UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

Before the Atomic Safety and Licensing Board

In the Matter of	)	
	)	
CAROLINA POWER & LIGHT COMPANY	)	Docket Nos. 50-400 OL
AND NORTH CAROLINA EASTERN MUNCIPAL	)	50-401 OL
POWER AGENCY	)	
	)	
(Shearon Harris Nuclear Power Plant,	)	
Units 1 and 2)	)	

AFFIDAVIT OF JACOB I. FABRIKANT

District of Columbia) SS:

Jacob I. Fabrikant, being duly sworn according to law, deposes and says as follows:

1. Q. State your name, occupation, and present position.

A. My name is Jacob I. Fabrikant. I am a physician and radiologist, research biophysics scientist, teacher and university professor in radiology and in biophysics at the University of California, San Francisco School of Medicine, University of California, Berkeley, and the Lawrence Berkeley Laboratory, University of California, Berkeley.

2. Q. What is the scope and purpose of your testimony?

A. The scope and purpose of my testimony is to respond to Joint Intervenors' Contention II(a) and (d) and a portion of II(b) and to Wells Eddleman's Contention 37B. In preparing this statement, I have considered both the specific language of Joint Contention II and Eddleman's Contention 37B and certain supplementary information provided by Joint Intervenors and Wells Eddleman in response to discovery initiated by Applicants. Both Contentions generally assert that the long-term somatic and genetic health effects of radiation releases from the Shearon Harris Nuclear Power Plant during normal operations, even where such releases are within existing guidelines, have been seriously underestimated for a number of stated reasons. The portions of these Contentions which I am addressing concern the health effects estimates in the Shearon Harris Draft Environmental Statement ("DES") as these are derived from the 1972 and 1980 Reports of The Committee on Biological Effects of Ionizing Radiation (BEIR I, BEIR III--full citations to references in this statement are set forth in Exhibit A).

3. Q. Briefly describe your education, including dates of degrees received, academic and other honors, professional societies and professional experience.

A. I hold a Bachelor of Science degree in chemistry and mathematics, McGill University (1952); a Doctor of Medicine degree and a Master of Surgery degree (1956), both from McGill University; and a Doctor of Philosophy degree in biophysics,

University of London (1964). I am a Fellow of the American College of Radiology (1978). I did post-doctoral training in surgery and pathology at Duke University Hospital, and trained in radiology at The Johns Hopkins Hospital. I am certified by the American Board of Radiology in diagnostic radiology, therapeutic radiology and nuclear medicine. I have been Professor and Head of the Department of Radiology and Chairman, Department of Diagnostic Radiology, McGill University Faculty of Medicine. I am presently Professor of Radiology, University of California School of Medicine at San Francisco; Staff Senior Scientist at Lawrence Berkeley Laboratory, University of California, Berkeley; Physician-in-charge of the Donner Pavilion, Cowell Memorial Hospital, University of California, Berkeley; and Professor and Member of the Graduate Physics Group, Department of Biophysics and Medical Physics, University of California, Berkeley. My professional and academic activities are devoted to patient care, primarily diagnostic and therapeutic radiology and nuclear medicine; to research in the radiological sciences, primarily cancer research; and to teaching in radiology and biophysics, primarily in the radiological sciences in the medical school and in the graduate school at the University of California. These are all documented in my curriculum vitae which is attached to this testimony as Exhibit B.

4. Q. Have you ever been appointed to or served or do you presently serve on any recognized national or international committees, commissions or groups dealing with the radiological sciences in general, and radiation and health in particular?

A. Yes. I have served on seven committees of the National Academy of Sciences--National Research Council, including the 1972 BEIR I, 1977 BEIR II and 1980 BEIR III Committees. I am a member of the Board on Research on the Effects on Radiation (BRER) of the National Academy of Sciences--National Research Council. I presently serve on the National Academy of Sciences Oversight Committee for the NIH-Prepared Radioepidemiologic Tables (OCRET). I am consultant to the National Academy of Sciences Board of Radioactive Waste Management. I was the Director of Public Health and Safety of the President's Commission on the Accident at Three Mile Island; I am on the Safety Advisory Board of Three Mile Island-2. I have served on advisory scientific committees of the President's Commission, the National Academy of Sciences, USPHS, NIH, NCI, BRH, NASA, American College of Radiology, the National Radiological Protection Board (NRPB) of Canada and England, and other scientific bodies dealing with radiation and health and cancer research. I am a member of the International Commission on Radiological Protection.

5. Q. Have you ever published in the scientific literature dealing with medicine, cancer research, radiation and health?

A. Attached to my statement as Exhibit C is a complete bibliography of my publications. My publications now number in excess of 250 scientific articles, reports, chapters, and reviews in the open literature. They are all in the fields of radiological sciences, medicine and surgery, radiobiology, radiation sciences and health, cancer biology, cancer research, and related disciplines.

6. Q. Briefly describe the BEIR Committee, NCRP, ICRP, the relationship between them and your personal participation in each committee.

A. The BEIR Committee is an expert scientific advisory committee on radiation and health effects of the National Academy of Sciences--National Research Council, viz., the Committee on the Biological Effects of Ionizing Radiation. The National Council on Radiation Protection Measurements (NCRP) is an expert scientific advisory committee on radiation and health effects chartered by the U.S. Congress in 1964 (originally dating back to 1929) with designated responsibility to collect and analyze scientific data and to develop recommendations about protection against radiation and on radiation measurements, quantities and units. The International Commission on Radiological Protection (ICRP) is the oldest expert scientific advisory body on radiation and health; it dates to 1928. The ICRP is represented by scientists from some 15-20 countries throughout the world with responsibilities to evaluate the health risks of radiation, particularly concerning

radioisotopes, occupational exposure, and medical applications, estimate the extent of these risks; and to recommend limits on radiation exposures to worker populations and the general population.

These advisory committees on radiation of international and national composition have, for many years, met and served effectively to discuss, to review, to evaluate and to report on three important matters of societal concern: (1) to place into perspective the actual and potential harm to the health of man and his descendants in the present and in the future from societal activities involving the use of ionizing radiations; (2) to develop quantitative indices of harm based on dose-response relationships to provide a scientific basis for the evaluation of somatic and genetic risk so as to better protect human populations exposed to low-level radiation; and (3) to identify the sources and levels of radiation which could cause harm, to assess their relative importance, and to provide a framework on how to reduce unnecessary radiation exposure to human population.

I was a member of the 1972 BEIR I Committee, and on the Subcommittee on Somatic Effects and the Subcommittee on Teratogenic Effects. I was Vice-Chairman of the 1977 BEIR II Committee. I was a member of the 1980 BEIR III Committee, on the Subcommittee on Somatic Effects, and Chairman of the Ad Hoc Committee for Estimating the Total Cancer Risk on Low-Dose, Low-LET, Whole-Body Radiation. I am on the ICRP, and a member

of Committee 1, which deals with risk estimation and all health effects of exposure to ionizing radiations.

7. Q. Please describe more specifically the selection and composition of the BEIR III Committee.

A. The BEIR III Committee, comprised of 22 scientists and assisted by numerous additional scientific consultants, was the scientific advisory committee of the National Academy of Sciences--National Research Council concerned with the health effects in human populations exposed to low levels of ionizing radiation. The Committee was composed of men and women selected for their expert knowledge and judgment in science and public policy. Members specialize in the broadest spectrum of scientific disciplines, including medicine, public health, epidemiology, biostatistics, genetics, medical genetics, population genetics, health physics, nulcear physics, radiology, endocrinology, nuclear medicine, mammalian radiobiology, experimental radiobiology, cell biology, physical biology, biophysics, pediatrics, reproductive biology, cancer biology, radiological sciences, and occupational health.

The criteria for membership was a record of scientific achievement and scholarly contribution to the member's scientific discipline and recommendation by his scientific peers in the related discipline. The Committee and its consultants were among America's most outstanding radiation scientists.

8. Q. What is the BEIR III Report?

A. The BEIR III Report (1980) is the consensus of deliberations of the BEIR III Committee and deals with the scientific basis for evaluating the health effects in human populations exposed to low levels of ionizing radiation. The report broadly encompasses two areas. (1) it reviews the current scientific knowledge--epidemiologic surveys and laboratory animal experiments--relevant to late effects of low-level radiation exposure of human populations, (2) it evaluates and analyzes these late health effects--both somatic and genetic effects--as risks from exposure to low-level radiation.

In its review and estimates, the BEIR III Committee took into account all potential factors influencing risk estimation. For example, for cancer risks and genetic risk estimations, observations are based on epidemiological surveys of exposed human populations and extrapolations from high doses to low doses; extensive research on laboratory animals and extrapolations from animal data to man; analysis of dose-response relationships, both from experimental and epidemiological data, and from mathematical and theoretical models of carcinogenesis and genetic and other effects; from known mechanisms of cell transformation, cell injury, cell lethality, chromosome injury, mutagenesis, and tissue injury; cell and tissue repair and recovery; in vivo and in vitro experiments; biological factors in mammals and in human beings, such as age and sex; physical factors and types of radiation, such as linear energy transfer and relative biological effectiveness. All these are detailed in the various sections of the BEIR III Report.

9. Q. What is the BEIR I Report?

A. In February 1970, the NAS-NRC Advisory Committee on the Biological Effects of Ionizing Radiations (BEIR) undertook a complete review and reevaluation of existing scientific knowledge concerning radiation exposure of human populations. The functions and activities of the BEIR I Committee (the first of the three NAS-NRC BEIR Committees that have issued reports during the past decade) were the same as those summarized above for the BEIR III Committee. The BEIR I Committee produced its report in November 1972: "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation" (BEIR I). In September 1976, the NAS-NRC and the BEIR Committee were asked to update the 1972 BEIR report on the basis of newly developed scientific information. The BEIR III Committee produced its report in July 1980 (the BEIR III Report).

10. Q. How have the BEIR reports been used?

A. The reports of the BEIR Committees have become the reference texts for the scientific basis for development of radiation protection standards and public health policy. The BEIR reports have become the basis of current Environmental Protection Agency estimations of potential health impacts from radiation. Further, the 1980 BEIR III Report risk estimates now provide the basis for the NIH-prepared Radioepidemiological Tables on Probability of Causation of Cancer-Induction required by the United States Orphan's Drug Act.

11. Q. You have mentioned other national and international organizations which have reviewed the health effects of low-level radiation. How do the BEIR reports compare with reports of these organizations?

A. The approaches, assessments and evaluations of potential health effects from low-level ionizing radiation in the BEIR reports are consistent with and confirmed by the observations and conclusions of all leading national and international scientific advisory committees and commissions concerned with radiation protection, standards and health. These include the ICRP (Report No. 26, 1977), the NCRP (Report No. 43, 1975; Report 60, 1980) and UNSCEAR (Report, 1977; Report, 1982).

12. Q. Joint Contention II and Eddleman Contention 37B allege that the somatic and genetic health effects as estimated in the Draft Environmental Statement related to the normal operation of Shearon Harris Nuclear Power Plant, Units 1 and 2 are underestimated. Do you agree?

A. No. The potential radiological impacts on humans in the DES are based on conservative approaches derived primarily from risk estimates contained in the 1972 BEIR report (for cancer) and the 1972 BEIR report and the 1980 BEIR report (for genetic ill-health). These reports, as I have indicated, were prepared by a group of America's preeminent experts in radiation, medicine and science, and the reports are consistent with the conclusions of all other recognized national and international organizations.

It is remarkable how many careful and detailed epidemiological and experimental studies have been made concerning the effects of ionizing radiation in man (see UNSCEAR, 1977; BEIR III Report). As new reports have been issued, these have supported the 1972 and 1980 BEIR reports; and these BEIR reports, thus, become increasingly compelling over time. Indeed, there is little doubt that the BEIR reports are presently considered the most scientifically reliable estimations of potential radiation risks of detailed health effects, cancer induction and genetic ill-health in populations exposed to low levels of ionizing radiation.

13. Q. Have you read and evaluated the sections of the Draft Environmental Statement which deal with population and occupational exposure and potential radiological impact on humans?

A. Yes.

14. Q. Do you agree with the conclusions of the NRC Staff as regards potential long-term somatic and genetic health effects resulting from the normal operation of the Shearon Harris Nuclear Power Plant, Units 1 and 2.

A. Yes.

15. Q. What are the potential health effects in human beings that may be associated with the normal or routine operation of a nuclear power plant, such as the Shearon Harris Nuclear Power Plant?

A. During the normal operation of a nuclear power plant, such as Shearon Harris, very low-level radiation is released to the environment. This is essentially all low-LET gamma and

beta radiation (one/billionth of the releases is high-LET radiation). See Whipple Affidavit at ¶ 13-16. Accordingly, any potential health effects, if they do occur in exposed human populations, would invariably occur from exposure to lower level, low-LET radiation. This is sparsely ionizing radiation at very low doses and dose rates.

Only two potential health effects can occur from exposure to low-dose, low-LET, ionizing radiation, namely, cancer and genetically-related ill health. The probability of occurrence of either cancer or genetic effects from routine operations is extremely small, as demonstrated in the cancer and genetic disease risk estimates in the DES.

There are other potential health effects that could occur from radiation exposure, e.g., cataracts and heart disease, but the dose levels from routine Shearon Harris operations would be far too low for any of these diseases to be potential health risks. No other known health effects are expected to occur following such low-level, low-LET radiation exposure.

16. Q. Have these two health effects, cancer-induction and genetic health effects, ever been observed to occur in humans or in human populations exposed to dose-levels equivalent to that of routine normal operation of a nuclear power plant, such as Shearon Harris?

A. No.

17. Q. Have they ever occurred after exposure to higher dose levels of radiation, higher than than the dose levels occurring with the routine operation of a nuclear power plant?

A. Only cancer-induction has occurred in human populations exposed to much higher dose levels, and this has only been demonstrated statistically, at dose levels of 100 rads or more. There are some reliable epidemiological data that indicate a statistical excess of cancer down to 50 rads. These dose levels are many orders of magnitude greater than that which will occur in association with the routine operation of a nuclear power plant.

Genetic health effects have never been demonstrated to occur in the progeny of any human populations exposed to ionizing radiation.

18. Q. Is it possible that no health effects may occur from the low-level radiation emissions from normal operation of nuclear power plants?

A. Yes. With respect to the range of potential effects, from the low-level radiation of the sort at a nuclear power plant, BEIR III reaches the following conclusion:

> Expectations based on linear extrapolation from the known effects in man of large doses delivered at high dose rates in the range of rising dose-incidence relationship may well overestimate the risks of low-LET radiation at low dose rates, and may, therefore, be regarded as upper limits of risk for low-level low-LET irradiation. The lower limit, depending on the shape of the dose-incidence curve for low-LET radiation and the efficiency of repair processes in counteracting carcinogenic effects, could be appreciably smaller (the

possibility of zero is not excluded by the data). (BEIR I, 1972, at 88; BEIR III, 1980, at 139)

19. Q. Briefly describe the BEIR Reports' treatment of potential genetic effects of low-level radiations in humans.

A. As indicated, genetic effects due to ionizing radiation have never been directly observed in man. However, they have been observed in laboratory animals. Estimations of the radiation risks of genetically related ill-health are based mainly on these laboratory animal observations--primarily from laboratory mouse experiments. Our knowledge of fundamental mechanisms of radiation injury at the genetic level is far more complete than for example, our knowledge of the mechanism of radiation carcinogenesis. This permits, conservatively, greater assurance in extrapolating information on genetic mutagenesis from laboratory animals to man.

The genetic disorders that may result from radiation exposure are: (1) those which depend on changes in individual genes (gene mutuation, or small deletions); and (2) those which depend on changes in chromosomes, either in total number or in gene arrangement (chromosomal aberrations). Gene mutations are expected to have greater health consequences than chromosomal aberrations. At a low level of exposure, the effect of radiation in producing either kind of genetic change is proportional to the dose. BEIR III's genetic health effects review and estimates are described in Chapter IV of the BEIR III Report and are summarized in Table IV-2, at p. 85.

