

UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

BEFORE THE ATOMIC SAFETY AND LICENSING BOARD.

In the Matter of)
PACIFIC GAS AND ELECTRIC COMPANY) Docket Nos. 50-275 O.L.
(Diablo Canyon Nuclear Power Plant,) 50-323 O.L.
Units Nos. 1 and 2)

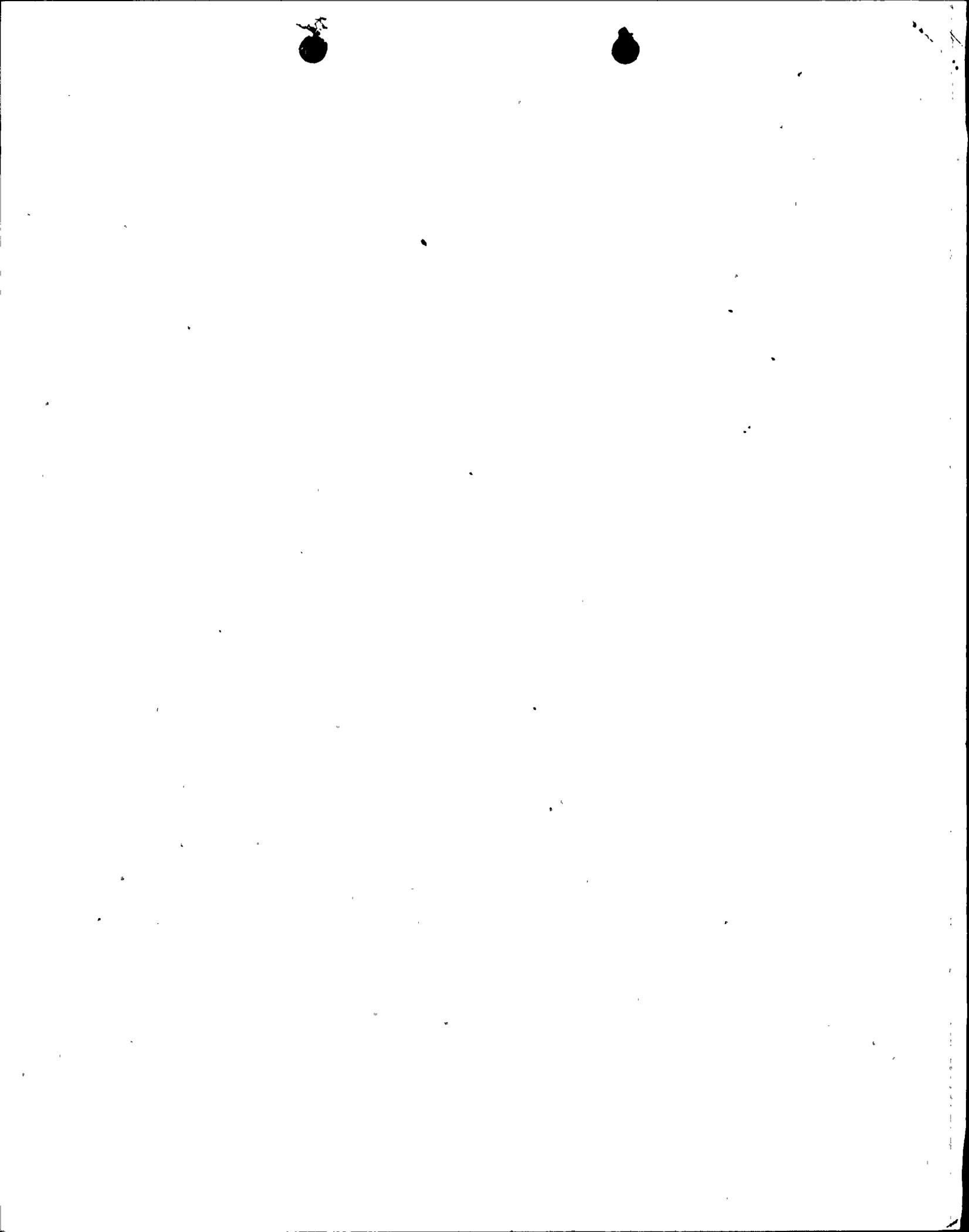
TESTIMONY OF ROLAND FINSTON
CONCERNING CONTENTIONS 4A, 4C, and 4D

12-8

1. I am a health physicist and am employed at Stanford University, Stanford, California as Acting Director of the Health Physics, Safety and Health Office and as a lecturer in Nuclear Medicine.

2. My professional qualifications include a Bachelor of Science in Physics from the University of Chicago in 1957; a Master of Science in Health Physics from Vanderbilt University and Oak Ridge National Laboratory in 1959; and a Doctor of Philosophy in Biophysics from Cornell University in 1965. I was an Associate Professor of Radiological Physics at Oregon State University in 1965-66, and I have been employed at Stanford University since 1966 as a health physicist. I have specialized in medical health physics and in this specialty have taught radionuclide dosimetry and have also been responsible for calculating the radiation dose to patients which results from purposely administered radiopharmaceuticals. My work in radionuclide

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dosimetry has involved analyses of the physical and biological properties of newly recognized radiopharmaceuticals; specifically, my research has involved the emissions, biological distribution, retention and resulting radiation dose to humans of such radiopharmaceuticals. For purposes of benefit/risk evaluations, I have also been involved in the evaluation of the somatic and genetic hazards posed by the resulting doses of such radiopharmaceuticals. I have done research on the effects of external radiation on metabolism in bones and used X-ray degradation to study the properties of DNA.

I am a member of the University's Human Use Radioisotope Committee which is also approved by the FDA as a Radioactive Drug Research Committee, and have been responsible for dose and risk calculations resulting from administration of radioactive materials in medical research. I have similar responsibilities for external irradiation from X-ray used in medical diagnosis.

3. The purpose of my testimony is to present the Intervenors' position regarding the inadequacy of the NRC Staff's Final Environmental Statement [FES] as it has assessed the environmental costs, doses, and effects of low level radiation as to (A) the buildup of concentration of radioisotopes in the food chain; (C) the somatic effects, including incidences of human cancers, leukemias, and infant mortalities and genetic



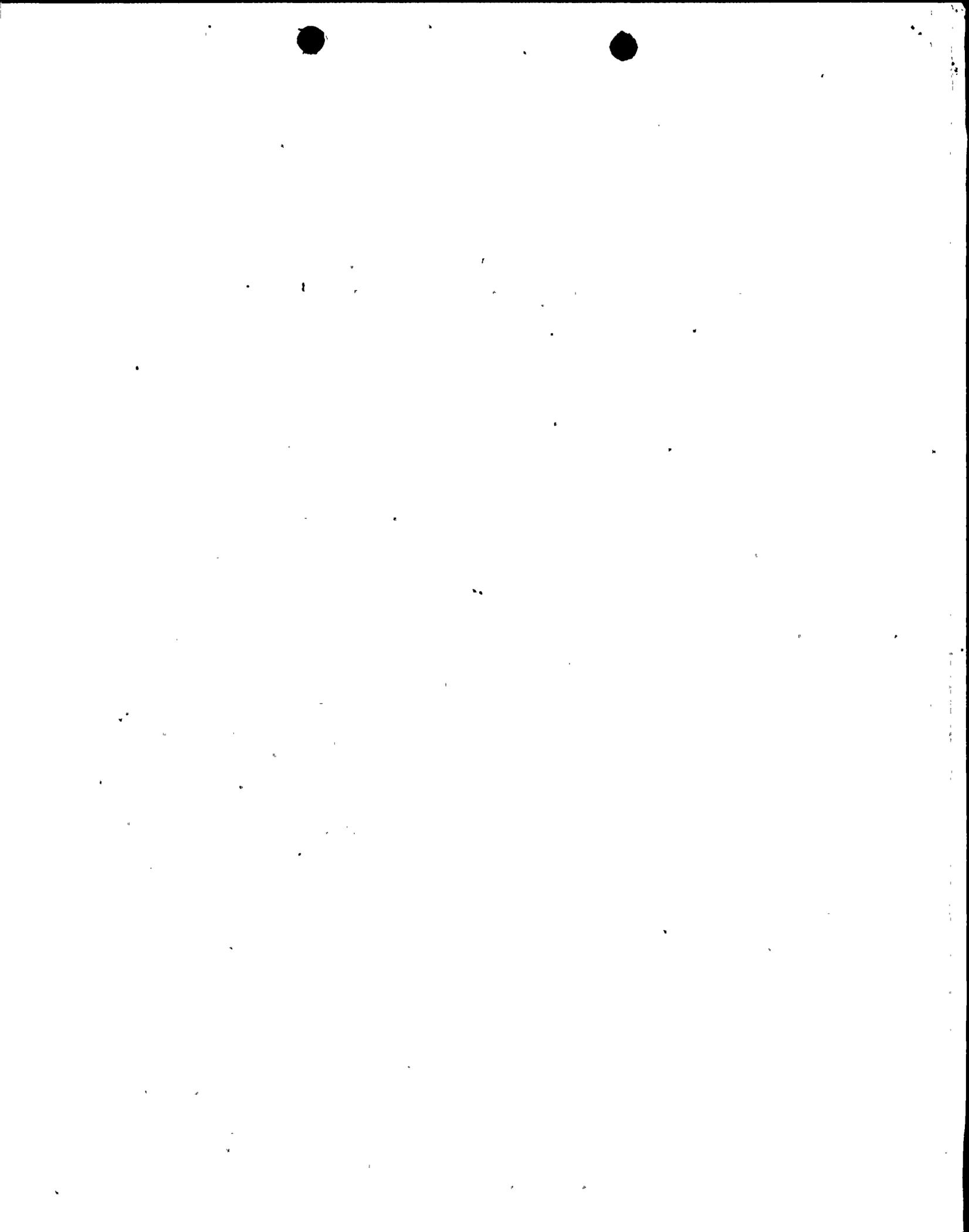
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effects of routine releases on the population within a 50-mile radius of the plant; and (D) the somatic and genetic effects on plant personnel including inadvertent ingestion of radioactive materials. My evaluation for each contention will be taken in turn.

