ORNL/TM-5257

Review of John W. Gofman's Reports on Health Hazards from Inhaled Plutonium

Chester R. Richmond

OAK RIDGE NATIONAL LABORATORY
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National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road, Springfield, Virginia 22161
Price: Printed Copy $5.00; Microfiche $2.25

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Contract No. W-7405-eng-26

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FEBRUARY 1976

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REVIEW OF JOHN W. GOFMAN'S REPORTS ON HEALTH HAZARDS FROM INHALED PLUTONIUM

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ABSTRACT

This document is a review of two reports prepared in 1975 by John W. Gofman on the subject of plutonium toxicity. Because Gofman's estimates of the calculated health effects from inhaled plutonium are significantly higher than those obtained from other analyses (including the risk estimates calculated by the National Academy of Science's Advisory Committee on the Biological Effects of Ionizing Radiation), it was decided to critically review Gofman's papers and supporting arguments.

This review concludes that Gofman's predictions of large numbers of plutonium-induced lung cancers from nuclear weapons testing are derived mainly from his incorrect assumptions about the effects of cigarette smoking on the retention of plutonium particulates in the tracheobronchial region of the lungs. It appears that Gofman's assumptions are considerably overstated and cannot be substantiated by currently available information. Also, Gofman's attempt to equate a given number of lung cancer deaths to a pound of plutonium tends to obfuscate rather than clarify attempts to derive risk estimates for inhaled plutonium.

INTRODUCTION

The following comments resulted from a review of two publications by John W. Gofman which are sponsored by the Committee for Nuclear Responsibility, Post Office Box 2329, Dublin, California 94566.

The first report is entitled, "The Cancer Hazard from Inhaled Plutonium," and is identified as CNR Report 1975-1-R and is dated May 14, 1975 (1). The second report is entitled, "Estimated Production of Human Lung Cancer by Plutonium from Worldwide Fallout," July 10, 1975, and is designated CNR Report 1975-2 (2). The R designation in CNR Report 1975-1-R represents a revision of an earlier version.

In essence, the basic arguments in the first of the Gofman papers (1) relate to the question of non-uniformity of radiation dose distribution in the lung and the question of impairment of normal physiological

clearance of the lung and damage to cilia because of smoking. Gofman, in his first paper, resorts to the use of 1 gram of ciliated respiratory epithelium as a target tissue at risk for carcinogenesis from plutonium exposure within the lung. Basically, he is reducing the volume of m (the mass of the lungs) from 570 grams to 1 gram or by a factor of 570. In the second paper (2), Gofman quotes his first paper as a reference source, "Since the lung cancers expected per microgram of plutonium inhaled are available," and references CNR Report 1975-1 (1). I think this point is especially interesting since the introduction to Gofman's first paper references the Tamplin-Cochran estimates for lung cancer from insoluble plutonium dioxide and then states that the problem of plutonium toxicity as regards the lung "has been clouded by needless polemic discussion of whether or not the 'hot particle' hypothesis (Geesaman) is correct." Gofman further states that the issue of carcinogenicity from insoluble plutonium dioxide particles in the lung can be approached in a straightforward manner without reference to "hot particle" theories. It would appear that Gofman is completely dismissing the hot particle arguments, yet it is not clear until one reads the paper that he obviously leans heavily as he derives his risk estimates upon the argument of a large reduction in the mass of the presumed critical target tissues within the lung. For example, on page 2 of reference 1, Gofman's step 2 requires the "analysis of the nature of the problem of non-uniform distribution of plutonium within the lung and the crucial problem of which cells in the broncho-pulmonary system are involved in human lung cancer production."

I find numerous problems with the exclusive use of the "relative risk method" for estimating the biological effects from irradiation of the lung or other tissue. Although there are instances where the effect of radiation may be multiplicative or even potentiating, the effect in most situations is no more than additive. Also, the concept of the "lung-cancer dose" leaves much to be desired. The dose-response relationship in this type situation is seldom, if ever, linear, yet the "lung-cancer-dose concept" requires a linear relation between dose and response.

