Nov. 1, 1982

UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

BEFORE THE ATOMIC SAFETY AND LICENSING BOARD

In the Matter of	)	
	)	
UNITED STATES DEPARTMENT OF ENERGY	)	
	)	Docket No. 50-537
PROJECT MANAGEMENT CORPORATION	)	
	)	
TENNESSEE VALLEY AUTHORITY	)	
	)	
(Clinch River Breeder Reactor Plant)	)	

NRC STAFF TESTIMONY OF MICHAEL A BENDER, Ph.D.
REGARDING CONTENTION 11(b)

Question 1: By whom are you employed, what is your position, and what is the nature of your work?

Answer 1: I am employed by the Brookhaven National Laboratory where I am Senior Scientist in the Medical Department. I am also employed by the United States Nuclear Regulatory Commission as a consultant through a contract between the Clinch River Breeder Reactor Program Office and the Brookhaven National Laboratory. At Brookhaven I conduct research on the genetic effects of radiation and other environmental agents, and on the molecular mechanisms involved. A statement of my professional qualifications is attached.

Question 2: What is the subject of your testimony?

Answer 2: My testimony addresses Intervenor's Contention number 11b:

"Neither Applicants nor Staff have adequately assessed the genetic effects from radiation exposure including genetic effects to the general population from plant employee exposure."

Question 3: Have you read and are you familiar with the Final Environmental Statement (FES) and the Supplement to the Final Environmental Statement (FESS) for the Clinch River Breeder Reactor?

Answer 3: Yes.

Question 4: Do you agree with the genetic effects estimates of the Staff that are presented in the FESS?

Answer 4: I am in agreement with the Staff's genetic effects estimates. There are, however, several ways to make such estimates, and I have independently estimated the genetic effects using as a basis the dose estimates supplied in the FESS (Sect. 5.7) and the genetic effects estimates made by the National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation as given in its Report "The Effects on Populations of Exposure to How Levels of Ionizing Radiation: 1980" (the BEIR III Report).

Question 5: What are the genetic effects of radiation?

Answer 5: Such genetic effects include both gene mutations and chromosomal aberrations, and by definition will be expressed only in the offspring and the more remote descendants of the exposed population. Though the production of genetic effects by radiation has not been demonstrated in humans, it is extensively documented in experimental organisms, and must surely occur in humans as well. Since radiation-induced genetic effects have not been demonstrated directly in humans, however, the estimation of the number to be expected as a consequence of a particular exposure presents some uncertainties.

In all organisms studied experimentally, mutations arise spontaneously, without any deliberate exposure to radiation or other mutagenic agents. While some of these spontaneous occurrences may be due to the

natural background radiation to which we are all exposed, it is clear that the vast majority arise from other causes, the nature of which is not as yet known. A striking feature of radiation-induced mutations, both genetic and chromosomal, is that the types observed are exactly the same as the types which occur spontaneously. None are novel or unique. Thus radiation simply increases the frequency of events which are occurring already in the population.

It is generally agreed that the majority of mutations, whether spontaneously arising or induced, are to a greater or lesser extent deleterious. Some produce dramatic effects on the health of the individual, shorten lifespan or interfere with normal embryonic development to produce congenital defects. Most mutations, however, have relatively minor effects, and many produce no detectable effect at all upon the individual's health or well being. A few human mutations are known to have both deleterious and beneficial effects, depending on the circumstances, and it is possible that many mutations fall in this category. Thus while an increase in human mutation rate must be considered undesirable, it must also be noted that much of the effect on affected individuals will be relatively minor and frequently undetectable.

Question 6: What aspects of radiation dose are important for your estimates?

