OUTLINE OF JACOB I. FABRIKANT'S TESTIMONY
ON
CONTENTIONS 42, 111, and 112

I. Dr. Fabrikant is a physician and radiologist, professor, and researcher who has served on many committees in the area of radiation and health and has published numerous articles on that subject.

II. The 1980 NAS-BEIR committee preferred a linear quadratic hypothesis, and its determination of the excess cancer risk per rem, of generally 1 in 10,000, does not appreciably differ from the 1977 UNSCEAR Report.

A. The current estimates of risks associated with exposure to low dose, low-LET radiation were adopted over 10 years ago.

B. In 1980 NAS-BEIR Committee placed emphasis on the methods of estimation of the cancer risk coefficient rather than emphasizing the numerical estimates derived.

C. The 1980 BEIR Committee reached an agreement on the magnitude of the risk associated with low levels of radiation exposure.

III. The scientific community usually endorses the linear dose-response model for radiation protection purposes because it is the most conservative.

A. The supralinear hypothesis does not fit the data for cancer induction following exposure to low dose, lot-LET radiation.

B. Low doses of low-LET radiation do not produce more cancers per unit dose than high doses.

C. More malignancies will not occur if a given number of person-rems is distributed among a greater number of people.

D. Because of the dose rate effectiveness factor, linear interpolation from high doses may over-estimate the effects of either low doses or doses delivered at low dose rates by a factor of 2 to 10.

IV. Some reports on epidemiological studies claim the linear hypothesis is not conservative but no expert advisory committee has agreed with such claims.

A. The Mancuso, Steward, and Kneale (1977) study on the cancer mortality rate for workers at the Hanford Nuclear Facility has serious flaws and their results are inconsistent with known radiobiological evidence.

B. The Najarian and Colton (1978) study on the Portsmouth Navel Shipyard workers also suffers from serious methodological flaws.

C. Bross' conclusion that susceptible subgroups in the general population exist which are especially sensitive to radiation damage is not supported by the evidence.

D. Bross and Driscoll's (1981) study on lung cancer deaths in the Portsmouth Navel Shipyars workers uses unconventional statistical methods and has methodological flaws.

E. Modan's study on Israeli children who had been treated with radiation for ringworm of the scalp has substantial dosimetry uncertainties.

F. Dr. K.Z. Morgan's claims that low level exposure to radiation is more hazardous per unit of absorbed dose than exposure to high doses at high dose rates fails to differentiate between the effects of high-LET and low-LET radiation.

G. Dr. Morgan's various publications and talks provide no new information on the risks of low dose, low-LET radiation exposure.

V. Reliable epidemiological studies exist that allow risk estimates to be made on the effects of low-dose, low-LET radiation.

A. Any changes that have been made in the scientific dose response hypothesis and the risk estimates are results of new knowledge derived from epidemiological studies and have not been made because those studies demonstrate that the risk per rad of low-LET radiation is greater at low doses than at high doses.

B. The NAS-BEIR Committee used the Hiroshima and Nagasaki atomic-bomb survivor studies as a chief source of data, especially for low to intermediate doses of radiation.

1. The Japanese atomic-bomb survivor study does not support the hypothesis that the cancer risk from low level exposure to radiation is greater than what was previously believed.

2. The Japanese atomic-bomb survivor studies will not solve the controversies surrounding potential low dose health effects but still will play an important role in testing alternative dose-response models because of the studies' population size and dose distribution.

3. The Japanese atomic-bomb survivor studies represent normal populations.

4. The dosimetry of the Hiroshima and Nagasaki studies currently is being reassessed, but the reassessment is not expected to yield significant changes in the low dose, low-LET cancer risks estimates reported by BEIR, UNSCEAR, and other councils.

VI. Sensitive subpopulations to cancer induction exist on the basis of age and sex, but present radiation protection guides already take into account the existence of these small, unidentifiable subpopulations.

VII. Dr. Fabrikant believes the NCRP recommendations on dose limitation for exposure to declared pregnant women provide adequate protection against potential teratogenic effects.

VIII. Skin cancer is not considered to be an especially radiogenic cancer.

IX. Calculating of internal dose and dose commitments over a 50-year period provides an adequate estimation of the dose commitment to an occupationally exposed worker.

X. Dr. Fabrikant believes that the present ICRP recommended standards of dose limitation for the general public and radiation worker populations are not without risk, but it will not be possible to detect any excess

instances of cancer or genetic ill-health, if they do occur, caused by such exposures because the doses are so small and because the probability of occurrence is so infrequent.

TESTIMONY OF JACOB I. FABRIKANT

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. 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, both from McGill University (1956); 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, University of Connecticut School of Medicine; and Professor 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 Biophysics Group, Department of Biophysics and Medical Physics, University of California, Berkeley. I devote all my professional and academic activities 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.

3. Q. Have you ever been appointed to or served on, 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, 1976 BEIR II, and 1980 BEIR III Committees. I served on the 1982 National Academy of Sciences Committee on a National Institute for Occupational Safety and Health ("NIOSH") study of the Portsmouth Naval Shipyard workers. I presently 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 (1979). I have served on scientific advisory committees of the President's Commission, USPHS, NIH, NCI, BRH, NASA, American College of Radiology, the 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. I am a member of the Nuclear Regulatory Commission's Harvard University Scientific Committee for the Reevaluation of the WASH-1400 Radiological Health Effects Model.

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

A. Attached to my testimony is a complete bibliography of my publications. My publications now number in excess of 200 scientific articles, reports, chapters, and reviews in the open literature. They are all in the fields of the radiological sciences, medicine and surgery, radiobiology, radiation sciences and health, cancer biology, and related disciplines.

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

A. The BEIR Committee is a standing expert scientific advisory committee on radiation and health effects of radiation of the National Academy of Sciences - National Research Council, viz., the Committee on the Biological Effects of Ionizing Radiations. The National Council on Radiation Protection and 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 and medical applications, estimate the extent of these risks, and recommend limits on radiation exposures to worker populations and the general population. These advisory committees on radiation of international and national composition have, for these 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 those 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 populations.

I was a member of the 1972 BEIR I Committee and was on the Subcommittee on Somatic Effects. I was Vice-Chairman of the 1976 BEIR II Committee. I was a member of the 1980 BEIR III Committee, on the Subcommittee of Somatic Effects, and Chairman of the Ad Hoc Committee to estimate radiation cancer risks of 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.

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

A. The scope and purpose of my testimony is to respond those portions of Contentions 42, 111, and 112 of the Rockford League of Women Voters (the "League") which are within my scientific and medical expertise. Contention 42 generally alleges that no assurance exists that Byron can be operated so that proper radiation exposure levels to the employees and workers are maintained. Contention 111 asserts that the in-plant monitoring systems at Byron are inadequate and that Byron's design base will not keep radiation exposure levels as low as is reasonably achievable. Contention 112

generally claims that Commonwealth Edison Company ("Edison") has not accurately assessed the effects of radiation exposure on plant workers.

In creating this testimony, I have considered both the specific language of the contentions and supplementary information which the League provided in response to discovery requests by both Edison and the NRC Staff. A basic thrust of these contentions to which this testimony responds is that new scientific data are available which demonstrate that occupational radiation exposures within the limits of applicable NRC regulations and in accordance with as-low-as-reasonably achievable ("ALARA") principles have more serious health effects than previously believed. This position is articulated in many different ways, but perhaps most clearly as follows:

> spreading out a given man-rem dose to more persons . . . [causes] more cancers because the lethal cancers per man-rem are more at low doses than at high doses . . . .