Contention 4A -- Buildup of Radioisotopes in the Food Chain.

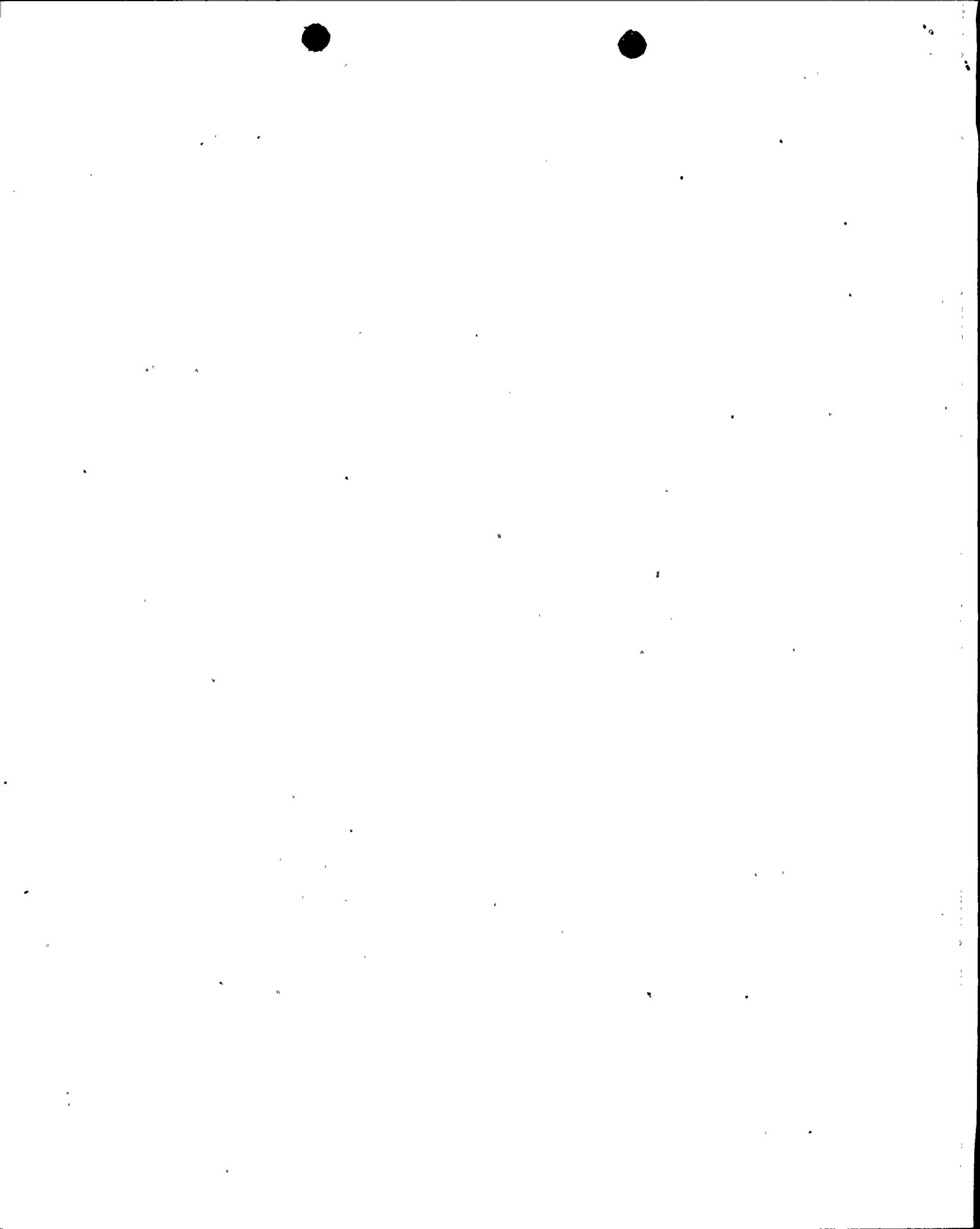
4. The Appendix I considerations (Letter from Edward Ketchen of the NRC Staff to James Geocaris, dated September 21, 1976, and repeated in the Supplementary Testimony of NRC Staff, Messrs. Parsont and Boegli; hereafter "Letter") appear to contain two anomalous values of estimated radioactivity releases which indicate, in one case, that releases may not be as low as readily achievable, and in the other, that calculated doses may be underestimated.

5. Specifically, it is calculated (Table 2 of Letter) that 710 Ci/year/reactor of H^3 are to be released as gases. This corresponds to 788 Ci/year/reactor if the value of 710 Ci/year/reactor is normalized to a 1000 MW plant operating at 100% capacity factor. In contrast, data from PWR units of similar size and manufacturer have been performing with much smaller H^3 releases. Reported releases from 6 reactors during the years 1973-74^{1/} have been averaging 1/20 as great as that estimated for the DCNGS units, which estimate was also normalized



for rating and capacity factor (Table 1). This conclusion was based on an average of the range of H^3 emissions from 6 reactors, from 4.35 to 94.2 Ci/year. Only the Zion reactors have approached, but still to less than 50%, the DCNGS estimate (normalized for rating and capacity factor). While this overestimate of release has resulted in an overestimate of dose, it is of concern to know why this is so. It is unclear, thus, whether the applicants intend to keep the gaseous releases of H^3 as low as readily achievable as required by 10 CFR Part 50, Section 50.34a. The experience at other similar operating reactors would seem to suggest that a lower release of H^3 is possible.

