing critique of the standards adopted by the AEC Task Group for Enewetak:

> 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). It is noteworthy that the AEC has not established that this standard is unduly conservative and 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.

I note that the DEIS recommends no remedial action for soils containing < 40 pCi or < 88 dpm Pu/g, averaged over the top 15 cm depth. This is much more than 44 times the Colorado interim standard (2 dpm per g in the top 1 cm) because 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. Thus the recommended standard for Enewetak is about 100 to several hundred times that adopted in Colorado.

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. 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. The typical burden of insoluble particles of respirable size (< 5.0 Um diameter) is about one gram in human adults. For this reason I would recommend that surface soils should not exceed about 1 pCi of $^{239}PuO_2$ and other insoluble alpha emitting particles per gram of insoluble particulates of respirable size in the airborne dust resulting from the disturbance and resuspension of surface soils. On this basis even the Colorado standard may give rise to excessive organ burdens.

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.)

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.

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. The range between 40 and 400 pCi/g is a wide one indeed and if 40 is too high, then to make decisions on a "case-by-case" basis within that range is to have no standard at all.

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.)

At any and all of these hearings, every effort should be made to elicit the widest possible range of information and opinion bearing upon the question. Once such standards are set, they should govern the planning and cleanup activities at Enewetak.

3.4 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, §§5.3.3.3 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 the environs of the Atoll. Ocean dumping, according the DEIS (Vol. I, § 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, § 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 yards. (Vol. I § 5.5.2.) If the soil standards are lowered as they should be, that volume will increase.

It is suggested in the DEIS that cost, legal, political and technical problems aside, the removal of contaminated soil and its replacement with clean soil may not "assure radiological safety" and may present "serious ecological damage of unknown proportions." (Vol. I, § 5.3.3.3.) We fully favor this conservative approach to these problems (just as we do when the question is one which may reduce the program cost, i.e., high soil contamination standards), 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. These studies should be commissioned immediately and prosecuted with all deliberate speed. In the meantime, complete soil removal and replacement should be adopted as the prime objective.

In addition, maximum effort must be made to overcome technical, legal and political impediments to off-Atoll disposal of contaminated soil.

3.5 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.

3.6. 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.

3.7. 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 Enewetak 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. It too must deal with the health effects of hot particles and all forms of low level radiation, with emphasis on internal emitters.

3.8. Unknown Concerns

We have tried to identify all the radiological needs of this program which require further attention, all with the ultimate safety of the People of Enewetak in mind, but we cannot be certain that we have done a complete job. Hence, we call upon the United States government to continue to assume the important responsibility of giving the best and most careful attention to these matters for the long range future.

4. Considerations Related to Cost

Funding requests for the initial phase of this program have been previously presented to the United States Congress. They did not receive very favorable or sympathetic consideration, to put it mildly, by the members of the House Armed Services and Appropriations Committees. In general, the objections related to the great cost of the entire program and evidenced a reluctance to commit the United States government to the first phase of a

program, the ultimate cost of which would be in the neighborhood of $49,000,000. Hence, the request was disapproved. In the House and Senate Interior committees to which the rehabilitation and resettlement phases were referred in a legislative package separate from the cleanup, sympathetic and favorable action was taken and $12,000,000 was authorized.

Notably absent from the presentations made to the Congress and from the inquiries of the Congressmen themselves was realization of the enormous benefit which (in the view of the United States) has been derived from the use of Enewetak Atoll for nuclear testing and related national security activities. In the Armed Services hearings, the total projected cost of this program was divided by the number of Enewetak people and the suggestion made that perhaps the money should simply be given to the people.

We do not have accurate figures for the total cost of the atomic energy program, the nuclear weapons testing program, nor for the amount of money actually spent for programs at Enewetak. But judging by figures we have seen (for example, Congress And The Nation, Vol. I, p. 262, Congressional Quarterly Service, 1965) indicate that the cost was on the order of several billions of dollars in the AEC budget, and that says nothing about the undoubtedly large sums contained in one or more places in the Defense budget. We will suggest a figure of, say, $50 billion for the sake of discussion. That represents the agreed minimum value to the benefit to the United States of the same activities, the effects of which must now be remedied. Beyond the dollar

value, the United States must assign a value to the benefit to national security of the testing program, however debatable that benefit may be in and of itself.

The cost of the direct benefits in this program for the Enewetak people, such as housing, community development, etc., are a very small fraction of the total, about $5,000,000. And even that portion of the total funding is directly attributable to their forced removal by the United States to make way for the testing program.

And as we have said before, the United States undertook trusteeship of the Micronesian Islands of its own free will (without consent of the Micronesians) and put Enewetak Atoll, the property of the trust, to its own use for the very nuclear testing which deposited the radioactivity.

This is the only perspective by which to consider and decide upon the outside cost limits of this program. The costs of the radiological and engineering cleanup of the Atoll are properly to be considered ordinary and necessary costs of the testing program. Indeed, the cleanup should have been planned from the beginning and funded and done at the end of the testing program about 1958.

The Enewetak People do not want money in any amount, they want and are entitled to their land, in safe and habitable condition.

In the presentation of future requests to the United States Congress, this general approach should be taken and the leadership of the people themselves should be called to testify.

"Case 3", outlined in Section 5.4.3, Vol. I of the DEIS, is offered as the preferred plan for cleanup and resettlement of the

Atoll. Essentially, it represents a compromise of cost, radiological and other factors, which will be far short of the theoretically ideal "Case 5". (Vol. I, § 5.4.5). Exclusive of contaminated soil and scrap disposal costs, the cleanup cost for Case 3 is $35.5 million and for Case 5 it is $81.6 million. Comparative soil disposal cost estimates are $7 million for Case 3 and $92.2 for Case 5.

We appreciate the political and practical realities of seeking sums on the order of $100 million from the United States Congress in these times of grave concern about the economy, but given the rationable stated above, it is Case 5 for which funding should be sought and for which funding should be given.

Finally, quite apart from any cost-benefit analysis of the nuclear testing program, as a result of a recent decision of the United States Court of Appeals for the Ninth Circuit (People of Saipan, etc. v. U.S. Dept. of the Interior, etc., 502 F.2d 90 (1974)), the obligations imposed by the Trusteeship Agreement under which the United States administers the Micronesian Islands has become legally binding and enforceable. Under the terms of Article 6 of the Trusteeship Agreement, the United States is required to "promote the economic advancement and self-sufficiency" of the Enewetak People; to "protect [them] against the loss of their lands and resources"; to "promote the social advancement" of the Micronesians; and to "protect [their] health." These are the express obligations. Beyond that, like any trustee, the United States bears implied duties to protect and promote the best interests of the beneficiary in every way.

Litigation by the beneficiary against the trustee to enforce these obligations would unseemly and costly. Every United States official involved, including members of the Congress, should freely and willingly undertake to fulfill them by planning, funding and conducting a cleanup, rehabilitation and resettlement program for the Enewetak People which approximates the ideal.

5. Conclusion

We have made a number of recommendations in the course of these comments to which we hope the program sponsors will give consideration in the preparation of the final impact statement. The recommendations relating to assessment of the radiological risk, if accepted, may or may not result in delay for the project as now planned. We hope not, but certainly the further study required and the development of soil, air and food contamination standards for plutonium may have a direct affect upon the initial cleanup phase. We urge the Defense Nuclear Agency to proceed with funding requests and planning for the base camp and to seek commitments from the United States Congress for the estimated cost of the program as a whole based on the "Case 5" projections. 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.

It bears repeating here that we are mindful of the immense amount of time, effort and money which has been devoted to development of this program to date by many officials in the Defense Nuclear Agency, the Atomic Energy Commission, the Department of

the Interior and its Trust Territory administration, to mention only the principal agencies. We are deeply grateful the professional and humanitarian commitment of all of these people and special appreciation is due Lt. Gen. Warren D. Johnson, Director, Defense Nuclear Agency for all that he has done and will continue to do.

Respectfully submitted by

Theodore R. Mitchell, Counsel
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DIVISION OF THE HUMANITIES
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October 29, 1974

Mr. Theodore R. Mitchell
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Dear Ted,

I have now read through the three volumes of the Draft Environmental Impact Statement dealing with the Clean Up, Rehabilitation, Resettlement of Enewetak Atoll-Marshall Islands. One thing that you have going for you is that the people of Enewetak wish to return home, and have been pressing for this return for years. Many of the stresses associated with the type of compulsory relocation that I have studied including the undermining of local leadership, are simply not present although I would suspect a carry-over from the past.

Another favorable factor has been the willingness of everyone involved to date (a) to listen to the local people (at least through their council of 12) and (b) to take into consideration their wishes in planning their return. On the other hand, any kind of settlement scheme involves stress to the settlers and as you note in your letter of October 11, little attention has been paid to the potential impacts of this stress.