20. Q. How does the BEIR III analysis of genetic risks in exposed human populations compare with the peer-reviewed scientific literature?

A. The BEIR III analysis of potential genetic disorders and genetic risks is in complete accord with the world's peer-reviewed scientific literature on radiation genetics, population genetics, cytogenetics, environmental mutagenesis, human genetics, and molecular genetics. (This is all documented in UNSCEAR 1972, 1977, 1982; ICRP, 1977; BEIR I Report, BEIR III Report; NCRP, 1975, 1980.) There have been no challenges in the peer-reviewed scientific literature to the genetics portions of the BEIR III Committee (Genetic Subcommittee) Report (1980).

21. Q. What is peer-reviewed literature, and why is that characterization significant?

A. Scientific articles published in the biological, physical, chemical and medical sciences are of three main types: proffered, invited and survey. Invited or survey papers, chapters, or books are usually published as the author wrote them with little or no process to question hypotheses or to verify stated results. Proffered papers in the scientific literature are reviewed by peers in the field selected by the editorial board of the scientific periodical, in order to evaluate and criticize the scientific quality and scholarship of the paper and its contribution to new knowledge, and based on these criteria -- scientific quality, scholarship, and new

knowledge -- recommend suitability for publication in the journal.

Peer-review does not mean that the article is scientifically correct or the hypothesis has been proven; however, it establishes a standard of professionalism. If an article has not been peer-reviewed, this means that the article has not been subjected to rigorous prepublication evaluation.

Q. Turning now to the risk of carcinogenesis from exposure to low level, low-LET radiation, what procedure did the BEIR Committee follow in estimating potential risks of radiation-induced cancer in exposed human populations?

A. Risk estimates are based on the data derived from examination of epidemiological surveys, together with theoretical, mathematical, and statistical considerations and with extensive experimental cellular, tissue, and laboratory animal studies. The chief sources of epidemiological data currently used for risk estimation are the Japanese atomic bomb survivors exposed to whole-body irradiation at Hiroshima and Nagasaki, the patients with ankylosing spondylitis and other patients who were exposed to partial body irradiation therapeutically or to medical diagnostic radiography and fluoroscopy, and various occupationally-exposed populations such as uranium miners and radium dial painters. With the above tools, the BEIR reports define dose-response relationships between radiation dose and observed cancer incidence, and thereby derive cancer risk coefficients in human populations exposed to low-level radiation.

23. Q. What are the various postulated dose-response curves for low-level radiation doses?

A. Analysis of a number of dose-incidence curves for cancer-induction in irradiated populations, both in humans and animals, has demonstrated that for different radiation-induced cancers, different forms of the same multicomponent dose-response curve can be defined. Simplifications of the complex model can occur by reducing the number of components which have the least effect on the form of the dose-response relationship in the low-dose range. Such simpler models, with increasing complexity, include the linear, the pure quadratic, the quadratic (with a linear term in the low-dose region), and finally, the multicomponent linear-quadratic dose-response form with a linear term and an exponential modifier. On the basis of microdosimetric and biophysical theory, for low-LET radiation at low doses, the linear-quadratic relationship alters so that the quadratic term becomes progressively unimportant, and the linear term is expected to be dominant.

A fourth model, one with a rapidly rising curvilinear dose-response form and a decreasing quadratic function (sometimes referred to as "supralinear" dose-response curve) has also been described. It is not used by and recognized organization for risk estimation for low-dose, low-LET radiation exposure, since there is no experimental evidence or epidemiological evidence that this dose-response relationship is appropriate for risk estimation.

24. Q. Prior to 1980, what was the position of the BEIR Committee regarding the shape of the dose-response curve for low-level radiation doses?

A. The 1972 BEIR I Committee considered it scientifically appropriate to adopt a no-threshold linear hypothesis of the dose-response to estimate the cancer risk at very low-level (low-LET, whole-body) radiation exposure. It was assumed the same proportional risks are present at low levels as at high levels of radiation.

25. Q. Has the position of the BEIR Committee changed regarding the shape of the dose-response curve for cancer-induction?

A. Yes. The BEIR III Committee examined all possible dose-response relationships for estimation of carcinogenic risk in human populations exposed to low-level, low-LET radiation. The most compelling scientific and epidemiological evidence led the BEIR III Committee to select and apply the multi-component linear-quadratic no-threshold dose-response form (with the linear term in the low-dose region). To estimate carcinogenetic risk of low-dose, low-LET whole-body radiation, the linear quadratic dose response relationship was considered the preferred model, that is, it would lead to the most accurate risk estimate. The linear dose response model was determined to be the most conservative and would lead to overestimates of risks. See BEIR III Report at 139. This change from the 1972 BEIR report was based upon the available epidemiological surveys,

experimental and cell culture evidence and current microdosimetric theory. Of the 22 Committee members, all except two accepted the preferred approach. Of the two members on the Committee disagreeing with the preferred approach, one preferred a quadratic model with much lower risk estimates and one preferred the linear model. No one advocated supralinearity.

26. Q. What dose-response curve is used in the DES for cancer induction, and is this conservative?

A. The DES utilizes the linear dose-response model from BEIR I for estimating potential carcinogenic risks. The DES in so doing is conservative, that is, it tends to overestimate potential health effects. The BEIR I Report, based on information then available, used the linear dose-response relationship for cancer induction from low-dose, low-LET radiation. Based on more complete information, the BEIR III Committee adopted as its preferred model, the linear-quadratic relationship, which predicts lower estimates of cancer-induction than does the linear model. By using a linear dose-response relationship, the DES uses a method which overestimates the risk leading to cancer risk estimates which are <u>higher</u> than the scientific community generally believes is warranted by analysis of the scientific data.

27. Q. Joint Intervenors' Contention II and Eddleman Contention 37B depend heavily on the reports of Mancuso, Stewart and Kneale, Gofman, Bross, Bertell and Morgan. Did the BEIR Committee or other committees take into account the reports and positions of these individuals?

A. Yes. All these matters have been considered by the 1980 BEIR Committee, the NCRP (Report 64, 1980) and the ICRP. Each has been dealt with carefully in the various Committee reports or in the open literature where appropriate. Individually and collectively, the statements and interpretations in the reports referenced by Joint Intervenors principally claim that the carcinogenic risk per rad at low doses and dose rates is greater than observed at high doses and dose rates so that the radiation effects at low doses in man would be substantially greater than estimated by the linear hypothesis. The uniform conclusion has been that these reports are unconvincing and utterly fail to establish that the linear hypothesis is anything other than conservative (BEIR III Report; NCRP, 1980). The experimental and epidemiological data simply do not support the use of a supralinear dose-response model for radiation carcinogenesis following low-LET exposure. In rejecting this model, the GAO Report (1981) comments as follows:

> The analysis of a few recent epidemiological studies [e.g., Mancuso, Stewart and Kneale and Bross] have been cited by some [e.g., Bertell, Gofman and Morgan] to support the supralinear model. These studies, however, have been seriously criticized on statistical and methodological grounds.

Further, no evaluations in the peer reviewed literature of any recent reports on epidemiological studies suggest in any manner that the linear hypothesis is not conservative.

Besides favoring supralinearity, Bertell--relying principally on Bross--also suggests low-level radiation causes

a host of diseases independent of cancer and genetic effects. This contention likewise has been rejected by the scientific community.

28. Q. What are the principal studies of the authors on the points that you have referenced?

A. The following reports have been interpreted by their authors, or a few other people, to set forth the points above made: Mancuso, Stewart and Kneale, 1977; Kneale, Mancuso and Stewart, 1978, 1981; Bross and Natarajan, 1972, 1977; Bross, Ball and Falen, 1979; Bross and Driscoll, 1981; Morgan, 1975; Bertell, 1977;,1979; and Gofman, 1969, 1970a, 1970b, 1971, 1979, 1981.

29. Q. Let's look at these in turn. What data were the basis of the Mancuso, Stewart and Kneale (1977) study?

A. Mancuso, Stewart and Kneale (1977) conducted an epidemiological survey of the cancer mortality rate for the workers at the Hanford Nuclear Facility at Richland, Washington between 1943 and 1971. Their report was based on the work experience of 24,939 male workers with 3,520 certified deaths (death certificates) and an unspecified number of female workers with 412 certified deaths. Their preliminary report came out in 1977 and primarily was an analysis of the cancer mortality data for the 3,520 male deaths for which death certificates were available. In that report, the authors claimed that their analysis demonstrated a greater number of

radiation-induced cancers than the linear dose-response hypothesis would indicate. Leading epidemiologists and statisticians have, however, widely criticized this analysis because of its serious deficiencies in methodology, formulations, and conclusions. In addition, other analyses of the data have been performed which show little or no radiation induction effect. See NCRP, 1980; Hutchison, et al., 1979; NAS-BEIR, 1980; Reissland, 1978; GAO, 1981; Anderson, 1978; Mole, 1978; and Gilbert and Marks, 1979; Marks and Gilbert, 1978.

30. Q. What are the serious methodological flaws in the Mancuso, Stewart and Kneale (1977) analysis?

A. The methodological flaws in that report involve inadequacies of radiation dosimetry, confounding factors which could have caused cancer in the workers in the absence of radiation exposure, selection bias, and inconsistencies with the spontaneous incidence of cancer in the exposed population. Their report does not give the actual individual radiation doses of the Hanford workers who died of cancer received, but instead only provides mean cumulative radiation doses. Their analysis also did not consider the calendar year in which the cancer began in the individual and in the study population. It made no correction for the fact that the United States population as a whole had an increasing number of cancers of the types observed in the Hanford workers during the study period. Thus, this study's conclusion that an increase in cancer occurs

with increasing dose accumulations over increasing time fails to take into account that a similar increase in cancer incidence in the entire United States population occurred, even in the absence of increasing doses of radiation.

Gilbert and Marks (1979) and Hutchison, et.al. (1979) have analyzed the same data used in the Mancuso, Stewart, and Kneale (1977) report and concluded that cancer of the pancreas and multiple myeloma are possibly associated with the work experience of the study population. In these studies, no radiation relationship exists for lymphatic cancers or cancers of the blood-forming tissues other than multiple myeloma. Thus, no excess of leukemias existed which experience, such as in the Japanese atomic bomb survivors, suggests should have been most observable where radiation is a factor.

Because the recorded radiation doses in the Hanford workers were very small, perhaps only a few rads, the very low cancer-doubling dose estimates reported by Mancuso, Stewart and Kneale (1977) are spurious. Numerous scientists have strongly disputed those doubling-dose estimates because their values are inconsistent with known and established radiobiological evidence. If the estimated small dose in the worker population actually caused a doubling of the spontaneous rate of cancers, natural background radiation in the United States would produce more than the actual number of cancer cases observed in the entire population. Such a result is impossible. As a result of these criticisms, Mancuso, Stewart and Kneale (Kneale,

Stewart and Mancuso, 1981) apparently have modified their estimates of cancer doubling doses and presently are quoting a mean doubling dose of 15 rem with a range of 2 to 150 rem in the worker population. This mean doubling dose and the lower range, however, still are inconsistent with existing knowledge and experience in cancer epidemiology and statistics.

Recently, Darby and Reissland (1981) have presented a more detailed standard analysis of the Hanford data in which the observed death rates were examined for trends with increasing radiation dose, and the total numbers of observed deaths were compared with those expected from the United States national mortality data. Overall, Darby and Reissland (1981) found no evidence that shows that the currently used risk estimates are too low. That conclusion supports the conclusions of earlier reanalyses of the Hanford data by Gilbert and Marks (1979), Marks and Gilbert (1978), and Hutchison, et al. (1979), and an assessment by the BEIR III (1980) Committee, the NCRP (1980) and a study by the General Accounting Office (GAO, 1981).

31. Q. What conclusions has Bross made in his Bross and Driscoll, 1981; Bross and Natarajan, 1972, 1977; and Bross, et al., 1979 studies?

A. Bross claims that the risk for cancer-induction to pregnant women and all adults following diagnostic X-ray exposure, which is a low-level radiation exposure, is greater than the risk at high doses and at high dose rates. Bross also

claims that he has identified susceptible subgroups in the general population which are specially sensitive to radiation damage.

32. Q. Do you agree with Bross' observations?

A. No. Bross' belief that specially sensitive subgroups exist is derived from his analysis of the Tri-State Leukemia Survey (Graham, et al., 1966; Gibson, et al., 1972) wherein he studies what he termed certain "indicators of susceptibility" (e.g., viral infections, bacterial infections and allergy) shown by the leukemic child from birth until diagnosis of leukemia. Bross concluded "the apparently harmful effects of antenatal irradiation are greatly increased in certain susceptible subgroups of children possessing the indicators associated with a slightly higher intrinsic risk of leukemia" (Bross and Natarajan, 1972).

Reanalysis of Bross' observations (Smith, et al., 1973) shows, however, that children with leukemia are simply more prone to viral and bacterial infections and allergies before the clinical onset of the leukemic disease. Thus these "indicators" characterize the disease itself and do not relate to the child's inherent susceptibility or sensitivity to leukemia. The occurrence of these "indicator" diseases as part of the pre-leukemia phase of leukemia in children is well known in pediatric medicine and in clinical hematology. Analysis of Bross' data shows that the incidence of these "indicator"

diseases before the clinical onset of leukemia is the same in children who had received no irradiation in utero as those who had. Bross' hypothesis, that a susceptible portion of the population exists that has a higher risk of leukemia, also has been challenged on the grounds that Bross' methods do not allow the identification of susceptible individuals ahead of time and, therefore, do not allow his thesis to be tested (Smith, et al., 1973).

More recently, Bross has claimed that the relatively small radiation exposures (in the millirad range) from diagnostic X-rays in adults significantly increases the risk of leukemia (Bross, et al., 1979). In coming to this conclusion, it appears that Bross erroneously assumes that, in the absence of diagnostic X-rays, the incidence of heart disease and leukemia on the general population would be zero. Of course, this is not the case. Also, below the ten rad exposure point, his "dose-response" curves of adults exposed to diagnostic X-rays are flat. This suggests that a threshold exists in the dose-response relationship contrary to his contention elsewhere. A more conventional relative risk analysis recently done by Boice and Land (1979) found that Bross' conclusions were not justified on statistical, dosimetric or logical grounds.

Bross also erroneously assumes that the relative risks are fixed and that the "percentage of the population affected" varies with the dose, i.e., he assumes that the basic

response variable is the proportion of the irradiated population affected by radiation rather than the dose. Conventional relative-risk analysis assumes that everyone is affected and that the relative risks vary with dose. The reason for Bross' unconventional methodological approach is unclear. This position taken by Bross in his 1979 study appears to be at odds with his earlier paper (Bross and Natarajan, 1972) in which he postulated the existence of a sensitive, fixed size subgroup of people whose relative risk of leukemia increased rapidly with increasing X-ray dose.

In addition, the leukemia risk (or "percent affected") in Bross' analysis increases dramatically only in males and only after a large number of diagnostic X-rays. Females, however, appear to be unaffected. No radiation dosimetry was performed in the Tri-State Survey, but the cause-effect relationship is obscured because if a person is receiving very large numbers of diagnostic X-rays --40 or more within 10 years--it implies that a disease state is present and perhaps is deriving from heart disease or a preleukemic sensitivity to infections.

Further interpretations of the Tri-State leukemia study data introduced by Bross (Bross and Natarajan, 1972, 1977; Bross, et al., 1979; also Bertell, 1977, 1979) have subsequently been severely criticized in scientific literature (Smith, et al., 1973; Land, 1977, 1979; Oppenheim, 1977; Boice and Land, 1979; Rothman, 1977; MacMahon, 1972; Hamilton, 1979) as have the conclusions Bross has drawn.