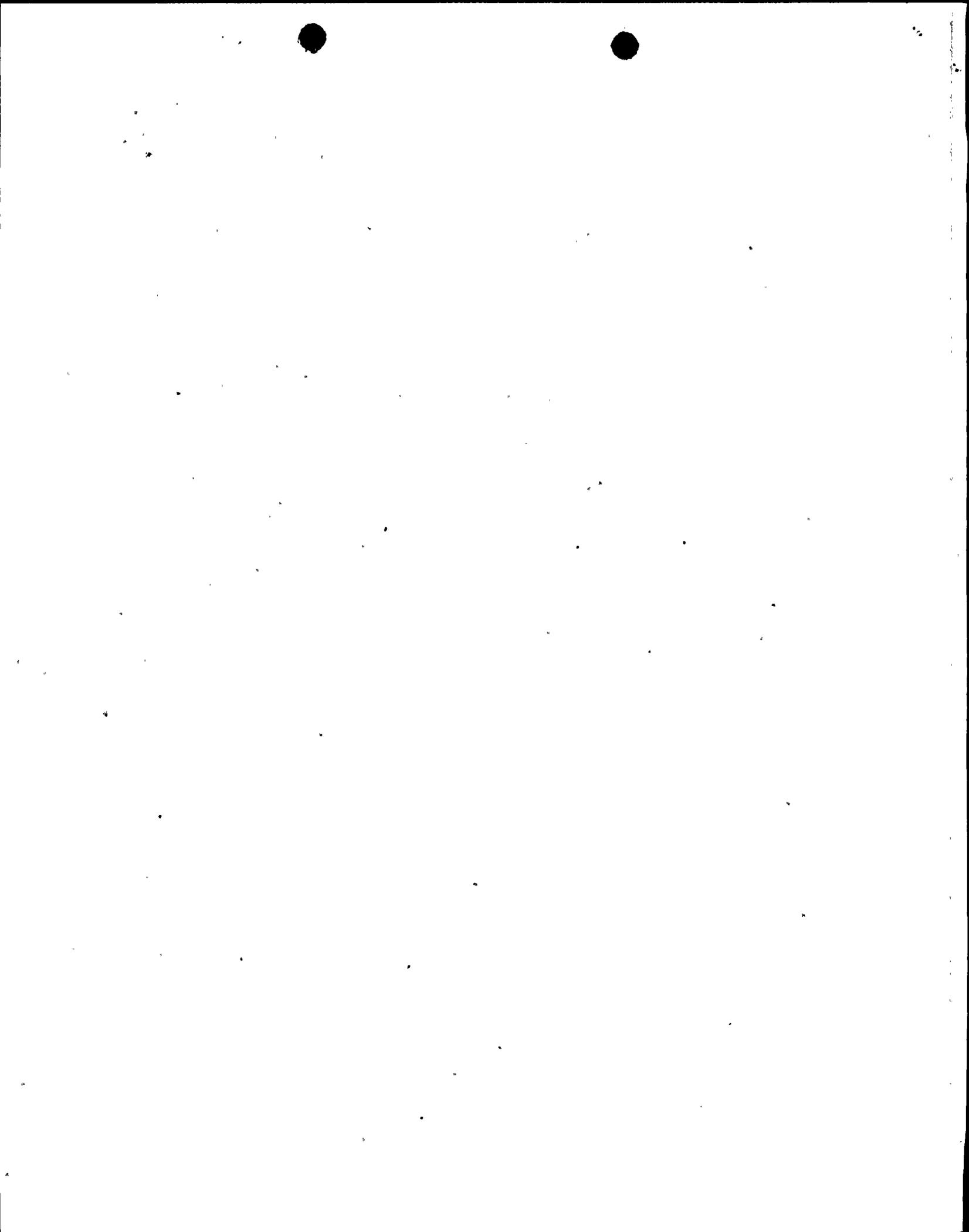
6. The other anomaly concerns the release of fission products, corrosion and activation products to liquid effluents. Performance data for 8 PWR's operating in the years 1973-74^{1/}, normalized to rating (1000 MW) and capacity factor (100%), show releases 5 times greater than the figure estimated for the DCNGS units, also normalized to rating and capacity factor. The five-fold difference (0.38 vs. 1.88 Ci/year) was calculated (Table 1) by averaging the releases at eight PWR's, a range from 5.61-0.33 Ci/year/reactor, and by comparing it to the estimate given in Table 2 of the Letter. In fact, one PWR (Indian Point 2) released 30 times more fission products and corrosion and activation products to liquid effluents than are estimated for the



DCNGS. (This unit was deleted from the above analysis on the assumption that this represents abnormal operations). It would appear, then, that the estimate of 0.34 Ci/year/reactor is unrealistically low in comparison to other reactors whose emissions have been calculated. A realistic release value would in turn result in a realistic dose, which is needed to adequately assess the impact of low level emissions that the DCNGS will have on the food chain and consequent population doses.

7. Furthermore, my analysis of performance of PWR's indicates that with increasing age of PWR plants, environmental releases increase. This conclusion is based on my examination of the emission data for other PWR's reported over several years. I have requested the emission data from the NRC for 1975 in order to more thoroughly evaluate this trend; conflicting reports as to the availability of that data have been received and have thereby delayed my obtaining it. Another consultant has been contacted by the Intervenors and will obtain the data prior to the hearing so that an analysis of this observed phenomenon can be presented.

At any rate, neither the FES nor the calculations pursuant to App.I appear to have taken this phenomenon into account in indicating dose levels over the years, and in this respect, the assumptions for the DCNGS reactors may be unduly optimistic.



Contention 4C -- Somatic and genetic effects on Population
within 50 miles

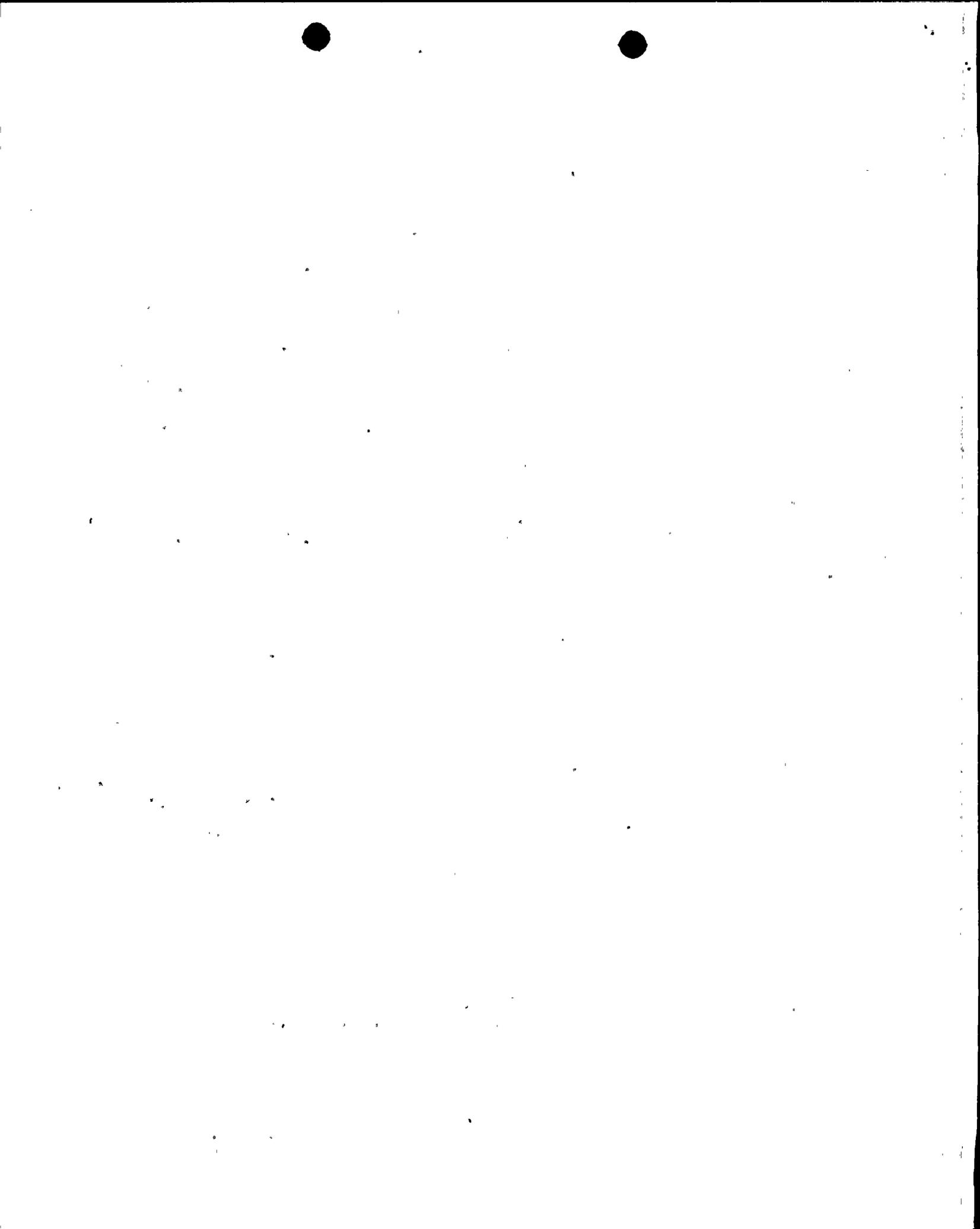
8. The NRC consultant's analysis of the background radiation and corresponding rate of cancer deaths on the 260,000 person population living within a 50-mile radius of the DCNGS is incorrect. It was assumed^{2/} that the background radiation for the 260,000 person population was 30,000 man-rem/year. Data specific to the region (Santa Barbara being the closest area for which figures are available) indicate that the combined internal and external dose of radiation is 82 mrem/year, comprised of 41 cosmic, 23 terrestrial, and 18 mrem/year internal radioactivity^{3/}. The population's corresponding background radiation is 21,000 man-rem/year ($82 \text{ mrem/year} \times 260,000 \text{ persons} = 21,000 \text{ man-rem/year}$). The effect of this lower background radiation figure is to make the relative impact of low level radiation from the DCNGS higher than that originally offered in the Goldman Testimony. Accordingly, the FES should consider data which is more pertinent to the geographical area in which the reactor is located, and take into account the comparative effect of the DCNGS against a lesser background radiation rate.

