UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

Before the Atomic Safety & Licensing Board

IN THE MATTER OF	:	Docket No. 50-272
PUBLIC SERVICE ELECTRIC & GAS COMPANY	:	(Proposed Issuance of Amendment to Facility Operating License No. DPR-70)
(Salem Nuclear Generating Station, Unit No. 1)	:	

RESPONSE TO THE ATOMIC SAFETY AND LICENSING BOARD ORDER DATED FEBRUARY 22, 1980

The Intervenor, Township of Lower Alloways Creek hereby submits the testimony of Dr. David B. Fankhauser in response to the Atomic Safety & Licensing Board Order dated February 22, 1980 which provides for submission of evidence to the following question:

> "In the event of a gross loss of water from the spent fuel storage pool at Salem 1, what would be the difference in consequences between those occasioned by the pool with the expanded storage proposed by the Licensee and those occasioned by the present pool.

Dr. David B. Fankhauser's qualifications are attached to his written testimony.

Respectfully submitted,

CARL J. VALORE, SPECIAL NUCLEAR
COUNSEL FOR THE INTERVENOR
TOWNSHIP OF LOWER ALLOWAYS CREEK

April 9, 1980

UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

Before the Atomic Safety and Licensing Board

In the matter of	:	
PUBLIC SERVICE ELECTRIC & GAS COMPANY (Salem Generating Station Unit #1)	:	Docket No. 50-272

CERTIFICATE OF SERVICE

I hereby certify that copies of Dr. David B. Fankhauser's testimony response to the Board Order dated February 22, 1980

in the above captioned matter have been served upon the attached list by deposit in the United States mail, at the Post Office in Northfield New Jersey with proper postage thereon, this 9th day of April, 1980.

CARL J. VALORE Special Nuclear
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Dated: April 9, 1980

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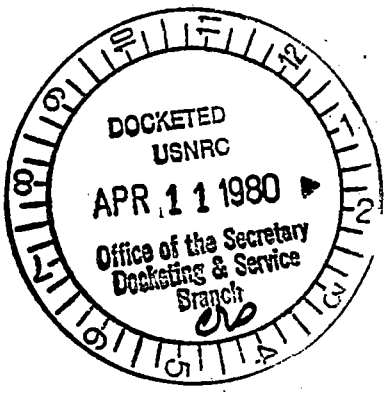
HEALTH EFFECTS OF A POSTULATED SPENT FUEL POOL FIRE AT THE SALEM NUCLEAR POWER STATION.



by

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March 31, 1980

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I. INTRODUCTION

Significant quantities of radioactive materials are projected to be released and dispersed as a result of a Zirconium fire in the Salem spent fuel pool following a gross loss of cooling water (Richard E. Webb; Review of Draft Testimony). This paper deals with the scope of somatic and genetic effects of the resulting radiation exposure. It is important to note at the outset that only three isotopes will be considered: Strontium-90 (SR-90), Iodine-131 (I-131), and Cesium-137 (Cs-137). These isotopes are particularly significant because they would be gaseous under accident conditions and are important bioligically. Actual exposure calculations will be performed only for Cs-137. Because the accident scenario proposed by Dr. Webb's testimony clearly carries the potential for violent explosion, additional analysis should be performed to assess the impact of particulate contamination as well.

II. ACCIDENT PARAMETERS

The capacity of the Salem spent fuel pool with assemblies re-racked amounts to approximately 1,100 fuel assemblies. The storage time for the assemblies ranges from freshly stored to thirty years old. Therefore, except for the fresh assemblies, most of the short-lived fission products will have decayed to relatively low levels. Table I lists the characteristics of the three isotopes considered in this paper. Sr-90 and Cs-137 will be present in large quantities in all assemblies, while I-131 will be found primarily in the recently added assemblies.

During the course of the accident in which cooling water is lost from the spent fuel pool, decay heat will build up. Once the temperature in the dry pool reaches 900 C, the Zirconium cladding will undergo a self-sustaining fire. The heat from combustion will vaporize the isotopes under consideration, and they will be easily dissipated through any breach in containment. Note that only Sr-90 has a boiling point above 1000 C.