33. Q. Has Bross performed any other studies?

A. Yes. Recently, Bross and Driscoll (1981) took data from the Najarian-Colton (1978) survey and concluded that the Portsmouth Naval Shipyard workers sustained very large numbers of lung cancer deaths as a result of exposure to low-level radiation (Bross and Driscoll, 1981).

34. Q. Do you agree with that observation?

A. No. This Bross study has many methodological errors and uses unconventional statistical methods. Bross and Driscoll (1981) also make unsubstantiated claims on the existence of subpopulations which are super-sensitive to radiation exposure. Furthermore, in their attempt to reanalyze the data from the Portsmouth Naval Shipyard study, Bross and Driscoll (1981) claim that the official publication of Rinsky, et al. (1981) was purposely misleading and underestimated the lung cancer risk. By regrouping selected data for lung cancer, which do not appear in the Rinsky, et al. (1981) paper, Bross reached the conclusion that above the 1 rem exposure range, with more than a 15-year follow-up, a two-fold increase of lung cancer exists. This would mean an excess of 189 deaths per million persons exposed per year per rem compared with the ICRP (1977) and BEIR III Report estimates of about 1 lung cancer death per million persons exposed per year per rem. Because no detailed denominators, basis for expected cases, or host

factors are given or corrected for in the Bross' analysis, his conclusions cannot be evaluated nor substantiated. Finally, smoking was not examined in any detail as an important confounding factor in Bross' analysis.

35. Q. Has any other study been made of the Portsmouth Naval Shipyard workers?

A. Yes. The final report of the U.S. Department of Health and Human Services, Public Health Service Centers for Disease Control, National Institute for Occupational Safety and Health (NIOSH)'s Epidemiologic Study of Civilian Employees at the Portsmouth Naval Shipyard, based on a total cohort of 24,545 civilian white males employed at the Portsmouth Naval Shipyard between 1952 and 1977, is now available (Rinsky, et al., 1982).

36. Q. What were the findings of that study?

A. The report found no excess deaths due to malignant neoplasms or due specifically to neoplasms of the blood and blood-forming tissues (leukemias) in civilian workers at the Portsmouth Naval Shipyard. This NIOSH study found no relationship between exposure to radiation and mortality from any cause among the worker population when compared to the United States white population. Furthermore, no excess in leukemia mortality was observed in the radiation-exposed population when compared to the non-radiation-exposed employees of the Portsmouth Naval

Shipyard. National Academy of Sciences--National Research Council (NAS-NRC, 1982) scientific advisory committee has reviewed this report and took no exception with the NIOSH study findings.

37. Q. Will you comment on Bertell's work?

A. Her two relevant papers (Bertell 1977, 1979) on low-level radiation (which are not peer-reviewed in the scientific literature) are neither scientifically nor medically sound. She claims, based principally on the earliest and preliminary data of the Mancuso, Stewart and Kneale Hanford study, that low-level radiation effects are substantially underestimated. She further claims, based on Bross' work on the Tri-State Leukemia Survey, that risk per rad of low dose X-radiation is greater than generally believed and that the linear hypothesis underestimates low-dose risk. She further suggests that many diseases other than cancer and genetic effects have been caused by low-dose radiation.

To the extent her work is based on analyses by Mancuso, Stewart and Kneale and by Bross, her position fails for all the same reasons that the original works by their authors failed. Further, her work has been seriously criticized by Gilbert and Marks (1979) of Batelle Pacific Northwest Laboratories (two noted epidemiologists and biostatisticians), and by Parkinson and Fair (1979) of Oak Ridge National Laboratory (two noted industrial health physicists). They totally

discredit her work, finding erroneous claims, selection of data, bias, misinterpretation of the Hanford cancer data and Tri-State Leukemia data, misstatements and distortions of facts, lack of support by references to the scientific literature, and drawing of speculative conclusions based on no experimental or clinical data. She, for instance, lists numerous illnesses and diseases (including allergies, heart disease and diabetes) and implies all are radiation-induced and/or radiation-associated and/or radiation-related, but she offers no scientific evidence for these claims. Parkinson and Fair conclude that Bertell's 1979 article was "a collection of unfounded claims and distortions of fact which should not appear unchallenged in the technical literature.... the author's [Bertell's] contentions are unsupported by references to the literature and the stated effects, of course, should be regarded as pure speculation unless the author can cite experimental data."

Bertell clearly is not an expert in the field of low-level radiation. She has done no original analyses of the Hanford and Tri-State Leukemia surveys. Rather, she merely draws her own conclusions from the works of others without a full understanding of the data. She is a poor statistician and has no training in medicine, nor in the radiation sciences, nor in genetics, nor in epidemiology, nor in health physics, nor in radiological health, nor in radiation protection, nor in radiobiology, nor in cancer biology, nor in any of the scientific-medical disciplines she addresses in her work.

Bertell (1979) claims that she has been intimately involved in the analysis of the Tri-State Leukemia data for over a decade and with a large team of researchers. It is surprising that in all this time, she has failed to publish a single peer-reviewed article in the scientific literature as a leading author or as a co-author with Gibson, et al., Graham, et al., Lilienfield and his colleagues, or Bross and his colleagues, that is, with those scientists who have published the Tri-State Leukemia Survey studies. Bertell notes, in her 1979 article, no academic or professional affiliation (only a street address is given), and she acknowledges no professional colleagues. One has to conclude that her claims are without substance or merit, and that she has made no scientific contribution to the field of the radiation sciences concerned with human populations and health.

38. Q. Would you comment on Gofman's reports?

A. Gofman and his colleagues (1969, 1970a, 1970b, 1971) wrote three papers and one book on radiation and health between 1969 and 1971; none appeared in the peer-reviewed scientific literature. The papers and book purported to provide radiation cancer estimates that, if correct, showed then-current Federal Radiation Council (pre-EPA) guidelines for maximum "allowable" radiation dose would result, if reached by all in the U.S. population, in over 100,000 extra cancer deaths per year (or 34.3% over the current rate). (Gofman et al., 1970b) The BEIR I

1972 Committee reviewed the Gofman et al. (1971) analysis, data, methodology, and basis for the estimates, and severely criticized their erroneous generalizations and assumptions (see BEIR I at 183-188).

Since that time, Gofman has published papers and articles on radiation and health, but only one has appeared in the peer-reviewed scientific literature (Gofman, 1979) and that was an early reanalysis of the Mancuso, Stewart and Kneale (1977) Hanford study. Gofman's analysis did not take into account the many criticisms of the Mancuso, Stewart and Kneale (1977) report and drew the same conclusions as did the original authors. Rejection of the Mancuso, Stewart and Kneale (1977) report, as discussed above, constitutes rejection of Gofman's article to the same effect.

In 1981, Gofman wrote a book (not peer-reviewed) setting forth his views. Gofman, unfortunately, appears to have learned little in the past decade. In his book, he repeats the same errors as in his 1969 to 1971 reports for which BEIR I (1972) criticized him. Among the many examples of Gofman's erroneous generalizations and assumptions, as criticized in BEIR I Report and utilized by Gofman in his book are the following: (1) In the 1969-1971 reports, Gofman contended that all forms of cancer show closely similar doubling-doses and closely similar percentage increases in cancer mortality rate per rad (see BEIR I at 1983). This has been proven wrong (BEIR I Report; BEIR III Report). Nevertheless, Gofman (1981)

uses this erroneous generalization in his current book. (2) In 1969-71, Gofman assumed that youthful subjects require less radiation to increase the mortality rate by specified fraction than do adults. (See BEIR I Report at 183). This has been proven wrong (see BEIR I Report; BEIR III Report; UNSCEAR, 1977). Gofman (1981) continues to use this erroneous generalization in his current book. (3) In 1969-71, Gofman assumed that there is no dose-rate effect for any type of radiation-induced malignancy (see BEIR I Report at 183). This is incorrect (see BEIR I Report; BEIR III Report; NCRP, 1980; UNSCEAR, 1977). Gofman (1981) continues with that assumption in his current book.

Gofman's 1981 risk estimates, themselves, also repeat Gofman's earlier errors. Thus, both in his early work and in his present book, Gofman used epidemiological surveys to argue his case. Analysis of certain data on radiation-induced neoplasia in the light of these assumptions led Gofman to construct a table (Gofman et al., 1970) of age-specific sensitivity of cancer induction. This table has been modified in his current book (Gofman, 1981), but remains essentially the same. His table of fixed relative risk estimates are applied to a hypothetical case of exposure using three models differing in the lengths of latency period and periods of expression of the cancer.

In his most "pessimistic" case, which makes certain erroneous assumptions, he predicts an annual radiation-induced

cancer mortality rate of over 100,000 cases, or a 34.3% increase in the present rate. In the most "optimistic" case, he predicts an increased cancer mortality rate of almost 10,000 cases or a 3.1% increase. The BEIR I Committee (1972) carefully analyzed all of Gofman's approaches (which still remain the basis for much of his current estimates in his 1981 book) and step-by-step proved they were wrong. (see BEIR I at 183-188).

Finally, certain manipulative procedures introduced by Gofman (1971) and continued in his 1981 book make substantial differences in the size of his estimates. As stated in BEIR I Report at 188, "The crucial difference in terms of predicting the annual excess cancer deaths between these [BEIR I Report] estimates and those used by Gofman et al. (1971) is the length of the plateau region [i.e., length of expression of the excess cancer in the exposed population] following exposure in utero." (at 188). The conclusion of the BEIR Committee was that "the figures generated by Gofman et al. (1971) are overestimates: the reasons for their overestimates are: (i) an overestimation of the relative risk of solid tumor induction following irradiation of 0-9 year olds by a factor of 4-5, and by a factor of 10 for all other ages; (ii) the unreasonable assumption of a life-long plateau following in utero irradiation." (BEIR I Report at 188). Gofman continues to use these generalizations and assumptions to construct tables which overestimate risk in his current book (1981).

39. Q. What is Morgan's position on low-level radiation and do you agree with it?

A. Morgan in his major article (1975) claims that low-level exposure may be more hazardous per unit of absorbed dose than exposure to high doses at high-dose rates. I do not agree with Morgan's assessment. In his article, he did not differentiate between the effects of high-LET and low-LET radiation. As a consequence, Morgan does not demonstrate that his claim holds true for low-LET radiation. Indeed, in his study, he emphasized the potential effects of high-LET radiations at high doses from internally deposited radioisotopes. Thus, Morgan's analysis does not provide any information on the low-level dose range of low-LET radiation exposure.

Since 1975, Morgan has written a number of additional articles, letters to the editor, and the like, contending that health effects from low-level radiation are being underestimated. However, in these articles, he simply is interpretating other people's data; he provides no information or data based on his own research. Increasingly, he has been placing his principal reliance on the work of those who have been thoroughly discredited (primarily Mancuso, Stewart and Kneale and Bross). Morgan has provided no information not already known to the ICRP (1977), NCRP (1975 and 1980), UNSCEAR (1977) and BEIR (1972 and 1980) Committees; and among the thousands of scientific references in these major reports, there is not one reference to Morgan as contributing scientific

knowledge to cancer or genetic risk estimation from low-level, low-LET radiation. Morgan's articles afford no basis for questioning the conclusions in these reports.

40. Q. Are any of the studies, reports, papers, or talks mentioned above, or any similar work, sufficiently reliable to justify an alteration of the conclusion that the linear, no-threshold hypothesis adopted in the DES is conservative?

A. No. The claims of higher risks from the low-dose levels described by Bross, Mancuso, Stewart, Kneale, Gofman, Morgan, Bertell and others have become the subject of considerable public debate and controversy. As I have explained, examination of their work and their reports to date does not support these claims. In addition, neither their data nor their claims are new to the scientific community or to international and national groups and councils constituted to provide expert advice on radiation and health, such as the ICRP, NCRP, BEIR Committee, and UNSCEAR. Most of the referenced authors' papers pre-date the scientific reports which analyze all the available data (e.g., UNSCEAR, 1977; BEIR I and III Reports; NCRP, 1980) and are quoted and reviewed in these reports.

Some scientific papers have been published or presented at meetings in the past year (1982-1983). However, these do not provide new information and, indeed, have never been published in the open literature through the peer-review process. They simply give the authors' current personal interpretation of old data which have been available for years or decades. The claims by the current authors do not argue

effectively against the conservatism associated with the linear hypothesis, the dose and dose-rate effectiveness in the human being for repair of radiation injury at low doses and dose rates, or the contention that the risk per rad of low-dose, low-LET radiation is less than at high doses.

No paper claiming any such effect goes unnoticed by the scientific community or goes without critical evaluation. Committee 1 of the ICRP continually reviews all epidemiological surveys, including all studies on human population groups exposed to low doses. The Committee continues to conclude that the available information on the role of low doses of radiation in cancer induction in humans does not provide any new indication of a true relationship for doses below 10 rad or low-LET radiation and that the results of new epidemiological studies of occupationally exposed groups are consistent with previously available information.

41. Q. Joint Contention II (a)(1) alleges that the long-term somatic and genetic health effects of radiation releases from the facility during normal operations, even where such releases are within existing guidelines, have been seriously underestimated ... [because] ... (a) the work of Mancuso, Steart, Kneale, Gofman, and Morgan establish that the BEIR-III Report (1980 ...) (1) incorrectly understood the latency periods for cancer ... Do you agree?

A. No. The BEIR III Report explains fully the scientific and epidemiological factors involved in the concept of "latency period" for cancer induction. "Latency period" means the period of seeming inactivity between the time of exposure

of tissue to an injurious agent and the appearance of a response in a form that can be diagnosed medically. The BEIR III Report also describes in detail the use of latency periods and follow-up intervals in the BEIR III Report and in the epidemiological studies of exposed human populations. The BEIR III Committee's treatment is in accord with all radioepidemiological studies and analyses and with the reports of the ICRP (1977), NCRP (1975, 1980); GAO (1981); UNSCEAR (1972, 1977, 1982), and all other national and international agencies and committees concerned with the radiological protection and cancer risk estimation in human populations.

42. Q. In answer to Interrogatories II-3 and II-5, Joint Intervenors state that authorities are questioning the use of the concept of "latency period" for cancers induced by radiation and that the whole concept of latency period is coming under question. Do you agree?

A. No. The concept of "latency period" in cancer induction as used in the BEIR III Report is well established. Latency period is common to all diseases (chicken pox, flu, etc.), and it exists for cancer from whatever cause. Joint Intervenors offer no reference to support their outrageous comment. In answer to Interrogatory II-55, they state they do not have the source for this representation. They do reference Morgan's 1975 article but add this information is only "tangentially relevant". In fact, the article is not relevant at all and in no way suggests the standard meaning of latency period has been called into question.

43. Q. How was the concept of latency period used by the BEIR III Committee in its estimation of risk coefficients for cancer induction in human populations exposed to low-level ionizing radiation?

A. The 1980 BEIR III Report described in considerable detail the methods used in the section, "Estimating the Total Cancer Risk of Low-Dose, Low-LET, Whole-Body Radiation" (at 176) in general, and in the subsection "Requirements for Calculating Cancer-Risk Estimates" (at 191) in particular. (I was chairman of the BEIR III subcommittee responsible for this section in the Report). There are several important parameters that influence calculations of cancer risk, one of which is the minimal latent period. Maximum latent period in particular situations can also be pertinent. These various parameters must be age- and sex-specific to be applied to the demographic model, and this was done in the BEIR III Report.