League's Response to the NRC Staff's Interrogatories, p. 42-3.

7. Q. Describe the characteristics of ionizing radiation which are associated with routine exposures to radioactivity by workers in nuclear power plants in the United States.

A. Under normal operating conditions, the radiation resulting from the routine release of radioactive isotopes is primarily gamma radiation, but beta, alpha, and neutron radiations also exist in very minute amounts.

8. Q. In the scientific community, what are generally regarded as high, low, and intermediate doses of radiation?

A. The NCRP (1980) Report provides a reasonable working definition: "low" doses of sparsely ionizing radiation are arbitrarily defined as 0-20 rads; "high" doses exist at "150-350" rads; "intermediate" doses are defined as between the two; and "ultra high" doses are anything greater than 350 rads.

9. Q. What is the definition of low-LET radiation and how does it differ from high-LET radiation?

A. Linear energy transfer ("LET") is defined as the average amount of energy lost per unit of ionizing particle spur-track length. Low-LET radiation is sparsely ionizing radiation and is characteristic of electrons, x-rays, and gamma rays. Low-LET radiations are those encountered primarily in the routine operation of nuclear power plants.

High-LET radiation is characteristic of alpha particles and fast neutrons. These are commonly encountered in the operation of high energy accelerators.

10. Q. What are the observed biological effects of low-LET radiation in human beings?

A. Briefly, low-LET radiation can affect the cells and tissues of the body in three important ways. First, if the damage is caused in the DNA molecule and occurs in one or a few cells, such as those of the blood-forming tissues, the irradiated cell can occasionally transform into a cancer cell, and, after a period of time, there is an increased risk of cancer developing in the exposed individual. This biological effect is carcinogenesis; and the health effect, cancer. Second, if the embryo or fetus is exposed during gestation, injury can occur in the proliferating and differentiating cells and tissues, leading to abnormal growth. This biological effect is teratogenesis; and the health effect, developmental abnormality in the newborn. Third, if the macromolecular lesion occurs in the reproductive cell of the testis or the ovary, the hereditary genome of the germ cell can be altered, and the injury can be expressed in the descendants of the exposed individual. This biological effect

is mutagenesis; the health effect, genetically related ill-health.

There are a number of other important biological effects of ionizing radiation, such as induction of cataracts in the lens of the eye or impairment of fertility, but these three important delayed or late biological effects - carcinogenesis, teratogenesis and mutagenesis - stand out as those of greatest concern.

11. Q. How have these biological effects been observed?

A. A considerable amount of scientific information is now known from epidemiological studies of exposed human populations and from laboratory animal experiments. Furthermore, the scientific evidence indicates that any exposure to such delayed or late radiation, even at low levels of dose, carries some risk of such health effects. And as the dose of radiation increases above very low levels, the risk of these delayed or late health effects increases in exposed human populations.

12. Q. What are the observed health effects of low-LET radiation on human beings?

A. A number of important observations on the late health effects of low-LET radiation have now emerged, about which there is general scientific agreement.

These observations are based primarily on evaluation of epidemiological surveys of exposed human populations, on extensive research in laboratory animals, on analysis of dose-response relationships of carcinogenesis, teratogenesis and genetic effects, and on known mechanisms of cell and tissue injury *in vivo* and *in vitro*. Cancer-induction is considered to be the most important late somatic effect of low-dose, low-LET ionizing radiation. The different tissues appear to vary greatly in their relative susceptibility to cancer-induction by radiation. Influences affecting the cancer risk include: age at the time of irradiation and at the time of the expression of the disease, sex, and radiation factors and types such as LET and relative biological effectiveness.

Effects of growth and development in the irradiated embryo and fetus have been observed and these effects are related to the gestational stage at which exposure occurs. It appears that a threshold level of radiation dose and dose rate may exist below which gross teratogenic effects will not be observed.

Estimation of the radiation risks of genetically related ill-health are based mainly on laboratory animal observations - primarily from laboratory

mouse experiments - because of the paucity of data on exposed human populations. Genetic effects due to ionizing radiations have never been directly observed in human beings. Our knowledge of fundamental mechanisms of radiation injury at the genetic level is far more complete than, for example, of mechanisms of radiation carcinogenesis, thereby permitting greater assurance in extrapolating information on genetic mutagenesis from laboratory animals to man.

13. Q. At what level of radiation doses have these health effects been observed?

A. Epidemiological surveys of exposed human populations are highly uncertain in regard to the forms of the dose-response relationships for radiation-induced cancer in man. This is especially the case for low-level radiation. It has been necessary to estimate human cancer risk from low radiation doses primarily from observations of relatively high doses, frequently greater than 100 rads. While radiation-induced cancer in man has been observed at levels below 50 rads, the epidemiological surveys are too uncertain to provide reliable dose-response data. Surveys of developmental abnormality in the newborn demonstrate teratogenic

health effects in the 10-19 rad dose range. Genetic effects in exposed human populations have never been demonstrated, even after high-level exposure.

14. Q. Describe the relationship between a rem and a rad.

A. A rad is the unit of absorbed dose of radiation = 100 ergs/gram. The rem is the unit of dose equivalent (used in radiological protection) = absorbed dose (in rads) times quality factor times distribution factor times any other necessary modifying factors; it represents a quantity of radiation that is equivalent - in biologic damage of a specified sort - to 1 rad of 250-kVp x-rays.

15. Q. Do you agree with the position expressed in Contentions 42, 111, and 112: that the health effects of occupational exposure to radiation from routine operation of nuclear power plants in accordance with NRC regulations and ALARA are greater than set forth in the current scientific reports by standard setting bodies?

A. No.

16. Q. How does the most recent NAS-BEIR Committee Report express the health effects of low-dose exposures to low-LET radiation?

A. Table V-4 of the 1980 BEIR Report is a table which lists "Comparative Estimates of Lifetime Risk of Cancer Mortality Induced by Low-LET Radiation - Excess Deaths per Million, Average Value per Rad by Projection Model, and Type of Exposure." To compare the risk estimates with those of the 1972 BEIR Report and the 1977 United Nations Scientific Committee on the Effects of Atomic Radiation ("UNSCEAR") Report, the NAS-BEIR (1980) Report found it convenient to express the risk estimates as cancer deaths per million persons (including both sexes) per rad of continuous lifetime exposure. In this form, the risks were based on average values per rad recieved over a lifetime and are not estimates of the excess for a single dose of 1 rad. For continuous lifetime exposure to 1 rad/year, the relative-risk projection in the 1980 BEIR Report is 182 excess cancer deaths per million persons exposed per rad, and the absolute-risk projection is 67 deaths.

The 1980 NAS-BEIR Committee preferred a constrained linear-quadratic, rather than the linear, dose-response model for low-dose, low-LET, whole-body radiation and preferred not to assume a fixed relationship between the effects of high- and low-LET radiation. The BEIR Committee chose to express

the incremental cancer risk estimates as the number of excess cancers or of excess cancer deaths in an exposed population of one million people followed from the onset of exposure to the end of life. These numbers also may be expressed as percentages of the number of cancers normally expected for a population cohort of that size over the period under consideration and in the absence of any additional radiation exposure. The expression of excess cancer risk per rad generally is avoided in the published 1980 BEIR Report tables (Chapter V) because it would suggest a commitment to the linear hypothesis that some members of the Committee wished to void because they believe that the effect per rad is most probably variable: that is, an increasing function of dose in the region from zero rads up to the point where cell-killing becomes important.