Because of the numerous uncertainties in estimating or calculating radiation dose for human exposure to alpha radiation, such as in the case of uranium miners, and the lack of lung effects in humans exposed to plutonium in occupational situations, we must lean heavily on experimental animal data. A summary of information concerning plutonium-induced lung cancer in experimental animals in which the incidence of lung cancer is related to cumulative mean dose to the lung is given in WASH-1359 (3). The relationship which includes information for rodents, dogs and rabbits given various forms of plutonium is definitely non-linear. In addition, a recent publication entitled, "Radiation Carcinogenesis" (4), considers the question of observed dose-response relationships in experimental animals in some detail. It would appear that the true form of most dose-response curves for cancer induction is probably sigmoid. High-LET radiation dose-response curves are more nearly linear than those for low-LET radiation. However, many investigators feel that the data currently available are inadequate to allow one to determine which, if either, of the two dose-response curves is characteristic for high-LET radiation (4).

LUNG CANCER RISK ESTIMATES

The risk estimators used by Gofman are variants of the doubling dose which were the basis for some of the arguments he and A. R. Tamplin used several years ago (5,6). We know, however, that the spontaneous cancer incidence or mortality rate for a given kind of cancer is not the same for males and females in various countries at any given time. For example, the naturally occurring age-adjusted mortality rates for a malignant neoplasm of the lung, bronchus and trachea vary from a low of approximately 3 per year per 100,000 females in Portugal to about 78 per year per 100,000 males in Scotland (7). Although some might argue that cofactors, cocarcinogens, climatic conditions and smoking habits might interact with radiation in an additive or synergistic manner, I and others do not think it is reasonable to use a risk estimating system that allows for large differences in the predicted effect per unit dose

because of factors such as sex and nationality. I should also point out, however, that several organizations have in the past used the doubling dose concept in their considerations of radiation hazards. For example, the 1970 report of the National Academy of Sciences Radiobiological Advisory Panel--Committee on Space Radiation (8) used a modification of the doubling dose in estimating potential hazards to astronauts.

The Gofman article entitled "The Cancer Hazard from Inhaled Plutonium" (1) argues about the use of the absolute and relative risk methods as used by the BEIR Committee of the National Academy of Sciences (9). Actually, the BEIR report considered Gofman's doubling dose concept, as well as information concerning both the absolute and relative risk methods. The BEIR report did not unequivocally support either of the two methods. It is also interesting to note that Gofman compares his relative risk value of 2% increase in the natural incidence of lung cancer per rem per year with a value of 0.5% increase per rem per year as given by the BEIR report. Using this comparison, Gofman argues that only a factor of 4 exists between the BEIR estimate and his estimate. Actually, the relative risk estimate used by the BEIR report is documented as 0.29%. Therefore, the difference between the BEIR and the Gofman relative risk estimates is a factor of about 7 rather than a factor of 4. This difference results from the statement in the BEIR report (page 156) that in the final analysis, it is possible that the relative risk for lung cancer will reach 0.5% or higher.* Gofman obviously chose to use the value of 0.5% from this conditional statement instead of the 0.2% or 0.29% value recommended by the BEIR report.

Gofman's relative risk factor of 2% per year per rem derives from earlier reports with A. R. Tamplin that define the "doubling dose" for lung cancer as 50 rem (6). If one uses a quality factor (Q) of 10 for alpha particles, the Gofman-Tamplin doubling dose for lung cancer becomes 5 rad.

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*The BEIR report also notes a value of 0.2% (p. 171) for the relative risk (% increase in deaths per rem) for "all other cancers" which includes all cancers but leukemia.

The footnote on page 4 of Gofman's paper (1) relates the relative risk method to the spontaneous occurrence rate of the particular cancer under consideration. Apparently cancers resulting from smoking are considered as being "spontaneous" and perhaps also those resulting from other carcinogens. Indeed, if they were not so considered, one would expect the "radiation induced" cancers to be the same for groups of equal size. However, if lung cancers resulting from smoking and other causes are regarded as being part of the "spontaneous" cancer incidence, one might ask why the population is not further subdivided according to other carcinogens. It is difficult to formulate a correct definition of what is meant by "spontaneous occurrence rate" in Gofman's definition and hypothesis.