Because my predictive theory deals primarily with compulsory relocation at the time of forced removal, rather than 28 years later!, I will have to cast the net wider (which of course is a much more risky business) and deal with settlement schemes in general, compulsory resettlement being an extreme example of this more general category. As I am sure you are well aware, the history of settlement schemes throughout the world is a grim one -- with probably over 90% being unsuccessful from the point of view of both settlers and settlement authorities. It is hard to imagine a more difficult task that creating from scratch new communities, which are both socially and economically viable. Though the situation is more favorable when these people are willing participants, in the Enewetak case no settler selection is possible since everyone who wishes to return -- young and old, conservative and progressive, hard working and lazy, must

APPENDIX I

be accommodated. In commenting on the Impact Statement I wish to discuss in sequence (1) Housing (2) Social Services (3) the Economic System and (4) Social Factors associated with settlement. Let me emphasize right now that (1) and (2) are by far the easiest to handle -- and (1) and (2) represent the greatest strengths of the Impact Statement. But while it is relatively easy to provide improved housing and social services, it is much harder to create viable land and water use systems -- indeed it is here that most settlement schemes fail. And it is much harder to handle the social factors associated with settlement as well as the institutional factors dealing with the inter-relationships between settler institutions and those of the agencies involved in their future -- all of which must be viewed as part of a single (and very complex) social system.

(1) Housing. Though Holmes and Narver should be complimented on the extent to which they have taken into consideration the stated desires of the Enewetak people and their system of land tenure in proposing house types, as I understand the situation, the people have yet to live in houses of the type proposed. If so, we must distinguish between what they think they want and what subsequently they decide they want after living in the new houses for a complete year. I strongly urge that a small number of pilot houses be built for at least some of those involved in the initial cleanup operation, so that the people will have a chance to assess their strengths and weaknesses -- to work the bugs out of them, so to speak, before the main construction program tends to rigidify their family structure and social organization in concrete for years to come. One thing that planners and architects tend to forget when providing housing in permanent materials, is that discrete structures in non-permanent materials provide more flexibility. Before pouring concrete one should try to anticipate some of the implications which inevitably will arise (and which will have an impact on the peoples' lives) and make corrections where desirable. Problems of maintenance also need to be anticipated in advance and local people trained to maintain their own structures.

A major problem associated with many settlement schemes relates to provision and maintenance of adequate water supplies. Though the plans incorporated in the reports look good to me, I just want to mention this general difficulty for the record, and to emphasize the need to provide the simpliest facilities possible in terms of (1) peoples' needs and (2) their hopes -- with the second factor being far less important than the first. I have seen too many projects where people, after several years, must fall back on inadequate local water supplies simply because government-provided facilities are inadequate to start

with, or because costs for their maintenance are not provided, or because local people are not trained to properly use and maintain them. While I was very favorably impressed by the thinking on water supplies within the reports, I wonder if enough thought has gone into problems concerning their long term maintenance.

(2) Social Services. While impressed again by the thoroughness with which the desires of the local people have been taken into consideration, it is hard to comment on social services without knowing more about the breakdown of the population itself. None of the reports tell us much about the current educational and literacy status of the people, and about their goals for self and children -- other than to return to Enewetak. Though obviously their expectations for imported items has gone up during their 28 years of exile, what about their occupational desires, and especially the occupational desires of the younger people? One thing that bothered me about the reports is that while four room schools are proposed for both the driEnewetak and driEnjebi, nothing is written about the type of education system proposed for these schools and the type of teachers to be recruited. Let me generalize this comment to all types of service personnel, since I was also concerned about the lack of attention paid, under agriculture and fishing, to extension personnel, let alone to the relationship of the different types of service personnel to each other. I am raising here the fundamental question as to what different categories of people will be willing to do, occupationally, once they return and how best to facilitate their future economic and social independence and development.

(3) Viable Land and Water Use Systems. The Master Plan was based on the assumption that all the islands in the atoll could be used for subsistence and cash crop agriculture -- with a total available acreage of approximately 1000. As a result, however, of the AEC Task Force recommendations, this total has been cut to a maximum of 722 usable acres for a current population of over 400 people. Bearing in mind the poor quality of the soil and the rapid rate of population increase, it seems to me absolutely essential that the people retain access to Ujelang Atoll. Even then the available land area on a per capita basis is considerably less than that utilized by the people prior to their first relocation. The situation is worrisome and points up the need (a) to obtain the best possible seed for coconuts for both subsistence and cash crops purposes, with the search bearing in mind the major advances in productivity that have occurred on research stations in the Ivory Coast and in the Phillipines. (b) to push mariculture hard while keeping the means of production strictly in local hands so as to spread employment. Equipment (outboards for example) should be

standarized and kept as simple as possible (seagull type engines vs Johnstones). A number of interesting case histories come to mind here including the lobster cannery which is the principle employer among the several hundred islanders on Tristan da Cunha in mid-Atlantic who were moved from their home after a volvanic eruption in 1961 and returned there later in the 1960s. (c) provide a first rate unified extension service (d) ensure a dependable and sufficient water transport service and pier and port facilities to connect Enewetak to neighboring islands (including Ujeland and the relevant market centers). (e) actively attempt to diversify the economy, always bearing in mind local desires, interests, needs and expectations. Especially attractive is the suggestion that the function of the Eniwetok Marine Biological Laboratory (which apparently will continue under AEC sponsorship) be expanded to include technical assistance to the people. Couple this with the possibility of a Community College for the Marshalls which would use the facilities already present on Enewetak, and one has one way of providing a unified extension service while possibly broadening the economic base of the people. Such possibilities however need be carefully evaluated concerning the extent to which the people will actually be involved and the extent to which they will actually profit. This caution applies even more to the development of a tourist industry which even at best is a mixed blessing on small islands.

It seems to me that the future of the people of Enewetak depends on the extent to which the people regain their independence and the extent to which their atoll can become economically self-sufficient. It is my impression that the authors of the Defense Nuclear Agency report do not understand how much recommended Case 3 alters the assumptions on which the original Master Plan was based. This alteration also has major implications for social factors as I hope to show below.

(4) Social Implications of Settlement. Depending on whether they are driEnjebi or drīEnewetak, the present move home will represent the fifth or sixth time that the people of Enewetak have been moved since 1944. Since the original move was compulsory, and hence falls within the scope of my own research, I suspect that it was accompanied by a great deal of stress, which, for analytical purposes, can be divided into psychological, physiological and socio-cultural stress. According to my own model of how people respond to compulsory relocation, this stress (or transition) period does not come to an end until (a) the people once again get back on their feet economically or at least reach the position that they held before relocation, and (b) feel at home in their new habitat. Since neither of these factors applies to the people of Enewetak after nearly 28 years, I would suspect that the older people (that is, those who were old enough to remember the trauma

associated with the original moves) are still under stress. What this means, however, is hard to access at a distance since my theory applies primarily to the months and years immediately preceding and following forced removal. All I can say is that the mental and physical health of the people should be carefully assessed before their shift home and before they are involved in major new ventures -- ventures which would require radical changes in their activities and life style. I say this since the theory predicts that populations undergoing forced removal behave as if a social system was a closed system; that is they change no more than they have to in order to continue doing what they did in the past and the changes which occur are incremental rather than sudden. The insistence of the people through out all these years that they be allowed to return "home" is consistent with the theory here. But once the people get home and the euphoria of having "won" fades, what then? What can be expected when they begin to settle down with three times the number of people on an idealized homeland which can be only partially utilized. With these questions in mind, I would like now to consider three points.

(1) It is very important to recall that approximately 80% of the population is under 30 years of age according to the population figures. In other words, the large majority of the people will either have no memory at all or only a vague memory of life on Enewetak. It is this age bracket which strikes me as a major unknown. To what extent do the Council of 12 really speak for them? To what extent do they wish to return to the life style of their parents and grandparents? I can not answer this question at a distance, in large part because the Enewetak population within the three volume Impact Statement is treated as if it was homogeneous. But I doubt very much that such is the case, a doubt that is reinforced by the odd statement in the reports -- for example, "A number of people have been exposed to education away from Enewetak and have developed strong tastes for imported foods and other luxuries" and the people have "achieved a good understanding of the behavior and values of Americans, and several have distinguished themselves in government and mission schools." In assessing the impacts of the return on the people I suspect we need at least differentiate from the very beginning between the older 20% and the remainder.

(2) Compulsory resettlement projects always run the risk of the relocatees developing a dependency relationship with the relocating authorities. I would suspect that a strong sense of dependency characterizes the older people from Enewetak and that this will continue during the next decade. Even if the dependency does not already exist, most of the people are going to be dependent on outsiders for years to come simply because it will take at least seven years to

prepare lands for planting, to plant them and then to harvest the resulting tree crops. Should the cash cropping of coconuts proceed according to schedule only then will the people begin receiving what Holmes and Narver hope will be an annual cash income of perhaps $40,000 or slightly less than $100 per capita in terms of present population. In the meantime the people will have to use their trust fund (which currently produces $60,000 per annum in income or somewhat less than $150 per capita) to provide for their external needs and to depend on the U.S. government and other donors. Reliance on both the trust fund and on further external assistance continues and increases the risk of a dependency relationship which can be expected to make subsequent development more difficult. Already the people have acquired a taste for outside staples which apparently on occasion can make up as much as 80% of the diet. These include rice, flour, sugar, tea, canned meat, and fish; in other words the usual foods that low income people desire after they come into closer contact with the outside world. So we have the combined problems of rising expectations and dependency, both of which have to be taken into consideration in planning subsequent development for the atoll. Neither makes the task easy. Once the euphoria of regaining the homeland passes, disallusionment may well come, along with new demands on the United States (which of course continues to bear the responsibility for the original move) to provide for the people. Looking to the future, very careful planning and plan execution will be required if the people are not to continue as wards of the government.