The 1980 BEIR Committee linear-quadratic estimates do not differ appreciably from those in the 1977 UNSCEAR Report. These values indicate that the lifetime excess cancer risk per rem is generally 1 in 10,000 and may be considerably less.

17. Q. Does the scientific community now believe that the risks of low-dose, low-LET radiation exposure are much greater than was thought a few years ago?

A. No. The current estimate of the risks associated with exposure to low-dose, low-LET radiation was adopted by the BEIR Committee in 1972. In addition, the present protection guides for maximum dose limitations were set by the ICRP over two decades ago.

18. Q. Are there any risk estimates of health effects from low-dose, low-LET radiation as great as 10^{-3}, or one chance in one thousand, that have been endorsed by any radiation protection standard setting commission or council?

A. No.

19. Q. Did the 1980 NAS-BEIR Committee state that its numerical estimates of the cancer risk coefficients for exposure to low-dose, low-LET radiation were exact?

A. No. The 1980 BEIR Committee, in its report (NAS-BEIR 1980), emphasized that the numerical estimates for risk coefficients for low-dose, low-LET radiation are imprecise and should not be considered as firm numerical values because of numerous uncertainties. The quantitative estimation of the

carcinogenic risk of low-dose, low-LET radiation is subject to numerous uncertainties. The greatest of these involves the shape of the dose-response curve. Other uncertainties include the length of the latent period, the RBE for fast neutrons and alpha radiation relative to gamma and X radiation, the period during which the radiation risk is expressed, the model used in projecting risk beyond the period of observation, the effect of dose rate or dose fractionation, and the influence of differences in the natural incidence of specific forms of cancer. In addition, uncertainties are introduced by biological risk factors, e.g., the effect of age at irradiation, the influence of any disease for which the radiation was given therapeutically, and the influence of length of followup. Moreover, these uncertainties, unlike sampling variation, cannot be summarized in probabilistic terms. Instead, their collective influence denies any great credibility to estimates that can be made for low-dose, low-LET radiation. Therefore, the 1980 NAS-BEIR Committee placed emphasis on the methods of estimation rather than on any numerical estimates derived thereby.

20. Q. Did the 1980 BEIR Committee reach an agreement on the magnitude of the risk from low-level radiation exposure?

A. Yes. The lowest dose levels of low-LET radiation for which estimates of excess cancer risk were calculated included a single exposure (whole-body) to 10 rads to a population of 1 million people and continuous exposure to 1 rad per year for periods up to a lifetime in a population of 1 million people. The 1980 BEIR Committee agreed that the epidemiological data available were not sufficiently reliable - either with regard to the size of the study populations or to the completeness of radiation dosimetry - for estimation of excess cancer risks at dose levels below those cited. The excess cancer risk estimates were calculated using four dose-response relationships: linear, linear-quadratic, a modified form of the linear-quadratic, and a pure quadratic, and were presented as an envelope of risks. In this envelope, the linear relationship presented the highest risk, the quadratic the lowest, and the linear-quadratic relationships intermediate between the two. There was no disagreement that this envelope of risks adequately described the range of dose-response relationship at low doses.

21. Q. Why does the scientific community usually endorse the linear dose-response model for radiation protection purposes?

A. Although the weight of the experimental evidence generally favors the linear-quadratic dose-response model for low-LET radiation (NCRP, 1980), extrapolation from animals to humans is always hazardous, and for example with breast cancer, the human data provide fairly strong support for the linear model. Moreover, all of the models fit the data with approximately the same success and a conservative model is preferred when the level of uncertainty is high and when human life and health are at stake. The linear model also has an advantage in that the scientific uncertainty about dose-response models chiefly concerns the region lying below the linear regression line. The simplicity and ease of application of the linear model are important advantages. Furthermore, because the use of the linear model does not require observations over a wide range of doses, it obviates the necessity for depending so heavily on the experience of only one epidemiological survey, such as the Japanese atomic-bomb survivors study. The linear model is also a more flexible tool, permitting use to be made of all available

epidemiological data representing different exposure situations and populations.

Application of the linear model for radiological protection of workers in the workplace or the general population is prudent. Under this model, the risk of radiation-induced cancer remains the same derived from the population collective dose equivalent -- in other words, the risk is the same whether 100,000 people receive a dose of 1 rem, 10,000 people receive a dose of 10 rems, or 1,000 people receive a dose of 100 rems.

22. Q. Following exposure to low-dose, low-LET radiation, does the supralinear hypothesis fit the data for cancer induction better than the linear hypothesis?

A. No. The GAO Report (1981) describes alternative dose-response models and comments as follows on the supralinear model (see Chapter 2, page 37, GAO, 1981):

> The square root of dose-response model (such as the supralinear dose-response model as the most representative) predicts a 40 percent increase in radiation-caused-cancer if radiation exposure doubles. According to this model, lower doses of radiation are much more harmful than predicted by the linear model. The analysis of a few recent epidemiological studies have been cited by some to support the square root model. These studies have been seriously criticized on statistical and/or methodological grounds.

For a more detailed analysis of many of those studies, see the answers to questions 29 through 43 of this testimony.

23. Q. Do low doses of low-LET radiation produce more cancers per unit dose than high doses?

A. No. The best reliable evidence available to the scientific community strongly suggests that the linear model probably tends to over-estimate the risk of most radiation-induced cancers in man as a result of exposure to low-LET radiation. The linear model can be used to define the upper limits of the risk associated with exposure to low-LET radiation and implies that these risks rise proportionately as the amount of the dose increases.

24. Q. Will more malignancies occur if a given number of person-rems are distributed among a greater number of people?

A. No. This is another way of saying that the risk per rem at low doses of low-LET radiation is greater than for high doses and that the linear, no-threshold dose-response relationship is not conservative because it underestimates the risk at low doses. No convincing scientific evidence exists for these conclusions.

The consideration of the processes of repair and recovery of radiation injury in the cells and tissues of the body and of dose-rate effectiveness factors (NCRP, 1980) leads to the conclusion that the linear hypothesis generally overestimates the risk of low-LET radiation exposure at low doses. In experimental systems, the risk per unit dose of low-LET radiation for cell killing, for the induction of chromosome aberrations, mutations, teratogenic effects, tumor formation, and for the shortening of life 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 intermediate-to-high doses and dose rates generally overestimates the risk of low-LET radiation at low doses and low-dose rates. This observation also has been reported by the ICRP (1977), NCRP (1980), and UNSCEAR (1977, 1982).

25. Q. What is a dose-rate effectiveness factor?

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

effects (e.g. cancer induction) 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 (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).

Although extensive data from human beings permit reasonable risk assessments to be made for exposures to intermediate-to-high doses of low-LET radiation, these data are not adequate to demonstrate conclusively that a dose rate effect either does or does not exist. The experimental evidence from many different biological effects, including carcinogenesis, and for many species of animals in support of a dose rate effect is so extensive, however, that it would be extraordinary if such dependence did not apply to the same endpoints in the human being as well. Because of the complexity

and wide spectrum of the tumorigenic responses to radiation in the experimental animal, however, NCRP Report No. 64 (1980) is reluctant at the present time to go beyond providing a range of factors within which a single factor for the total yield of tumors in man after exposure of the whole body probably would lie. The DREF range is estimated to be 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.