9. The National Academy of Sciences (BEIR) report of 1972 actual estimate of the most likely rate of cancer deaths was 152-204 deaths/million man-rem, and not 89, as is stated in the

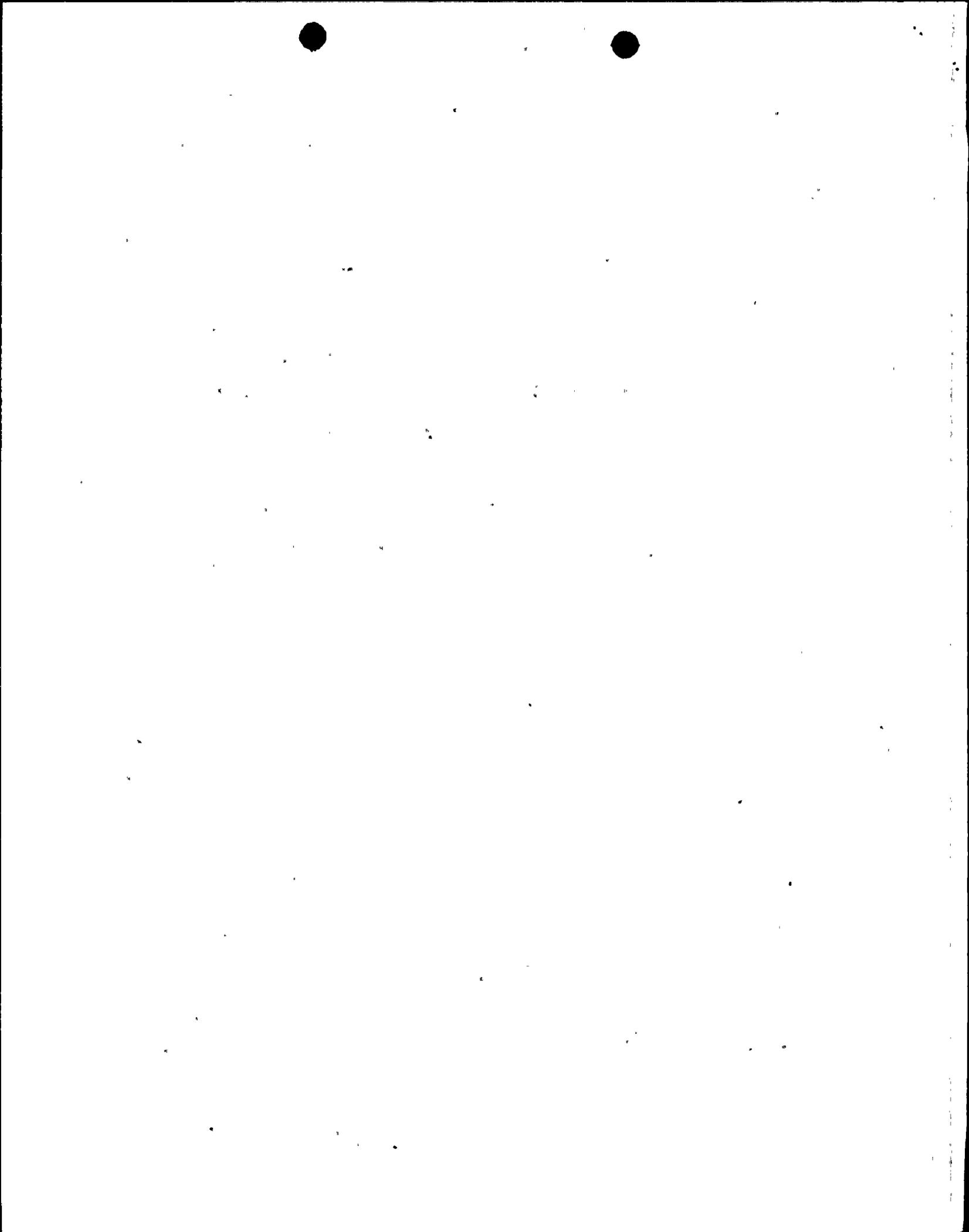


Goldman Testimony at p. 4.^{4/} . . . The NRC study relied on in the Goldman Testimony only based its figure of 89 on the BEIR Study; available data should be considered rather than an extrapolation or interpretation of that data. Accordingly, using the figures of 152-204 cancer deaths/million man-rem due to low level emissions from the DCNGS, together with the dose computed for Diablo Canyon effluents (0.3 man-rem/year, Letter, Enclosure 4) the risk is still increased by a factor of at least 2 from that reported in the Goldman Testimony.^{2/} (0.3 man-rem/year x 152 to 204 cancer deaths/million man-rem = 5.3×10^{-5} risk, double the figure of 2.7×10^{-5} given at p. 4 in the Goldman Testimony^{3/}). The risk factor of 5.3×10^{-5} (as properly calculated, using correct BEIR data) is not conservative, as the Goldman Testimony notes, p. 4, but in fact is recognized as the best estimate currently available. In fact, the BEIR best estimate of the number of cancer deaths per million man-rem may be non-conservative by a factor of 2, if the relative risk model applies. That model would predict twice as many cancers to occur from a given dose than does the absolute model^{4/}. The effect of "conservatizing" the BEIR data is to further increase the risk factor of 5.3×10^{-5} by a factor of 2, i.e., to 1.1×10^{-4}

10. Moreover, the conclusion that radiation absorbed at millirem per day levels is one-fifth as damaging as when absorbed at rem per day levels is considered by the EPA to be not a prudent



choice for central risk estimates^{5/}. The basis for the conclusion in the Goldman Testimony that the risk of radiation at low level emission rates is proportionately less than at higher, i.e. rem per day, levels is the conclusion that "molecular repair and reconstitution of initial 'lesions' can take place...such that the yield of 'effects' per unit dose is lower than when the dose or rate is high..." (Goldman Testimony at 5). This conclusion is surprising, since the concept of reconstitution of radiation lesions in genetic cells at low doses and dose rates ascribed by Goldman to S. Abrahamson is interpreted oppositely by the Environmental Protection Agency^{6/}. That report indicates that in mouse oögonia the reduced genetic effects observed are due to cell death rather than to cellular repair mechanisms. In other words, fewer cells indicate genetic mutation at low levels of radiation, because those cells which would contribute to genetic mutation do not survive. This conclusion in no way indicates that low level emissions are somehow "less damaging" than high levels, an indication which is relied upon in the Goldman Testimony. It is improper, therefore, to reduce a risk factor by five simply because the source is a low-level emitting source. The EPA report would support the conclusion that the risk factor at low level emission rates is as great as high level (i.e. rem per day) emission rates. Accordingly, the risk factor of 1.1×10^{-4} is the factor arrived at using conservative data. Although the radiation risk may still be negligible (i.e. 444 cancer deaths increased to 444.00011 deaths due to