(3) Another potential problem concerns future relationships between driEnjebi and driEnewetak simply because the former cannot occupy their former island or indeed their traditional section of the atoll. Rather they will find themselves relocated quite close to their neighbors. Although I note that distinctions between the two populations have been reduced to the extent that the 12 man council is now elected at large from all the people, and that the large majority of the population have been brought up as members of a "single community," nonetheless the present plan to relocate the driEnjebi on Medren and Japtan puts them in the relationships of 'relocatees' to the driEnewetak "hosts" which raises the possibility of the type of deteriorating relationships which all too frequently characterizes hosts and relocatees in other settlement schemes, especially where the two communities find themselves in competition for scarce resources, resources to which the hosts traditionally held claims.

At this point there is little more that I can say without further knowledge. In conclusion, however, let me say that there are sufficient social and economic problems connected with the entire relocation effort to justify

a well-thought out, longterm program for "monitoring" events from this day forward -- in hopes of anticipating problems before they arise and easing those that inevitably do arise. If I can be of further assistance alone such lines, please let me know.

With best wishes.

Yours sincerely,

Thayer Scudder
Professor of Anthropology

gsh
enclosure

P.S. I enclose an article which summarizes the impacts of compulsory relocation of people moved in connection with big dam projects which may be of some use to you. No, I have not seen Tobin's thesis nor do I have easy access to it. If you can get me a copy I would much appreciate it.

Basic Considerations in the Assessment of the Cancer Risks and Standards for Internal Alpha Emitters

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January 10, 1975

(Statement presented at the public hearings on plutonium standards sponsored by the United States Environmental Protection Agency, Denver, Colorado, January 10, 1975.)

APPENDIX II

1. Introduction: The adequacy of the biomedical basis of standards for occupational and public exposure to plutonium and other internal alpha emitters have been widely discussed[1-5] and seriously questioned[6-8]. The serious uncertainties in the cancer risks attributable to internal alpha emitters must be resolved before we are irretrievably committed to a nuclear energy program. This is a matter of immediate concern in the western suburbs of Denver due to plutonium and americium contamination of surface soils in public areas around the Rocky Flats Plutonium Plant[9]. Many other localities are similarly affected by transuranium element contamination and its attendant cancer risks.

Recent controversy regarding the adequacy of plutonium standards has centered on several aspects of the problem of the cancer risks attributable to inhaled plutonium oxide particles, including such questions as which organ and how small a tissue volume constitutes the "critical" organ (i.e., that experiencing the highest cancer risk), and whether the average alpha radiation dose to the critical organ or the tumor risk attributed to a given number of individual hot plutonium oxide particles provides the best guidance for the assessment of risks and standards for plutonium. Geesaman[6] has discussed possible mechanisms of cancer induction by hot particles and concludes that the tumorigenic risk may be as high as 1/2000 per particle for submicron particles of plutonium oxide. A recent examination of hot particle risks by Tamplin and Cochran[8], based largely on the Geesaman study, led these authors to recommend that the occupational MPLB (maximum permissible lung burden) be reduced by a factor of 115,000, to a value of 0.14 pCi. A recent study[10] was carried out by Bair, Richmond and Wachholz at the request of the U.S. Atomic Energy Commission with the specific objective of providing an updated review of the evidence bearing on the problem of uniform vs

nonuniform alpha radiation dose distribution in the lung. The authors of this study take exception to the conclusions and recommendations of Geesaman, Tamplin and Cochran[6,8] and conclude that

> "the nonuniform dose distribution of plutonium particles in the lung is not more hazardous and may be less hazardous than if the plutonium were uniformly distributed and that the mean dose lung model is a radiobiologically sound basis for establishment of plutonium standards."

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] 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.

On the basis of a brief review of the known effects of alpha interactions with cells (below) it will become evident that alpha radiation induced cancer in mammals and man must be brought about by subjecting a large number of living cells to a limited number of alpha interactions. Thus, in principle, the highest risk would be associated with a uniform distribution of the alpha dose, in accordance with the conclusion of Bair et al. However, in fact, we are almost always concerned with a highly irregular tissue distribution of alpha emitting particles. For hot particles, the tumor incidence must be due to the low dose irradiation of a large number of cells by a very small fraction of the hot particle burden. And for long term exposures, unacceptably high tumor risks appear to be associated with picocurie burdens of internal alpha emitters. This serious possibility calls for a drastic downward revision of permissible

exposure standards for inhaled plutonium. It also is possible that the critical health effects for inhaled 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.

2. Tumor Production: The interactions of various types of radiation with living cells and their mutagenic effects have been widely investigated, with results which have been reviewed and summarized by Lea[15], Muller[16] and others. When alphas interact with the chromosome or its genes in the nucleus of a 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. Thus, with regard to the production of irreversible structural changes in cells the relative biological effectiveness of alpha radiation, compared to X-rays and γ-rays, increases markedly at lower dose rates and over longer periods of exposure.

For alpha interactions with cell nuclei, most of the structural changes are lethal and lead to the mitotic death of the cell at the next or subsequent cell division[17,18]. However, as Lea[15] and others have pointed out, some cell nuclei experience only minor structural changes

(chromosome inversions, duplications, translocations, deletions, etc.) and remain viable. However, although only a very small fraction of alpha interactions give rise to viable mutated cells, these survive to proliferate, whereas cells which suffer lethal changes are eliminated from the cell population. Thus in the case of long-term exposure of tissue to internal alpha emitters at low dose rates there is a cumulative increase in the population of cells which have survived one or more chromosome structural changes. However it is equally obvious that a cell whose nucleus is subjected to repeated alpha interactions within the mean life of the cell has only a negligible chance of survival.

It is likely that the production of a radiation-induced tumor begins with the formation of a single malignant cell characterized by a combination of two or more chromosome changes and/or gene mutations. 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. This is consistent with the two-mutation and multiple-mutation theories of cancer[20,21] based on the age distribution of cancer in man. 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 $R^2t^2(t/\tau_c)$, where R is the alpha dose rate, t is the time of exposure, and τ_c is the mean life of the normal cell and singly mutated cell. The term (t/τ_c) represents the influence of the growth of the clone of the singly-mutated cell on the long-term risk.

This tumor risk relationship makes it abundantly clear that a linear

extrapolation to low dose rates is not only not conservative for alpha radiation induced tumors, but rather that there is a marked inverse dose-rate vs risk relationship. 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 $^{238}PuO_2$ 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 activity 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.

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 $R^2t^2(t/\tau_c)$, 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, τ_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].

3. <u>"Hot" PuO_2 Particle Risks</u>: If the above tentative conclusions are correct, then the same considerations must apply in the assessment of tumor risks for hot particles. In this connection a preliminary consideration of the influence of specific alpha activity and particle size of the hot alpha emitting particles is in order.

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. Such a dramatic difference in the chemical behavior

of two isotopes of plutonium is seriously inconsistent with the negligible influence of isotope effects on the chemical kinetics of heavy elements. Thus it seems necessary to explain this apparent solubility difference on physical grounds. The specific activity of the $^{238}PuO_2$ particles (~80% $^{236}PuO_2$ and ~20% $^{239}PuO_2$) was about 220 times that of $^{239}PuO_2$. 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. 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 ~10^4 atoms. The poorer structural integrity of the $^{238}PuO_2$ particles may give rise to an increase in the size range of the ejected fragments. 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]. Also, such small ablation fragments may exhibit a much higher mobility in tissue than that of 0.1 to 1.0 μm diameter, the size range of particles used in most animal inhalation experiments. This greater mobility for very small ablation fragments in tissue may explain the observed more rapid rate of translocation for $^{238}PuO_2$ than for $^{239}PuO_2$ from the lung to the liver and bone[32,33].

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. This process also would proceed at a rate proportional to specific activity and to particle surface area. In this

case the dissolved plutonium would diffuse away from the hot particles. 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. 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.

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. In this case the inhaled particles involve smaller particles and a correspondingly larger surface area. The observed more rapid rate of translocation to other organs can be attributed variously to the higher mobility of the smaller particles, or to the higher rate of surface ablation (or dissolution) for the increased surface area, or both. The higher tumor incidence can be attributed to the fact that the greater mobility and wider redistribution of the $^{238}PuO_2$ microspheres and their breakdown products subject a much larger number of cells to a limited number of alpha interactions.