26. Q. Do any recent reports on epidemiological studies claim that the linear hypothesis is not conservative?

A. Yes, but all reports of expert scientific advisory comittees, including the NCRP, ICRP, BEIR, and GAO (1981) committees, disagree with such claims. Current scientific evidence does not support those claims. Although some of those studies and reports may be worthy of further investigation, they currently are not considered convincing enough to argue effectively against the conservatism of the linear hypothesis for effects of exposure to low-dose, low-LET radiation in man.

In addition, none of those reports are new to the scientific community or to international and

national groups or councils constitued to provide expert advice on radiation and health, such as the ICRP, NCRP, NAS-BEIR Committee, or UNSCEAR. Most of those reports pre-date the scientific reports which analyze all the available data (e.g., UNSCEAR, 1977, 1982; NAS-BEIR, 1980; NCRP, 1980) and are quoted and reviewed in these reports. Although some scientific papers have been published or presented at meetings in the past year, they do not provide new information but rather give the authors' current personal interpretation of old data which have been available for years or decades. Many of these have never been published in the open literature through the critical peer-review process.

Furthermore, 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 published epidemiological surveys on exposed populations, including all studies on human population groups exposed to low doses. The Committee continues to conclude that the available information on low-dose radiation and cancer induction in humans is insufficient to enable conclusions to be drawn regarding the dose-response relationship for doses below 10 rads of low-LET radiation. The results of new

epidemiological studies of occupationally exposed groups are consistent with previously available information.

27. Q. Please describe these studies which challenge the conservatism of the linear hypothesis.

A. The following reports are among those which have been interpreted by their authors, or a few other people, to indicate that the currently applied radiation risk estimates, primarily based on the ICRP (1977), UNSCEAR (1977, 1982), and NAS-BEIR Committee (1980) reports, underestimate the risks of low-LET radiation exposure at all dose levels: Mancusco, Stewart, and Kneale, 1977; Bross and Natarajan, 1972, 1977, 1979; Bross and Driscoll, 1981; Najarian and Colton, 1978; and Morgan, 1975.

28. Q. Please describe the basis for your knowledge of these studies.

A. I have not personally reviewed the raw data which underlies these studies. All of these studies, however, have been discussed in considerable detail in meetings of the NAS-BEIR, ICRP, and other scientific committees to which I belong; I am therefore familiar with these studies. Distinguished members of those committees have in fact reviewed the raw data underlying those studies and

reported their findings to the committee. I personally critically reviewed the studies themselves and contributed to the scientific and other committee reports which analyze and criticize these studies in great detail. In many instances, the methodological and statistical flaws are apparent from the study itself.

29. Q. What data were the basis of the Mancuso, Stewart, and Kneale (1977) epidemiological survey?

A. Mancuso, Stewart, and Kneale (1977) studied 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, statistical

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; Gilbert and Marks, 1979; and Darby and Reissland (1981).

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 did not give the actual individual radiation doses the Hanford workers who died of cancer received but instead only provided 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 accumulated 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, when the incidence of cancer in the general population is plotted against increasing time.

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 or cancers of the blood-forming tissues other than multiple myeloma. Thus, no excess of leukemias existed which experience, such as with 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 esimates reported by Mancuso, Stewart, and Kneale (1979) 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 United States population. Such a result is, of course, impossible. Thus, if the Mancuso, Stewart, and Kneale (1977) cancer-doubling doses are correct, something other than radiation was the cause of the observed cancers in the Hanford workers. As a result of these criticisms, Mancuso, Stewart, and Kneale have modified their original estimates of cancer-doubling doses in the range of 2-7 rads and presently are quoting doubling doses of 2-150 rads in the worker population. The latter range, however, still is inconsistent with existing knowledge and experience in cancer epidemiology and statistics.

31. Q. What did the Najarian and Colton (1978) study involve?

A. Najarian and Colton (1978) studied employees at the Portsmouth Naval Shipyard in New England which has serviced nuclear-powered ships since 1959. In their initial report, the authors estimated that 20,000 people had been employed at the shipyard in

the past twenty years and that twenty per cent of those workers were exposed to radiation. By studying the workers' death certificates from 1959 to 1977, they estimated that 1,450 former shipyard employees died under the age of 80. The authors then telephoned relatives of the decedents to determine whether these former employees were radiation-exposed workers. They obtained telephone information on 525 cases, and relatives of 146 of these former employees claimed the deceased probably was exposed to radiation at the Portsmouth Naval Shipyard. By comparing this information to the mortality rate of United States white males in 1973, the authors concluded that the observed numbers of cancers and leukemias in the selected worker population were considerably greater than those expected. The actual number of cancers and leukemias was, however, quite small, and the conclusions based upon those numbers are erroneous.

32. Q. Does that study also suffer from serious methodological flaws?

A. Yes. Much of the criticism of the Najarian and Colton (1978) study already has been recorded. The flaws in that study include bias in worker selection, worker history, radiation dosimetry,

and confounding factors such as exposure to other carcinogens in the workplace. See Hamilton (1979).

Even Najarian and Colton (1978) listed the important inadequacies of their survey in their report. Their study was an analysis of cancer deaths only and provided no information on the total worker population at risk. A significant bias in the information supplied by relatives exists because this was recall information. The study did not incorporate data on how long the employees worked at the shipyard, how long the nuclear workers were exposed to radiation, or the amounts of radiation the workers received. Dosimetry data were not provided. The authors did not consider any confounding factors such as the carcinogenic effects of other toxic agents, including asbestos, smoking, or industrial solvents, which could have acted along with radiation to cause the apparent excess deaths from cancer and leukemia either in an additive fashion or through a multiplicative mechanism.

Further serious statistical and methodological inadequacies in the Najarian and Colton (1978) survey exist (Hamilton, 1979). For example, to exclude the effects of carcinogens other than radiation, the authors should have shown that the

cancer frequencies in the study population increased with increasing radiation exposure. Information on the lifetime-accumulated doses of former employees, however, was not available. More importantly, the radiation work at the Portsmouth Naval Shipyard began only in 1959. It is unlikely that changes in overall cancer frequency induced by radiation would appear before a minimum latent period of 10 years after the initial exposure or after 5 years for leukemia. These are roughly the minimum latent periods for cancer and leukemia induction in other studies of exposed populations. Thus, the Najarian and Colton (1978) data analysis should be divided into two periods: cancer deaths occurring during the period from 1959-1969 when radiation effects would not be expected to appear, and cancer deaths occurring from 1970-1977 when radiation effects might be expected to appear.

Of all of the Portsmouth Naval Shipyard workers who died between 1959-1969, about 25% had cancer listed on their death certificates as the cause of death. Only thirty-three radiation workers died during this period, however, and about 40% of their deaths were recorded as due to cancer. For all shipyard workers who died between 1970-1977, approximately 25% had cancer listed as the cause of death. Of the 113 radiation workers who died

during the 1970-1977 period, about 40% were due to cancer -- no more than during the previous ten year period. Thus, no differences existed in the incidence of cancer deaths in all workers or for the radiation workers during the two periods. Najarian and Colton's conclusions, therefore, are not concordant with well-established medical epidemiological data on the effect of the latency period of cancers on the expression of risk of radiation-induced cancer. This absence of the apparent latent period effect casts considerable doubt on any conclusions by Najarian and Colton (1978) and others who have chosen to cite these conclusions as evidence that very low-level doses of radiation contributed to the unexplained high numbers of cancer deaths among the radiation workers (Reissland and Dolphin, 1978).