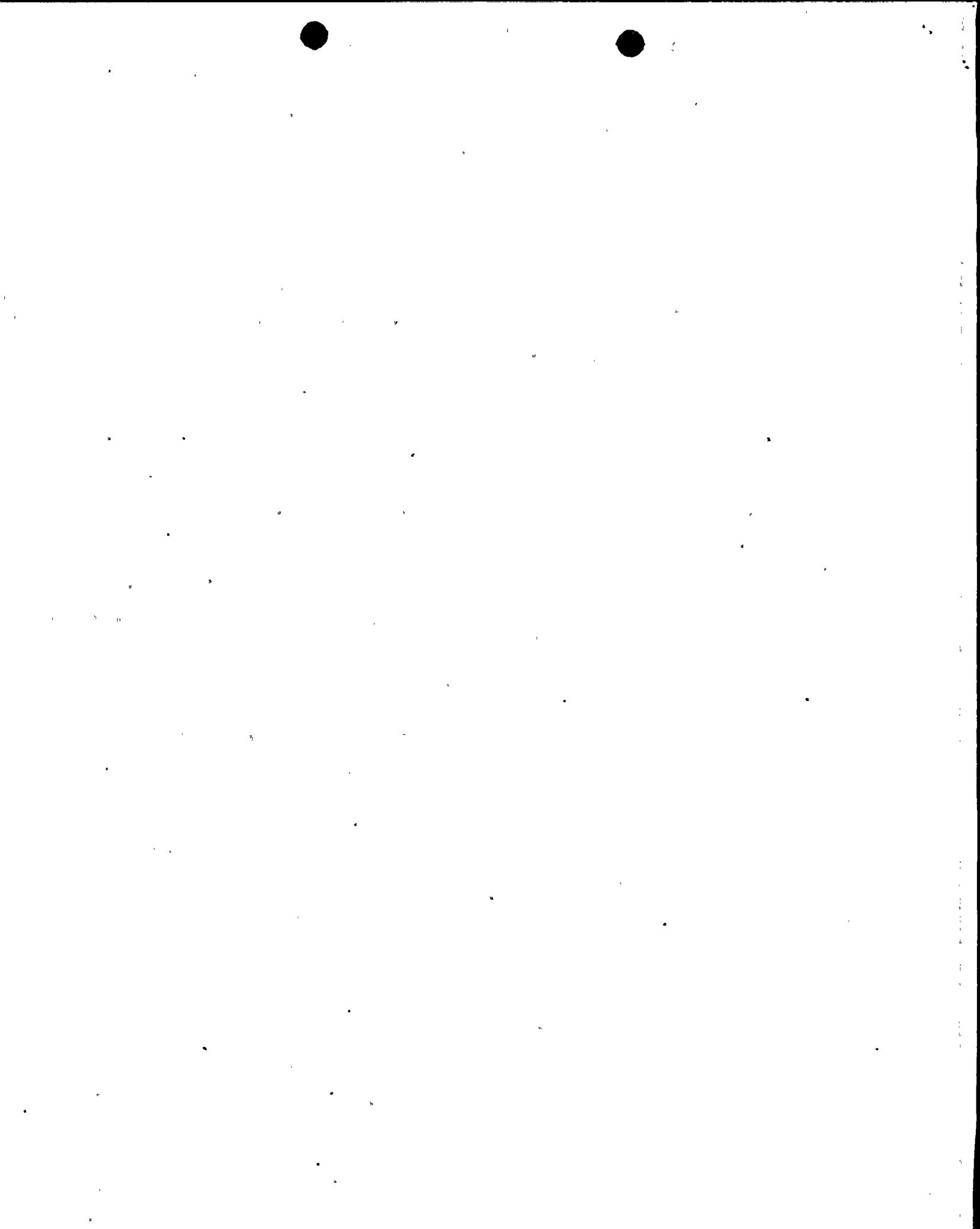


operation of the DCNGS), the effect of this increased risk factor, when combined with emissions from other sources (accidents, waste, transportation) should be considered in the FES.

11. There is, moreover, evidence which indicates that risk factor at low levels and low doses may in fact be higher than the factor derived linearly from high doses experience^{7/}. For three different human tumors (thyroid carcinoma, leukemia, and breast cancer), the risk estimates at low doses or at low dose rates are either the same as, or in some cases, perhaps higher, than risk estimates derived from high doses. This information indicates that the 1.1×10^{-4} risk factor for DCNGS may be too low and that the cancer deaths may be correspondingly higher than predicted.

12. More troubling is the NRC consultant's and Staff's myopic risk evaluation brought about by standardized calculations of annual doses, without taking regard for the total impact of long-lived emissions.

Table 2 of the Letter indicates that the release of Carbon-14 for Diablo Canyon will be 8 Ci/year/reactor. The approach taken by the NRC Staff to calculate the radiation dose per one year's decay of Carbon-14 is inadequate, because this figure only calculates the dose from only 0.012% of the radioactive atoms released by the plant. This vastly underestimates the total radiological impact of the long-lived radionuclide, whose average life



is 8,270 years. The concept of dose commitment has been recognized by an international expert committee as the appropriate means of evaluating the hazards of long-lived nuclides released to the environment^{8/}. The NRC also has adopted the concept (10CFR Pt. 50 App. I, Sec. II-A). For example, the Environmental Protection Agency (EPA) calculates that Carbon-14 is the major contributor to serious health effects of the nuclear fuel cycle, and may be responsible for 88% of the effects attributable to that cycle^{9/}. Using the EPA estimate, it can be shown that as the result of nuclear generation of two gigawatt-years of electric power (one year's operation of both DCNGS reactors at 90% capacity) there will be 3 serious health effects perpetrated in the first 100 years of Carbon-14 radioactive decay. Extrapolation beyond the first century is uncertain; but an upper limit, based on physical decay alone, would be 250 health effects to the complete decay. Future generations will pay the toll as the result of each year's routine release at DCNGS. The FES has therefore not adequately assessed the health effects attributable to this long-lived radionuclide.

Contention 4D -- Somatic and Genetic Effects on Plant Personnel.

13. The FES inadequately evaluates the impact of low level radiation emissions as to the somatic and genetic effects on personnel working at the plant. It is not clear from the Staff's



materials how the figure of 500 man-rem per year per unit for occupational exposures of plant employees was computed. Based upon operating experience during the years 1969-1974, a figure of 1.45 man-rem per Megawatt-year for occupational exposures of PWR plant employees would seem more accurate for the DCNGS reactors^{10/}. (The range is from 0.66 to 2.39 man-rem/megawatt year; the 1975 figure is 0.74 man-rem/Megawatt year)^{11/}. The dose to workers at the DCNGS station is therefore computed as follows:

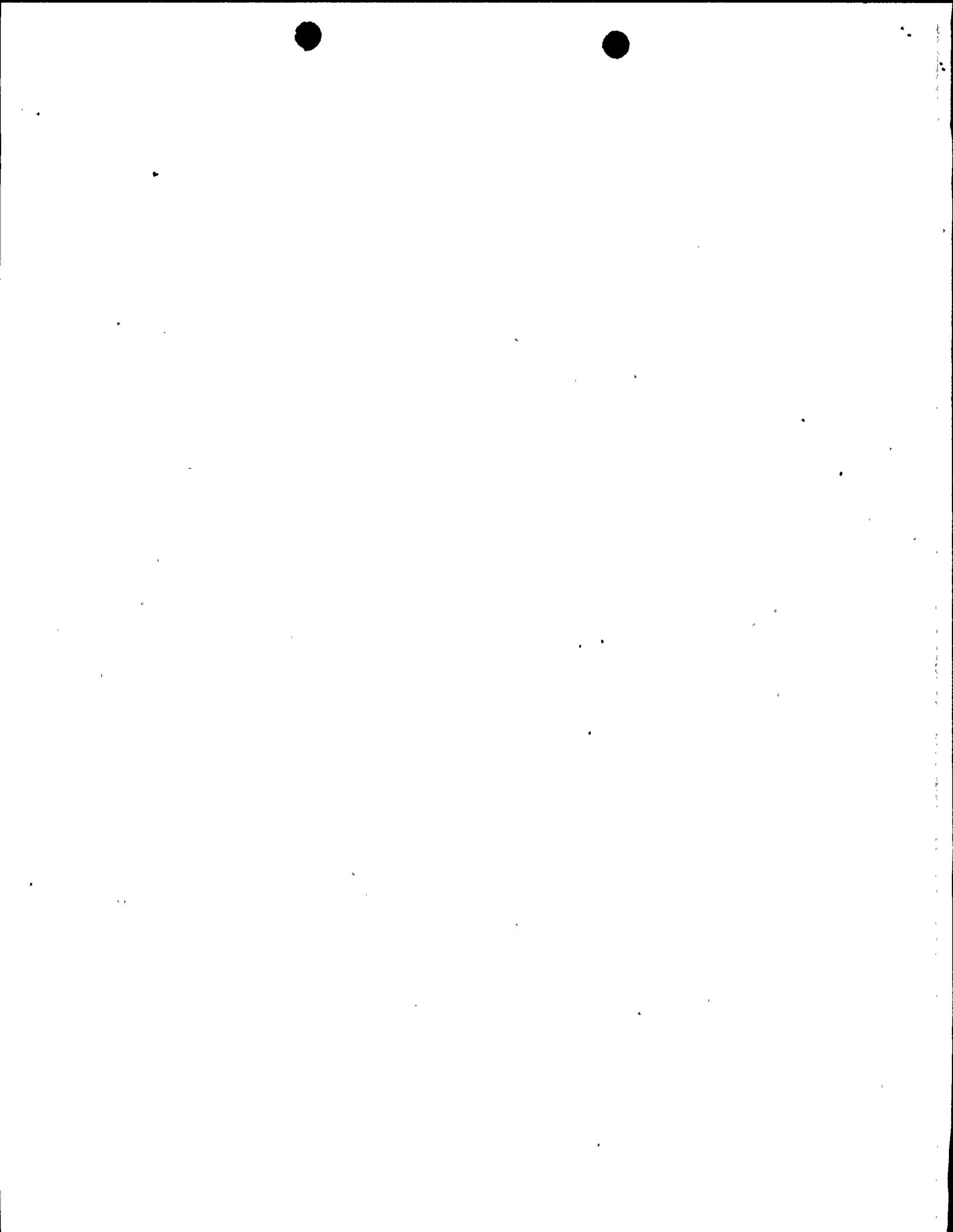
$$\begin{array}{rclcl} 2190 \text{ Megawatts} & \times & .85 & \times & 1.45 \text{ man-rem/} \\ \text{(capacity of site)} & & \text{(DCNGS estimated} & & \text{Megawatt-year} \\ & & \text{capacity factor)} & & \\ \hline = & 2700 \text{ man-rem/year/site.} & & & \end{array}$$