Answer 6: For the purpose of genetic hazard estimation, only doses received by the reproductive cells or their precursors need be considered. Furthermore, only the doses accumulated by these cells prior to conceiving a child are of concern. Obviously, exposures accumulated in other cells or tissues cannot produce effects which may be passed on to the

next generation, nor can those accumulated by persons who will not reproduce again result in inherited effects. The concept of "genetically significant dose" (GSD) is a convenient means of dealing with genetic hazards. Where detailed information on population structure and dose distribution is available, the GSD may be calculated by taking the sum of the gonadal doses weighted by the probability of future reproduction for each age group. Fortunately, since such detailed information is not available for future populations such as that of concern in connection with the Clinch River Breeder Reactor (CRBR), and acceptable "GSD" may be derived by estimating the whole body dose accumulations in man-rem for the population of interest and assuming that the population is a stable one, for which the average age at reproduction (i.e., at the birth of the middle child) is thirty years.

For the purpose of radiation protection and hazard estimation, doses are expressed in units of rem, or "roentgen equivalents, man." Radiations of different physical quality produce different levels of biological effect per physical dose unit (rad). The effectiveness of a particular radiation type in relation of a standard reference, usually either X or gamma rays, is termed its relative biological effectiveness, or RBE. Thus alpha particles, for example, have a higher RBE than less highly ionizing radiation. The RBE of a given radiation is allowed for in calculation of rem doses, so that doses from radiations of all types can be pooled, and no further allowance need be made for radiation quality for the purpose of hazard estimation.

Another property of some radiation types with high RBE values, such as alpha particles, is that they have a limited penetrating power, or range. Where the range in tissue is only a few micrometers, as for

example in the case of plutonium alpha particles, only the radiation actually arising in the gonad can produce any exposure of germ cells or their precursors. This is taken into account in the calculation of GSD from actual gonadal doses, but is not where the whole body dose is used as an estimate of gonadal dose. Because few radionuclides concentrate in the gonads (i.e., the gonad is rarely the critical organ), the use of whole body dose in genetic hazard estimation is most likely to lead to an overestimation of gonadal dose, and thus, of genetic effect. It should be noted that this is the case for the actinide elements, and especially so for the transuranic radionuclides such as ^{239}plutonium which will be present in the CRBR fuel.

Question 7: What is the relationship between radiation dose and genetic effects?

Answer 7: Radiation genetic hazard estimates are made on the basis of an assumption called the "linear hypothesis"; i.e., that there is a linear relation between dose and effect, and that it makes no difference, at least within the range of dose of interest, how the dose is distributed among the population. It is this assumption which makes it possible to estimate effects from population man-rem doses. Under the linear hypothesis the same genetic effect would result if a population of one million persons each received one millirem per year or if one thousand people in the population each received one rem per year while the rest received no dose; in either case the population dose is 1000 man-rem per year, and the effect is simply proportional to the population dose (obviously there are limits to the applicability of this idea, for a 1,000 rem whole body dose to one person in our population would kill him, and no genetic effect could possibly result).

The applicability of the linear hypothesis to genetic effects estimation for populations exposed to low-level chronic radiation is supported by both experimental evidence and radiobiological theory. The linear hypothesis is thus a conservative basis for hazard estimation. The data available on radiation-induced genetic effects is all for much higher doses and dose rates, and for these circumstances both radiobiological theory and experimental evidence strongly suggest that the dose-effect relationship for acute doses is greater than linear, that is, that there is an increasing increment in effect per increment of dose as the dose increases. Downward linear extrapolation from the lowest dose for which data are available to the spontaneous background level will inevitably in such a case lead to an overestimate of effect for all dose levels in between.

Question 8: Why have you chosen to use the BEIR III Report as a basis for your calculations?

Answer 8: Over the years a number of national and international groups of experts have attempted to estimate the genetic effects likely to result from increases in human population radiation exposure, of which the most recent is the National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation. I have adopted their 1980 Report and the so-called BEIR III estimates because I served on the Committee and am thus more familiar with it than with other reports. The estimates given in the BEIR III Report, though not made specifically for the purpose of evaluating the consequences of the operation of nuclear facilities, constitute a suitable, and in my opinion, the most appropriate basis for estimating the genetic effects likely to result from operation of the CRBRP. It must be emphasized, however, that any numerical estimates of

genetic hazards of radiation exposure at the very low dose rates anticipated are simply conservative estimates of the upper credible limits of risk. Such estimates cannot be considered reliable point estimates.