PHYSIOLOGY AND DYNAMICS OF LUNG CLEARANCE IN HUMAN SUBJECTS

Gofman claims that one of the effects of smoking is functional impairment or removal of cilia in the upper respiratory tract, which in turn results in a prolonged retention of plutonium dioxide particulates within the lung. Thus, instead of using a value of a few days for the clearance of plutonium particulates for the upper respiratory tree as recommended by the ICRP (10), Gofman selects a value of 500 days for the clearance half period and further states that the selection of such a value would not be at all conservative. The 500-day half-time value used by Gofman is recommended by the ICRP for clearance of plutonium deposited in the non-ciliated alveolar structures (10). Gofman's reasoning on this point is incorrect, and we do not agree with his use of a 500-day clearance time for the upper respiratory tree. For example, if this were true, the lungs of many heavy smokers would obviously become rapidly filling reservoirs for all sorts of atmospheric contaminants and particulates and perhaps, more important, if large regions (whatever anatomical reference this might have) were severely damaged by loss of cilia resulting in extremely long clearance half-times, the affected individuals would most probably drown in their own fluids.

Gofman, on page 24 of reference 1, admits that we simply don't know if the 500-day half-time for clearance of plutonium particles because of impaired ciliary function is reasonable. He further states that it is just as reasonable to expect an even larger (his emphasis) retention time as it is to hope for a shorter retention time. This I find to be an extraordinary statement.

Many examples exist on the state of our knowledge on the effects of smoking on respiratory physiology and anatomy (11-17). It is instructive, however, to consider a report by Albert et al. (18) on the bronchial deposition and subsequent clearance of aerosols in human subjects, some of whom were cigarette smokers and some of whom did not smoke. This and other studies suggest that there is little long-lasting effect of cigarette smoke on bronchial clearance time in man.

Ciliary action is but one of several mechanisms that work together to keep the airways clear. In fact, it is common practice to refer to the "mucociliary escalator" as the prime lung clearance mechanism. Gofman ascribes all clearance phenomena to ciliary action. Doubtless, smoking has some effect on clearance mechanisms but not to the extent assumed by Gofman. It is also known that clearance may be accelerated for certain smoking conditions rather than slowed down.

It is also clear that national and international radiation protection organizations have been aware of the potential effects (obviously not considered to be as severe as postulated by Gofman) from practices such as cigarette smoking on models used in radiation protection. In fact, the data on risk estimates for lung cancer as a result of radiation as given in the BEIR report (9) clearly show (page 150) that the summary of risk estimates for bronchial cancer is for adults only with cigarette smoking assumed to be characteristic of these populations. Page 150 of reference 9 shows the value of 0.29 for the relative risk (% increase in rate per year) per rem of mean bronchial radiation dose. The same table gives the absolute risk as one lung cancer case per 10^6 persons per year per rem.

VITAL STATISTICS DATA FOR LUNG CANCER IN HUMAN BEINGS

It is instructive to consider the vital statistics data for lung cancer as given in NCI Monograph 33 (19). For example, the age-adjusted death rate shows that the incidence of cancers of the bronchus, trachea and lung specified as primary sites (International Classification of Disease Code 162) for male whites in the United States has been essentially plateaued and unchanged since approximately 1960 at a value of approximately 20 deaths per year per 100,000 individuals. The data for non-white males may be rising slightly since 1960, but at a much slower rate than that observed during the preceding decade, perhaps in response to a myriad of socio-economic factors. It should be pointed out, however, that no similar leveling off effect has been observed for ICD 163 (lung, unspecified as primary or secondary) or for all cancers of the respiratory tract (ICD 160-164). For these categories, the incidence rate continues to increase throughout the period 1950-1967 (19). According to Gofman's predictions, the lung cancer rate should continue to climb.*

It is also instructive to look at the increasing rate of lung cancer for individuals in the United States for periods of time prior to the advent of plutonium availability in the early to mid-1940's. For example, data are also available on the time trends in cancer mortality rates, by site and sex, for the period 1930-1970 (7). These data show an increase in the rate of lung cancer for the decade prior to 1940, as well as several decades beyond 1940.