Minimal latent period is the interval between cause of a neoplastic disease and detection of a statistically significant excess of cancers. Maximum latent period is the time between exposure to a causative agent and the point after which no statistically significant excess of cancer is observed. The BEIR III Report conclusions regarding minimum and maximum latent period are as follows:

> The minimal latent period for most radiation-induced cancers is long--10 years or more after exposure. For some types--cancers arising after <u>in utero</u> irradiation, leukemia in children or adults, and bone cancer after exposure to radium-224 alpha radiation--excess cancers have been observed within 2-4 yr after

> irradiation. Moreover, there is evidence that the increased risk of leukemia and bone cancer does not persist indefinitely, but becomes negligible 25-30 yr after the end of irradiation [maximum latent period]. For all the other radiation-induced cancers reviewed in Appendix A, the minimal latent period is 10 yr or more, and there is as yet no indication that the increased risk of cancer eventually declines. There are, however, no epidemiologic studies in which followup was carried out to the end of life for the entire population cohort. Hence, any projection of risk over the lifetime of exposed persons involves considerable uncertainty. (BEIR III Report at 191, 192, 193)

The last sentence means that on information available, cancer risks from an exposure to radiation may or may not continue for a lifetime, and the projection that it so continues (that is, that there is no maximum latent period) may be conservative and overestimate risk. The approach in the BEIR III Report is to utilize a maximum latent period only for leukemia and bone cancer where this phenomenon has been observed. For other diseases, BEIR III conservatively assumes that risk continues throughout a lifetime.

With respect to minimum latent period, I wish to emphasize that the term is a definition of an observed fact. Whether or not a definition is given to the phenomenon, the time between dose and observed effect must be taken into account in any projections, including development of dose/response curves; otherwise, the projections would be demonstrably spurious.

44. Q. In answer to Interrogatory II-4, Joint Intervenors explain how they believe the BEIR III Report incorrectly understood the latency period. Do you agree?

A. No. It appears to be Joint Intervenors who understand nothing about the latency period. They suggest underlying epidemiological studies have not been pursued for a sufficiently long period of time. They are wrong. First, all available, reliable epidemiological data in the scientific literature being analyzed in the world's scientific community was made available to the BEIR III Committee for use and analysis. In some cases, the most current data was made available to the Committee before the authors submitted the data for publication. Therefore, the longest possible periods of follow-up were used. Second, as indicated, except where the data clearly showed a particular disease had a maximum latent period, the BEIR III Committee assumed there was no maximum latent period and conservatively assumed that the cancer risks for a particular exposure continues throughout an individual's lifetime.

Joint Intervenors also reference Note j (pg. 199) to Table V-14 of the BEIR III Report (concerning breast cancer), and contend BEIR III uses an incorrect latent period for breast cancer. The supporting reference is to "Rossi, in criticizing the report (at pgs. 278-279 thereof)." Joint Intervenors appear confused. First, pages 278-279 are a part of the Appendix to BEIR III Report and not of any dissent thereto. Hence,

the Report specifically includes the very pages Joint Intervenors contend should have been included. Secondly, while Rossi does dissent (BEIR III Report at 254-260), in his dissent, he never mentions breast cancer, breast cancer risks estimates or latency periods. Beyond this, Rossi's principal dissent is that the BEIR Committee's preferred dose response curve for low level, low-LET radiation overestimates risks. This is totally antithetical to Joint Intervenors' position. Third, the reference Note j has little to do with latency period and instead concerns adjustment for age at exposure to apply a uniform model for estimating risk of cancer incidence. This adjustment had no effect on the total risk estimate in the Table.

45. Q. Joint Contention II (a)(2) alleges the long-term somatic and genetic health effects of radiation releases from the facility during normal operations, even where such releases are within existing guidelines, have been seriously underestimated because (a) the work of Mancuso, Stewart and Kneale, Gofman and Morgan establish that the BEIR III Report ... (2) considered only expressed dominant genetic defects rather than recessive genetic defects .... Do you agree?

A. No, and again Joint Intervenors don't seem to understand their own contention. From whatever cause, approximately 10% of all liveborn will suffer from some form of serious spontaneously-arising genetic disease. These defects fall into four well-defined categories -- single gene dominants, recessive diseases, chromosomal derangements and irregularly inherited diseases. The last category accounts for

approximately 90% of all genetic health effects. "Recessive diseases" account for 1% of genetic health effects. Recessive diseases are scientifically defined in genetics as "diseases the result of both parents contributing the same defective genes to the offspring. Joint Intervenors, not knowing the difference apparently, essentially define "recessive diseases" as including all defects that are not single gene dominants. Joint Intervenors' definition, thereby, includes chromosomal derangements and irregularly inherited diseases as well as the properly recessive diseases.

Taking Joint Intervenors' concern as they have misdefined it, they still are wrong. The BEIR III Report extensively discusses, evaluates and estimates the risks for genetic effects from exposure to low-level radiation. The risk estimates are summarized in Table IV-2 at pg. 89. Furthermore, these conclusions are in accord with the UNSCEAR reports (1977, 1982). The UNSCEAR reports devote huge sections to the most comprehensive reports on potential genetic effects of low dose radiation that exists in the scientific literature. The approaches and estimates in the BEIR III Report are exactly the same as those in the UNSCEAR reports (1977 at 505-507 and Tables 48 and 50 at 538 and 539).

A full explanation of potential risk estimates in human genotypes that confer increased susceptibility or resistance to DNA damage and a thorough discussion of the implications of these findings for risk estimation, are included in

the BEIR III report (1980). They are also discussed at length in UNSCEAR 1977 and UNSCEAR 1982.

46. Q. Of the authors referenced by Joint Intervenors in the text of Contention II, which have written extensively, on the topic of genetic defects from low-level radiation? What are their qualifications and expertise in this respect?

A. So far as I am aware, neither Mancuso, Stewart and Kneale nor Morgan have extensively discussed genetic effects. Morgan (1978) does mention genetic effects, but not in any systematic fashion and without analysis of potential risk estimation. Contrary to Joint Inventors' Contention in answer to Interrogatory II-59, there is no "box on genetic damage" in this article. Gofman in his book addresses genetic defects.

The referenced authors, including Gofman, are not radiation geneticists, human geneticists, cytogeneticists or population geneticists. They have never trained, done research in, or published in the scientific literature in the areas of genetics indicated. Their names in any event do not appear under this topic in the references cited in the encylopedic UNSCEAR reports (1977, 1982) or the three BEIR reports.

Gofman in particular is a physician who never practiced medicine, and a Ph.D. in the area of nuclear chemistry. His interests originally were in the biophysical properties of uranium series elements, such as uranium-233. From 1960-70, the last decade of his research career, his interests were in the field of lipoproteins and cardiovascular

(heart) disease. He is not known to have done any research work in radiation genetics of which I am aware.

47. Q. With respect to Gofman (1981), has he carefully and correctly considered the issue of genetic effects?

A. No. Gofman in his book argues that the genetic sections of the BEIR III Report and the UNSCEAR 1977 Report are grossly in error. An examination of his analysis shows that his conclusions readily can be rejected.

Gofman is particularly in error as regards the genetic risk estimates on a number of counts, the most important of which appears to be a lack of understanding of the science of genetics. He has made a number of assumptions and generalizations in genetics which are incorrect. Some important ones are identified in the following excerpt, with which I agree, from a Nuclear Regulatory Commission - Harvard University Report on Revision of the Radiological Health Effects Model (1982-1983) presently in draft form (genetics section), written by the members of that Committee specializing in genetics:

> Gofman's [in his 1981 book] disagreements [with the genetic section of the BEIR III Report (1980) and the UNSCEAR Report (1977)] lie largely (though not entirely) in two areas: irregularly inherited diseases and chromosomal anomalies. For the irregularly inherited diseases, he disputes two factors entering into their estimation: the current incidence of genetically related ill health, and the mutational component of such diseases. With respect to current incidence, Gofman argues [in his book] that the BEIR III estimates are "probably 3 to 5 times too low, because important diseases of adulthood with a

genetic component are simply not listed by various quasi-governmental committees." We believe that the estimate of "at least 9%" actually comprises a substantial fraction of such diseases occurring later in life generally appear to have a smaller genetic component, so their incidence would be increased less by increased mutation rates. Certainly Gofman' upper limit guess of five times the 9% actually observed up to age 21 is unacceptable.

With respect to the mutational component of such diseases, Gofman argues that the estimate of 5-50% adopted by BEIR III or of 5% adopted by UNSCEAR are the product of "sheer, unsupported speculation," and adopts a value of 100% in his own calculations. Such a value is simply incompatible with basic mendelian genetics, however; 100% is the value for the regularly inherited diseases, and the value for irregularly inherited diseases must by definition be less. We believe values even as high as 50%, the BEIR III (1980) upper bound, are in fact the upper bound for a limited fraction of such disease (Denniston and Crow, [1982]).

Gofman's arguments [in his 1981 book] regarding chromosomal anomalies involve three separate types: deletions, translocations and nondisjunction. He argues that since most deletions are too small to be detected by conventional cytogenetic techniques, and so "far more important than is commonly recognized." This ignores completely the fact that estimates of doubling doses [of radiation] are based mainly upon mouse specific locus mutation data, and these mutations include the small deletion class. Most of the mutant alleles are homozygous lethal and a large fraction are indeed large enough to be detected cytogenetically.

The disagreement over translocations lies in Gofman's miscalculation [in his 1981 book] from published studies made in [mouse] males exposed at high dose rate. Gofman [1981] fails to take into account the dose rate reduction factor [DREF], the

> fact that the transmission of translocations in females is extremely low, and the observation that the probability of recovering an unbalanced segregation product from a translocation is only about 6%, although recent unpublished data could raise this figure to about 10%. When these appropriate corrections are made, the doubling dose for translocations is about 100 rem, not the as low as 3 rem Gofman calculates [in his 1981 book].
>
> Gofman's [1981] argument regarding radiation-induced chromosome 21 nondisjunction is even less acceptable. After noting the extremely equivocal evidence for any such effect at all, Gofman [1981] simply adopts a lower limit value of 3 rem. This, of course, implies that all trisomy-21 is radiation-induced, and is in unacceptable conflict with the evidence.

I would conclude by saying that the areas of the biological and medical sciences of genetics are an extremely complex science involving many disciplines in science, and require an extensive effort in training, research, and experience. The fields move rapidly and require scientific interaction so that new knowledge is applied correctly. Gofman expe-rience in the field nor does he interrelate with any of the scientific, medical, population, molecular, or environmental geneticists in the field. One could hardly regard him as an expert in the field of genetics or a scientist who in any way can challenge on the basis of scientific and experimental data the conclusions drawn by the many geneticist-scientists of UNSCEAR and BEIR Committees who have devoted a professional lifetime to the science of genetics.

48. Q. In answer to Interrogatory II - 13, Joint Intervenors argue that Gofman points out that for "irregularly inherited disorders" the human mutational component used in the BEIR I and BEIR III Reports were "arbitrarily" chosen by the Committee to be 5-50%. Does this relate to recessive diseases?

A. It has nothing to do with recessive diseases.

49. Q. Do you agree with the comment?

A. No. The BEIR III Committee evaluated the uncertainties involved in the light of currently known medical genetics, and concluded the following:

> The uncertainties involved in relating the incidence of irregularly inherited disorders to recurrent mutation remain too great to permit any narrowing of the range of the mutational component used in BEIR I, 5-50%. These uncertainties enter into the calculated range of increase in the equilibrium incidence of these disorders resulting from an additional 1-rem/generation increase in population exposure (Table IV-2). The current incidence of irregularly inherited disorders is approximately 90,000/million liveborn offspring. The increase expected at equilibrium would be about 20-900 per million liveborn. (BEIR III at 87)

The decision was not "arbitrary" or permissive in any way, but based on the most current knowledge and information available in human population and medical genetics and the expert knowledge of these Committee geneticists and their colleagues throughout the world.

It should be noted that UNSCEAR determined the mutational component for irregularly inherited diseases to be a flat 5%. BEIR III's range, accordingly, should be viewed as a very conservative range.

As for Gofman's criticism, it reflects his lack of understanding of genetics. As indicated above, Gofman adopts a mutational component value of 100% in his calculations; and this is simply impossible in mendelian genetics. Furthermore, Gofman's view is unsupported by references to the genetic literature. His contentions, therefore should be regarded as personal opinion and pure speculation.

50. Q. What is mutational component and why can it not be 100% for irregularly inherited diseases?

A. Mutational component, slightly simplified, is the proportion of time a genetic disorder is expressed in progeny. By definition, a genetic disorder which is expressed in 100% of the progeny is a regularly inherited (dominant) disorder. Necessarily, an irregularly inherited disorder must be expressed in the progeny less than 100% of the time and, accordingly, must have a mutational component of less than 100%.

51. Q. Bertell refers to "mild mutations" in humans, and expects these to be expressed in terms of risk estimation in human populations exposed to very low levels of ionized radiation. Do you agree?

A. No. Both BEIR I and III carefully considered mild mutations and concluded it was not appropriate to include a factor in genetic risk estimates for these in man. Bear in mind that no genetic defects have been observed in humans from low or high level radiation. Mild mutations have not even been observed in laboratory mice, the most appropriate laboratory

experimental mammal for assessing radiation-induced genetic effects for risk estimation. They have, however, been observed in the fruit fly. While this fruit fly effect is worthy of attention, as BEIR III noted, the Committee properly did not purport to estimate risk to man, because these "minor mutations" have been identified only in the fruit fly, they cannot be determined statistically, there is evidence that they occur very infrequently, and they are eliminated from the population through genetic mechanisms. (BEIR III Report at 103-104)

52. Q. Would "mild mutations" add measurably to the risk if included?

A. No. At best, mild mutations would be expressed in the population at the rate of recessive disorders. Even if these estimates were in error by an order of magnitude (an extremely conservative estimate), this still would result in no more than an additional 0.1 genetic disorder per 10 million liveborn in all future generations. Given the uncertainties defined in the BEIR III Report and the UNSCEAR Reports, the contribution of mild mutations to the genetic risk of low-level radiation such as that from normal operation of the Shearon Harris plant would be extremely small indeed.

53. Q. Is Bertell's (1979) commentary on "mild mutations" professional and competent?

A. No. Bertell's approach in her 1979 paper is to start with Bross' concept that radiation exposure at very low levels

in the 1 rad range down to 10 mrad shows an increased susceptibility to allergies, heart disease and diabetes, derived from his interpretation of the Tri-State Leukemia Study. Bross' conclusions have been extensively criticized and thoroughly discredited in the scientific literature, as discussed above. Bertell then, with no scientific evidence, relates Bross's "disease indicators" of increased susceptibility to "mild mutations" induced in the exposed population. She then concludes, without scientific basis, that increased incidences of pneumonia, dysentery, asthma, hives, eczema, and rheumatic fever (here she quotes Bross and Natajaran, 1977) are due to mild mutations randomly occurring in the germ cells of previously exposed parents. She states (at 399, Bertell, 1979): "These latter evidences of mild germ cell mutation are closely related to the adult indicators of susceptibility noted above." This conclusion is without scientific basis, and one that is solely her own opinion. Thereafter, in her paper, she makes no attempt to produce a method to estimate the effect of "mild mutations" on long-term somatic health effects. She provides no methodology for use either in checking her conclusions or utilizing them.

54. Q. Joint Intervenors II (a) (3) alleges that the long-term somatic and genetic health effects of radiation releases from the facility during normal operations, even where such releases are within existing guidelines, have been seriously underestimated ... [because] ... (a) the work of Mancuso, Stewart and Kneale, Gofman, and Morgan establish that the BEIR III Report ... (3) failed to use a supralinear response rather than a threshold or linear-or-less model to determine low-level radiation effects. Do you agree?