When radiation dosimetry data were made available to Najarian and Colton, a number of serious inconsistencies in their analysis became apparent. For example, one-third of the leukemia cases reported in their original paper had no history of radiation and another one-third had negligible levels of exposure. With the new dosimetry data, statistical analyses showed no significant differences in the cancer incidence in the different exposure levels.

33. Q. Do any other significant factors exist which cast considerable doubt on the conclusions made by Najarian and Colton (1978)?

A. Yes. The list of chemical and physical agents probably present at the Portsmouth Naval Shipyard during the past 25 years includes over 40 potentially harmful chemicals. The common occupational carcinogens affecting the health of workers in the United States are quite well known. The presence of so many chemical carcinogens in the workplace underscores the difficulty in assessing the effects of low levels of radiation in this and other nuclear worker populations.

34. 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, 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).

35. 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 male 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. A National Academy of Sciences - National Research Council (NAS-NRC, 1982) scientific advisory committee has reviewed this report, and the committee did not disagree with the NIOSH study findings.

36. Q. What conclusions has Bross made in his 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 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 especially sensitive to radiation damage.

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

A. No. Bross' belief that especially sensitive subgroups exist is derived from his analysis of the Tri-State Leukemia Survey (Graham, et al., 1966; Gibson, et al., 1972) wherein he studied what he termed as certain "indicators of susceptibility" (e.g., viral infections, bacterial infections and allergies) 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). Re-analysis of Bross' observations (Smith, et al., 1973) showed, 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 in the general population would be zero. Of course, this is not the case. Also, below the ten rad exposure level, his "dose-response" curves for adults exposed to diagnostic X-rays are flat. This suggests that a

threshold exists in the dose-response relationship. Indeed, a more conventional relative risk analysis recently done (Boice and Land, 1979) found little or no increase in the risk of leukemia from a small number of diagnostic X-rays in Bross' selected Tri-State study populations.

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 (Bross et al., 1979) 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. 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) have subsequently been criticized in the scientific literature (Smith, et al., 1973; Land, 1977, 1979; Oppenheimer, 1977; Boice and Land, 1979, Rothman, 1977; MacMahon, 1972; Hamilton, 1979), as have the conclusions Bross has drawn.

Although small subpopulations which are abnormally sensitive to radiogenic cancer apparently exist, the evidence does not support Bross' conclusions that such groups may exist and be identified on the basis of the presence of certain diseases or on the existance of conditions other than age or sex. See also my response to question 55 of this testimony.

38. Q. Has Bross performed any other similar radiation study analyses?

A. Yes. Recently, Bross took data from the Najarian and Colton 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).

39. Q. Do you agree with that observation?

A. No. This Bross report has many methodological errors and uses unconventional statistical methods. Bross and Driscoll (1981) also make unsubstantiated claims on the existence of subpopulations which are especially sensitive or susceptible to radiation damage. Furthermore, in their attempt to re-analyze 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 a factor of 20 to 200. 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 people exposed per year per rem compared with the ICRP (1977) and NAS-BEIR (1980) estimates of about

1 lung cancer deaths per million people 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 or substantiated. Finally, smoking, the leading cause of lung cancer, was not examined in any detail as an important confounding factor in Bross' analysis.

40. Q. What conclusions can be drawn from Modan's study of Israeli children who had been treated with radiation for ringworm of the scalp (Modan, et al., 1977)?

A. Certain human thyroid tumor data derived from that study (Modan, et al., 1977) appear to show that the risk coefficients at low doses are greater than or equal to the risk coefficients at high doses and high dose rates. Substantial uncertainties, however, exist in the dosimetry. Any interpretation of the low-dose thyroid cancer effect in the Modan study (Modan, et al., 1977) must consider the possibility that: (1) imprecise irradiation techniques or restless children could have resulted in direct thyroid exposure; (2) pituitary irradiation may have increased the number of thyroid cancers; and (3) the radiation may have interacted with other factors such as

ethnic background, nutritional deficiencies, or goiter to alter the risk. Also, Modan's conclusions must be balanced against the lack of thyroid tumors in other similar series and the lack of such an increase in thyroid tumor incidence in children in Utah who apparently received much larger thyroid doses from fallout radioiodine than those reported in the Modan series.

41. Q. Are you familiar with Dr. K. Z. Morgan's conclusions about low-level radiation effects?

A. Yes. Dr. Morgan claimed that low-level exposure may be more hazardous per unit of absorbed dose than exposure to high doses at high dose rates (Morgan, 1975). He has persisted in that claim since then.

42. Q. Do you agree with those claims?

A. No. In his assessment, Dr. Morgan did not differentiate between the effects of high-LET and low-LET radiation. As a consequence, Dr. Morgan has never demonstrated that his claim holds true for low-LET radiation. Indeed, in his study he emphasized the potential effects of high-LET radiation at high doses from internally-deposited radioisotopes. Thus, Dr. Morgan's analysis does

not provide any information on the low-level dose range of low-LET radiation exposure.

43. Q. Do Dr. K. Z. Morgan's publications and talks provide any new facts or new information on the risks of low-dose, low-LET radiation exposure?

A. No. I have reviewed the publications listed in the League's Response to Edison's Second Set of Interrogatories, pages 9-1 to 9-3. Dr. Morgan's papers, articles, and talks provide no information not already known to the ICRP (1977), NCRP (1980), UNSCEAR (1977, 1982), and NAS-BEIR (1980) committees. Dr. Morgan is a well-known health physicist who has been a member of national and international radiation and health committees, and his writings and statements do not go unnoticed. Most of his writings and statements listed are his own opinions, unreviewed by his scientific peers, and frequently merely interpret other people's data or reports and analyses by authors who have been thoroughly discredited (e.g., Mancuso, Stewart and Kneale, 1977; Kneale, et al., 1978; Kneale et al., 1981; Bross and Natarajan, 1972, 1977; Bross, et al., 1979; Stewart, et al., 1980).

44. Q. Have the changes in scientific hypotheses and cancer risk estimates in recent years occurred

because of the existence of new scientific information that the risk per rad of low-dose exposure to low-LET radiation is greater than at high doses?

A. No. Changes in the scientific dose-response hypotheses and the risk estimates are results of new knowledge derived in the last ten to fifteen years from epidemiological studies of exposed human populations and new scientific laboratory observations. Quantitative epidemiology based on current statistical methods is a relatively new science of medicine. Until such studies were instituted with sufficiently large study populations, for periods of 10 to 20 years or more, and with sufficient information on accurate radiation dosimetry, risk estimation for excess cancer induction could not be precisely determined. Therefore, after many years of follow-up, long latent periods, and new insight into the mechanisms of radiation carcinogenesis, greater reliability and precision of risk estimation can be obtained. Even with these great advances in our knowledge, however, no new information derived from either laboratory experiments or epidemiological studies has reliably demonstrated that the risk per rad of low-LET radiation is greater at low doses than at high doses. On the contrary, the evidence is

compelling that for exposure to low-LET radiation, the risk per unit dose at low doses generally appears to be less than at high doses (see NCRP, 1980; NAS-BEIR, 1980) because of the more efficient repair mechanisms of cells and tissues of the body at low doses and low-dose rates and because of dose rate effectiveness factors (NCRP, 1980).

45. Q. Do reliable epidemiological studies exist that allow risk estimates to be made on the effects of low doses of low-LET radiation?