One can also consider the time trends in the lung cancer incidence rates by site, race and sex for surveys conducted by the National Cancer Institute in 1937, 1947, and 1969 (7). The annual incidence rates for white males for these three time periods per 100,000 individuals are

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*Gofman claims that about 10,000 people annually may be now dying throughout the world from plutonium-induced lung cancer (presumably as the primary site) related to nuclear weapons fallout and the total number irreversably committed to lung cancer death may rise to about 1,000,000 people in the Northern Hemisphere.

13.7, 29.5, 68.9 respectively. Thus, the 1947 cancer rate for white males was 2.15 times the 1937 level. Put another way, the lung cancer incidence during the decade following 1937 increased by slightly more than a factor of 2. However, for the two decades plus period between 1947 and 1969, the lung cancer incidence increased by a factor of 2.34. Therefore, if anything, we appear to be witnessing a relative reduction in the rate of lung cancer development as a function of time since 1947.

EXPOSURE OF OCCUPATIONAL WORKERS TO PLUTONIUM

It is of interest that more than half of Gofman's paper on the estimated production of human lung cancers by plutonium from worldwide fallout (2) considers two groups of occupational workers who were exposed to plutonium, namely the Los Alamos Manhattan Project workers (20-22) and the Rocky Flats workers (23). Gofman's major argument as regards the Manhattan Project workers is that it is not unreasonable that no cancers of the lung were reported by Hempelmann et al. (21) as a result of medical follow-up studies covering the period from the early 1940's until the last comprehensive medical tests in 1972.

Gofman believes that the nonoccurrence of lung cancers among the plutonium workers from the Manhattan Project (21) and the Rocky Flats fire (23) does not offer any strong indication that his present hypothesis is incorrect. Neither, however, does the absence of lung cancer offer anything to corroborate Gofman's hypothesis.

Hempelmann et al. (22) used vital statistics data (19) to estimate the probabilities of death from certain "normally occurring" cancer types over a 65-year period from age 20 to 85 for the Manhattan Project workers (21). Total probability per 100,000 deaths for ICD Code 162 (bronchus, trachea and lung specified as primary) was 3,023 for United States white males. Thus, for the group of 25 men, we would expect 0.76 death from lung cancer and should not assume a priori that the appearance of a lung cancer (should one occur) in the group means it is radiation-induced.

Ultimately, Gofman predicts a total cumulative incidence of 5.24 lung cancers as the life-time expectation for the Manhattan Project workers

reported by Hempelmann et al. (21). Of course, only time will provide the answer to this particular situation, and we should obviously study this cohort and others until death. According to vital statistics data as given in NCI Monograph 33, the age specific lung cancer death rate is approximately 115 deaths per year per 100,000 white males for age 65-74 whereas the corresponding rate is 34 per year per 100,000 individuals between the ages of 50-54 years (19). The difference for the two age groups is but a factor of about 4.

Gofman appears to like the ICRP estimate of 100 years for the retention half-time of plutonium in bone (24) as the basis for his argument that the original lung burden of the Manhattan Project plutonium workers, as extrapolated from urine bioassay measurements made in 1971, was in serious error. It is instructive, however, to consider the amount of plutonium retained in the skeleton after 30 years if the biological half-life is not 100 but 1,000 years. The amounts retained for half-lives of 100 and 1,000 years are 81.2 and 97.9% respectively at 30 years, the total amount of plutonium lost from the bone over the entire 30-year period being 18.8% (1.9×10^{-5} per day) for the 100 years biological half-life case and 2.1% (1.9×10^{-6} per day) for the 1,000 year half-life case. The excretion fraction at 30 years based upon the Langham power function equation for plutonium retention is about 2.1×10^{-6} per day. Recent data from the Los Alamos Scientific Laboratory suggest that the plutonium excretion rate for human subjects becomes fairly constant with time after the initial rapid drop during the early years following exposure (25).

The 100-year retention half-time for plutonium in bone (24) is based upon a log plot of observed retention half-times for 5 mammalian species (including man) as a function of body weight. The actual value for man from the original Langham work is 200 years and the value extrapolated to man from the least squares analysis of the other data points (man excluded) is about 40 years.