The correctness of the above interpretation is reinforced by the results of the Los Alamos ceramic sphere experiments reported by Richmond et al.[12,13] and further discussed by Bair et al.[10]. In these experiments 2000 zirconium oxide microspheres of 10 μm diameter, each set containing a specified amount of plutonium, were injected into the lungs of groups of experimental animals. The total plutonium per microsphere ranged from 0.07 to 1.6 pCi of ^{239}Pu and from 4.3 to 59.4 pCi of ^{238}Pu, with identical activity for each of the 2000 microspheres in each of eight animal exposure groups of 70 animals per group. The local dose rate,

averaged over the small tissue volume within 40 μm from the surface of the ceramic microspheres is ~17,000 rads per year for the 0.07 pCi microspheres, or ~200,000 alpha disintegrations per year within each microgram of irradiated tissue. The dose rate is correspondingly higher around the microspheres of greater activity. Less than one milligram of tissue, only one millionth of the lung, is subjected to these massive radiation doses.

The limited biological response obtained in these experiments is consistent with expectations based on Barendsen's results[17,18]; the small population of cells within the alpha range around the microspheres experience so many alpha interactions that they all receive chromosome structural changes that result in their mitotic death. The 10 μm diameter microspheres are immobile in tissue. Also their specific alpha activity is so low compared to pure PuO_2 that their surface recoil ablation and dissolution rates are negligibly low. Thus in these experiments there is no large population of cells which are subjected to a limited number of alpha interactions, as is the case for Sanders crushed $^{238}PuO_2$ microsphere experiments[11]. Richmond and Voelz[12] observed only two lung tumors (at 9.5 months and 12 months in animals exposed to 2000 ceramic microspheres of 0.42 pCi ^{239}Pu per microsphere) for a total of ~10^6 hot particles. 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. Such protons have energies of about 100 KeV and a range about 4 times that of the alpha particle. Thus these secondary protons irradiate 63 times as many lung cells at correspondingly much lower doses. It is unlikely that the two tumors observed in these experiments can be attributed to X-rays or γ-rays from plutonium for reasons discussed by Warren and Gates[35,36].

4. Critical Health Effects: It is widely recognized that inhaled insoluble alpha emitting particles deposited in the lung are, in part, translocated via the phagocytic action of macrophages to the lymph nodes and to other sites in the reticuloendothelial system, and also via blood leucocytes to the liver, spleen and bone marrow. Recent experiments with inhaled plutonium make it evident that the pattern and rate of translocation of plutonium from the lung to other sites is highly dependent on particle size and specific activity, with more rapid transport of the smaller and more active particles. Thus, it is far from obvious whether the lung, lymph nodes, liver, bone or other organ, or fraction thereof, should be taken as the critical organ or critical tissue site.

It has long been known that those tissues in which there is more active cell division suffer the earliest and most 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. 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].

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].

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. Elkeles[41-43] has observed anomalously high concentrations of alpha activity at the calcified plaque sites. 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. 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]. However atherosclerotic plaques have been directly induced in human arteries by intensive irradiation with X-rays and radium[45]. There is a high incidence of early coronaries among cigarette smokers, with a mortality rate for males who smoke two packs or more daily that is 2 to 2.5 times that of non-smokers but at a mean age of death some 10 to 16 years earlier.[46] For all these reasons it is proposed that inhaled insoluble alpha emitting smoke particles are very likely to be the mutagenic agent which gives rise to atherosclerosis in cigarette smokers. 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. Attention should be addressed to industrial and combustion product aerosols which contain uranium oxide, thorium oxide and lead-210, as well as to plutonium oxide from nuclear industry, nuclear accidents and fallout from atmospheric nuclear tests.

The first and most obvious place to look for such effects is among past and present plutonium workers. 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]. 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 most of 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 but 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. Which 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]. 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. 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. 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 plutonium, or fractions thereof, is totally unjustified. 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.

5. Discussion: The published evidence, reviewed above, clearly indicates that a linear extrapolation to lower doses and dose rates is not conservative for internal alpha emitters. The initial effects of alpha interactions with cell chromosomes are irreversible and thus will vary linearly with alpha dose rate. 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. Therefore the tumor incidence per alpha disintegration must increase with decreasing dose rate. 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 increases.

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.

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 which insoluble 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.

It also is 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.

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. 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. If so, fallout plutonium and alpha emitting contaminants must already be contributing to increased health risks and life shortening to the general public. 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). Fallout levels from past atmospheric nuclear tests have given rise to plutonium organ burdens of ~0.5 pCi/kg of lung tissue and ~0.7 pCi/kg of liver tissue in the general public[56]. 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.

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.

For the estimation 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 the ultimate 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 exercice. There is a more direct approach which sould give more reliable estimates. Lewis et al[57] show that the adult lung burden of nitric acid-insoluble particles increases almost linearly with age, with about 1.5 grams per kilogram of lung tissue at age 60. It seems reasonable to assume that individuals chronically exposed to soil dust and urban dusts will acquire just such burdens of the insoluble constituents in the respirable size fraction of dust particles (i.e., particles less than ~5 μm diameter). It should be noted that PuO_2 particles are highly insoluble and friable.

Experiments in the Rocky Flats area also have shown that about one-third of the airborne plutonium which has been resuspended from soil surfaces by wind action falls within the respirable particle size range. However only a very small fraction of the bulk surface soil is made up of insoluble particles of respirable size. 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. 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. Thus the Colorado interim 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.

There are, of course, a number of considerations which make it inappropriate to equate the effects of a given burden of low specific activity alpha emitting cigarette smoke particles with the same amount of alpha activity in hot particles. The Los Alamos experiments[12,13] make it evident that most of the alpha dose from "hot" particles of PuO_2 is wasted in the excessive irradiation of cells within the alpha range of the hot particle surface. 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 of larger numbers of cells with scattered protons (an effect that may be significant for very hot particles).

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. 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 $^{239}PuO_2$. 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.

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.

It also should be noted that americium-241 is present in association with plutonium contamination in the Rocky Flats area and in nuclear test areas. In addition, curium isotopes as well as americium-241 will be present in high concentration in the nuclear fuel mixture from fission and breeder reactors which use plutonium fuel. The chemical behavior of americium and curium in the environment will give rise to their substantial uptake in the biosphere and the food chain. Thus the ingestion of americium and curium, their uptake from the gastrointestinal tract, and their accumulation in the liver and skeletal tissue of mammals and man will give rise to additional serious health risks. These contaminants will be relatively more serious than plutonium inhalation in some environments, particularly in vegetated areas of moderate to high rainfall, where soil resuspension processes are not effective.

6. Recommendations: It is urged that the U.S. Environmental Protection Agency consider and act upon each of the following recommendations which are called for (a) in order to provide an improved basis for the assessment of health risks and standards for plutonium and other actinides and (b) to provide a higher degree of protection from the effects of internal alpha emitters for occupational groups and the general public by adopting more conservative interim standards for plutonium exposure.

(1) Initiate a comprehensive interagency research program to assess the health risks of inhaled alpha emitting particles, with special attention to both "hot" particles and insoluble particles of low activity per particle (Some pertinent studies have been proposed to the EPA[58].)

(2) Conduct a comprehensive epidemiological health study of all past and present plutonium workers, and of all other groups which have been exposed to the inhalation of plutonium at levels significantly above fallout plutonium.

(3) Call upon the National Cancer Institute and the National Heart and Lung Institute to apply an appropriate fraction of their resources to assess the role of inhaled alpha emitting particles on the incidence of human cancer and heart disease.

(4) Adopt more conservative occupational standards for plutonium. A reduction of present air concentration and lung burden standards by a factor between 100 and 1000 appears to be in order. Better protection should be provided for younger employees and groups exposed to possible inhalation of finely divided and higher specific activity plutonium.

(5) Maintain public exposure levels of plutonium and other alpha emitters to the practical minimum. In my view this would limit public exposure to airborne dusts not exceeding 0.5 picocuries of alpha activity (about one alpha disintegration per minute) per gram of nitric acid insoluble

particulates of respirable size. This level would result in the accumulation of adult organ burdens about equal to that from fallout plutonium[56]. On this basis the Colorado interim standard may be at least 10 times too high.

(6) Call for a full disclosure of all past plutonium spills and accidental releases and conduct appropriate surveys and cleanup operations.

(7) Develop standards for americium and curium, with particular attention to their distribution in the food chain and their uptake from the gastrointestinal tract.

(8) Give immediate attention to current plans of the U.S. Department of Defense and the U.S. Atomic Energy Commission to resettle Enewetak Atoll. The high levels of plutonium and americium on these islands and in the lagoon sediments are likely to give rise to tragic health effects on this small native population group.

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APPENDIX III

COMMENTS ON THE

DRAFT ENVIRONMENTAL IMPACT STATEMENT

LIQUID METAL FAST BREEDER REACTOR PROGRAM
(WASH-1535)

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PREFACE

Four comments are attached.

Comment #1, ACCIDENTS

Comment #2, ESTIMATION OF THE HEALTH EFFECTS OF PLUTONIUM AND OTHER ALPHA-EMITTING TRANSURANICS

Comment #3, DIVERSION AND SAFEGUARDS OF FISSIONABLE MATERIALS

Comment #4, GENERAL AND IN SUMMARY

With the possible exception of #2, these comments are generic in nature. For a draft statement of this physical extent, detailed comment would be nearly prohibited by personal limitations of time and resources. This dilemma is not encountered here since generic comment seems indicated. Treatment of acne can be sensibly deferred when the patient shows systemic failure.