A. Yes. In fact, it is remarkable how many careful and detailed epidemiological studies have been made on the effects of ionizing radiation in man (see UNSCEAR, 1977, 1982; NAS-BEIR, 1980). Good information on the probability with which cancer is likely to be induced by moderate absorbed doses in the region of 100 rads and, in a few cases, by much lower doses of tens of rads is now available. This information allows risk estimates to be made with reasonable confidence for ten or more individual body organs or tissues, with the likelihood that these are the more sensitive ones to cancer induction, and an adequate estimate can be made of the total risk of cancer induction following uniform whole-body irridation.

46. Q. What use of the Hiroshima and Nagasaki atomic-bomb survivor studies was made by the NAS-BEIR committee?

A. The chief sources of data used in the NAS-BEIR Report (1980) were the populations exposed to whole-body irradiation in Hiroshima and Nagasaki, patients with ankylosing spondylitis treated with x-rays in England and Wales, other patients who were exposed to partial-body irradiation therapeutically or for diagnostic reasons, and various occupationally-exposed populations such as uranium miners and radium-dial painters. But most epidemiologic data do not systematically cover the range of low to intermediate doses, and the Japanese data appear to be fairly strong for that dose range. Analysis in terms of dose response must, therefore, rely heavily on the Japanese atomic-bomb survivor data.

The comparatively large neutron component of the dose in Hiroshima and its correlation with gamma dose, however, limit the relevance of the more numerous Hiroshima data for the estimation of risk from low-LET radiation. The Nagasaki data, for which the neutron component of dose was very small, are weaker for doses below 100 rads. Thus, it is necessary to obtain the maximum benefit from the Hiroshima data. In any analysis of the Japanese

data that attempts to separate the effects of neutrons and gamma rays, however, the gamma-ray coefficients (low-LET radiation) are determined mainly by the Nagasaki data.

47. Q. Do the epidemiological studies of either the Japanese atomic-bomb survivors or the ankylosing spondylitis patients of England and Wales support the hypothesis that the cancer risk from low-level exposure to radiation is greater than what was believed previously?

A. No. It is recognized that neither epidemiological survey involves a study population that will necessarily provide a better understanding of the carcinogenic effects of low-level radiation (see Beebe, 1981a, 1981b; Smith and Doll, 1981). They represent, however, the two most important epidemiological studies existing of large populations exposed to higher levels of radiation. These two studies are important because the irradiated populations are sufficiently large enough and the radiation doses are sufficiently high enough to provide reliable statistical analyses.
They are not, however, epidemiological studies of low-level radiation cancer effects. The Japanese atomic-bomb survivor study has a cohort study

population (Life Span Study sample) of 110,000 survivors with radiation doses estimated as high as 400 to 600 rads (kerma) (Beebe, 1981a). The mean dose estimated for the leukemia patients in the atomic-bomb survivors is 86 rads (Beebe, 1981b). The British ankylosing spondylitis patient population under study comprises 14,560 persons, and the estimated mean bone marrow dose is 321 rads with some patients receiving thousands of rads to certain tissues (Smith and Doll, 1982). These are not low-level radiation exposures.

48. Q. How will the Japanese atomic-bomb survivor studies and the British ankylosing spondylitis studies contribute to the scientific knowledge regarding the effects of low-level radiation?

A. The epidemiological investigations of the Japanese atomic-bomb survivors and the British ankylosing spondylitis patients will never solve the controversies surrounding the potential low-dose health effects no matter how long these studies continue. Direct estimates of the low-dose risk in these studies will and should be made, but their statistical instability gives them little practical value for purposes of radiological protection. The Japanese atomic-bomb data also cannot reflect

the influence of dose-rate and dose-fractionation which are especially important in analyzing the potential cancer risks of low-doses in occupationally exposed workers or the general public. Although the effect of a single dose of ionizing radiation will always be of great importance, some of the most important questions being asked in radiological protection involve continuous low dose and low dose rate exposure. The continuation of the Japanese atomic-bomb survivors and the British ankylosing spondylitis patient studies will, however, play an important role in testing alternative dose-response models. No other populations of such size and dose distribution exist that could be expected to assist in defining with greater precision dose-response relationships in the low-dose region.

49. Q. Do the two studies discussed above represent normal populations?

A. The issue of whether the populations studied are "normal" is not important to the scientific results obtained from the study. To claim that such important epidemiological studies are incorrect or spurious because they do not involve "normal populations" implies that either other populations exist that do not contain any confounding factors

or the limitations of the present radiation epidemiological studies are not taken into consideration by the scientists or the national and international committees and groups concerned with radiation and health. Neither situation is true.

50. Q. Does evidence exist to support the statement that the atomic-bomb dosimetry in the Hiroshima and Nagasaki studies needs to be reassessed?

A. Yes. Criticism of the T-65D dosimetry system used in the Japanese atomic-bomb survivor epidemiological studies since 1967 has occurred recently. The entire basis for the dosimetry is now under restudy in several of the national laboratories of the Department of Energy and in other scientific laboratories. The preliminary findings indicate that a major reduction in the neutron component of the Hiroshima doses may be required with some compensating increase in the gamma component. Any changes in the Nagasaki doses, however, would be far less extensive.

51. Q. Does this mean that the reevaluation of the Japanese data will show that the risk estimates for cancer are grossly underestimated?

A. No. Until the present uncertainties of the Japanese atomic-bomb survivor data are resolved, no precise

statement can be made as to the effect of any of the necessary changes in risk estimation. The present indications are, however, that low-dose, low-LET radiation excess cancer risk estimates will not change uniformly for the various effects. Instead, those risk estimates may stay the same in some instances, may increase by less than 10 percent in other situations, and may double under the linear or linear-quadratic models of dose-response in very limited situations.

52. Q. If there is a change in the risk estimates calculated from the Japanese atomic-bomb survivor studies, would you expect a significant change in the low-dose, low-LET cancer risk estimates reported by BEIR, UNSCEAR and radiation-protection standard-setting commissions and councils?

A. No. The excess cancer risk estimates for the largest proportion of the organs and tissues of the body exposed (cited in the NAS-BEIR (1980), UNSCEAR (1977, 1982), ICRP (1977), and NCRP (1980) reports) are not based solely on the Japanese atomic-bomb survivor studies. Instead, they are based primarily on a large number of epidemiological studies of human population groups exposed to medical radiation or to occupational radiation. These latter two groups are studied with their own individual dosimetry. The Japanese data only

augment these. Almost all excess cancer risk estimates for the different organs and tissues of the body (except for bone cancer and bone marrow cancers) are based on linearity of dose-response and, therefore, are conservative (NAS-BEIR, 1980). In the NAS-BEIR (1980) Report, the cancer risk estimates for low-dose, low-LET whole-body exposure are based primarily on the Nagasaki survivor leukemia data, and those dosimetry estimates may not change appreciably and perhaps not at all.

53. Q. How will this reassessment of the Japanese atomic-bomb dosimetry data affect the risk coefficients for radiation carcinogenesis among the Japanese atomic-bomb survivors?

A. At the present time, it is not known. It would be premature to attempt reanalysis of observed effects until the revision of dose estimation is completed. This revision is expected to last two years or more and will include reassessments of individual shielding factors and organ dose calculations.

It is recognized, however, that any changes in the Nagasaki doses in the reassessment will be far less than those of the Hiroshima doses and possibly will result in negligible effects on the Nagasaki estimates of carcinogenic risk in the

exposed atomic-bomb survivors (Bartlett, 1982).