This is also similar to the annual dose which will be contributed by DCNGS workers to the gene pool dose (adjustment must be made for expected child-bearing as a function of age). It is important to compare the contribution of this dose to the natural background dose and to the other major source of radiation, the genetically significant medical X-ray dose. As stated previously, the background dose for the 260,000 person population living within a 50-mile radius of the plant is 21,000 man-rem per year. The addition of 2,700 man-rem per year to that background dose is genetically significant; in fact, it represents more than a twelve percent increase in the dose of radiation to the gene pool. The FES and



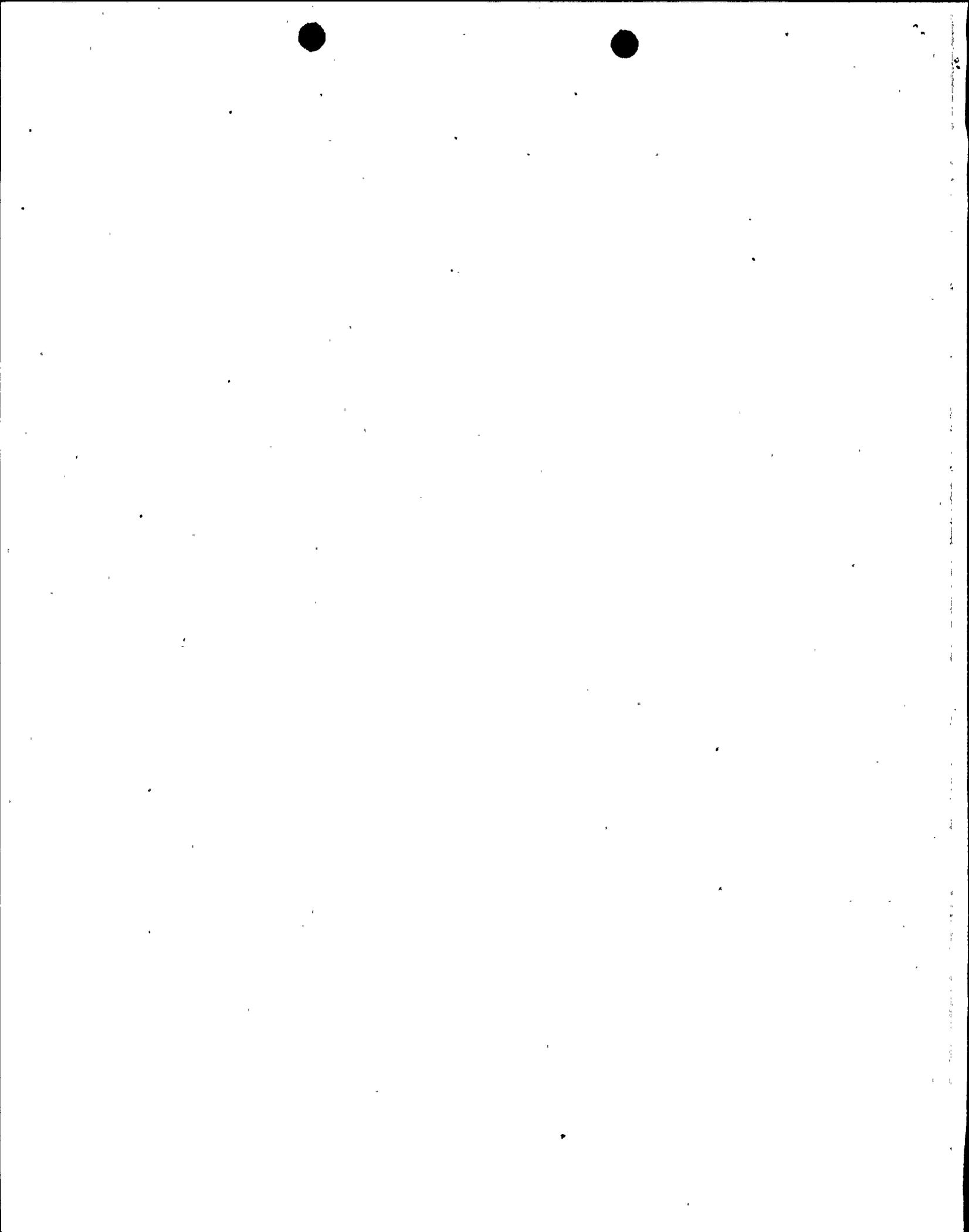
documents reviewed subsequent to the publication of the FES have inadequately assessed the impact of a twelve percent increase in the gene pool radiation. Furthermore, the genetically significant medical X-ray dose to such a population (260,000 persons) is 5,200 man-rems per year^{12/}. An addition of 2,700 man-rem/year on top of this genetically significant medical X-ray dose to the indicated population represents a fifty percent increase in man-made exposure. The belief that between 33 and 66% of the medical X-ray-caused gene pool dose may be unnecessary has led to a national effort by FDA and the radiological community to implement gonadal shielding programs^{13/}. Hence it follows that an increase equal to 50% of medical X-ray in the DCNGS local population's gene pool dose is a significant environmental effect which the NRC Staff must consider before contending that the FES and amendments are adequate.

14. The dose rate dependence of mutational effects of irradiation is the subject of active scientific debate. The estimates of rate dependence found in the BEIR Report^{4/} and relied upon by the Staff in its testimony have been criticized by the Environmental Protection Agency as being perhaps 140 to 220 percent too low^{6/}. Using this fact, and the fact that the 500 man-rem/year per unit is low by a factor of 2.7 (see paragraph 13 above, which computes the occupational exposure at 2,700 man-rem/year per site, which is 2.7 times the 1000 man-rem/year per site claimed), the 20



genetic "effects" per generation at equilibrium calculated by Goldman (Testimony at p.7) may actually range from 54 to 173. That is, 20 genetic "effects" multiplied by the 2.7-fold increase in occupational radiation results in 54 genetic "effects", which may go as high as 173 genetic "effects" if the EPA analysis of the BEIR data is correct. Properly compared with the spontaneous incidence of 15,000 to 25,000 "effects" in a population of 1/4 million, or 260,000 (Goldman Testimony at 7, 60,000 to 100,000 effects per million x 1/4), the increase in genetic disease is from 0.2% (54 effects/25,000 effects) to 1.1% (173 effects/15,000 effects). These genetic effects due to occupational exposure from the plant are certainly significant enough to merit evaluation in the FES by the Staff.

15. The genetic effect of low dose radiation on humans is no longer a matter of conjecture in light of recently published observations of a four-fold increased incidence of severe mental retardation and a statistically significant increased incidence of Down's Syndrome in populations exposed to natural background radiation levels of 1.5 to 3 rems/year^{15/}. This exposure rate is similar to that experienced by 11% of reactor workers^{10/11/}. Therefore, since the incidence of severe mental retardation may be increased in the children of a population so exposed, the effect is significant enough to be considered in the FES.



16. Finally, the impact of occupational exposure on the health of plant workers should be considered in the FES. Using BEIR^{16/} estimates, it is expected that one additional death from delayed effects of radiation cancer will occur for each 6,000 man-rem. Over a twenty-year period of operation, nine workers would be expected to have patent or occult lethal malignancies engendered in their bodies as the result of the estimated occupational exposure consequent to the operation of the DCNGS. Among a work force averaging 1,000 at the site, such a toll should not be overlooked, even if it is not statistically demonstrable above the "background" level of cancer deaths.