Comment #2, ESTIMATION OF THE HEALTH EFFECTS OF PLUTONIUM AND OTHER ALPHA-EMITTING TRANSURANICS

The estimate of lung cancer incidence associated with the inhalation of plutonium (or other transuranics) in particulate form is a critical factor, along with source terms and resuspension, in defining the probable impact of the LMFBR's plutonium based fuel-cycle. This subject is discussed in Section 4.G.5 "Particle Lung Dose Effects" of WASH-1535. I quote the first sentence from that section:

> "The estimates of lung cancer incidence associated with the inhalation of transuranics used in this report are based upon a calculation of the average radiation dose delivered to the lung and application of tumor incidence estimates for the uniformly irradiated lung as estimated in the BEIR report."[1]

This cited basis, and hence the derived estimates, are indefensible.

Section 4.G.5 acknowledges "that 'insoluble' particles of radioisotopes, when deposited in tissue, provide focal spots of high radiation dose rates close the the particle," so there is no presumption that the exposure by particulates of plutonium is uniform. The deep respiratory tissue of the lung is made up of 10^8 alveoli. Each aveolus is a complexly organized unit of tissue. If an insoluble alpha-emitting particulate is deposited in this tissue some 10 to 100 alveoli will be exposed. A crude measure of the nonuniformity of this exposure is that at most about one-millionth of the lung's alveoli are affected by a single particulate.

The significance of the preceding is that in the actual lung exposure by an alpha-emitting particulate, the energy of the ionizing radiation is deposited in a very limited volume of tissue, and hence that the actual radiation dose to lung tissue scaled roughly a million times

larger than the dose associated with an averaging of the equivalent radiation energy over the entire lung.

A multiplicative difference of a million in a significant physical quantity generally suggests a qualitative difference. Suppose, for example, that the problem were to estimate the effects of small projectiles on human organisms. Suppose that the projectiles weigh 1/2 ounce and have a velocity of 1000 ft/sec. Note that the effect of the projectile depends on the energy, and note that a 6 ton vehicle moving at 1 mile per hour has similar energy. There is experience with humans stopping slow moving vehicles by exerting strenuous counterforces. Using this experience the effect of the projectiles on humans is inferred to be oxidation of the biological fuel necessary to do the work of stopping the vehicle. But this reasoning is manifest nonsense. Even though the energies involved are similar, a fast moving rifle bullet is quite different from a truck weighing a million times more and moving at a one-thousandth the velocity. The former dissipates its energy in the local disruption of tissue, the latter leads to the ordered and non injurious oxidation of biological fuel. The end results become very different as the physical characteristics of the situation change, and a new biological phenomenon intercedes. Obviously the way to estimate the effects of rifle bullets is either from past experience that is explicitly applicable, or alternatively, to calculate the effects considering the physical characteristics of the rifle bullet and knowledge of the biological and physical characteristics of the human organism.

This nonsense example has much the same logical structure as the method of estimating hot particles effects set forth in Section 4.G.5 of

WASH-1535. There, by introducing a fictitiously large mass of exposed tissue, the calculated dose becomes commensurately small. In passing from the real situation in which a hot particle irradiates 10 to 100 alveoli, to the fictional situation in which the ionizing radiation from the hot particle is averaged over 10^8 alveoli, the dose scale has decreased by roughly a factor of a million.

Living tissue shows extensive intra-cellular and inter-cellular organization. Several regimes of biological response would be expected as physical characteristics of exposure are varied. Carcinogenic response to whole organ exposure by non acute doses of radiation will fall in one of these regimes, and this will be a regime in which there is human experience. From the physical characteristics of plutonium aerosols, from the lung deposition experience with aerosols, and from the lung clearance experience with plutonium particulates, it can be inferred that at least one class of particles exist which subject lung tissue to an exposure associated with a different carcinogenic response regime. This is because other biological phenomenon has intervened.

For hot particle exposure that phenomenon is mitotic death of cells, i.e., loss of the cell's ability to divide. There is an extensive literature on the subject. Radiologically induced mitotic death is, in fact, the basis for treating malignant tissue with ionizing radiation, and is the cause of most acute symptoms consequent to radiation exposure. Even though the intercession of extensive mitotic death of cells must inevitably place certain particulate exposures in a different response regime from whole lung, non acute exposures, a compelling argument might be made that the carcinogenic response in the former case is necessarily

less than the carcinogenic response in the latter. This argument would appear to have merit since mitotic death of cells, of well as reducing the general viability of the tissue, would also reduce the number of irradiated cells with carcinogenic potential. Usually implicit in this argument is a conceptualization of all radiation carcinogenesis as a single-cell, direct-injury process.

To confirm this argument, there is a respectable literature in which carcinogenesis is described as occurring after doses of radiation that are sufficiently local as to not be organism lethal, and that are sufficiently high for the fraction of mitotically competent cells to be greatly reduced, i.e., to 1% or less. Unfortunately, in at least some of these experiments, carcinogenesis is inversely related to the fraction of mitotically competent cells, i.e., cancer induction in the regime where mitotic competence is greater than 1% is small compared with the cancer induction in the regime where mitotic competence is much less than 1%.

There are several points to be made here. Loss of mitotic competence and carcinogenesis are two indices of radiation effect in tissue. They cannot be independent, and their relationship can tell us something about some radiation carcinogenesis.

Mitotic competence is not generally related in a linear way to carcinogenic response. Moreover, it is a major anomaly that an increased carcinogenic response is observed in dose regimes associated with greatly reduced mitotic competence. It is difficult to reconcile this result with any single-cell, direct-effect origin for radiation induced cancer.

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decreased with mitotic competence, then estimates of carcinogenesis based on a fictitious averaging of a local inhomogeneous dose over a much larger volume would be necessarily conservative. Since radiation carcinogensis can, and in fact, does increase to anomalously large values while the mitotic competence becomes vanishlingly small, the fictitious averaging of dose over larger volumes is not necessarily conservative. Instead it would appear that an intense local dose of ionizing radiation can be a more efficient carcinogen than a diffuse tissue exposure with the same type of ionizing radiation and the same total energy. The above then implies that averaging of dose over larger volumes may be far from conservative.

It is obvious that as a local exposure becomes more intense, a stage must finally be reached where the carcinogenic efficiency of the exposure (on a per unit energy basis) is reduced. This is not pertinent to previous arguments. It would, however, be important to know the characteristics of the most carcinogenicly efficient exposures.

The following excerpt taken from the BEIR report (p. 95) summarizes the state of knowledge concerning the causation of cancer (emphasis added):

> "Although the mechanisms of carcinogenesis, or of radiation carcinogenesis in particular, are not fully known, available information implies that most, if not all, types of cancer develop as a result of the combined effects of multiple factors. These causative factors may include: prezygotic (inherited) mutations of chromosomal aberrations, which can spread during development to many kinds of cells; somatic cell mutations or chromosomal aberrations, which can be acquired at any time after conception; changes resulting from the action of viruses; and changes in systemic growth factors (e.g., depressed immune competence, hormonal imbalance) and in local tissue regulation (disorganization, damage), such as may result from diseases other than cancer or from advancing age (1).
>
> "Although point mutations, chromosomal abberations, and other changes at the cellular and molecular level may require only small doses, tissue disorganization and

gross disturbances in physiology are unlikely without larger doses (2).

"Of the many types of changes which radiation can cause in cells or tissues, none is considered to be unique for radiation. Many, if not all, such changes can presumably result from a variety of other agents."

This summary view on carcinogenesis is compatible with the ideas leading to the conclusion reached earlier, that fictitious dose averaging to larger tissue masses need not be conservative. The possibility of various modes of carcinogenesis is acknowledged, and in particular, mention is made of a pathway mediated by tissue disruption.

Disease profiles are highly species specific. Cancer is no exception. Gross characteristics are obviously highly species specific also. A rat and a mouse are distinct and yet incredibly similar. The gross tissue differences are articulated out through subtly different informational resonances amongst cell populations, - the collective behavior being phased ultimately, though perhaps remotely, by the genetic controls of the cells. Not to belabor this point unnecessarily, - cancer profiles are species specific; gross characteristics and, of course, genetic material are also species specific. Collective detuning of tissue, by tissue disruption seem as acceptable an origin for the tissue instabilities of cancer as does an isolated single cell event.

Return now to the problem of risk estimates associated with radioactive particulates in human lungs. Most of what has been said earlier in this comment has been general, and has been aimed at showing that there was no inherent conservatism in the method of estimating cancer risks set forth in the first sentence of 4.G.5, and that moreover the method could be far from conservative. The conclusion could as well be applied to lymphatic tissue or to bronchial tissue.

Having this background notice that human lung tissue has a well known carcinogenic potential under a number of situations, including exposure to ionizing radiations; and that in the Hanford dog study induction of lung cancer was observed after exposure to plutonium aerosols. These are a sufficient basis to establish plutonium induced lung cancer as a legitimate concern for humans.

The following is a review of the official guidance for estimating the carcinogenic effects from exposure to radioactive particulates.