A. No. As discussed at some length above, the BEIR III Committee considered the supralinear model and unanimously chose not to use it for health effects under low-dose, low-LET radiation exposure since the scientific experimental and epidemiological evidence in no way showed that a supralinear model should be used for cancer-induction or genetic ill-health risk estimation in human populations exposed to low-dose, low-LET whole-body radiation. This is in accord with the scientific and epidemiological observations of the ICRP (1977), NCRP (1975, 1980), UNSCEAR (1972, 1977) and GAO (1981) Reports. Using the supralinear dose-response model, as based on the arguments of the findings of Mancuso, Stewart and Kneale in their analysis of the Hanford study, and on those of Morgan, Bertell, and Bross would argue that (a) for low-dose, low-LET radiation exposure, the risk per rem at low doses and dose rates is greater than at high doses and dose rates, (b) there is no repair from radiation injury at low doses and dose rates of low-LET radiation, and (c) that the linear hypothesis is not conservative. The scientific evidence is compelling that these three arguments are grossly incorrect (BEIR I and III Reports; ICRP, 1977; GAO, 1981; NCRP, 1975, 1980; UNSCEAR, 1972, 1977, 1982).

55. Q. Why do you say the linear dose is conservative?

A. The best available evidence to the scientific community strongly suggests that the linear model probably tends to

overestimate the risk of most radiation-induced cancers in man as a result of exposure to low-LET radiation. The linear model thus defines the upper limits of the risk associated with exposure to low-LET radiation and shows that the risks rise as the amount of the dose increases.

The consideration of the repair and recovery of radiation injury in the cells and tissues of the body and of dose-rate effectiveness factors (NCRP, 1980) leads to this conclusion. In experimental systems, the risk per unit dose of low-LET radiation for cell killing, for the induction of chromosome aberrations, mutations, and for other effects consistently depends upon both the magnitude of the dose and its temporal distribution. In general, the dose-response curves for low-LET radiation for late (carcinogenesis) and genetic effects increase in slope with increasing dose and dose rate. Thus, linear interpolation between the naturally-occurring spontaneous incidence and the incidence observed following exposure at intermediate-to-high doses and dose rates generally overestimates the rise of low-LET radiation at low doses and low-dose rates.

56. Q. You have mentioned the dose-rate effectiveness factor. What exactly is that?

A. The existence of dose-rate effectiveness factors has long been recognized from clinical experience and from studies of both genetic and somatic effects in experimental animals.

From the studies on somatic effects in animals (NCRP, 1980), the effectiveness per unit dose of low-LET radiation for cancer induction is lower at low doses and low-dose rates than at high doses and high-dose rates. The effectiveness per unit dose of high- vs. low-dose and dose rate exposure ranges from a factor of about 2 to about 10. In other words, linear interpolation from high doses (150 to 350 rads) may overestimate the effects of either low doses (0-20 rads or less) or of any dose delivered at dose-rates of the order of 5 rad per year or less by a factor of 2 to 10. This factor is referred to as the Dose Rate Effectiveness Factor (DREF). (NCRP, 1980)

Existing human data do not demonstrate conclusively that a dose rate effect does or does not exist in man. However, the experimental evidence from many different biological effects, including carcinogenesis and genetic effects, and for many species of animals in support of a dose rate effect is so extensive that it would be extraordinary if such dependence did not apply to the same endpoints in the human being as well. The NCRP (1980) concludes that the DREF range is 2 to 10 when the actual absorbed dose is 20 rads or less or when the dose rate is 5 rads per year or less. The UNSCEAR (1977) Report uses a factor of 2.

57. Q. Do the references in Joint Intervenors' answer to Interrogatory II-18 change your opinion regarding supralinearity?

A. No. I have already responded to the matter of Bross and Morgan, and the other principal authors cited by Intervenors. The risk estimates of the BEIR III Report to which the response refers is assessing the risk of low-dose, low-LET, whole-body iradiation. There is general scientific agreement that no scientific evidence exists that low-LET dose-response relationships support a supralinear model. On the contrary, they define, almost universally, a linear-quadratic model (see NCRP, 1980, BEIR III Report, UNSCEAR, 1977, 1982).

The remaining references by Intervenors -- Rossi and Radford in their dissents from the BEIR III Report, plus ICRP No. 18 (1972) and Potton (1981)--do not support Intervenors. As discussed earlier, for emissions from a nuclear power plant in routine operation the only issue is the effect of low level, low-LET radiation. In the BEIR III Report, Rossi supported the linear-quadratic model for low-level, low-LET radiation effects, but indicated that these led to <u>overestimates</u> of risk; Rossi wanted less emphasis placed on the linear model and more on the quadratic model. Indeed, the member of the BEIR Committee who most vehemently objected as regards the use of supralinearity for low-dose low-LET radiation was Dr. Harald Rossi. Radford likewise did not favor a supralinear curve, and Joint Intervenors' quotes out of context do not change this fact. He simply preferred that the <u>linear</u> (not supralinear) dose relationship be stressed more than it was. He favored, I might add, the linear quadratic relationship for leukemia and bone narrow cancers. (BEIR III Report at 244-45, 250)

The ICRP (1972) Report deals with high-LET radiations only and primarily with genetic mutagenesis in experimental systems, particularly microorganisms, fungi, plants, insects, and to a limited extent laboratory mammals (mice). No dose-response relationships or risk estimates are mentioned. ICRP risk estimates for low-dose, low-LET radiation are found in ICRP No. 26 (1977). The estimates therein are fully consistent with the BEIR reports.

The Potton (1981) paper has nothing to do with risk estimation and health effects of low-dose radiation exposure. It is a radiobiological paper on the proposed biological model for intestinal and skin cell depletion following exposure to 1200 rads, and 2700 rads of acute x-rays! No dose-response models for health risks are mentioned.

58. Q. Joint Contention II(b) states in part that "The long-term somatic and genetic health effects of radiation releases from the facility during normal operations, even where such releases are within existing guidelines, have been seriously underestimated... [because] (b) Insufficient consideration has been given to the greater radiation effects resulting from internal emitters due to ... underestimation of the health and genetic effects of alpha, beta and neutron radiation on DNA, cell membrances, and enzyme activity." Do you agree?

A. No. Based upon Intervenors' Answers to Interrogatories II-20, 22 and 24, apparently Joint Intervenors' attack in this Contention is on the BEIR reports. There is no merit to Intervenors' contention.

First, the BEIR I and III Reports thoroughly and correctly explain and apply current knowledge concerning

radiation effects, including effects on DNA, cell membranes and enzyme activity where available. The BEIR reports follow the nationally and internationally accepted approach in this respect. All radiation protection standards take into account fundamental scientific and experimental evidence at the cellular level, as well as epidemiological survey data. To the extent such data and reliable evidence can be used to support or change risk estimates, they are so used. However, when such data or evidence is incomplete or contradictory or cannot be reliably extrapolated to health effects on humans, it would be scientifically and medically inappropriate to do so, and it is not done. This is the approach in ICRP, 1977; UNSCEAR, 1977; 1982 and NCRP 1975, 1980. It was the approach in BEIR III.

Thus, in BEIR III, DNA and cellular data studies were utilized as one element in the genetic effects review and risk estimates. Likewise, study of the cells and DNA played an important role in the Committee's assessment of the dose reduction effectiveness factor discussed above (see also NCRP, 1980). I would add that the DREF research presently is the most promising area of research in mammalian cell radiobiology (see detailed reports of NCRP 1980, and UNSCEAR 1977, 1982 at 489-496, 500 to 505 and 570-579). On the other side of the coin, where cell studies did not warrant risk adjustment, they were not so used in BEIR III. For instance, after a review of the scientific literature, the BEIR III Committee determined not to accept the presentation of Sternglass that health

effects were being underestimated because of postulated effects on cell membranes. (BEIR III Report at 464-469)

In addition to properly utilizing DNA, cell membranes and enzyme activity information correctly, where available, as a general matter, BEIR III also correctly analyzed and utilized such information as it may have specifically related to alpha, beta or neutron radiation. Most important in this regard are studies of the effects of alpha and neutron radiation, which is high-LET radiation. Relevant studies of the effects of high-LET radiation on all biological systems were considered carefully in BEIR III and the other major reports (NCRP, 1975, 1980; UNSCEAR 1972, 1977, 1982; ICRP, 1972, 1966, 1977) since these are the bases for determining RBE factors and quality factors in experimental radiobiology and radiological protection (see NCRP, 1980 at 154).

59. Q. Would you describe more generally the state of knowledge regarding DNA, cell membrane and enzyme activity, and their use in risk estimation?

A. Effects on DNA are well studied in the scientific literature. It is the basis of all understanding of cell lethality and cell death, cell transformation and carcinogenesis, and cell (genetic) mutagenesis. High-LET radiation studies are the basis of RBE estimations for risk coefficients and Q (quality) factors for radiological protection standards. These effects, as discussed above, are taken into account in the assessment of radiation risk and dose-equivalent commitments to the general population in radiation protection standards.

Effects on cell membranes have been discussed in my prior answer concerning Sternglass' presentation on cell membranes. It is important to note that the risk factors for genetic effects are estimated from a knowledge of the mechanisms in lower animals extrapolated to man. For cancer, risk coefficients are estimated mainly from a knowledge of epidemiological data of the incidence of cancer in human populations exposed to intermediate- and high-dose radiation and extrapolated on the basis of theory, laboratory animal and other experiments and mathematical models. As a medical necessity, damage to biologically active micromolecules mediate all mechanisms of carcinogenesis or genetic effects. Further, some form of biological alteration at the subcellular level must act as a mediator and this occurs in the DNA, in the essential cell membrances (proteins and lipids) and in the enzymes (proteins) of the cell essential for cell survival and repair. However, it is not essential that the precise nature of these mechanisms of biological alteration at the protein or lipid levels be known in order to estimate the risk of health effects and genetic effects of radiation in human populations. What will be achieved is a better understanding of the mechanisms involved in changes leading to cancer and genetic effects and in understanding how these injuries are repaired. (NAS, 1981).

At the molecular level, it is not solely the quality (e.g., high or low-LET) of ionizing radiation that is the main factor in radiation effects, but the radiant energy interacting

with the atomic matter. At this biophysical level of the cell, the biological and biochemical changes due to the radiant energy imparted by the incident ionizing particle share common pathways leading to injury. What does occur, in general, is a greater amount of damage per unit dose with high-LET radiation versus low-LET. These differences in RBE are taken into account where appropriate in the BEIR Reports when risk estimation is determined for purposes of assessing potential health (cancer) and genetic effects.

60. Q. Joint Intervenors provide support for Contention II(b) as we have been discussing it, principally in answer to Interrogatories II-20, 22 and 24 and in the references in the Contention itself. Do these comments and references cause you to change your opinion?

A. No. What they do reflect is confusion of issues on the part of Joint Intervenors. Before commenting on the points for which Intervenors offer citation, let me observe that the uncited comments are totally puzzling in the context of Joint Intervenors' Contention II(b). Thus, Joint Intervenors reference, as apparently pertinent, radiation as a cause of reducing the effectiveness of the immune system and mutating bacteria and viruses. Doses in the thousands of rads are required to produce these effects and I cannot fathom the relation of these events to routine emissions from a nuclear power plant. Further, some terms used by Joint Intervenors, for instance, "greater specific radiation", make no scientific sense.

As for Joint Intervenors' references, they either support BEIR III, are irrelevant or are wrong. This is most significant with respect to what appears to be Joint Intervenors' greatest concern, namely, the greater relative biological effectiveness of high-LET radiation over low-LET radiation. Joint Intervenors' comments on this point at once are supportive of BEIR III and irrelevant to DES. Joint Intervenors postulate an RBE for high-LET radiation of 10 to 20. I would agree. The numbers are obtained from BEIR III and therefore support BEIR III! Further, high-LET radiation characteristics are irrelevant to consideration of health effects on routine nuclear power plant operations. Such releases amount to one/billionth of all the releases, and, accordingly, do not affect health risk estimates.

Joint Intervenors also quote a comment from BEIR III that more study is needed concerning induction damage in model membrane systems. This is irrelevant to Joint Intervenors' Contention. First, the referenced section of the BEIR III Report deals only with experimental studies on biochemical events following exposure to low dose, low-LET, external x-radiation and gamma radiation. It has nothing to do with internal emitters and does not deal with alpha, beta or neutron radiation (the matters of interest in Contention II(b)). Second, Joint Intervenors apparently believe low-level radiation cancer risk estimates in BEIR III are obtained by determining cellular effects and projecting health effects

therefrom. However, as indicated in my prior answer, for cancer, we rely on epidemiological studies and other information to extrapolate from high dose data to potential low dose effects. In short, while cellular information is certainly valuable in medical terms of cancer mechanisms, diagnosis, and cure, it is not essential to cancer risk estimates in BEIR III.

As with the quote to the BEIR III Report, the balance of Joint Intervenors' references do not address and are irrelevant to the effects of alpha, beta and neutron radiation on DNA, cell membranes and enzyme activity, and also are irrelevant to the relation of these to internal emitters. For instance, the Joint Intervenors reference Little's research. The referenced research does not deal with antibody formation; it deals with mouse fibroblast cell cultures grown in vitro, the use of x-radiation to induce cell transformation in vitro, and the relationships between cell killing and cell transformation. Furthermore, Little's hypothesis, drawn from his research studies using X-rays, is directed to the relationships between DNA repair mechanisms and cell transformation. Joint Intervenors' reference, therefore, does not deal with alpha, beta and neutron radiation effects on DNA, cell membranes and enzyme activities or on the relation of these to internal emitters. To me as a professional scientist, I must say that it is disturbing to find this kind of repeated misrepresentation on reference to the scientific literature.

61. Q. Joint Contention II(b) cites the references in Eddleman 37 (10), and this in turn references two papers in Health Physics as supporting the position set forth, namely, Health Physics 34 (1978) at 353-360 and 433-438. Do you agree these citations support Joint Intervenors' Contention II(b)?

A. No. The Intervenors surely chose the wrong citations, since both papers were written by well-known, outstanding radiation scientists who have argued strongly against the application of supralinearity to low-dose, lot-LET radiation exposure. The paper by Bond, Meinhold and Rossi (both Bond and Meinhold are senior members of ICRP and NCRP)(1978) deals with the RBE and Q factor for X-rays compared to gamma rays for low doses and dose rates. They found that the effect per rad can differ by a factor of 4, with gamma rays much less effective than X-rays. Thus, risks estimated for low-LET gamma radiation would be overestimated if based on medical X-ray exposure epidemiological studies, and the potential health risks would be substantially less. The greatest amount of radiation released under normal operations from Shearon Harris will be low-LET radiation including gamma radiation. (Bond was the chairman of the NCRP Committee which wrote Report No. 64 (NCRP, 1980), the strongest scientific support extant for linear-quadratic model for cancer induction and genetic effects.)

The second paper is a well-known report by Rossi and Mays (1978) (both are senior members of NCRP and BEIR III) that deals solely with leukemia risk from neutrons in the Japanese atomic-bomb survivors. This report played an important role in

the BEIR III Report, since both Rossi and Mays were outstanding and prominent scientists on the BEIR III Committee. Thus, its findings are accounted for in the BEIR III risk estimates.

Neither the Bond, Meinhold and Rossi (1978) paper, nor the Rossi and Mays (1978) paper has anything to do with experimental or theoretical effects on DNA, cell membranes, or cell enzyme activity.

62. Q. Joint Contention II(d) alleges "The long-term somatic and genetic health effects of radiation releases from the [Shearon Harris] facility during normal operations, even where such releases are within existing guidelines, have been seriously underestimated ... because ... (d) substantial increases in cancer mortality rates have been observed in the vicinity of nuclear facilities [ref] Sternglass, 'Cancer Mortality Changes Around Nuclear Facilities in Connecticut,' February 1978." Do you agree?

A. No. Such changes have never been observed or reported in the scientific peer-reviewed literature in the United States or in the Western world. No reports of such changes have been reported in the world literature cited in the BEIR III Report, UNSCEAR (1977, 1982), or NCRP (1975, 1980).

63. Q. Joint Intervenors cite the studies of Sternglass in support of their Contention II(c). Do you agree that the studies of Sternglass are a reliable source of authority?