A very recent analysis of occupational exposure and radiation carcinogenesis makes this a matter of concrete significance; it may no longer be a matter of extrapolation^{17/}. In a study of Hanford radiation workers, epidemiological methods have revealed an association between radiation exposure and reticulo-endothelial system neoplasms, breast cancer, pancreatic tumors, and lung cancers. The dose experience of these workers is no different from that experienced by today's nuclear plant employees. The study further reveals that the amount of radiation necessary to double cancer incidence (to i.e., add a number of cancers equal to the spontaneous incidence of cancer) is less than 10 rads in these four tissues. This information is significant in light of the fact that the average annual exposure to a plant worker is 0.8 rads^{11/}. The FES should, therefore, consider the increased danger of cancer death among plant workers.

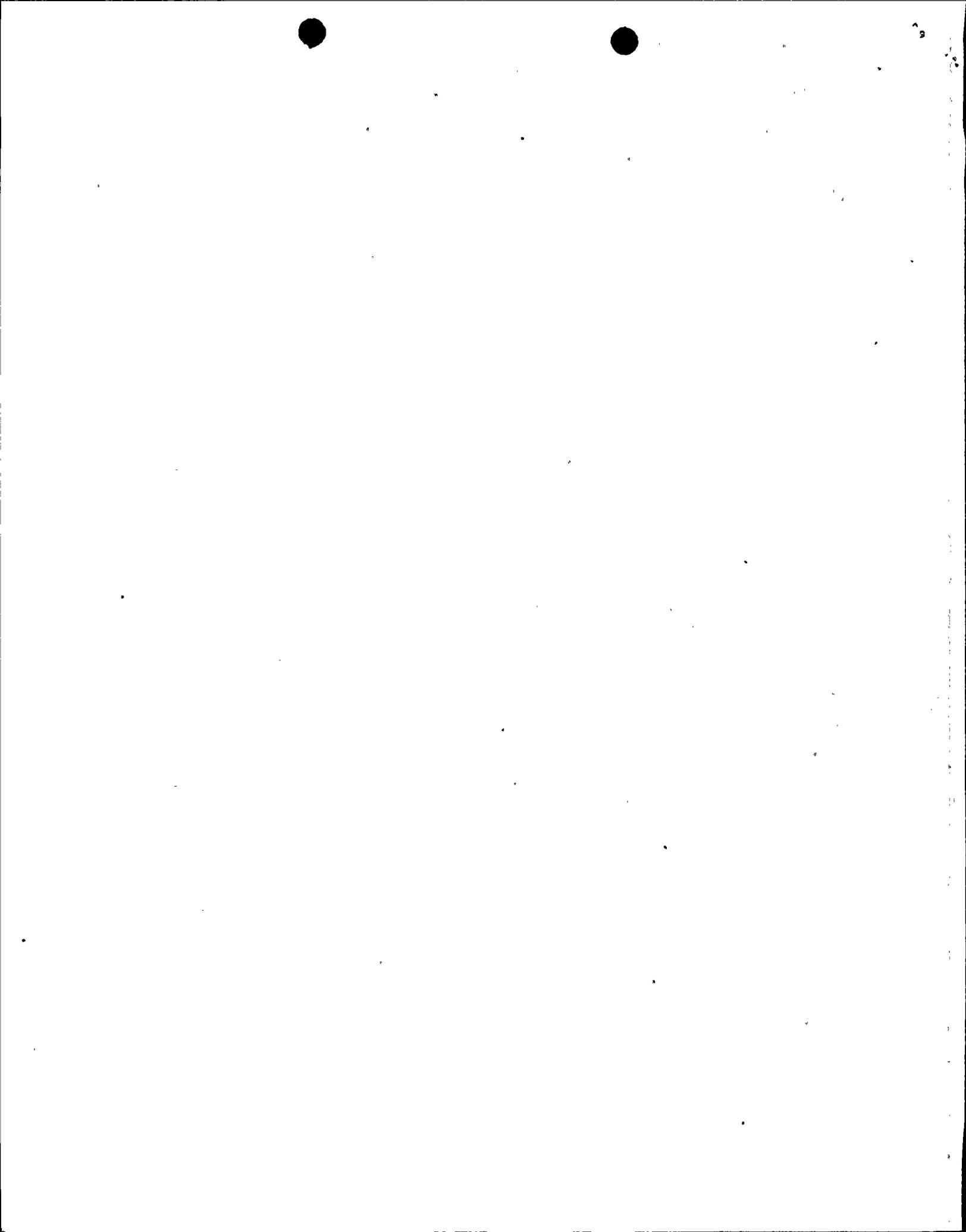


TABLE 1

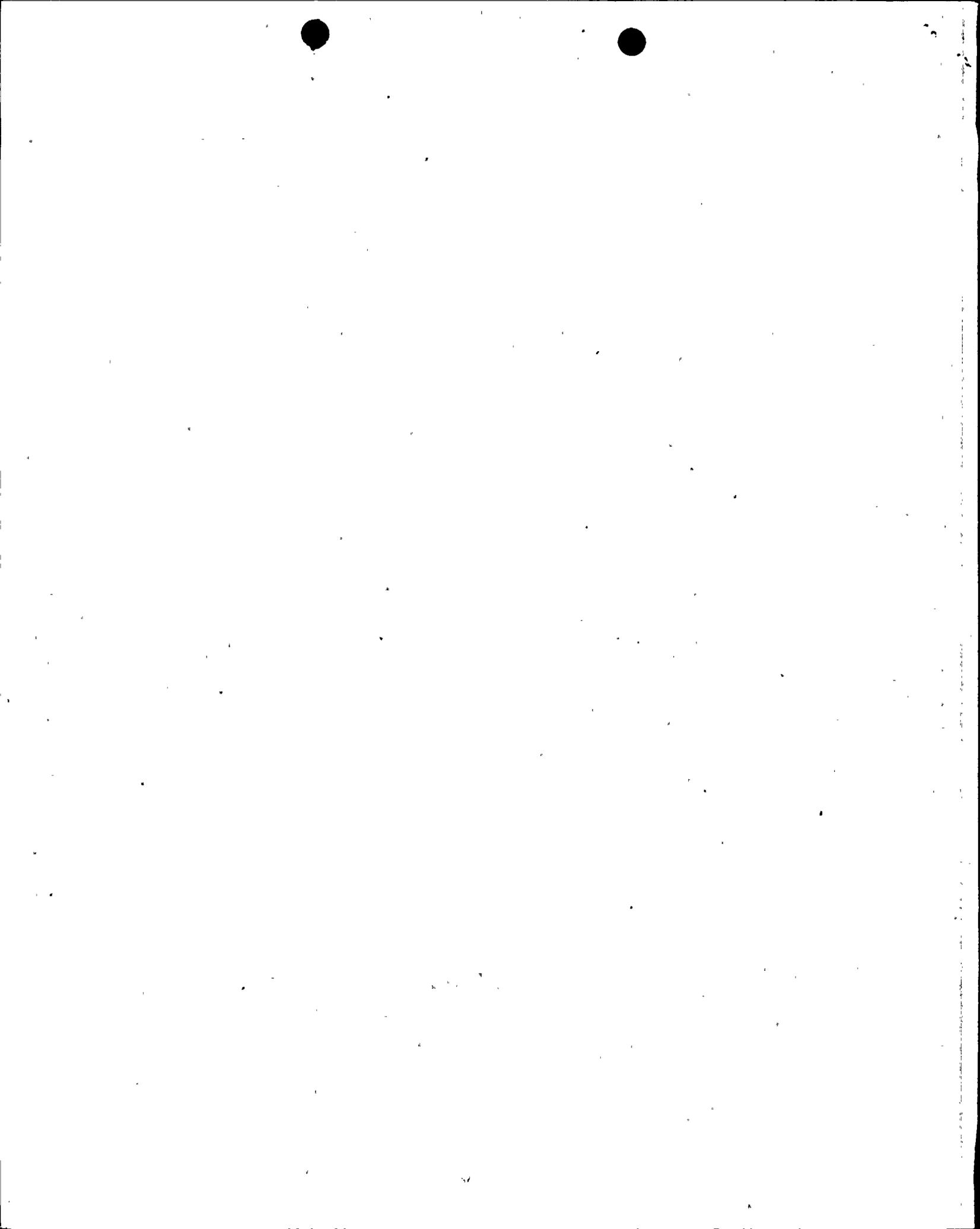
NORMALIZED TABLE OF ANNUAL RADIOACTIVITY RELEASES IN LARGE WESTINGHOUSE PWR'S^{a/}