Question 9: If your estimates are upper limits, are they then conservative ones?

Answer 9: It is my opinion that the BEIR Report estimates of genetic effects are conservative ones, and likely to overestimate the actual effects. This opinion has several bases. First, as I have already stated, the linear hypothesis is likely to overestimate effects. Second, a paper has appeared since the BEIR III Report (Shull, Otake and Neel, Science 213 (1981) 1220-1227) that suggests that the sensitivity of humans of the induction of genetic effects by radiation may well be less than the BEIR III estimates. Because, as already mentioned, our attempts to detect genetic effects in irradiated human populations, notably among the offspring of survivors of the atomic bombings of Hiroshima and Nagasaki, have all failed to demonstrate statistically significant increases, genetic effects estimates such as those in the BEIR III Report rely largely upon data from extensive experiments with mice. From these data are derived a "doubling dose"; that dose which will produce as many extra mutations as occur naturally in the absence of any added radiation exposure. This doubling dose, or actually its reciprocal, the relative mutation risk per unit dose, is then used to estimate the genetically-related ill health to be expected in each generation. Shull, Otake and Neel have noted that though the results of a number of individual investigations to detect genetic effects at Hiroshima and Nagasaki have failed to reveal statistically significant increases, there are small numerical

excesses. Making the assumption that they are indeed real, the result of parental radiation exposure, these authors have calculated a doubling dose. This doubling dose is substantially higher than the lower end of the range of from 50 to 250 rem adopted by the BEIR III Report on the basis of the mouse data, suggesting that the BEIR III estimates are if anything on the high side. Nevertheless, I have adopted the BEIR III estimates as a basis for my calculations of the genetic effects, as an upper credible limit, to be anticipated in connection with operation of the CRBR.

Question 10: What are the BEIR III genetic effect estimates?

Answer 10: The BEIR III Report (page 85) estimates that exposure of a population to 1 rem per 30-year generation would result in an increase in total genetic effects in the first generation of between 5 and 75 cases of genetic effects of all kinds affecting health per million live births. As stated by the BEIR III Committee this represents an increase of between 0.005 and 0.07 percent over the 106,000 children with such effects expected among the one million children born to the same population if there were no added radiation exposure. Many of the genetic effects produced will not, however, be expressed in the first generation but will appear in later generations. The Report estimates that if the population continued to receive 1 rem per generation over enough generations for genetic equilibrium to be established, the number of additional genetic effects would ultimately level off at between 60 and 1,100 per generation, or between 0.06 and 1.0 percent of the current spontaneous incidence. Though the BEIR III Committee did not consider the case of a radiation exposure of a population for one single generation, the equilibrium estimate is actually numerically equal to the genetic effects

arising in all future generations over all times as a result of a 1 rem exposure for a single generation.

Question 11: How have you converted these estimates to specific estimates for CRBR?

Answer 11: The BEIR III estimates are for a population of unspecified size and makeup. All that is specified is that all members who reproduce receive an average accumulated dose of 1 rem during the assumed 30 year interval between their own conception and that of their own children. Obviously, the number of man rem to the whole population is undefined, since some of any population will already have had their children, and others though of reproductive age will not for one reason or another have children. Thus in order to make my estimates I have assumed that the hypothetical BEIR III population and the population living within a 50 mile radius of the CRBR have the same age, sex and reproductive characteristics. I have further assumed that the 50 mile population estimated to number 910,000 persons in the year 2010 (FESS, Sect. 5.7.2.8) approximately reproduces itself, and that there will be one million live births in each generation.