A. No. For more than a decade, Sternglass has made unsupported and unproven allegations and claims that he has shown that radiation exposure from nuclear power operations has resulted in an increase in mortality, principally infant mortality. (Sternglass 1969, 1970, 1971, 1977, 1978). His

conclusions and unscientific methodology have been uniformally discredited by the scientific community. For Sternglass' population studies, his approach and the problems have remained constant over time. First, unscientifically, he predetermines a conclusion and selects data to support that conclusion. Second, he relies on data unsuitable, even if he were proceeding in good faith, for cancer studies.

64. Q. What is the problem with the data Sternglass uses?

A. Sternglass has used various statistical methods based on vital statistics data in various geographical regions to attempt to prove that very low-level radiation doses in the regions of nuclear power plants have caused excessive diseases and mortality. However, it has been clear for some time that this type of survey or study, depending as it does on death record data aggregated crudely by geographical region, does not constitute a sufficient basis for deciding whether one or another type of environmental factor such as dose levels in the region of background radioactivity or low-level radiation emitted during the normal operation of a nuclear power plants related to cancer rates. As a test of the effect on cancer risks of low-dose, low-LET protracted exposure radiation, this approach using crudely aggregated death record data does not appear to be fruitful in the United States within the framework of variations in background radiation, the very small increments of radiation emitted from normally operating nuclear

power plants, and exposure of populations insufficiently large to provide data that would be statistically useful (BEIR III Report at 469 to 471).

65. Q. What is the validity of Dr. Sternglass' conclusions? Would you discuss further his objectivity?

A. The BEIR I Committee reviewed the Sternglass allegations and his reports available to it (only one of his papers (1963) appeared in the peer-reviewed scientific literature; none of his papers on health effects of low-level radiation has been published in the peer-reviewed scientific literature since 1963). The Committee concluded the following in its 1972 Report:

> The evidence assembled by Sternglass [1969a, 1969b, 1970a, 1970b, 1971] has been critically reviewed by Lindop and Rotblat [1969] and by Tompkins and Brown [1969]. It is clear that the correlations presented in support of the hypothesis depend on arbitrary selection of data supporting the hypothesis and the ignoring of those which do not. In several regards, the data used by Sternglass appear to be in error ....In short, there is at the present time no convincing evidence that the low doses of radiation in question are associated with increased risk of mortality in infancy. Hence, for the purposes of this report, no estimates of risk are considered to be applicable. (at 178, 179).

As a result of Sternglass' repeated allegations, misrepresentations of facts and distorted scientific perspective, the president-elect and all living past presidents of the Health Physics Society (including Karl Morgan) unanimously

signed and issued in July 1971 the following statement at the 16th Annual Meeting of the Health Physics Society, thereby publicly rejecting Sternglass' allegations and criticizing his past papers:

> On the third such occasion since 1968, Dr. Ernest Sternglass has, at an annual meeting of the Health Physics Society, presented a paper in which he associates an increase in infant mortality with low levels of radiation exposure [from discharges from nuclear facilities]. The material contained in Dr. Sternglass' paper [Sternglass, 1971] has also been presented publicly at other occasions in various parts of the country. His allegations, made in several forums, have in each instance been analyzed by scientists, physicians and bio-statisticians in the Federal government, in individual States that have been involved in his reports, and by qualified scientists in other countries. Without exception, these agencies and scientists have concluded that Dr. Sternglass' arguments are not substantiated by the data he presents. The United States Public Health Service, the Environmental Protection Agency, the States of New York, Pennsylvania, Michigan and Illinois have issued formal reports in rebuttal of Dr. Sternglass's arguments. We, the President and Past Presidents of the Health Physics Society, do not agree with the claim of Dr. Sternglass that he has shown that radiation exposure from nuclear power operations has resulted in an increase in infant mortality.

Furthermore, Sternglass appeared before the BEIR III Committee and presented a number of arguments to substantiate his claim that the low-level radiation risks in man are seriously underestimated. Two arguments stand out. One was his cell-membrane argument, discussed earlier. In the other,

Sternglass alleged that fallout from Chinese nuclear bomb testing in 1976 led to an increased amount of radioactivity in milk in some areas of the United States (see BEIR III Report at 463). He concluded that there was an increase in infant mortality in the eastern-seaboard states from Delaware to New England shortly after these events--an increase that he ascribed to the radioactiviity. He also stated that his preliminary data demonstrated that this situation obtained for infant mortality increases around nuclear reactors in Connecticut and other sites, but presented no data. Although Sternglass stated that his analysis of the Chinese bomb radioactivity was incomplete, the BEIR Committee received no further data on this subject. The Committee concluded that the alleged associations claimed by Sternglass did not fit the time course for radioiosotope movement into the cow-milk food chain; nor was there clear evidence of a universally applicable change in infant mortality rates. Thus, the BEIR III Committee did not believe that the allegation was substantiated. (See BEIR III Report at 463, 464).

Sternglass' methodological manipulations of vital statistical data to cause fear arising from false allegations continues. Following the TMI accident, he claimed that an increase in hypothyroidism in newborns occurred in central Pennsylvania the year after the accident as compared with the year before the accident; he alleged that this was due to the large amounts of radioiodine released during the accident. In

each year, the numbers of cases were very small, less than 10. The report was investigated by the Pennsylvania Department of Health and the USPHS. All of Sternglass' allegations were proven wrong. What Sternglass had done was to cite from vital statistics reports (in fact, looking at the wrong columns and citing the wrong numbers), and failing to state that the numbers of cases in the two compared years were well within the range of cases occurring for a large number of years before the accident. Had he chosen a different year before the TMI accident, using the same dishonest method and logic, Sternglass could very well have "proven" that the TMI accident and the associated radioiodine releases could have caused a decrease in infant hypothyroidism in central Pennsylvania!

66. Q. How do Sternglass' Millstone studies compare with the approaches you have just described?

A. These studies are as good examples as any of the long standing problems with Sternglass' approach in his population studies. First, his Millstone conclusions are based on aggregations of vital studies crudely compiled by region. As I have indicated, this is an inappropriate procedure, even if done in good faith.

Second, his conclusions are wrong and his procedure is unprofessional. In his Millstone population study (1978), Sternglass used polemics to push a particular point of view, selected only facts suggesting that view, was illogically inconsistent and failed to consider alternative explanations,

including the possibility of random occurrences. Further, the 1978 Millstone report, referenced by Joint Intervenors, is based on an earlier Sternglass report concerning elevated-levels of strondium-90 and cesium-137 around the Millstone Plant. This earlier report selectively picked data points and drew erroneous scientific conclusions from his misinterpretation of official, documented records of environmental strondium-90 and cesium-137 levels in the atmosphere and in milk.

(For documentation of these rebuttals and criticisms, see letters and report critiques from Environmental Protection Agency Administrator, Douglas M. Costle, August 9, 1978; Nuclear Regulatory Commission Chairman Joseph M. Hendrie, January 18, 1978, incorporating the review of Professor Marvin Goldman, Director of Radiobiology Laboratory, University of California, Davis, May 31, 1978.)

67. Q. In your opinion, is Sternglass an expert in the field of low-level radiation effects?

A. No. Given his lack of professionalism and the weaknesses in his methodology, there is no way Dr. Sternglass could be viewed as an expert in low-level radiation effects.

68. Q. In answer to Interrogatory II-32, Joint Intervenors reference studies by the U.S. Public Health Service and by Carl Johnson as also showing elevated radiation health effects around a nuclear power plant. Would you comment?

A. I am not aware of any U.S. Public Health Service study showing elevated radiation health effects around nuclear facilities. On the contrary, an important scientific report, by NIOSH, a division of USPHS, indicates the very opposite. The USPHS (Rinsky et al., 1982) report demonstrates no excess of cancer in the nuclear workers over a 25 year occupational period at the Portsmouth Naval Shipyard. I would note, further, on this topic, that in January 1983, Enstrom (1983) reported no excess of any health effects in the general population resulting that from the radiation levels emitted from a normally operating nuclear power plant (San Onofre, California) in the United States.

As for the Johnson report (197), this is irrelevant for two reasons. First, his report is on plutonium effects. Plutonium is a high-LET emitter. All such emitters account for no more than one/billionth of the fatal releases from a nuclear power plant and their characteristics therefore will have no impact on health effects estimates from routine operation of a nuclear power plant.

Second, Johnson, as with Sternglass, adopted wholly inappropriate statistical procedures and ignored important environmental factors known to impact the outcome of his analysis. His study was considered and rejected as being without scientific merit by the ICRP Committee I. The BEIR III Committee also considered Johnson's work. Because of the serious methodological flaws in his statistical analyses,

incorrect statistical procedures, lack of understanding of the biopathways in man of the transuranic elements, and failures to correct for demographics and population variables, the BEIR Committee regarded the Johnson's conclusions as invalid and without meaning; it was rejected by the Committe. The study, as well, was reviewed and rejected on similar grounds by the Office of Radiation Programs of the EPA (see letter to Johnson of April 9, 1979 from the Office of Radiation Programs).

69. Q. Do you understand that Contention 37B adds any points concerning health effects not covered in Contention II?

A. No, except that Contention II is limited to cancer and genetic effects, while Contention 37B expressly raises the possibility of more diseases being caused by emissions from a nuclear power plant. In their answer to Interrogatory II-67, Joint Intervenors suggest Contention 37B also is intended to raise the issue of pain and suffering.

As to pain and suffering, I think it is safe to say that we all recognize that disease or illness, whatever the cause, is accompanied by pain and suffering. Whether the cause is radiation or something else, of course, the pain and suffering from illness or disease will be the same.

As for the contention that many diseases may be caused by the releases of a nuclear power plant in routine operation, I cannot agree. It will not occur as a medical and scientific fact, and Joint Intervenors' reference to the contrary--Bertell--completely lacks credibility.

There are two classes of radiation-associated diseases that must be considered. The first are those which potentially arise when the effects take place in one or a few cells and appear in an irradiated population as tumors or hereditary effects. This class includes only cancer and genetically-related ill-health. These diseases have the following characteristics: the incidence is related to dose, the incidence increases with increasing dose, and there is no threshold of dose-response.

The second class of radiation-associated diseases--all others than cancer and genetic effects--are those which potentially arise when the effects take place in many cells simultaneously and appear as tissue or organ damage in individuals in an irradiated population. This latter class of diseases all have a threshold dose-response relationship, that is, the diseases will not appear unless the dose is above a particular threshold. (BEIR III Report; ICRP, 1977, 1983; UNSCEAR 1977, 1982; NAS-FREIR, 1981; NCRP, 1980). The elevated dose is required before injury to the groups of cells or tissue is sufficient for the disease to appear. Characteristic threshold doses for typical tissue injury range, for example, from 500 rads for cataract induction to 4,000 rads for heart disease to up to 10,000 rads for muscle atrophy (UNSCEAR 1982, Table 8 at 631).

Because it is generally agreed that there is no threshold dose for cancer and genetic effects, these at least in theory

might be caused by the dose received from the very low levels of radiation released from nuclear power plants in routine operation. However, for all remaining diseases the threshold doses are many times greater (by orders of magnitude) than dose levels from releases during the normal operation of nuclear power plants such as Shearon Harris. Therefore, no disease, other than cancer and genetic disorders, can be considered as potential health effects in the normal operation of a nuclear power plant.

Eddleman's reference to the contrary is to Bertell, who in turn is relying on Bross. Not only does Bertell not deal with the above scientific facts, but she is not an expert in the field and her conclusions and methodology as well as Bross', as I have discussed earlier, have been completedly discredited and rejected.

70. Q. Among the authors cited by Joint Intervenors, who has attempted to calculate risk coefficients and risk factors of the sort developed in the BEIR reports and relied upon in the DES for cancer risks estimates?

A. The factors relied upon in the DES are for whole body, low-LET radiation. Only Gofman has attempted to calculate such factors from identifiable data.

71. Q. Have any of the other authors referenced by Joint Intervenors proposed cancer risks estimates or proposed an error factor for the BEIR or similar reports?

A. Numbers have been offered, but without development that would then make usable as alternatives to BEIR estimates.

Bertell once said the Mancuso/Stewart/Kneale data showed an error factor from the cancer risks estimates generally accepted of 4 to 16. However, Bertell, as she later acknowledged (1979a), was relying upon observations in a preliminary (1976) Mancuso/Stewart/Kneale statement which were not included by Mancuso/Stewart/Kneale in their actual report (1977). Morgan (1978) says the ICRP number (roughly similar to the BEIR figures) is off by a factor of 4-5 but he offers no calculation, no reference, and no scientific or epidemiologic basis of support whatever. Finally, Bross offers an error factor in the Portsmouth Naval Shipyard data survey of 20-200 for lung cancer (Bross and Driscoll, 1981). However, there is no way to transfer Bross' lung cancer factor (which is based on numerous errors) into some sort of factor for whole-body radiation, which is the pertinent consideration in the DES. In short, only Gofman even proports to be calculating (as opposed to simply creating) a whole body, low-dose risk estimate.

72. Q. Assume Gofman's analysis of cancer risks is correct and the BEIR estimate relied upon in the DES is wrong. How would this effect the DES estimate of cancer from Shearon Harris Nuclear Power Plant in routine operation?

A. The difference would be insignificant. The DES based its estimates on the linear dose-response of BEIR I. Based on this standard, the principal DES estimates are 0.008 excess cancer deaths/RRY in the general population and 0.04/RRY in the work force. These numbers are extremely small in comparison with the spontaneous incidence occurring naturally in these populations.

Assuming the most "pessimistic" approach, and that we use Gofman's worst case estimates, e.g., his analysis of the Hanford data, supralinearity of dose response, shortest possible latency effects, no repair or recovery from radiation injury, and expression of excess cancer risks from in utero exposure for an entire lifetime, then Gofman's cancer risk estimates would be approximately 40% greater (i.e. increase) per rad of exposure over that in BEIR I. (Gofman 1981 at 218) Applying this error factor to the DES numbers, we have with Gofman's factors 0.0112 cancer deaths/RRY in the general population and 0.056/RRY in the work force. The small differences are arithmetically insignificant in comparison with spontaneously occurring effects in the general population; they both fall well within probability uncertainties; they are infinitesimal; and, the calculations do not exclude a zero effect in either situation. Accordingly, whether BEIR risk estimates or Gofman's risk estimates are used, there is no significant difference in the DES (1983) estimates for potential cancer effects arising from the radiation released during the normal operation of the Shearon Harris Plant.

73. Q. Among the authors referenced by Joint Intervenors and Eddleman, who has attempted to calculate risk of genetic defects from low-level radiation?

A. Only Gofman.

74. Q. Assume Gofman's analysis is correct and the BEIR III estimates are wrong. How would this effect the DES estimate of genetic defects from the Shearon Harris Nuclear Power Plant?

A. The difference would be insignificant. The BEIR III estimate at equilibrium is approximately 60 to 1100 genetic disorders per million liveborn following 1 rem per 30-year generation (see Table IV-2, BEIR III Report at 85). The equivalent Gofman (1981) estimate is approximately 191 to 20,000 genetic disorders (see Table 76, at 849, Gofman, 1981). Calculating the risk by the relative-mutation-risk method for equilibrium estimates (see BEIR III Report at 95), the DES (1983) arrives at a figure per RRY (based on BEIR III Report estimates) of 0.1 potential genetic disorders in all future generations of the exposed population. This may be compared with a minimum expected number of spontaneous genetic abnormalities in the exposed population of 193,000. Using the Gofman (1981) risk estimates, the number for potential genetic disorders would be 7.58 times greater, or 0.76 potential genetic disorders in all future generations. The small differences are arithmetically insignificant in comparison with the very large incidence of spontaneously occurring genetic disorders in the population in all future generations; they both fall well within the probability uncertainties; they are infinitesimal; and, the calculations do not exclude a zero effect in either situation. Accordingly, where BEIR III's or Gofman's risk estimates are used, there is no significant difference in the DES (1983) estimates for potential genetic disorders arising from the radiation released during the normal operation of the Shearon Harris plant.