III. BIOLOGICAL SIGNIFICANCE OF STRONTIUM, IODINE AND CESIUM.

Strontium is chemically similiar to calcium. It is concentrated at each step up the food chain (notably in milk) becoming incorporated into bone in man. (Cronkite, in Schwartz, p. 191). Once deposited there, radioactive decay exposes bone, and more importantly, bone marrow to irradiation. Bone marrow exhibits a high degree of sensitivity to radiation, being exceeded in sensitivity only by lumphoid cells and the gonads. (See Table 4).

Iodine is rapidly accumulated in the thyroid gland where it is stored and incorporated into thyroxine. This growth hormone usually carries three or four atoms of Iodine per molecule. 98% of the Iodine in the body is concentrated in the thyroid and the kidney retains 97% of the remaining 2% within the body. (Ganong, p. 250). Any absorbed radioactive isotopes of Iodine will deliver a dramatically concentrated dose to this gland. Due to bioaccumulation, seemingly small quantities of radiiodine in the environment are concentrated in milk and result in a high dose to humans. Milk winds up being laden with both I-131 and Sr-90 and exposes nursing infants and children in particular. This group is notably more sensitive to radiation than adults.

Cesium belongs to Group I of the periodic series. Like Potassium (of the same Group) it exhibits a strong affinity for muscle and nervous tissue. (Silver, p. 305). Although muscle is relatively resistant to radiation, the high energy gamma radiation (660 Kev) released during decay results in whole body exposure and notable gonad exposure. (Tubis & Wolf, p. 272).

The significance of these three fission products in terms of human exposure is underscored by experience gained as a result of inadvertant exposure of the Rongelapese natives in the Marshall Islands to fallout from U.S. weapons testing.

Eight years after exposure 15/19 children who were younger than ten when exposed developed thyroid nodules. Furthermore, the exposed population carried 24 times more Sr-90 and 300 times more Cs-137 than the average U.S. citizen. (Behrens, et al, pp. 308-339, and Rahn, pp. 192-193).

IV. RADIOLOGICAL SCOPE OF SALEM SPENT FUEL ACCIDENT

In order to gain some perspective on the magnitude of the releases described in the previous section we can compare the sume of the three isotopes currently under consideration with the total released by the bomb dropped on Hiroshima. The three isotopes released due to the postulated accident comprise a total of 10^8 curies. (See Table I). In contrast, the number of curies released at Hiroshima is estimated at 15×10^6 (Berger, p. 48). Therefore, the fraction of releases from Salem in Table I amount to more than 6.5 times the radioactivity of the Hiroshima bomb. In fact, the postulated Salem releases assume even greater proportions in view of the substnatially shorter half-lives of the Hiroshima releases. The latter decay much more rapidly than the half-lives of Sr-90 and Cs-137. The Salem releases would be much more persistant in the environment, allowing more time for bioaccumulation and incorporation into human tissue.

Furthermore, much of the Hiroshima activity was propelled by the blast into the stratosphere where it did not expose the local population (Berhens, et al, p. 309). On the other hand, The Salem accident would result in a much more concentrated plume of activity. The heat from the Zirconium fire would not cause the contamination to rise to great heights. Enough height would be ensured to permit wind dispersal, and local weather conditions

would have a major effect on who would be exposed, and to how much. Figure 1 clearly illustrates, based on the path of fallout along the East Coast, that Salem releases could easily strike the major population centers of the region, particularly Wilmington (15 mi.), Philadelphia (39 mi.), and New York City (122 mi.). The clear implication is that there is a probability for uncommonly high levels of total person-rems for the quantity of radioactivity released.

DOSES ATTENDANT TO SPENT FUEL FIRE.

Dr. Richard Webb's draft testimony indicates the following results from his dose calculations for various locations around the Salem Plant:

Next to Plant, after 30 days:	2,000 rem/hr
1 mile from Plant, rainfall precipated Cs-137-specific dose:	between 44,000 and 1.9 $x10^6$/30 yr
Dose to average Philadelphian: (due to Cs-137)	1500 rems/30 yr.

I have independently calculated the Cs-137 doses to three metropolitan areas: Wilmington, Philadelphia and New York City. The dispersal factor used for this calculation was empirically derived from the dispersal relationships observed at Three Mile Island (TMI). There, the dose delivered was found by curve-fitting to be roughly proportional to the inverse square of the distance from the source ($1/R^2$)(NUREG-0558, p. A-2). This dispersal or diminution factor was applied to the 30 year dose from Cs-137 at 1 mile from the Salem facility as derived by Webb. It should be noted that localized contamination may be expected to be more severe where heavy deposition occurs. The results of these calculations are summarized in Table 5.