YEAR	Name of Plant	Date of Initial Criticality	MW	Cap. Factor (%)	Conversion Factor	A I R B O R N E Curies/Yr				LIQUID, Curies/Yr	
						Noble Gas	H ³	Particulates	Halogen	Mixed F & Ap ^{b/}	H ³
	Diablo		1060	85	1.11	2953	788	.0023	<u>.197</u> 1.14 ^{c/}	.38	788
1974	Indian Pt.2	9-73	873	43	2.65	14,800	52.7	.757	.763	<u>11.1</u>	127
1972	H.B.Robinson 2	IC 9-70 c/o 3-71	700	72	1.97	506.3	4.9	.0048	.048	1.63	798
1973				82	1.74	5390	4.35	.00007	.5	1.05	753
1974				78	1.83	4220	94.2	.00308	<u>.097</u> .0942	4.58	822
1973	Surry 1&2	5-72 3-73	788 ea	65	.975	844	41.3	.00097	<u>.04</u> .039	.0975	437
1974				43	1.475	10,133	89.1	.0612	<u>.241</u> ^{d/} .180	5.61	362
1973	Turkey Pt 3&4	10-72 6-73	725 ea	62	1.11	588	4.6	.0002	<u>.07</u> .07	.033	365
1974				62	1.11	5170	10.2	.244	<u>4.03</u> 3.83	178	643
1974	Zion 1&2	6-73 12-73	1100 ea	24	1.895	5670	<u>342</u>	.0038	<u>.03</u> .029	.019	<u>4.4</u>
NOTES:						TOTAL	47,321	643	6.2	26.07	4311
a/Data normalized to 1000 MW plant, 100% capacity factor. Data incl. only those years during which plant was in commercial operation for the full year.						AVERAGE	5,258	71.5	.69	2.9	479
						Total Anomalous Figure ^{e/}	<u>47,321</u>	<u>301.35</u>	<u>2.2</u>	<u>14.97</u>	<u>4307</u>
							<u>8</u>	<u>8</u>	<u>8</u>	<u>8</u>	<u>8</u>
b/ Fission and Activation Products.						Avg. w/o Anomalous Fig.	5,258	37.6	.28	1.88	538

c/ 1.14 is not the sum of .757+.763; original data in NUREG 0077 does not agree; 1.14 is considered a good estimate.

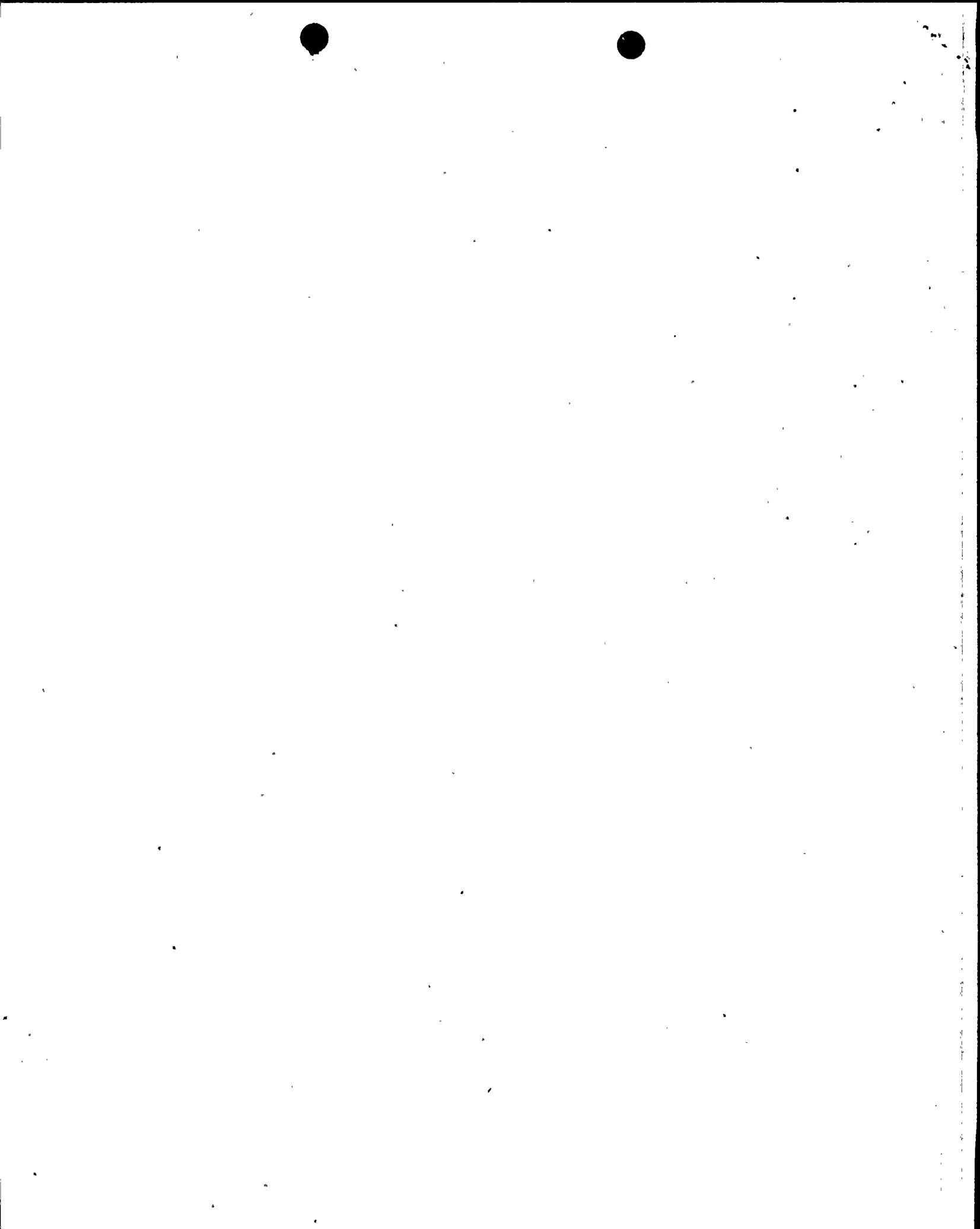
d/ .241 is probably correct; NUREG 0077 gives .14 for Halogens and Particulates, which is less than the sum of Halogens and Particulates reported in another place in NUREG 0077.

e/ Unusually large or small values (underlined) are deleted, assuming these are due to abnormal experience.



REFERENCES

- 1/ U.S. Nuclear Regulatory Commission, NUREG 0077 and NUREG 75/001.
- 2/ Testimony of Marvin Goldman for Nuclear Regulatory Commission, undated.
- 3/ Oakley, D.T., Natural Radiation Exposure in the United States. U.S. Environmental Protection Agency, Office of Radiation Programs (ORP/SID 72-1), 1972, p.36, 37, 50.
- 4/ National Academy of Sciences/National Research Council. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation ("BEIR" Report), NAS/NRC, Washington, D.C. 1972, p.168.
- 5/ Reactor Safety Study (WASH 1400): A Review of the Final Report. U.S. Environmental Protection Agency, Office of Radiation Programs, June 1976, p.2-5 to 2-8.
- 6/ Ibid, at p.2-11.
- 7/ J.M. Brown, "Linearity vs. Non-Linearity of Dose Response for Radiation Carcinogenesis" Health Physics, Vol. 31, No. 3, Sept. 1976, p.231.
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- 9/ U.S.E.P.A., "Draft Environmental Statement for a Proposed Rule-making Action Concerning Environmental Radiation Protection Requirements for Normal Operations of Activities in the Uranium Fuel Cycle", p.82 (1975).
- 10/ NRC; "Occupational Radiation Exposure at Light Water Cooled Power Reactors, 1969-1974", NUREG-75-032, (June 1975) p.7.
- 11/ NRC, "Occupational Radiation Exposure at Light Water Cooled Power Reactors, 1969-1975", NUREG-0109 (August 1976).
- 12/ U.S. Department of HEW, Food and Drug Administration, Gonad Doses and Genetically Significant Dose from Diagnostic Radiology, U.S. 1964 and 1970. Publication FDA 76-8034, April 1976.
- 13/ Federal Register 41, No. 143 p.30327-9, July 23, 1976.



- 14/ Reactor Safety Study (WASH 1400), supra, note 5 at 2-11.
- 15/ Kochupillai, N.; Verma, I.C.; Grewal, M.S.; and Ramalingsaswami, V., "Down's Syndrome and Related Abnormalities in an Area of High Background Radiation in Coastal Kerala", Nature 262, 60 (1976)
- 16/ BEIR Report, supra, note 4 at p.171.
- 17/ Mancuso, T.F., Stewart, A., Kneale, M.A., "Radiation Exposures of Hanford Workers Dying from Various Causes". paper presented at The Tenth Midyear Symposium of the Health Physics Society, Saratoga Springs, New York (Oct. 11-13, 1976).

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