I. "(210) The NCRP has arbitrarily used 10% of the volume of the organ as the significant volume for irradiation of the gonads. There are some cases in which choice of a significant volume or area is virtually meaningless. For example, if a single particle of radioactive material fixed in either lung or lymph node may be carcinogenic, the averaging of dose either over the lung, or one cubic centimeter may have little to do with the case. Use of significant volumes or areas must be looked on as one of the round off devices which in special cases must give way to detailed study."

NCRP Report #39
Basic Radiation Protection Criteria
January 15, 1971.
(emphasis added)

II.

40. The problems of high local concentration of dose are at their most severe with radioactive particulate material in the tissue, especially with α-emitters. Here the local dose can reach very high values even though the mean tissue dose may be very low. Certainly it cannot be assumed that linearity of dose and effect will hold at these high doses and dose rates. On the other hand, there may be a great deal of cell death, and particularly with α-emission, with its short and well-defined range, the number of affected but viable cells may be small compared with the number of killed cells. However, this ratio will depend on the size and activity of the particles, the extent to which they aggregate, and their movement within the tissue, and the movement of the cells past them.

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

42. From dog experiments being carried out in the United States and from limited studies in human subjects it has become clear that inhalation of plutonium particulates can lead to high concentrations of the particulate material in the pulmonary lymph nodes, and that the mean radiation dose to the pulmonary lymph nodes and indeed to lymphoid tissue as a whole is likely to be greatly in excess of that to the lung, which is at present regarded as the critical organ for inhaled particulate matter.

43. The actual dose ratios are not yet known with any precision but the Task Group understood that the mean dose to lymphoid tissue as a whole might exceed that to the lung by a factor of 10 or more, and, if the respiratory lymph nodes were alone taken into consideration, the factor could be 100 or perhaps much greater. The problem is whether lymphoid tissue as a whole or respiratory lymph node tissue in particular should be taken as the critical organ, and, if so, whether the dose limit (MPC$_a$) for plutonium in particulate form should be substantially reduced below the current value.

44. One is concerned here with the relative risks of particulate material (mainly α-emitting, but with some soft x-ray emission) deposited in lung and in lymph nodes, and any analysis based on dose determination encounters profound difficulties. Considering only the lymphoid tissue, there is first the problem of lack of knowledge of the sensitivity to radiation-induced malignant change of the reticulum cells present, and possibly also of lymphocytes themselves. Also any calculation of dosage distribution to the various elements of the lymphoid tissue would require information, at present unavailable, on such subjects as the degree of aggregation of the particulate material within the lymph nodes, its change with time and the movement of the particles within the lymph nodes. In addition there is very little reported work on the distribution of the material within the nodes, which could be obtained by autoradiographic study, or on the degree of fibrosis and other histological changes produced. Better data are also required on the residence times of the particulate material in the lymph nodes and the solubility of the particulate material over many years. Another factor to be taken into consideration, common to all particulate deposition and especially to those involving α-emitters, is that the number of cells irradiated is, for the same mean tissue dose, very dependent on the particle size. Finally, lymphocyte migration is a factor that should be taken into consideration.

45. In so far as mean dose calculations can be made the Task Group considered that they should, for consistency within the recommendations, refer to the whole lymph tissue and not only to the respiratory lymph nodes. However, until more information becomes available on some of the subjects mentioned above, little weight can be put on dose calculations. Data on tumour production from animal experiments are of more significance, and the results of present work with dogs, particularly those with lower amounts of plutonium, are awaited with interest. In the meantime, the Task Group are of the opinion that any immediate change in the dose limit for plutonium on the basis of risk to lymphoid tissue is not warranted.

ICRP Publication 14
Radiation Sensitivity and Spatial Distribution of Dose
(Publication 14 appears as a report of two Task Groups, and not as the official recommendations of the ICRP.)

III.

(20) In the case of non-homogeneous distribution of absorbed dose in the lung, an estimate of the Dose Equivalent to the whole lung, determined merely by the product of QF and the mean absorbed dose, may be greatly in error, but our full understanding of this problem must await further experimental evidence. In the meantime there is no clear evidence to show whether, with a given mean absorbed dose, the biological risk associated with a non-homogeneous distribution is greater or less than the risk resulting from a more diffuse distribution of that dose in the lung. When irradiation results from the inhalation of thoron or radon and daughter products, the relevant Dose Equivalent is that in the bronchial mucosa which is the tissue considered to be most heavily irradiated. Here the use of the whole lung would be an inadequate substitute for that of the irradiated tissue.

(23) Within the range of the Maximum Permissible Doses (see paragraph 37) specified for occupational exposure, when it is assumed that there is no threshold and that effects are linearly related to dose, it is justifiable to consider the average dose to the whole organ or tissue, although it is recognized that when more information is available, it will be more appropriate to use the mean dose for cells of any given type, as is already done when the bronchial mucosa is irradiated by daughter products of radon and thoron. The use of the mean dose has practical advantages in that the significant volume can be taken as that of the organ or tissue under consideration. In fact, this principle has necessarily been used already in calculating maximum permissible burdens of radionuclides in tissues. However, with extreme inhomogeneity of dose (for example, with particulate radioactive material of high specific activity) such a procedure may be inappropriate. This is a matter upon which further work is needed. Also, for external exposure of the skin, especially when the distance to the source is very short or when the exposed area is very small, it would not be appropriate to average the dose over the entire skin. Instead, it is recommended that the dose be averaged over an area of a square centimetre in the region receiving the highest dose; however, with very narrow beams of extremely high intensity, such as those used for X-ray analysis, the value of such an average dose may be misleading, and protection measures have to be based on qualitative considerations.

ICRP Publication 9
Recommendations of the
International Committee
on Radiological Protection
(adopted September 17, 1965).

The recommendations of the National Council on Radiation Protection and Measurement set forth in I, and the recommendations of the International Commission on Radiological Protection set forth in III, are explicit in offering no guidance.

II is a discussion of the hot particle problem taken from the report of an ICRP Task Group. It is not intended to give dispositive official guidance. The discussion is useful commentary, but inconclusive. The very conditional statement made in the first and second sentence of II (41) is not generally convincing.

With regard to the previously cited method of risk estimation described in the first sentence of 4.G.5, that section continues with the following supportive references:

> "This approach has been used by the Environmental Protection Agency in recent reports on the potential health consequences of the nuclear fuel cycle.[2-5] The approach leads to estimates comparable to those of Gavankar[6] following Thompson et al[7] based on linear non-threshold extrapolation of observations on beagle dogs administered $^{239}PuO_2$ aerosols."

As to the first, consensus in error may provide amiable agreement amongst federal agencies, but seems hardly a desirable basis for decisions involving the public health and safety. The observations on beagle dogs are discussed further on 4.G-117 and deserves separate consideration.

It requires pathological optimism to find reassurance in the results of the now completed Hanford beagle experiment. Dogs were given initial aerosol burdens of approximately 1-10 microcuries of $Pu^{239}0_2$. By nine years post-exposure the lung cancer response was virtually saturated and multicentric origins were noted in some dogs. Those receiving larger lung burdens greater than 10 microcuries died of pulmonary insufficiency within 4-1/2 years. Twenty-one dogs survived for more than 4-1/2 years,

and only one of these did not exhibit lung cancer at death. A relationship observed between initial lung burden and time to death with cancer has been often used to infer a threshold burden below which no life shortening of dogs would be expected. This is shown in Figure 4.G.10 on 4-G 118. Note that the fibrotic deaths there have no bearing on cancer incidence ...d inclusion of these points in the constructing extrapolated curves is a senseless exercise. Note also that the results are exhibited on a log-log graph which virtually obscures all differential detail. Most important, recognize the nature of the experiment, i.e., the lung burdens were large, the results were saturated, and the number of animals was small. The crude relationship observed between initial lung burden and time to death with lung cancer does not necessarily imply that a threshold burden exists for beagles. Quite to the contrary, the range of exposures above the inferred threshold burden may be interpreted as a region of saturated carcinogenic response, that is a burden regime in which lung cancer induction in a beagle population approaches 100% during a normal life span. The point is that the observed time to death is more likely related to the burden, through a population depletion effect, rather than through a burden dependent latent period. In the former interpretation appreciable cancer would be anticipated at lower burdens. This is again consistent with extensive observations of radioisotope-induced bone tumors in mice, which support the interpretation that "latent period is constant and that the apparent relationship between increasing dose and decreasing time to death with tumor is due to the effects of dose-level on survival and on tumor expectancy." (See Toxicity of Ra-226 in Mice," M. Finkel et al, in Radiation-Induced Cancer, IAEA, Vienna, 1969.)

The domain of this comment is broadened here in order to summarize

a specific concern with plutonium, and, to a lesser extent, other transuranics. Under a number of circumstances plutonium forms aerosols. The physical character of these aerosols is such that on inhalation by humans they are preferentially deposited in respiratory tissue. Because of slow clearance and because of their insoluble character, particles may experience long residence times in tissue. An appreciable mass fraction of the aerosol is usually associated with particles sufficiently large that small but physiologically significant volumes of tissue will be exposed to intense (i.e., organism lethal or greater) radiation doses within a meaningful physiological time. Studies of the effects of intense local radiation to skin and kidney tissue indicate that despite the near mitotic sterilization of the involved tissue, an enhanced carcinogenic response may occur, in the sense that energy dissipated in a limited volume may be far more carcinogenic than if the same type of radiation were to dissipate its energy over a much larger tissue mass. The question is then: do particulates of plutonium lead to exposures that have enhanced carcinogenic potential? If they do, then present standards can be in error by orders of magnitude.