The BEIR III estimates are for a population exposed to 1 rem per 30 year generation, or 0.033 rem per year. Under the above assumptions this is 33,333 man rem per year to the population. The annual whole body non-occupational dose which will be received by the CRBR 50 mile population is less than 0.09 man rem per year; the occupational dose is estimated to be 1,000 man rem per year (FESS, Table A 5.5). Because most of those occupationally exposed may be expected to be part of the 50-mile population, the total dose is thus about 1000.1 man rem per year. The ratio of the estimated 50-mile population dose to the BEIR III dose is

1000.1/33,333, or 0.03. Since the BEIR population is the same, the genetic effects to be expected are simply that fraction of the BEIR estimates, or between 0.15 and 2.25 cases in the first generation and between 1.8 and 33 over all time (from the BEIR III equilibrium estimates), assuming that the CRBR is operated for the entire 30 year generation time. Since 106,000 cases occur in each generation spontaneously, the first generation increase in risk caused by operation of the CRBR amounts at most to about 0.00002 percent. The percentage increase in risk per generation in subsequent generations would, of course, be even less.

Although the occupationally exposed are expected to be part of the 50-mile population, and their dose is properly included in the above estimates, it is also true that the risk on the part of occupationally exposed parents is voluntary, so for the first generation, at least, it is of interest to know the genetic risk from non-occupational exposure. Here the ratio of doses is 0.09/33,333 = 0.000003, and the maximum credible first generation estimate is 0.0002 cases, or an increase over the current incidence of about 0.0000002 %.

Question 12: What about effects in the population residing further than 50 miles from CRBR?

Answer 12: Genetic effects to be anticipated in the entire United States population as a result of operation of the CRBR may be estimated. The estimated total dose to the 280 million population projected for the year 2010 is about 1,170 man-rem (FESS, Table A 5.5). The ratio to the BEIR population dose is 0.035 and the first generation estimates based on the BEIR estimates are between 0.18 and 2.6 additional genetic effects, or between 2.1 and 39 over all time. Assuming for simplicity that the 2010

U.S. population just reproduces itself (i.e., 280 X 10^6 live births per 30 years), some 29 million spontaneous genetic effects would occur in the population during the same period, so operation of the CRBR would result, in the worst case, in an increased rate of affected births of about 8.8 X 10^{-6}%. To put it another way, the number of affected births would rise from 986,666.7 to not more than 986,666.8 affected births per year.

Question 13: Is it possible to estimate individual, rather than population risk?

Answer 13: Yes, the risk of genetic effects to be expected as a result of operation of the CRBR can indeed be considered from the point of view of the individual, rather than the population. Since the current incidence of genetic effects is 106,000 per million live births, the individual risk for each child a couple might have is about 11%. As a worst possible case we may consider a couple who are conceived at the time the reactor begins to operate, are born and live continuously at the fence line, who obtain their food and water from the area, and who have a child at the end of the reactor's lifetime of 30 years. The maximum annual whole body dose to such a person is estimated to be less than 0.44 millirem per year (from Tables A 5.2 and A 5.3 of the FESS, assuming very conservatively that the infant doses from milk continue through life). In thirty years this would add up to 0.013 rem. The BEIR III Committee estimate of a maximum of 75 affected births in the first generation for a population receiving 1 rem per generation and having one million live births amounts to an added risk of 0.008% per birth per rem. For a dose of 0.013 rem, the risk becomes approximately 0.0001%. The risk for our hypothetical couple's child would, then, rise from the current incidence figure of 10.6% to 10.6001% as a result of CRBR operation.

Question 14: Have you considered the possible genetic effects of possible exposure to radiation from plutonium and other transuranic elements?