75. Q. On a matter relating to Contention II(c), could you comment on Caldicott's qualifications?

A. Caldicott is a pediatrician whose concern has centered on the medical consequences of nuclear warfare. She has no training or experience in radiation physics, radiation biology, radiological health, radiological protection, nuclear energy sciences, nuclear physics, or nuclear engineering. She has no publications in the peer-reviewed scientific literature in these fields, and thus has done no "work" in these fields which could conceivably relate to Joint Contention II(c).

Jacob I. Fabrikant
JACOB I. FABRIKANT

Subscribed and sworn to before me
this 1st day of October 1983

Angela D. Black

My Commission expires July 14, 1987

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CURRICULUM VITAE

JACOB I. FABRIKANT

Birth	February 9, 1928	New York, New York

Education

1948-52	McGill University, Montreal Faculty of Arts and Science	B.Sc. (magna cum laude; Chemistry)
1952-56	McGill University, Montreal Faculty of Medicine	M.D., C.M.
1961-64	University of London, England Faculty of Science	Ph.D. (Biophysics)
1978	American College of Radiology	Fellow (F.A.C.R.)

Academic Appointments

1956-57	Duke University Hospital and School of Medicine, Durham	Intern in Surgery
1957	Duke University Hospital and School of Medicine	Assistant in Pathology
1957-58	Duke University Hospital and School of Medicine	Fellow in Surgery
1958-61	The Johns Hopkins Hospital, Baltimore	Resident in Radiology
1958-61	The Johns Hopkins University School of Medicine, Baltimore	Fellow in Radiology
1961-64	Department of Physics Institute of Cancer Research University of London, England	Advanced Fellow in Academic Radiology of the James Picker Foundation, National Academy of Sciences-National Research Council
1964-65	The Johns Hopkins University School of Medicine and School of Hygiene and Public Health, Baltimore	Advanced Fellow in Academic Radiology of the James Picker Foundation, National Academy of Sciences-National Research Council
1964-68	The Johns Hopkins University School of Medicine	Assistant Professor of Radiology
1964-70	The Johns Hopkins Hospital	Radiologist

Academic Appointments (cont.)

1965-68	The Johns Hopkins University School of Hygiene and Public Health	Assistant Professor of Radiological Science
1968-70	The Johns Hopkins University School of Medicine	Associate Professor of Radiology
1969-70	The Johns Hopkins University School of Hygiene and Public Health	Associate Professor of Radiological Science
1970-75	The University of Connecticut School of Medicine, Farmington	Professor and Head Department of Radiology
1973-75	The Royal Society London, England	Special Consultant for the Advisory Committee on the Biological Effects of Ionizing Radiations, National Academy of Sciences-National Research Council, U.S.A.
1973-75	Royal Postgraduate Medical School University of London, England	Picker Sabbatical Study Year James Picker Foundation National Academy of Sciences-National Research Council, U.S.A.
1973-75	Royal Postgraduate Medical School University of London, England	Visiting Colleague Department of Diagnostic Radiology
1973-75	Hammersmith Hospital Royal Postgraduate Medical School London, England	Honorary Consultant Radiologist Department of Diagnostic Radiology
1975-78	McGill University Faculty of Medicine Montreal, Canada	Professor of Diagnostic Radiology Department of Diagnostic Radiology
1975-78	The Montreal General Hospital Montreal, Canada	Diagnostic Radiologist-in-Chief Department of Diagnostic Radiology
1976-78	McGill University Faculty of Medicine	Professor & Chairman Department of Diagnostic Radiology Diagnostic Radiologist-in-Chief
1978-	University of California School of Medicine San Francisco, California	Professor of Radiology

Academic Appointments (cont.)

1978-80	University of California, Berkeley Donner Laboratory	Staff Scientist
1979	President's Commission on the Accident at Three Mile Island, The White House, Washington, D.C.	Director, Public Health and Safety
1980-	University of California, Berkeley Lawrence Berkeley Laboratory	Staff Senior Scientist
1980-	University of California, Berkeley	Professor, Graduate Biophysics, Department of Biophysics and Medical Physics

JACOB I. FABRIKANT

Academic and Professional Organizations

American College of Radiology, 1972- ; Member, 1972-78; Fellow, 1978-
Society of Chairmen of Academic Radiology Departments, Member, 1970-75; 1976-78
Association of University Radiologists, Member, 1967-
British Institute of Radiology, Member, 1961-
Society of Nuclear Medicine, Member, The Academic Council, 1970-78
Canadian Association of Radiologists, Member, 1975-80
    Committee on Basic Research, Member, 1975-78
The New England Roentgen Ray Society, Member, 1972-78
Radiological Society of Connecticut, Member, 1971-75
Radiation Research Society, Member, 1965-
    Councillor in Medicine, 1973-76
Association for Radiation Research (U.K.), Member, 1964-
American Association for the Advancement of Science, Member, 1966-75
New York Academy of Sciences, Member, 1958-70
American Institute of Biological Science, Member, 1968-75
The Johns Hopkins Medical and Surgical Association, Member, 1965-
Connecticut State Medical Society, Member, 1971-75
Maryland Medical and Chirurgical Society, Member, 1958-70
Society of Sigma Xi, Member, 1971-
Cell Kinetics Society, Member, 1978-
Alpha Omega Alpha Honorary Medical Society, Member, 1955-
Nu Sigma Nu Medical Fraternity, Member, 1953-

Academic Honors

Alpha Omega Alpha Honorary Medical Society, McGill University Faculty of Medicine, Canada, 1955-
Wood Gold Medal, McGill University Faculty of Medicine, Canada, 1956
Advanced Fellow in Academic Radiology of the James Picker Foundation, National Academy of Sciences-National Reserach Council, 1961-65
Special Consultant, Committee on the Biological Effects of Ionizing Radiations, National Academy of Sciences-National Research Council, The Royal Society, London, England, 1973-75
Picker Sabbatical Study Year Award of the James Picker Foundation, National Academy of Sciences-National Research Council, 1973-75
Visiting Colleague in Diagnostic Radiology, Royal Postgraduate Medical School, London, England, 1973-75
Fellow of the American College of Radiology (F.A.C.R.), 1978

Extramural Research and Education Review Committees

National Academy of Sciences-National Research Council, Division of Medical Sciences, Committee on Radiology, Member, 1967-74
U.S. Atomic Energy Commission, Division of Biology and Medicine, Consultant, 1968-75
National Science Foundation, Division of Developmental Biology, Consultant, 1970
State of Connecticut, Commission on Higher Education, Standing Committee on Accreditation, Connecticut Council on Higher Education, Consultant, 1971-73
Connecticut Cancer Epidemiological Program, Planning Committee, Member, Secretary, 1972-7
American Cancer Society, Connecticut Division, Board of Directors, Member, 1972-73
U.S. Energy Research and Development Agency, Consultant, 1975-76

JACOB I. FABRIKANT

Scientific Advisory Committees

Commission on Radiation and Infection, Armed Forces Epidemiological Board, Department of the Army, Liaison Member, 1965-66
Committee on Radiology, Division of Medical Sciences, Assembly of Life Sciences, National Academy of Sciences-National Research Council, Member, 1967-74
X-Ray Image Production and Related Facilities Advisory Committee, Department of Health Education and Welfare, United States Public Health Service, Member, 1968-69
Medical Radiation Advisory Committee, Bureau of Radiological Health, Food and Drug Administration, Department of Health, Education and Welfare, USPHS, Member, 1969-74
Long-Term Radiation Effects Advisory Committee, Bureau of Radiological Health, Food and Drug Administration, Department of Health, Education and Welfare, USPHS, Member, 1969-74
Neurology A Study Section, National Institutes of Health, Department of Health, Education and Welfare, Member, 1969-72
Committee on the Biological Effects of Ionizing Radiations, Division of Medical Sciences, Assembly of Life Sciences, National Academy of Sciences-National Research Council, (BEIR I),Member, 1969-72; Subcommittee on Somatic Effects, Member, 1969-72
Committee on the Biological Effects of Ionizing Radiations, Division of Medical Sciences, Assembly of Life Sciences, National Academy of Sciences-National Research Council, (BEIR II), Member, 1973-77; Vice-Chairman, 1973-77; Subcommittee on Medical Radiatio Chairman, 1973-77
Committee on the Biological Effects of Ionizing Radiations, Division of Medical Sciences, Assembly of Life Sciences, National Academy of Sciences-National Research Council, (BEIR III), Member, 1977-80; Subcommittee on Somatic Effects, Member, 1977-80; Ad hoc Subcommittee (Subcommittee on Somatic Effects), Chairman, 1979-80
Committee on Genetic and Carcinogenic Effects, Division of Radiotherapeutic Research, Commission on Radiation Therapy, American College of Radiology, Member, 1972-76
Committee on Medical Uses of Radiation and Radiation Exposure of Patients, National Radiological Protection Board, United Kingdom, Member, 1974-75
Associate Committee on Scientific Criteria for Environmental Quality, Subcommittee on Physical Energy, National Research Council, Canada, Member, 1976-78
Committee on Radiation Risks to Space Workers (Satellite Power Systems), National Aeronautics and Space Administration, Member, 1979-81
Committee on Federal Research into the Biological and Health Effects of Ionizing Radiation, National Institutes of Health, Department of Health, Education and Welfare, USPHS, Member, 1979-80
Board of Radioactive Waste Management, National Academy of Engineering, National Research Council, Consultant, 1981-82
Committee on Federal Research on the Biological and Health Effects of Ionizing Radiation, Division of Medical Sciences, Assembly of Life Sciences, National Academy of Sciences-National Research Council, Consultant, 1980-81
President's Commission on the Accident at Three Mile Island, The White House; Director, Public Health and Safety, 1979
International Commission on Radiological Protection, Committee 1 on Radiation Effects, Member, 1980-
Committee to Review Portsmouth Naval Shipyard Cytogenetics Protocol, Division of Medical Sciences, Commission on Life Sciences, National Aacdemy of Sciences-National Research Council, Member, 1982
Advisory Committee on Radiological Health Effects, Nuclear Regulatory Commission-Harvard University School of Public Health, Member,198
Oversight Committee on the Radioepidemiologic Tables, Division of Medical Sciences, Commission on Life Sciences, National Academy of Sciences-National Research Council, Member, 1983-
Board on Research on Effects of Radiation, Division of Medical Sciences, Commission on Life Sciences, National Academy of Sciences-National Research Council,

JACOB I. FABRIKANT

University Research and Education Review Committees

The Johns Hopkins Medical Institutions, Radiation Control Committee, Member, 1966-70
The University of Connecticut Health Sciences Center, Radiation Control Committee, Chairman, 1970-73
McGill University, University Senate, Senator, 1976-78
McGill University, Faculty of Graduate Studies and Research, Faculty Council, The Graduate Council, Councillor, 1975-78
McGill University, Faculty of Medicine, Postgraduate Training Committee, Member, 1975-78
McGill University, Faculty of Medicine, Department of Diagnostic Radiology, Postgraduate Training Committee, Program Director, 1976-78
University of California, Berkeley, Lawrence Berkeley Laboratory, Radioactive Drug Research Committee, Member, 1979- ; Chairman, 1981-
University of California, Berkeley, Committee on the Protection of Human Subjects, Member, 1983-

Visiting Professorships

Visiting Professor of Radiology, Bowman Gray School of Medicine, 1968
Visiting Professor of Oncology, Clinical Cancer Program, Georgetown University School of Medicine and Hospital, 1969
Visiting Radiation Biologist, American Institute of Biological Sciences, 1969-75
William O'Brien Professor of Radiation Science, University of Minnesota School of Medicine and Hospitals, 1970
Visiting Professor of Radiology, University of Vermont College of Medicine, 1970,1977,1978
Visiting Scientist, L.H.Gray Laboratory, Cancer Research Campaign, Mt. Vernon Hospital, England, 1971
Visiting Lecturer, Cambridge University Medical School, Addenbrooke's Hospital, Cambridge, England, 1971
Visiting Professor of Radiology, University of Southern Florida College of Medicine, 1973
Visiting Professor of Radiology, University of Montreal Faculty of Medicine, 1977
Visiting Lecturer, Oxford University Medical School, The Radcliffe Infirmary, Oxford, England, 1979, 1980, 1981
Visiting Lecturer, University of London, Institute of Cancer Research, London, England, 1979, 1981
Visiting Lecturer, Royal Postgraduate Medical School, Hammersmith Hospital, London, England, 1979
Visiting Scientist, National Radiological Protection Board, Harwell, England, 1979, 1981
Visiting Professor of Radiation Medicine, Brown University Division of Biology and Medicine, Providence, 1979,1980,1981,1982

Scientific Journal Review

Cell and Tissue Kinetics, 1968- ; Member, Editorial Board, 1972-
Investigative Radiology, 1973- ; Member, Editorial Board, 1973-76
Journal of the Canadian Association of Radiologists, 1976- ; Member, Editorial Board, 1976-78
McGill Medical Journal, 1952-56; Managing Editor, 1954-55; Editor, 1955-56

Cancer Research, 1968-
Journal of the National Cancer Institute, 1969-
Science, 1970-
Radiation Research, 1972-
New England Journal of Medicine, 1982-
Biology of Reproduction, 1970-
Radiology, 1970-
Cancer, 1971-
Medicine, 1970-
BioScience, 1970-
International Journal of Applied Radiation and Isotopes, 1973-

Hospital Appointments

1964-70	The Johns Hopkins Hospital Baltimore, Maryland	Radiologist
1970-73	University of Connecticut Hospital Hartford, Connecticut	Head, Department of Radiology
1973-75	University of Connecticut Hospital Hartford, Connecticut	Attending Radiologist
1970-73	Veterans Administration Hospital Newington, Connecticut	Acting Chief, Department of Radiology; Consultant in Radiology
1971-75	New Britain General Hospital New Britain, Connecticut	Consultant in Radiology
1971-75	William W. Backus Hospital Norwich, Connecticut	Consultant in Radiology
1972-75	Hartford Hospital Hartford, Connecticut	Consultant in Radiology
1972-75	Mount Sinai Hospital Hartford, Connecticut	Consultant in Radiology
1973-75	Hammersmith Hospital London, England	Honorary Consultant Radiologist Department of Diagnostic Radiology
1975-78	The Montreal General Hospital Montreal, Canada	Diagnostic Radiologist-in-Chief Department of Diagnostic Radiology
1975-78	The Montreal General Hospital Montreal, Canada	Director, Department of Diagnostic Radiology
1978-present	Cowell Memorial Hospital University of California, Berkeley	Physician
1978-present	University of California Medical Center, San Francisco	Radiologist, Clinical Faculty

JACOB I. FABRIKANT

Certification

1962 American Board of Radiology

Medical Licensure

1957 National Board of Medical Examiners (No. 36999)
1958 Maryland (No. D 1511)
1971 Connecticut (No. 14808)
1973-75 Great Britain
1976-78 Quebec, Canada (No. 76-033)
1978 California (No. G 36656)

Military Service

World War II, Veteran, United States Navy

Marital Status

Irene B. Fabrikant, Wife

B.Sc. (McGill University)
M.Sc. (McGill University, Bacteriology and Immunology)
Ph.D. (University of Maryland, Microbiology)

1966-70 Instructor, Department of Microbiology
University of Maryland School of Medicine

1970-75 Assistant Professor of Medicine, Department of Medicine
The University of Connecticut School of Medicine

1973-75 Honorary Research Fellow (Immunology)
Department of Zoology and Comparative Anatomy
University College, London, England

1975-78 Assistant Professor, Department of Microbiology & Immunology
Faculty of Medicine, McGill University, Montreal

1977-78 Executive Secretary, McGill University Biohazards Committee
McGill University, Montreal

1978-79 Research Fellow, U.S. Public Health Service, DHEW
Center for Disease Control, San Juan Laboratories, Puerto Rico

1979-80 Research Associate, University of California, Berkeley, School of
Public Health, Department of Biomedical & Environmental Health Sciences

1981- Associate Research Immunologist, University of California
School of Medicine, San Francisco, Cancer Research Institute

JACOB I. FABRIKANT

Exhibit C

BIBLIOGRAPHY

1. Fabrikant, J.I. The Osler Society. (Editorial) McGill Med. J. 24:128, 1955.

2. Fabrikant, J.I. The Dean. (Editorial) McGill Med. J. 24:180, 1955.

3. Fabrikant, J.I. A concept of the term "anxiety". McGill Med. J. 24:201-207, 1955.

4. Fabrikant, J.I. Pediatric problems in clinical practic. (Book Review) McGill Med. J. 24:114-115, 1955.

5. Anylan, W.G., Delaughter, G.D., Jr., Fabrikant, J.I., Sullenberger, J.W. and Weaver, W.T. The management of acute venous thromboembolism. JAMA 168: 725-729, 1958.