Notice that the emphasis here is on the anomalous hazard associated with a single particle; and that if any threshold is relevant, it is not a dose threshold since local exposures are large, but rather a possible volumetric threshold that must be exceeded by the physical extent of the exposure. Plutonium, as an insoluble aerosol-forming, long-lived alpha-emitter, constitutes a very special case of the low exposure problem.

In conclusion, it is indefensible to base estimates of cancer risk on the method of dose averaging over fictitiously large volumes. Similarly, estimates based on non conservative interpretations of the Hanford beagle results are highly suspect.

Attachment #1 to Comment #2

"Plutonium and Public Health," in
Electric Power Consumption and
Human Welfare, AAAS Committee
on Environmental Alterations,
August 11, 1972 (non-copyrighted).

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Oxford University Press

PLUTONIUM AND PUBLIC HEALTH

Donald P. Geesaman

Author's Note--June 1972

On May 11, 1969 a major fire occurred at the large Rocky Flats plutonium facility located northwest of Denver, Colorado, and operated for the AEC by the Dow Chemical Company. For description of this fire see AEC press releases M-121, May 20, 1969, and M-257, November 18, 1969.

Consequent to this fire E.A. Martell and S.E. Poet conducted a pilot study on the plutonium contamination of surface soils in the Rocky Flats environs. Their results suggested an off site contamination that was orders of magnitude larger than that which would have been expected from the measured plutonium releases in the air effluent of the facility.

In a letter of January 13, 1970 to Glenn Seaborg, then chairman of the AEC, and in a press release of February 24, 1970 by the Colorado Committee on Environmental Information, Martell et al. called attention to this anomalous contamination and expressed concern over its uncertain origin and over its significance to public health. In response the AEC fixed the probable origin of the off site contamination as wind dispersal of plutonium leaking from rusted barrels of contaminated cutting oil, and denied that cause existed for concern over hazards to public health (see AEC press release N-22, February 13, 1970).

It was my conviction that the AEC response provided a distorted

and inadequate representation of the possible hazards associated with the observed off site contamination, and that the imminent large-scale commercial introduction of plutonium gave this situation a precedential significance much greater than the already considerable significance of the situation itself.

In April 1970 a representative of the AEC's Division of Biology and Medicine and myself were invited to present our views at the University of Colorado. "Plutonium and Public Health" derives from the preceding history and should be so interpreted. The presentation was to a lay audience and was made with that expectation. Adequate referencing was added to the written text prior to its inclusion in Underground Uses of Nuclear Energy, Part 2, Hearings before the Subcommittee on Air and Water Pollution of the Committee on Public Works United States Senate, August 5, 1970.

As it stands the paper still represents a legitimate critique, and the recent emphasis on plutonium as a major energy source increases the relevance of the discussion. An updating would involve only incremental changes, and would generally supplement rather than disturb the substantive arguments of the original paper. Hence while such an updating is desirable, it is also of sufficient marginal value that it can be properly deferred at my discretion.

For those who are interested in reading the traditional AEC position on the subject I would suggest "Appendix 24 - Safety Considerations in the Operations of the Rocky Flats Plutonium Processing Plant", from AEC Authorizing Legislation Fiscal Year 1971 - Hearings before the Joint

Committee on Atomic Energy, Part 4, March 19, 1970.

Times have changed since May 1969. Then plutonium was regarded as a military substance and was accordingly given little public attention. Now it is much publicized as the energy source of the not too distant future. April 1970 was a time of transition, and I felt the strong presence of the earlier tradition, and the decision to speak was not an easy one for me. I have had no regrets.

D. P. G.

Plutonium and Public Health

For the sake of completeness let me give you some background on plutonium. It is an element that is virtually non-existent in the earth's natural crust. In the early 1940's it was first produced and isolated by Dr. Seaborg and colleagues; --Dr. Seaborg is presently Chairman of the Atomic Energy Commission. Plutonium has several isotopes, the most important being plutonium-239, which, because of its fissionable properties and its ease of production, is potentially the best of the three fission fuels. That is why it is of interest. Aside from its fissionable properties, plutonium-239 is a radioactive isotope of relatively long half-life (24,000 years), hence its radioactivity is undiminished within human time scales. When it decays, it emits a helium nucleus of substantial energy. Because of its physical characteristics, a helium nucleus interacts strongly with the material along its path; and as a consequence deposits its energy in a relatively short distance, --about four-hundredths of a millimeter in solid tissue. For comparison, a typical cell dimension is about 1/4 to

1/10 of that. A cell whose nucleus is intercepted by the path of such a particle suffers sufficient injury that its capacity for cell division is usually lost (Bardeen, A.W., 1962 and Bloom, W., 1959).

The cancer inducing potential of plutonium is well known. One millionth of a gram injected intradermally in mice has caused cancer (Lisco, H., et al., 1947); a similar amount injected into the blood system of dogs has induced a substantial incidence of bone cancer (Mays, C.W., et al., 1947), because of plutonium's tendency to seek bone tissue. Fortunately the body maintains a relatively effective barrier against the entry of plutonium into the blood system. Also, because of the short range of the emitted helium nuclei, the radiation from plutonium deposited on the surface of human skin does not usually reach any relevant tissue. Unfortunately the lung is more vulnerable.

Before I describe why this is, I'd like to say something about the characteristics of an aerosol. An aerosol is physically like cigarette smoke, or fog, or cement dust. Because of their small size, the particles comprising an aerosol remain suspended in air for long periods of time. If an aerosol is inhaled, then, depending on its physical characteristics, it may be deposited at different sites in the respiratory tree (Health Physics, 1966). Larger aerosol sizes are usually removed by turbulence in the nose, particles deposited in the bronchial tree are cleared upward in hours by the ciliated mucus blanket that covers the structure. This clearance system does not penetrate into the deep respiratory structures, the alveoli, where the basic oxygen-carbon dioxide exchange of the lung takes place. Smaller

particles tend to be deposited here by gravitational settling, and if they are insoluble they may reside in the alveoli for a considerable time. The problem is that, under a number of conditions (Anderson, B.V., et al., 1967; Fraser, D.C., 1967; Kirchner, R.A., 1966; Mann, J.R., et al., 1967; Stewart, K., 1963; Wilson, R.H. et al., 1967) plutonium tends to form aerosols of a size that are preferentially deposited in deep lung tissue. Plutonium dioxide, which is a principal offender, is insoluble and may be immobilized in the lung for hundreds of days before being cleared to the throat or to the lymph nodes around the lungs (Health Physics, 1966).

An aerosol is comprised of particles of many different sizes, and their radioactivity may differ by factors of thousands or even more. I will simplify the argument and say that there is a class of these particles, the largest ones deposited in the deep lung tissue, that can be expected to have a different potential of cancer induction than the particles of the smaller class. This is because they are sufficiently radioactive to disrupt cell populations in the volume of cell tissue which they expose (Geesaman, D.P., 1968a). An example might be a particle that emits 5000 helium nuclei per day. It would subject between 1 and 20 alveoli to intense radiation, sufficient to inflict substantial cell death and tissue disruption. For reference, the alveoli are the basic structural units of the deep lung. They are shaped and bunched roughly like hollow grapes 0.3 millimeter in diameter. Their walls are thin, a few thousandths of a millimeter, and they are a highly structured tissue with many cell types. Intense exposure of local tissue by a radioactive particle is referred to as the hot

particle problem. The question is: does such a particle have an enhanced potential for cancer? No one knows. One can argue that cancer cannot evolve from dead cells, hence a depleted cell population must be less carcinogenic. This is believeable, and must be true on occasion. The facts are, though, that intense, local doses of radiation are extremely effective carcinogens, much more so than if the energy were averaged over a larger tissue mass (Geesaman, D.P., 1968b) Furthermore, this can take place at high doses of radiation where only one cell in ten thousand has retained its capacity to divide. The cancer susceptibility of lung tissue to radiation has been demonstrated in many species; one can say in general that the lung is more susceptible to inhomogeneous exposures from particles and implants than it is to diffuse uniform radiation. Some very careful skin experiments of Dr. Albert have indicated that tissue disruption is a very likely pathway of radioactive induction of cancer after intense exposure (Albert, R.E., et al., 1967a, 1967b, 1967c, 1969). The experiments show that the most severe tissue injury is not necessary, nor even optimal, for the induction of cancer. When these notions are applied to a hot particle in the lung, the possibility of one cancer from 10,000 disruptive particles is realistic. This is disturbing because an appreciable portion of the total radioactivity in a plutonium aerosol is usually in the large particle component.