There exists other information, however, which suggests that Gofman's suggested correction factor is perhaps in the right direction. For example, at the July 1974 international meeting of the Radiation Research Society, it was reported that: "Results of the tissue sampling programs for occupationally exposed plutonium workers have also given the opportunity to compare

the body burden found at autopsy with that estimated during life on the basis of bioassay data. Almost without exception, workers in the United States and the United Kingdom have found less plutonium by a factor of approximately 10 at autopsy as compared with that amount predicted by bioassay data. Thus, it would appear that estimates of the body burden made during life are conservative in that they predict more plutonium than is actually present in the body. Because a considerable amount of data on this subject is now available, it might be profitable for responsible persons in the radiation protection areas to evaluate this finding in terms of current radiation protection practices and guides followed in the nuclear energy industry" (26). This statement was based upon the observations of several groups of researchers in the United States and the United Kingdom (27-28). The factor of approximately 10 may be somewhat on the high side based on information available in the recent Annual Report of the Los Alamos Scientific Laboratory's Biomedical and Environmental Research Program (25). Estimates of the body plutonium content as extrapolated from tissue analyses can be compared with the body burden estimates based upon urine radiochemical analyses for 16 individuals (25). The ratios of the body burdens (urine assay/tissue extrapolation) range from 0.5 to 155.0. Sixty-three percent of the body burden ratios have a value of 10 or less and 38% have a value of 5 or less.

Similar information obtained from nine autopsy cases in the United Kingdom yield body burden ratios (urine assay/tissue extrapolation) that vary from 1.2 to 8.3 (28). Similar information on the over-estimation of body burden from urine assay has been reported by Lagerquist et al. (27).

Incidentally, it is also interesting that Gofman did not mention a direct comparison of case No. 2 reported by Hempelmann et al. (21). In this specific instance, the agreement between the body burden based on urine bioassay or on the basis of assay of various tissues, including bone, was quite good. The plutonium systemic body burden based upon urine radiochemistry was about 0.03 μCi as compared with a value of about 0.016 μCi based upon actual measurements of plutonium in bone (and assumptions regarding the relative amounts in bone and liver). This particular case is interesting since it represents a period of almost 30 years after a contaminating event.

Gofman estimates that the Rocky Flats workers exposed to plutonium during a fire in 1965 will ultimately produce 19.3 lung cancers as a final corrected life-time expectation (2). I agree that this cohort should be studied very carefully for the rest of their lives. I do not agree with Gofman's inflated estimate of the number of lung cancers that will develop in this group of individuals, since the lung cancer estimate is based on incorrect assumptions developed earlier (1).

EFFECTS OF PLUTONIUM IN FALLOUT AND LUNG CANCER

In an earlier section of this critique, I pointed out that Gofman claims that thousands of the lung cancer deaths presently occurring throughout the world are the result of plutonium contained in fallout. I also pointed out that the age-adjusted lung cancer (ICD Code 162) death rate per 100,000 white males in the United States has been quite constant for the last fifteen years and that the rising incidence before 1960 was observed prior to 1945. In addition, observations on the static geographic distribution of cancers of the bronchus, trachea and lung (primary site) show that except for Nebraska and Illinois, increased death rates are scattered in coastal states with a general decrease in death rates in the central United States for male whites (19). The dynamic geographic distribution of lung cancer shown in NCI Monograph 33 indicates for male whites a clustering of states with relative increasing death rates, as well as a cluster of states with decreasing death rates in the central Midwest (19). These observations are not in concert with Gofman's predictions of an increasing incidence of lung cancer as a result of exposure of human populations to fallout containing plutonium-239.

Gofman also refers to the work of Bennett (29) in deriving his health effects estimates from fallout plutonium. However, Bennett uses parameters obtained from the ICRP to compute fallout plutonium body burdens, which he then compares with measured burdens determined from autopsy cases. I find it extremely interesting that the agreement is quite good in that Bennett computes plutonium body burdens of 2.6 pCi as compared with measured burdens of 3.2 pCi for Colorado-New Mexico autopsy cases (1970-1971). The values

for the lung in both cases are exactly the same and have the value of 0.3 pCi. I think the agreement is remarkable, especially considering the fact that Gofman accuses the ICRP and other organizations of not using correct values in their lung and other metabolic models.