Neutron and gamma tissue dose values as a function of distance from the hypocenters of the atomic-bomb explosions of Hiroshima and Nagasaki, however, have been recalculated by Loewe and Mendelsohn (1980; 1981) of the Lawrence Livermore National Laboratory. These calculations are supported by those of Pace (Oak Ridge National Laboratory), Scott (Scientific Applications, Inc.), and Bartlett (1982).

For values of neutron dose, the differences between the new estimates and the T-65D estimates can be broadly attributed to the effect of the moisture content of the air and to the methods of calculation, including the use of a different neutron energy distribution than presently known to be correct spectra. Humidity effects give rise to a factor of about two in reduction of neutron dose, the neutron spectra to the remainder. The reductions in dose due to the latter is greater in the case of Hiroshima.

Considerable uncertainties remain in the results of these new calculations, and firm conclusions at the present time are unwarranted (Bartlett, 1982). The reassessment is still in progress and will need to include recalculations of shielding factors for the effects of terrain and buildings,

a consideration of organ dose conversion factors, and the possible contribution to doses to persons from other sources. It is premature, therefore, to derive risk factors, RBEs, or dose-response functions using these new, but preliminary, dose estimates.

54. Q. Do all radiation epidemiological studies have uncontrolled variables such as those which exist in the atomic-bomb survivor studies?

A. Yes, but this will not necessarily reduce the value of the study. Investigators reporting their results, and those who depend on those results, should be aware of the possibility that host factors, such as age, sex, nutrition, infection, other illnesses, and carcinogens other than radiation, may be present and may influence the results in some unexpected way. The host or environmental factors may interact with radiation to exaggerate or minimize the effect of radiation or some characteristic associated with exposure to radiation may independently affect the normal expectation of the effect under study. For example, the results of the study of British ankylosing spondylitis patients treated by X-ray (Smith and Doll, 1982), valuable as they were seen to be from the time

they were first reported, were regarded with some reserve until it was shown that ankylosing spondylitis patients not treated by X-ray probably have a normal cancer experience (Smith et al., 1977) except possibly for cancer of the colon where some correlation between that cancer and ankylosing spondylitis exists through their mutual association with ulcerative colitis (Beebe, 1981b). These host and environmental factors do not, however, eliminate the value of the study as long as the researchers are aware of the possibility of their existance.

55. Q. Does scientific evidence support the theory that some people may have increased susceptibility or sensitivity to cancer induction by ionizing radiation?

A. Data on sensitive subpopulations provide one of the strongest direct pieces of evidence for the existence and importance of repair (and hence of dose-rate dependence) in radiation carcinogenesis in man. Data also aid in the identification of small population groups which apparently are abnormally sensitive to radiogenic cancer. Differences in sensitivity to radiogenic cancer obviously occur as a function of age and of sex, and therefore, sensitive subpopulations do exist

on this basis. Additionally, Bross has claimed that such groups may exist on the basis of other conditions or the presence of certain diseases, such as allergy prone or virus infection of a mother while an individual was in utero, but Bross' claims have been shown to be unsupported by the data.

A notable development during the past decade is the increasing recognition that human genotypes exist that confer both increased susceptibility or resistance to DNA damage and increased cancer risk after exposure to carcinogenic agents, including ionizing radiation. The role of constitutional susceptibility to cancer induction is not well enough understood, however, for it to be used as a factor to modify risk estimates (NAS-BEIR, 1980). The risk estimates developed for the BEIR Report (1980) are averages for large populations that include many genotypes. Thus, it is unlikely that the present risk estimates would be notably altered if data representing very small subsets of abnormally radiosensitive persons could be recognized and excluded from the calculations of the NAS-BEIR (1980) Committee cancer risk estimates. If population subsets can be identified as having a substantially greater risk of radiation carcinogenesis, and at the present this has not been

the case, their risk will require separate estimation. At the present time, the incidence and sensitivity differentials of these diseases, particularly for ionizing radiation, appear to be so low that even their increased sensitivity to radiation would not be likely to detectably influence the dose-effect response of a large, random population containing a normal number of such individuals. Accordingly, present radiation protection guides already take into account both the existence of small, unidentifiable susceptible sub-populations and the existence of various susceptibilities of the tissues and organs of the body to cancer-induction.

56. Q. Does the potential risk of teratogenic damage to the developing child in utero justify a prohibition against women of childbearing age and young men working jobs that could potentially expose them to radiation?

A. No. Teratogenic effects, by definition, can occur only in the developing embryo and fetus. Therefore, teratogenic effects cannot occur unless a woman is pregnant. I believe the NCRP recommendations on dose-limitation for exposure to pregnant women in the workplace of 500 millirem for the entire gestation period provide adequate protection

against potential teratogenic effect. Because men cannot become pregnant, teratogenic effects cannot be considered relevant to radiation protection of young men who are occupationally exposed.

57. Q. Is skin cancer, particularly malignant melanoma, considered to be an especially radiogenic cancer?

A. No. The skin is not considered to be a sensitive organ to radiation-induced cancer. Doses of radiation as high as 1500 to 2000 rads are required before malignant lesions will occur in the skin. Such doses are far beyond the levels of exposure a nuclear power plant worker can expect to receive in his entire lifetime (NAS-BEIR, 1980; UNSCEAR, 1977, 1982; ICRP, 1977).

In addition, malignant melanoma is one skin cancer in which ionizing radiation is not considered to be a risk factor (Austin et al., 1981). The epidemiological study at the Lawrence Livermore National laboratory (Austin et al., 1981) has demonstrated no causal relationship between radiation exposure and the increase of malignant melanoma in workers. These scientists studied over 5,000 employees of the Lawrence Livermore National Laboratory and noted a statistically significant excess of malignant melanoma among male workers. The cancers were, however, found to be unrelated

to either the length of employment or to the type of radiation exposure. It was highest among the group of employees classified as chemists. The study did not give detailed exposure comparisons, but the authors attributed the excess malignant melanoma of the skin to the life style of the exposed persons and not to the radiation.

58. Q. Do internal dose and dose commitments that are calculated over a 50 year period provide an adequate estimation of the internal dose and dose commitments that an occupationally exposed worker will receive?

A. Yes. This 50 year period was established by the NRC and represents an appropriate length of time the average person works in a lifetime. This period is used for the general population as well. Because the rate of accrual of a committed dose in the worker population tends to decrease over time, the percentage of a person's total dose which he would receive after the 50 year period would be quite small. In addition, the average age of the general population, the likelihood that a worker will not be employed at the same job for more than 50 years, the normal operating lifespan of a nuclear power plant, and the probability that a person will not live his entire life in the same

geographical location, all make it extremely unlikely that large numbers of people will be continually exposed to radiation, in the workplace or in the areas surrounding a normal operating nuclear power plant, for periods of more than 50 years in their lifetime.

59. Q. In your opinion, what levels of low-LET radiation exposure are sufficiently low as to properly protect the health and safety of the workers at the Byron Station?

A. Epidemiological studies primarily involve studies of people after exposure to intermediate-to-high doses and dose rates. Linear interpolation of dose-response between the naturally-occurring spontaneous incidence and the incidence observed following exposure at intermediate-to-high doses and dose rates generally overestimates the risk of low-LET radiation at low doses and low dose rates and may be considered as an upper bound. The ICRP (1977), NCRP (1975), UNSCEAR (1977, 1982), and NAS-BEIR (1980) reports all incorporate this observation.