VI. SPECTRUM OF DOSE RELATED HEALTH EFFECTS

VI. A. Acute Somatic Effects.

The effects of acute whole body exposure to greater than 100-200 rads are well established as outlined in Table 2 (Merck Manual, pp. 1729-1733). The median sickness dose is that dose which produces obvious signs of acute radiation poisoning in ½ of the exposed persons. It is placed at 175 rads (Behrens et al., p. 65). The dose at which ½ of the exposed persons die is termed the Lethal Dose$_{50}$ (LD_{50}) and is placed at 450 rads. Doses in excess of 600 rads are uniformly fatal. In those exposures

which are sub-lethal, there is a characteristic interim period after initial nausea and vomiting in which the individual is apparently well. However, the hematopoeitic effects become pronounced with massive hemorrhaging 3 to 6 weeks later leading to infection and anemia.

Delayed effects resulting from exposures of less than 450 rads are summarized in Table 3. Doses below 100 rads produce blood effects, but are not generally fatal (Bond et al., p. 156). Carcinogenesis, number 9 in Table 3, will be dealt with separately below in section VI.B.

The various tissues of the body exhibit a marked variability of sensitivity to radiation. Table 4 lists the tissues in order of decreasing sensitivity. It is a general rule that the most rapidly dividing cells show the greatest sensitivity. It is also important to note that the gonads are the second most sensitive tissue, a fact which bears directly on the estimation of genetic effects.

The fetus exhibits increased sensitivity as compared to the adult, presumably due at least in part to the high rate of proliferation of his cells. Microcephaly has been observed in about 25% of fetuses irradiated with several hundred rads (BEIR Report, pp. 74 & 75).

Exposure to radioiodine during childhood has been shown in the Rongelapese to lead to atrophy of the thyroid, with attendant features of hypothyroidism. The 9 million curies of I-131 would pose an extraordinarily increased risk to children and the unborn. An excessive number of hypothyroid infant

have recently been born in the three counties surrounding TMI. The probability of this occurring is, according to a recent article in Nature, $1/10^5$ years. It therefore seems likely that it is in some way associated with the TMI accident (Nature, 283:807, 28 Feb. 1980).)

VI.B. CARCINOGENESIS

Carcinogenesis is a delayed effect of radiation exposure which has received a great deal of attention (see Shapiro, pp. 260-264 for a tabulated summary.) Numerous uncertainties plague the precise determination of the dose-effect. Individual variability in sensitivity is pronounced, particularly with regard to age. Two notable examples of this phenomenon are (1) the maximum sensitivity of women to radiation as measured by the induction of breast cancer is 6.5x higher in adolescent women than the average for all ages (BEIR, p. D-1). (2) The maximum sensitivity of the very young as measured by leukemogenesis is 17x greater if the exposure occurs prenatally rather than postnatally (Upton, in Hiatt et al., pp. 477-500).

An additional factor which tends to lead to an underestimation of dose-effect for carcinogenesis is the latent period between exposure and the appearance of the malignancy. Five to 20 years is a common length, with greater

latent periods occasionally being observed (Bodmer & Cavalli-Sforza, pp. 176-177). John W. Gofman has pointed to the above factors and also mentioned short comings of the absolute risk estimations as presently derived and the low estimates of thyroid cancer mortality due to the protracted course of thyroid cancer. He suggests a figure of 7,200 cancers/10^6 person-rems. On the other hand, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) does not take into consideration many of the above factors and suggests a figure of 450 cancers/ 10^6 person-rems (UNSCEAR, section H. pp. 411-412). This latter figure was used recently by the NRC in assessing the health effects of the TMI accident (NUREG-0558, p. D-1). I have used the latter figure to estimate the lower bounds of the induced cancers, but have also included the projections based on the Gofman figure as probably being more realistic.

VI.C. GENETIC EFFECTS

There are also considerable difficulties in estimating genetic effects (Salthe, p. 233, Newcombe, 1978, and Neel, 1978). Neel specifically emphasizes that his estimates of the absolute minimum contribution of mutation to disease will "increase... rather dramatically in the next decade..." showing many previous estimates to have been conservative. He postulates that roughly 14.7% of conceptions will have serious disease potentialities maintained by background mutation pressure.