Let me demonstrate what I mean. Suppose a man received a maximum permissible lung burden for plutonium, and suppose roughly 10% of the mass of the burden was associated with the most active class

of particles deposited (that is those emitting several thousand helium nuclei per day). This is reasonable. There would be something like a thousand of these particles and each would chronically expose 1 to 20 alveoli to intense radiation. If the risk of cancer is like 1 in 10,000 for one disruptive particle, then the total risk in this situation is one in ten, i.e., one man in ten would develop lung cancer.

Put another way, about 1 cubic centimeter of the lung is receiving high doses of radiation. It would not be surprising if intense exposure of such a localized volume led to a cancer one time in ten. The question is: if the individual volumes are separated from each other, is substantial protection afforded? No one knows. It is much easier to find two cancers using 50 exposures of 1 cubic centimeter each, than it is to find a couple of cancers in 50,000 single particle exposures. Certainly the length scales of injury are long enough that a disruptive carcinogenic pathway cannot be disregarded for isolated hot particles (Geesaman, D.P., 1968b).

One can look to the relevant experience for reassurance. In an experiment done at Hanford by Dr. Bair and his colleagues, beagle dogs were given $Pu^{239}O_2$ lung burdens of a few hundred thousandths of a gram (Bair, W.J., et al., 1966; Ross, D.M., 1967). At 9 years post exposure, or after roughly half of an adult beagle life span, 22 of 24 deaths involved lung cancer, usually of multiple origin. Five dogs remain alive. For comparison, these exposures are about 100 times larger than the present maximum permissible burdens in man.

There are two unsatisfactory aspects of this experiment. First,

because all of the dogs are developing cancer, it is impossible to infer what would happen at lower exposures: simple proportionality does, however, suggest that present human standards are too lax by at least a factor of ten. Second, because the radiation dose is large, with tissue injury almost killing the dogs; and because large numbers of particles are involved, often acting in conjunction; it is improbable that the risk from disruptive particles can be inferred. And after all, this is what we need to know, since almost all human exposures will involve hot particles acting independently, and if there is a risk from these particles, it will be additive throughout the population; --there will be no question of a threshold burden; and there will be a possibility that a man with an undetectable burden of a few particles will develop a cancer as a consequence. For the exposures of concern, 1000 people with 100 disruptive particles each will suffer as many total cancers as 10,000 people with 10 particles each, or as 100 people with 1000 particles each.

Human experience does not give us the answer either. Plutonium has been around for 25 years, and people have been exposed. In 1964 through 1966 contractors indicated an average total of 21 people per year with over 25% of a maximum permissible burden of plutonium (Ross, D.M., 1968). Three out of four of these exposures derived from inhalation. To be reasonably useful, the documentation of exposure must go back more than 15 years, because of the latent period for radiation induced cancer. In recent years documentation has improved greatly, but from early days there is pitifully little of relevance to the hot particle problem in the lung.

Since I have mentioned maximum permissible lung burdens, you are aware that there is official guidance. I would like to comment on it. The maximum permissible lung burden is established by equilibrating the exposure from the deposited radioactive aerosol with that of an acceptable uniform dose of x-rays. The International Commission on Radiological Protection indicates this may be greatly in error, and specifically states in its publication 9, "In the meantime there is no clear evidence to show whether, with a given mean absorbed dose, the biological risk associated with a non-homogeneous distribution is greater or less than the risk resulting from a more diffuse distribution of that dose in the lung." (ICRP, 1966). They are effectively saying that there is no guidance as to the risk for non-homogeneous exposure in the lung, hence the maximum permissible lung burden is meaningless for plutonium particles; as are the maximum permissible air concentrations which derive from it.

So there is a hot particle problem with plutonium in the lung, and the hot particle problem is not understood, and there is no guidance as to the risk. I don't think there is any controversy about that. Let me quote to you from Dr. K. Z Morgan's testimony in January of this year before the Joint Committee on Atomic Energy, U.S. Congress (Morgan, K.Z., 1960). Dr. K.Z. Morgan is one of the United States' two members to the main Committee of the International Commission on Radiological Protection; he has been a member of the committee longer than anyone; and he is director of Health Physics Division at Oak Ridge National Laboratory. I quote: "There are many things about radiation exposure we do not

understand, and there will continue to be uncertainties until health physics can provide a coherent theory of radiation damage. This is why some of the basic research studies of the USAEC are so important. D.P. Geesaman and Tamplin have pointed out recently the problems of plutonium-239 particles and the uncertainty of the risk to a man who carries such a particle of high specific activity in his lungs." At the same hearing, in response to the committee's inquiry about priorities in basic research on the biological effects of radiation, Dr. M. Eisenbud, then Director of the New York City Environmental Protection Administration, in part replied, "For some reason or other the particle problem has not come upon us in quite a little while, but it probably will one of these days. We are not much further along on the basic question of whether a given amount of energy delivered to a progressively smaller and smaller volume of tissue is better or worse for the recipient. This is another way of asking the question of how you calculate the dose when you inhale a single particle." (Eisenbud, M., 1970). He was correct; the problem has come up again.

In the context of his comment it is interesting to refer to the National Academy of Sciences, National Research Council report of 1961 on the Effects of Inhaled Radioactive Particles (U.S. NAS.NRC.1961). The first sentence reads, "The potential hazard due to airborne radioactive particulates is probably the least understood of the hazards associated with atomic weapons tests, production of radioelements, and the expanding use of nuclear energy for power production." A decade later that statement is still valid. Finally let me quote Drs. Sanders, Thompson, and

Bair from a paper given by them last October (Sanders, C.L., 1970). Dr. Bair and his colleagues have done the most relevant plutonium oxide inhalation experiments. "Nonuniform irradiation of the lung from deposited radioactive particulates is clearly more carcinogenic than uniform exposure (on a total-lung dose basis), and alpha-irradiation is more carcinogenic than beta-irradiation. The doses required for a substantial tumor incidence, are very high, however, if measured in proximity to the particle; and, again, there are no data to establish the low-incidence end of a dose-effect curve. And there is no general theory, or data on which to base a theory, which would permit extrapolation of the high incidence portion of the curve into the low incidence region." I agree and I suggest that in such a circumstance it is appropriate to view the standards with extreme caution.

There is another hazardous aspect of the particulate problem in which substantial uncertainty exists. In case of an aerosol depositing on a surface, the material may be resuspended in the air. This process is crudely described by a quantity called a resuspension factor which is remarkable in that it seems generally known only to within a factor of billions (Kathren, R.L. 1968). Undoubtedly it can be pinpointed somewhat better than this for plutonium oxide, but the handiest way to dispatch the problem is to say there is some evidence that plutonium particles become attached to larger particles and are therefore no longer potential aerosols. Unfortunately there is also evidence that large particles generate aerodynamic turbulence, and are hence blown about more readily, and on

being redeposited tend to knock small particles free. In relation to this, I'd like to give you a little subjective feeling for the hazard. There is no official guidance on surface contamination by plutonium. Two years ago, in an effort to determine some indication of the opinions of knowledgeable persons with respect to environmental contamination by plutonium, a brief questionaire was administered to 36 selected LRL employees (Kathren, R.L., private communication). All were persons who were well acquainted with the hazards of plutonium. The group consisted of 16 Hazards Control personnel, primarily health physicists and senior radiation monitors. The remainder were professional personnel from Biomedical Division, Chemistry, and Military Applications, who had extensive experience with plutonium. I had nothing to do with the survey, nor was I one of the members who was queried. The conjectured situation was that their neighborhood had been contaminated by plutonium oxide to levels of 0.4 microcuries per square meter. For reference, this value is roughly ten times the highest concentration Dr. Martell found east of the Rocky Flast Dow Chemical facility (Martell, E.A., 1970), --and bear in mind that a factor of ten is a small difference relative to the large uncertainties associated with the hazards from plutonium contamination. Several questions were asked. One was, would you allow your children to play in it? 86% said No. Should these levels be decontaminated? 89% said Yes. And to what level should the area be cleaned? 50% said to background, zero, minimum, or by a reduction of at least a factor of 40. This has no profound scientific significance, but indicates that many people conversant of the hazard are not

blase about the levels of contamination encountered east of Rocky Flats.

Finally I would like to describe the problem in a larger context. By the year 2000, plutonium-239 has been conjectured to be a major energy source. Commercial production is projected at 30 tons per year by 1980, in excess of 100 tons per year by 2000. Plutonium contamination is not an academic question. Unless fusion reactor feasibility is demonstrated in the near future, the commitment will be made to liquid metal fast breeder reactors fueled by plutonium. Since fusion reactors are presently speculative, the decision for liquid metal fast breeders should be anticipated and plutonium should be considered as a major pollutant of remarkable toxicity and persistence. Considering the enormous economic inertia involved in the commitment it is imperative that public health aspects be carefully and honestly defined prior to active promotion of the industry. To live sanely with plutonium one must appreciate the potential magnitude of the risk, and be able to monitor against all significant hazards.

An indeterminate amount of plutonium has gone off site at a major facility 10 miles upwind from a metropolitan area. The loss was unnoticed. The origin is somewhat speculative as is the ultimate deposition.

The health and safety of public and workers are protected by a set of standards for plutonium acknowledged to be meaningless.

Such things make a travesty of public health, and raise serious questions about a hurried acceptance of nuclear energy.

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