The summary and conclusion section of Gofman's paper (1) states that there are 7.83×10^9 "lung cancer doses" per pound of plutonium. He neglects to point out in this section, however, that this estimate--if true--is per pound of plutonium deposited in the lung. The words deposited in the lung make a great difference because only a small amount of the plutonium released into the environment from atmospheric nuclear weapons tests has appeared in mankind (about 10^{-8}).* Otherwise, the unsuspecting reader might incorrectly calculate 10^{13} to 10^{14} lung cancer deaths for the five to seven tons of plutonium produced as weapons fallout. (There are about 3×10^9 people on earth.)

Perhaps another observation is of interest as regards fallout contamination of human subjects. It has been estimated by several individuals that approximately 0.3 to 0.4 Megacurie of fallout plutonium has returned to the earth's surface (24,29). By assuming the 0.4 MCi value, one can calculate that approximately 10^{-8} has found its way into the 3×10^9 people on earth. The 0.4 MCi (4×10^5 Curies) is equivalent to approximately 7 tons of plutonium-239.

The contemporary body content of each individual on earth is roughly 5 picocuries (24,31). Thus, we can estimate that approximately 15×10^{-3} Curie or 0.25 gram of plutonium-239 has found its way into all of mankind (5×10^{-12} Curie $\times$ 3×10^9 people). It can also be estimated that only about 10^{-8} of the earth's inventory has found its way into mankind (15×10^{-3} Curie/4×10^5 Curies).

For simplicity, one can assume that all the plutonium entered man via the lungs. THUS, TO OBTAIN ONE POUND OF PLUTONIUM DISTRIBUTED THROUGHOUT THE BODIES OF ALL MANKIND, ONE WOULD NEED TO START WITH 10^8 POUNDS OR ABOUT 500,000 TONS OF PLUTONIUM RELEASED TO AND DISTRIBUTED

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*Or about 10^{-17} of the amount released has found its way to man. A similar value was reported by Bair and Thompson (30).

THROUGHOUT THE ENVIRONMENT!!! This amount would correspond to about 9×10^{-9} Ci per person.

We have seen no cases of lung cancer in the Manhattan Project workers, some of whom have chest burdens of plutonium in the neighborhood of 10^{-8} Ci some 30 years after exposure (21). Autopsy studies in the United States currently indicate approximately 4×10^{-13} Ci as the contemporary lung burden of plutonium from fallout which is 25,000 times smaller than the 10^{-8} Ci for the plutonium workers. On this basis alone, we would not expect to see disastrous biological consequences of the order predicted by Gofman for plutonium in nuclear fallout.

Gofman calculates that 116,000 people in the United States and about 1,000,000 people in the Northern Hemisphere have been irreversibly committed to plutonium-induced lung cancer as the result of fallout from nuclear weapons testing. For purposes of comparison and to place the issue into proper perspective, it is interesting to calculate--by Gofman's methods--the number of people who would die of lung cancer from naturally occurring alpha radiation in the environment.

In his step 1 calculation, Gofman claims that one lung cancer death (LCD) can be equated to 1,310 man-rem (or 7.6×10^{-4} LCD per life-time per man-rem). The LCD is claimed to result from the deposition in the lung of 10.8 μg (0.66 μCi) ^{239}Pu. After making adjustments for estimates of the quantity of critical tissue irradiated and the effects of smoking on lung clearance, Gofman calculates the LCD for smokers and nonsmokers to be 0.058 μg (0.0036 μCi) and 7.3 μg (0.45 μCi), respectively. Note that the final LCD value for smokers is a factor of 186 lower than that obtained in his step 1 calculation. However, Gofman developed the use of LCD's per pound of plutonium between his step 1 and step 3 calculation. This makes it difficult to compare his predicted effects with those from absorbed radiation dose arising from sources such as background radiation.

To simplify the comparison with background radiation (alpha), one can use Gofman's estimate of one LCD per 1,310 man-rem. Barr (32) recently estimated, on the basis of information contained in the 1972 United Nation's Scientific Committee on the Effects of Atomic Radiation (33), that the average burden of naturally occurring alpha-emitting

radionuclides in the U.S. population is about 0.1 Ci. This quantity delivers about 10^7 man-rem per year (lung dose) to the U.S. population or about 50 millirem per person per year. Thus, using Gofman's value of one LCD per 1,310 man-rem, we would expect 7,633 LCD's from the 10^7 man-rem annual radiation dose to the U.S. population from natural background (alpha). Over the 30-year period which Gofman uses to accrue 116,000 LCD's from nuclear weapons fallout, one would calculate that 229,000 LCD's (30 × 7,633) would occur.