6. Anylan, W.G., Baylin, G.J., Fabrikant, J.I. and Trumbo, R.B. Studies in coronary angiography. Surgery 45:8-18, 1959.

7. Fabrikant, J.I. Colostomy--A short review. Il. Quart. 2:23-33, 1959.

8. Sullenberger, J.W., Weaver, W.T., Fabrikant, J.I. and Anylan, W.G. A study of the pressor effects of serotonin and its possible role in massive thromboembolism. Surgical Forum 9:127-130, 1959.

9. Fabrikant, J.I. Reflections on illness. Il. Quart. 3:6-8, 1959.

10. Fabrikant, J.I., Anlyan, W.G., Baylin, G.J. and Trumbo, R.B. A comparison of various techniques for a safe and reliable method of coronary arteriography. Surgical Forum 9:233-237, 1959.

11. Fabrikant, J.I., Anlyan, W.G. and Creadick, R.N. The management of radiation injuries to the intestines. South. Med. J. 52:1186-1191, 1959.

12. Fabrikant, J.I. The ileal bladder. Il. Quart. 3:43-47, 1959.

13. Fabrikant, J.I., Anlyan, W.G., Baylin, G.J. and Trumbo, R.B. A comparison of techniques for visualization of the coronary arteries. Amer. J. Roentgenol. Rad. Therapy and Nuclear Med. 81:764-771, 1959.

14. Fabrikant, J.I. The wet colostomy. Il. Quart. 4:1-5, 1959.

15. Koehler, P.R., Fabrikant, J.I. and Dana, E.R. Gastric retention during oral cholecystography due to underlying lesions of the stomach and duodenum. Surg. Gynec. and Obstet. 110:409-412, 1960.

16. Fabrikant, J.I., Anlyan, W.G. and Creadick, R.N. Management of intestinal injuries caused by pelvic irradiation. Modern Med. 28:117-118, 1960.

17. Fabrikant, J.I. An improved ileostomy appliance. AMA Arch. Surg. 89:416-418, 1960.

18. Anlyan, W.G., Baylin, G.J., Fabrikant, J.I. and Trumbo, R.B. Studies in coronary arteriography. (In) Year Book of Radiology, Eds., Holt, J.F., Whitehouse, W.M., Jacox, H.W. and Kligerman, M.M., pp. 123-125, Year Book Medical, Chicago, 1960.

19. Fabrikant, J.I. Specialists at your service: The radiologist. II. Quart. 5: 29-32, 1961.

20. Fabrikant, J.I., Richards, G.J., Jr., Brack, C.B. and Goodwin, P.N. A vaginal applicator for radium therapy of carcinoma in the vagina. Radiology 77:987-989, 1961.

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22. Fabrikant, J.I. Reflections upon illness. Nursing News 12:3-5, 1961.

23. Fabrikant, J.I., Anlyan, W.G., Baylin, G.J. and Isley, J.K. Isotope studies for the evaluation of venous disease of the lower extremity. J. Nuclear Med. 2:136-148, 1962.

24. Koehler, P.R., Fabrikant, J.I. and Dickson, R.J. Observations on the behavior of testicular tumors with comments on racial incidence. J. Urol. 87: 577-579, 1962.

25. Fabrikant, J.I., Richards, G.J., Jr., Tucker, G.F., Jr. and Dickson, R.J. Contrast laryngography in the evaluation of laryngeal neoplasms. Amer. J. Roentgenol., Rad. Therapy and Nuclear Med. 87:822-835, 1962.

26. Fabrikant, J.I., Richards, G.J., Jr., Tucker, G.F., Jr. and Dickson, R.J. Aid to diagnosis of laryngeal cancer. Modern Med. 31:212, 1962.

27. Fabrikant, J.I. Radiological changes in experimental animals following the administration of bone-seeking radionuclides. (ab) Intern. Congr. Radiation Res. 2:212, 1962.

28. Fabrikant, J.I. Cellular response and cell population kinetics under continuous irradiation. Radiologic changes in bone following irradiation. (In) James Picker Foundation, Annual Report, pp. 23-25, New York, 1962.

29. Fabrikant, J.I. and Dickson, R.J. Contrast cinefluorographic studies of the larynx. (ab) Intern. Congr. Radiology 10:261, 1962.

30. Fabrikant, J.I. and Dickson, R.J. Clinical observations on radiation carcinogenesis. (ab) Intern. Congr. Radiology 10:243, 1962.

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32. Fabrikant, J.I. Regenerating liver. (In) Report of the Institute of Cancer Research: Royal Cancer Hospital, Annual Report, p. 122, London, 1963.

33. Fabrikant, J.I., Richards, G.J., Jr., Brack, C.B. and Goodwin, P.N. Vaginal applicator for radium therapy of carcinoma in vagina. (In) Year Book of Radiology, Eds., Holt, J.F., Whitehouse, W.M., Jacox, H.W. and Kligerman, M.M., p. 315, Year Book Medical, Chicago, 1963.

34. Fabrikant, J.I. Studies of cellular reponse and cell population kinetics under continuous irradiation. (In) James Picker Foundation, Annual Report, pp. 26-27, New York, 1963.

35. Fabrikant, J.I. Cell proliferation studies in normal, continuously irradiated and malignant tissues. Regenerating liver. (In) Report of the Institute of Cancer Research: Royal Cancer Hospital, British Empire Cancer Campaign for Research, Annual Report 41:152-153, 1964.

36. Fabrikant, J.I. and Smith, C.L.D. Radiographic changes following the administration of bone-seeking radionuclides. Brit. J. Radiol. 37:53-62, 1964.

37. Fabrikant, J.I. and Roylance, P.J. Cinefluorographic anatomy of the larynx and hypopharynx. Proc. Anat. Soc. Great Britain and Ireland 33:25, 1964.

38. Fabrikant, J.I. Investigation of cellular reponse and cell population kinetics in tissues under continuous irradiation. (In) James Picker Foundation, Annual Report, pp. 28-29, New York, 1964.

39. Fabrikant, J.I. Studies of cell proliferation in the regenerating liver and the effect of prior continuous irradiation. Ph.D. Thesis, University of London, 1964.

40. Fabrikant, J.I., Dickson, R.J. and Fetter, B.F. Mechanisms of radiation carcinogenesis at the clinical level. Brit. J. Cancer 18:459-477, 1964.

41. Fabrikant, J.I. and Dickson, R.J. The use of cinefluorography for the radiological examination of the larynx and hypopharynx in cases of suspected carcinoma. Brit. J. Radiol. 38:28-38, 1965.

42. Fabrikant, J.I. and Roylance, P.J. Cinefluorographic functional anatomy of the normal and diseased larynx. J. Anat. 99:209, 1965.

43. Fabrikant, J.I. and Koburg, E. Röntgen-Kontrastuntersuchungen von Larynx und Hypopharynx in Verbindung mit Bildverstärkung. HNO Wegw. f. fach. Praxis 13:16-19, 1965.

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46. Fabrikant, J.I. Cell cycle of regenerating hepatocytes after continuous irradiation. (ab) Intern. Congr. Radiation Res. 3:79, 1966.

47. Fabrikant, J.I. Cell population kinetics in the regenerating liver in normal and continuously irradiated mice. (ab) Intern. Congr. Radiation Res. 3: 80, 1966.

48. Fabrikant, J.I. Radiation-induced chromosome aberrations in the regenerating liver under continuous irradiation. (ab) Intern. Congr. Radiation Res. 3: 80, 1966.

49. Fabrikant, J.I. and Dickson, R.J. Use of cinefluorography for radiologic examination of larynx and hypopharynx in cases of suspected carcinoma. (In) Year Book of Cancer, Eds., Clark, R.L. and Cumley, R.W., pp. 384-387, Year Book Medical, Chicago, 1966.

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51. Fabrikant, J.I. The effect of prior continuous irradiation on the G_2, M and S phases of proliferating parenchymal cells in the regenerating liver. Radiation Res. 31:304-314, 1967.

52. Fabrikant, J.I. Radiation sterilization in man. JAMA 200:201-202, 1967.

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54. Fabrikant, J.I. The accumulation of chromosome damage under continuous low dose-rate exposure. Radiology 88:767-774, 1967.

55. Fabrikant, J.I. The ileal bladder. Colorado St. Dept. Public Health, Suppl., pp. 1-4, Denver, 1967.

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58. Fabrikant, J.I. Cell proliferation in the regenerating liver of continuously irradiated mice. Brit. J. Radiol. 40:487-495, 1967.

59. Fabrikant, J.I. and Wisseman, C.L., III. Cell proliferation in normal and malignant tissues. I. In vitro incorporation of thymidine-H^3. (ab) Radiol. Soc. N. Amer. 37:41, 1967.

60. Fabrikant, J.I. Cell proliferation in the regenerating liver and the effect of prior continuous irradiation. Radiation Res. 32:804-826, 1967.

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62. Fabrikant, J.I. The analysis of cell population kinetics in a conditional renewal system under continuous irradiation. (In) Russell H. Morgan, A Tribute. Eds., Fabrikant, J.I. and Donner, M.W., pp. 93-100, du Pont, Wilmington, Delaware, 1967.

63. Fabrikant, J.I. Kinetic analysis of hepatic regeneration. Growth 34:311-315, 1967.

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66. Fabrikant, J.I. and Wisseman, C.L., III. In vitro incorporation of tritiated thymidine in normal and neoplastic tissues. Radiology 90:361-363, 1968.

67. Morreels, C.L., Jr., Cherry, J. and Fabrikant, J.I. Masquerader. (ab) Modern Med. 36:103, 1968.

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70. Fabrikant, J.I. Cell proliferation during lymphopoiesis in normal and continuously irradiated mice. (ab) (In) Symposium on the Effect of Radiation on Cellular Proliferation and Differentiation, IAEA, Vienna, SM-103/41, 1968.

71. Fabrikant, J.I. Radiation effects on a conditional cell renewal systrem under continuous low dose rate exposure. Amer. J. Roentgenol., Rad. Therapy and Nuclear Med. 102:811-821, 1968.

72. Fabrikant, J.I. Cell proliferation in normal and malignant human tissues. (In) James Picker Foundation, Annual Report, 1967, New York, pp. 44-45, 1968.

73. Fabrikant, J.I. Cell proliferation during lymphopoiesis in normal and continuously irradiated mice. (In) Symposium on Effects of Radiation on Cellular Proliferation and Differentiation, SM-103/41, pp. 1-24, IAEA, Vienna, 1968.

74. Fabrikant, J.I. Influence of cell cycle stage on radiation response in vivo. (ab) Assn. Univ. Radiologists, 16:27, 1968.

75. Fabrikant, J.I. Cell proliferation in the regenerating liver of continuously irradiated mice; effect of a radiation-free interval. Brit. J. Radiol., 41:369-374, 1968.

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77. Hoopes, J.E. and Fabrikant, J.I. Objective evaluation of cleft palate speech. Plast. Reconstr. Surg., 42:214-224, 1968.

78. Fabrikant, J.I. The kinetics of lymphoid cell proliferation under continuous irradiation. (ab) Radiol. Soc. N. Amer., 54:149, 1968.

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80. Fabrikant, J.I. Cell proliferation during lymphopoiesis in the thymus of continuously irradiated mice. (In) Effects of Radiation on Cellular Proliferation and Differentiation, pp. 269-393, I.A.E.A., Vienna, 1968.

81. Fabrikant, J.I., Wisseman, C.L., III, and Vitak, M.J. The kinetics of cellular proliferation in normal and malignant tissues. II. An in vitro method for incorporation of tritiated thymidine in human tissues. Radiology 92:1309-1320, 1969.

82. Fabrikant, J.I. Studies on cell population kinetics in regenerating liver. (In) Human Tumor Cell Kinetics, Nat. Cancer Inst. Monogr. No. 30:169-183, 1969.

83. Fabrikant, J.I. and Cherry, J. The kinetics of cellular proliferation in normal and malignant tissues. III. Cell proliferation in the larynx. Ann. Otol., Rhinol., Laryngol., 78:326-341, 1969.

84. Hoopes, J.E., Dellon, A.L., Fabrikant, J.I. and Soliman, H. The locus of levator veli palatini function as a measure of velopharyngeal incompetence. Plastic Reconstr. Surg., 44:155-160, 1969.

85. Fabrikant, J.I. and Foster, B.R. The kinetics of lymphoid cell proliferation during radiation lymphomogenesis in C57BL mice. Radiation Res., 39:544, 1969.

86. Fabrikant, J.I. and Cherry, J. The kinetics of cellular proliferation in normal and malignant tissues. V. Analysis of labeling indices and potential doubling times in human tumor cell populations. J. Surg. Oncol., 1:27-51, 1969.

87. Fabrikant, J.I. Size of proliferating pools in regenerating liver. Exp. Cell Res., 55:277-279, 1969.

88. Fabrikant, J.I. Radiation response in relation to the cell cycle in vivo. Amer. J. Roentgenol., Rad. Therapy and Nuclear Med. 105:734-745, 1969.

89. Fabrikant, J.I. Studies on cell population kinetics in radiation leukemogenesis. Assn. Univ. Radiologists 17:88, 1969.

90. Fabrikant, J.I. and Foster, B.R. Cell cycle of lymphocytes in mouse thymus. Die Naturwissenschaften 57:567, 1969.

91. Fabrikant, J.I. Cell proliferation in continuously irradiated mammals; Effect of age. (In) Radiation Biology of the Fetal and Juvenile Mammal, Sikov, M. and Mahlum, D.D., eds. USAEC, CONF. 690501, pp. 621-628, Oak Ridge, Tenn., 1969.

92. Hoopes, J.E., Dellon, A.L., Fabrikant, J.I., Edgerton, M.T. and Soliman, A.H. Cineradiographic definition of the functional anatomy and pathophysiology of the velopharynx. (ab) Intern. Congr. Cleft Palate, 42, Houston, Texas, 1969.

93. Fabrikant, J.I. The kinetics of cellular proliferation in the seminiferous epithelium under continuous irradiation. XII Intern. Congr. Radiology 12:98, 1969.

94. Fabrikant, J.I. Radiation effects on lymphopoiesis under continuous low dose-rate exposure. Radiology 93:887-893, 1969.

95. Hoopes, J.E. and Fabrikant, J.I. Objective evaluation of cleft palate speech. (ab) Cleft Palate J. 6:181, 1969.

96. Fabrikant, J.I. Research on cell proliferation in normal and malignant human tissues. (In) James Picker Foundation Annual Report, 1968, pp. 45-50, 1969.

97. Fabrikant, J.I. The kinetics of cellular proliferation in normal and malignant tissues. VIII. Studies on cell population kinetics in normal, inflammatory and neoplastic tissues in man. (ab) XII Intern. Congr. Radiology 12:442, 1969.

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