On this basis, some potential risk of carcinogenic or genetic effects of low-dose, low-LET radiation exposure exists which is estimated to be no greater

than a lifetime risk of one in ten thousand per average rem to large populations and could be much less (ICRP, 1977; NCRP, 1980; UNSCEAR, 1977, 1982; NAS-BEIR, 1980). This is a conservative estimate, and the existing epidemiological data will not support any greater risk estimate. Quite apart from current NRC or EPA regulatory positions, no epidemiologic data exist to support any suggestion that maximum levels of 25 mrem (whole-body) and 75 mrem (thyroid) per year are necessary to protect the public health.

The present ICRP (1977) recommended standards of dose-limitation - 0.5 rem per year for the general public and 5 rem per year for the radiation worker population - are not to be considered without risk. Such a concept does not exist, because the "linear, no-threshold hypothesis" does not accept the possibility that no risk exists. The practical questions of concern, however, are whether these current recommended standards adequately protect the general public and worker populations taking into account all factors, including a comparison of risks that society normally accepts in every day life.

The question of interest for this proceeding is: For the radiation worker population in industry, will delayed or late health effects occur at

levels of exposure in the range of 0.5 to 5 rems per year? The NAS-BEIR (1980) Committee concluded that delayed health effects could occur in those radiation workers who are exposed to an amount of radiation close to the maximum permissible dose during their entire occupational lifetime.

Such health effects, however, are unlikely to ever be observed in these occupationally exposed populations. This is so for at least two reasons. First, as stated previously, the current risk estimates for radiation protection standards, recommended by the ICRP and NCRP, are based on the linear hypothesis which is considered to represent the upper bound of the risk; the scientific evidence is compelling that the actual risk would be considerably less even for an accumulated dose in the range of 100-150 rems working lifetime of low-dose, low-LET radiation exposure at low dose-rates. Furthermore, any estimation of risk based on the linear hypothesis does not take into account known biological processes of cell and tissue repair and recovery following exposure to low levels of low-LET radiation delivered at low dose-rates. Accordingly, and as mentioned previously in this testimony, a dose rate effectiveness factor of from 2 to 10 can be predicted to

decrease the potential risk due to these normal processes of cell and tissue repair and recovery. Second, one must keep in mind that any potential incremental risk of cancer-induction or genetic ill-health due to exposure to low-LET radiation at dose-rates between .5 and 5 rems per year would be extremely small when superimposed on the present risks to the population of spontaneous cancer induction or genetic ill-health due to natural causes and in the absence of any excess radiation. Hence, it would not be possible to actually detect any excess incidences of cancer or genetic ill-health, if these do occur, caused by exposures in the range described, because the radiation doses are so small and the probability of occurance is so infrequent.

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

JACOB I. FABRIKANT

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

JACOB I. FABRIKANT

Academic Appointments (cont.)

1978-80	University of California, Berkeley Donner Laboratory	Staff Scientist Research Medicine and Radiology
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

JACOB I. FABRIKANT

Academic and Professional Organizations

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

Academic Honors

Alpha Omega Alpha Honorary Medical Society, McGill University Faculty of Medicine, Montreal, 1955-
Wood Gold Medal, McGill University Faculty of Medicine, Montreal, 1956
Advanced Fellow in Academic Radiology of the James Picker Foundation, National Academy of Sciences-National Research 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, England, 1973-75
Fellow of the American College of Radiology (F.A.C.R.), 1978

Visiting Professorships

Visiting Professor of Radiology, Bowman Gray School of Medicine, 1968
Visiting Professor of Oncology, Clinical Cancer Program, Georgetwon 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 Professorships (Continued)

Visiting Professor of Radiology, University of Vermont College of Medicine, 1970, 1977-78
Visiting Scientist, Gray Laboratory, Cancer Research Campaign, Mt. Vernon Hospital, England, 1971
Visiting Lecturer, Cambridge University Medical School, Addenbrooke's Hospital, England, 1971
Visiting Professor of Radiology, University of Southern Florida College of Medicine, 1973
Visiting Professor of Radiology, University of Montreal, Faculty of Medicine, Montreal, 1977
Visiting Lecturer, Oxford University Medical School, The Radcliffe Infirmary, Oxford, England, 1979
Visiting Lecturer, University of London, Institute of Cancer Research, London, England, 1979
Visiting Professor of Radiation Medicine, Brown University, 1979

Scientific Advisory Committees

Commission on Radiation and Infection, Armed Forces Epidemiological Board, Liaison Member, 1965-66
Committee on Radiology, Division of Medical Sciences, National Academy of Sciences-National Research Council, Member, 1967-74
X-Ray Image Production and Related Facilities Advisory Committee, DHEW, USPHS, Member, 1968-69
Medical Radiation Advisory Committee, Bureau of Radiological Health, DHEW, USPHS, Member, 1969-74
Long-Term Radiation Effects Advisory Committee, DHEW, USPHS, Member, 1969-74
Neurology A Study Section, National Institutes of Health, DHEW, Member, 1969-72
Committee on the Biological Effects of Ionizing Radiations, National Academy of Sciences-National Research Council, Member, 1973-;
Vice-Chairman, 1973-77;
Subcommittee on Medical Radiation, Member, 1973-77
Subcommittee on Somatic Effects, Member, 1977-
Ad hoc Subcommittee on Somatic Effects, Chairman, 1979-
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 the 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 (Space Powered Satellite), National Aeronautics Space Administration, Member, 1979-
Committee on Federal Research into the Biological Effects of Ionizing Radiation, National Institutes of Health, DHEW, Member, 1979-
President's Commission on the Accident at Three Mile Island, The White House, Washington, D.C.; Director, Public Health and Safety, 1979
International Commission on Radiological Protection, Committee 1 on Radiation Effects, Member, 1980-
Committee on NIOSH Portsmounth Naval Shipyard Workers Study, National Academy of Sciences-National Research Council, Washington, D.C., 1982-

JACOB I. FABRIKANT

Extramural Research and Education Review Committees

National Academy of Sciences-National Research Council, Committee on Radiology, Division of Medical Sciences, 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, Secretary, 1972-73
American Cancer Society, Connecticut Division, Board of Directors, Member, 1972-73
National Academy of Sciences-National Research Council Assembly of Life Sciences, Division of Medical Sciences, Consultant, 1972-75
U.S. Energy Research and Development Agency, Consultant, 1975-76
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

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-
Biology of Reproduction, 1970-
Radiology, 1970-
Science, 1970-
Medicine, 1970-
BioScience, 1970-
Cancer, 1971-
Radiation Research, 1972-
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- Research Associate, University of California, Berkeley, School of
Public Health, Department of Biomedical & Environmental Health Sciences.

BIBLIOGRAPHY

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

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

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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.

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

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

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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.

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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.

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

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

61. Fabrikant, J.I., Peterson, W.E. and Donner, M.W. Biographical note. (In) Russell H. Morgan, A Tribute. Eds., Fabrikant, J.I. and Donner, M.W., pp. xi-xii, duPont, Wilmington, Delaware, 1967.

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76. Fabrikant, J.I., Vitak, M.J. and Wisseman, C.L., III. The kinetics of cellular proliferation in human tissues. IV. Nucleic acid synthesis and the cell cycle in relation to normal, inflammatory and neoplastic growth. (ab) Radiol. Soc. N. Amer., 54:47, 1968.

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79. Knudson, D.H. and Fabrikant, J.I. Radiographic evaluation of x radiation induced bone tumors. (ab) Radiol. Soc. N. Amer., 54:180, 1968.

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