Answer 14: Yes. The estimates I have given actually include the effects attributable to radiation from plutonium and other transuranic elements simply because the whole body rem dose estimates used include the dose contribution from them. As I have already noted, the use of the rem unit includes an allowance for the high biological effectiveness of alpha particles such as those from plutonium. However, my use of whole body dose estimates (in lieu of gonadal dose estimates) must surely result in an overestimation of the genetic effects to be anticipated from plutonium, and possibly from the other transuranics as well. The plutonium in the CRBR fuel elements will be in an insoluble form. Most would enter the bodies of those exposed through the gut, and only a very small fraction would be absorbed. Very little of the plutonium entering the circulatory system would become located in the gonads. According to Richmond and Thomas (Health Phys. 29 (1975) 241-250), about 5×10^{-4} of the systemic burden will be taken up by the testis in males, and only about 1×10^{-4} by the ovaries in females. Furthermore, though studies of the genetic effects of plutonium in mice have only been undertaken recently, what results are available so far tend to confirm that the effects are no greater than would be predicted on the basis of the RBE for the plutonium alpha particle and the radionuclide's distribution and retention in the gonad (Grahn, et al., Radiation Res. 67 (1976) 587-588; Lunning, Frolen and Nielson, Mutation Res. 34 (1976) 539-542; Searle, et al., Mutation Res. 41 (1976) 297-301). Thus all of the available evidence indicates that the genetic effects of plutonium and other transuranics are adequately, and indeed quite conservatively, accounted for in the estimates I have presented.

Question 15: What is your final conclusion regarding the genetic effects likely to result from operation of the CRBR?

Answer 15: I have estimated that the genetic effects resulting from operation of the CRBR will, as an upper limit, be about 0.004 case among the one million births to the 50 mile population in the first generation from non-occupational exposure for 30 years and about 2.25 cases from occupational exposure for the 30 year plant lifetime. The Staff central estimate of about 0.3 case over all future generations from occupational and non-occupational exposure for one year when adjusted to a common basis (i.e., 30 years' exposure) results in 9 genetic effects, which is within the range of values I have calculated (i.e., 2.1 to 39 genetic effects over all time, as stated in my response to Question 12). Among the one million births over the same period 106,000 "spontaneous" cases are expected without the CRBR. Such an increase is not only very small, but would certainly not be detectable. Furthermore, the actual increase is, in my opinion, very likely to be smaller, possibly much smaller, than the upper limit estimates. I therefore conclude that the genetic effects from operation of the CRBR will be so small as to constitute a negligible impact upon human health and welfare.

MICHAEL A BENDER

PROFESSIONAL QUALIFICATIONS

I am presently Senior Scientist in the Medical Department of the Brookhaven National Laboratory, where I devote most of my time to research on the genetic effects of radiation and other mutagenic and carcinogenic agents, on the molecular mechanisms involved in the production of chromosomal aberrations in human and other vertebrate cells, and to the study of the molecular lesions involved in certain inherited human diseases which are characterized by sensitivity to radiation and a predisposition to develop cancer.

I hold a Bachelor of Science Degree in Zoology from the University of Washington, and the Ph.D. in Genetics from the Johns Hopkins University. I am a member of the American Society for Photobiology, The Radiation Research Society, the American Society for Cell Biology and the American Association for the Advancement of Science, and am a Counselor of the Environmental Mutagen Society. I was on the Editorial Board of CYTOGENETICS from 1962 to 1967 and Associate Editor of RADIATION RESEARCH from 1974 to 1977. I am presently on the Editorial Boards of MUTATION RESEARCH and RADIATION PROTECTION DOSIMETRY.

My professional experience totals approximately 25 years of research in radiation genetics and cytogenetics. I was a Senior Biologist and Group Leader in the Biology Division of the Oak Ridge National Laboratory for almost 12 years, carrying out research on the radiation sensitivity of human chromosomes and cells. In 1969 I joined the Faculty of the Vanderbilt University School of Medicine, where I continued my research and also did some teaching in Radiation Biology. In 1971 I accepted a two-year Professional Term Appointment as Geneticist with the U.S. Atomic Energy Commission, where I was responsible for evaluation of research programs in genetics. Following two years as Visiting Professor of Radiology at the Johns Hopkins University I moved to my present position at Brookhaven in 1975.

My experience includes work with the National Committee on Radiation Protection on the evaluation of the genetic hazards of radioactive isotopes, as well as membership on a number of National Academy of Sciences Committees concerned with radiation effects on human health, the most recent being the Committee on the Biological Effects of Ionizing Radiation (the BEIR III Committee) and the Panel on Reassessment of A-bomb dosimetry. I have published over 100 scientific papers, many dealing directly with the effects of radiation on humans and the evaluation of human radiation hazards.