It is also instructive to compare the predicted number of LCD's from weapons fallout or natural background (alpha) radiation with the total number of LCD's now recorded in the United States. The annual rate is about 84,000 LCD's per year, so one would expect about 2,520,000 LCD's over the same 30-year period, provided the rate did not change.

LOCATION OF LUNG CANCERS

Gofman assumes that lung cancers resulting from plutonium exposure will arise in the critical lung tissue (one gram) of the respiratory tract where he predicts physiological or anatomical impairment from cigarette smoke. We need to have a more careful assessment of the question of where plutonium-induced cancers develop in the lung. We have no relevant human data, so we must resort to experimental animal data. It does appear that lung tumors resulting from plutonium alpha irradiation may develop in the periphery of the lung rather than in the upper respiratory tract (34-38).

SUMMARY AND CONCLUSIONS

1. Gofman's contentions regarding the number of lung cancers that will be produced from plutonium-239 contained in nuclear fallout are greatly exaggerated, as are his underlying arguments which are based upon a variation of the doubling dose concept and speculations concerning impairment of lung clearance as a result of cigarette smoking.

2. The foreword of Gofman's first paper (1) clearly states (paragraph 2) that there "are certain critical voids in mankind's knowledge of the physical and physiological parameters which determine the dosimetry and thus we have made necessary assumptions which are all clearly identified." A careful analysis of the Gofman paper (1) shows that the assumptions, although clearly identified at times, are incorrect. Therefore, subsequent calculations of the number of cancer deaths from plutonium in fallout and from theoretical releases from the nuclear economy are not valid.

3. We estimate that Gofman's risk estimate differs from those of the National Academy of Sciences BEIR Committee by a factor of 4-10 for relative risk and perhaps a factor of 20 for absolute risk (which Gofman rejects).

4. Gofman's assumptions concerning the value used by the ICRP for lung clearance from the ciliated portions of the respiratory tract appear to be overstated and at variance with our knowledge of respiratory clearance mechanisms. Gofman attributes all clearance to ciliary clearance mechanisms and argues that smoking impairs clearance by destroying cilia.

   Gofman introduces a "correction factor" of about 100 at this point, making a total "correction factor" of from 400 to 1,000 if one uses relative risk models to predict the number of lung cancers from plutonium exposure.

   If one uses the absolute risk model, which Gofman rejects, Gofman's estimates of lung cancer risk would be high by a factor of roughly 2,000.

5. Gofman's concept of "lung cancer dose" stated in terms of "per pound of plutonium" is very misleading. Gofman states on several occasions (e.g., ref. 1, p. 9 and p. 26) that the lung cancer dose is related to "fatal lung cancers per pound of deposited plutonium."

In fact, in one instance the word "deposited" is underlined by Gofman for emphasis (ref. 1, p. 26). However, because of the rambling nature of Gofman's papers, this important point can easily be overlooked by many readers.

6. It is difficult to envision a pound of plutonium being deposited (and retained) in the lungs of man since the transport to man from plutonium released into the environment is so inefficient. Only 0.25 gram (0.00055 pound) of plutonium has gotten into all the earth's inhabitants from the approximately 7 tons (14,000 pounds) that were released during atmospheric testing of nuclear weapons. To get an entire pound deposited in mankind, we would need to release some 500,000 tons of plutonium to the environment!!! Gofman does not point out the large discrimination factor (10^8) representing the amount of plutonium in the environment as compared with the amount that gets incorporated into humans via inhalation.

7. Gofman assumes a release factor of 10^{-4} for the amount of plutonium that might find its way into the environment. Again, Gofman's estimate appears to be too large when compared with other estimates and, consequently, magnifies the calculated numbers of lung cancer deaths which would "result" from the release.

ACKNOWLEDGEMENT

This Review includes some comments from W. S. Snyder, J. A. Auxier and P. Nettesheim.

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