Man is diploid, receiving a set of genes from each parent. It is to be expected therefore that the majority of induced mutations will be masked. Only when the induced mutation is homozygous will it appear as a mutant. Current estimates are that only 2.5% of the recently induced mutations appear per generation (Casarett, p. 343). For this reason, a major impact on the gene pool would be required before it would be detected. The actual damage would be roughly forty times as great as the observed effects. It comes as no surprise therefore that pronounced genetic effects have not been observed in the single generation since the Hiroshima bombing. None-the-less, 100 rads delivered to the exposed population has led to a doubling in observed chromosomal abnormalities in their children. (Bodmer & Cavalli-Sforza, pp. 176-177). The amount of radiation to which our sex-cells are exposed has tripled in modern times, with as yet no clearly demonstrable effects (Singer, pp. 96-97).

Extrapolation from high dose experiments suggests that 1 rad to the parents/10^6 live births induces 8000 new mutations (Casarett, p. 343). At some time, all 8000 mutations will result in genetic death, the process taking many generations to completely "cleanse" the gene pool.

According to currently accepted models, the initiating step for both mutagenesis and carcinogenesis is the alter-

ation of DNA. Indeed, 90% of all known mutagens are carcinogens (Ames et al., and Mole in Duplan, p. 31-32). For this reason, in the absence of direct measurement of human mutagenesis, it is prudent to assume that doses to the gonads will be at least as efficient in mutagenesis as they are in carcinogenesis, and probably an order of magnitude more so since regulatory genes involved in controlling cell division obviously constitute a small fraction of the total genes in a cell.

With these reservations in mind, we will make an estimation of the minimum genetic effect from the postulated exposures. The number of live births per population center over the thirty year period can be estimated by multiplying the population times the crude birth rate for the U.S., 17.5/1,000/year (Mausner and Bahn, p. 135), times 30 years. (We assume for simplicity's sake, that the population will not change in size over the 30 year period.) The calculated number of live births is shown in table 6.

Other effects which cannot at present be quantified include the induction of mutant strains of pathogenic bacteria and viruses. The havoc created by new virulent strains of influenza virus is an example of this problem. Such variants are particularly troublesome since there exists no herd immunity, and therefore transmission occurs with great efficiency. New strains of agricultural diseases will also be anticipated as a result of increased environmental mutagens such as radiation.

VII. PROJECTED POPULATION DOSE-EFFECTS

We have not had the opportunity to examine comprehensive demographic and meteorologic data which would allow more specificity in making dose calculations. It is hoped that the board will permit additional submission of testimony on this subject. However, the scope of the impact can be appreciated by the use of the data assembled in this paper.

A person standing next to the facility would receive an invariably fatal dose within 20 minutes. Persons attempting to perform emergency procedures would do so at severe risk even in such a short exposure as 5 to 10 minutes. Likewise, persons within 1 mile of the facility for 24 hours after the accident would be expected to develop the full spectrum of acute radiation sickness outlined in Table 2.

Effects on the major urban centers would vary according to dispersal parameters, and populations should most certainly be evacuated before doses shown in Tables 5 and 6 were received. However, in the event that such relocations were not undertaken, the health of the exposed populations would be severely impacted. The lower bounds, as marked by low exposure, and using the UNSCEAR risk factor, would produce a total of more than 10,000 cancers in the three

metropolitan areas considered, this from Cs-137 alone. If Gofman's risk estimate is more accurate, for which he makes a persuasive argument, the effects would be catastrophic, with a total of 370 million cancer-dose-equivalents marking the upper bounds of these calculations.

The genetic effects are also projected to be substantial. As can be seen on Table 6, the number of genetic deaths is projected for the first generation to be between 24,000 and 10 million in the three urban areas. It must be remembered also that only about 2.5% of the total genetic deaths will occur in the first generation, and that ultimately, as many as 430 million deaths may be caused over many generations.

VIII. CONCLUSIONS

From these preliminary calculations, it is obvious that the postulated spent fuel accident at Salem would have severe, wide-ranging and long-lasting effects. Major relocations of millions of persons would be advisable due to the projected health effects accruing to unrelocated populations. Evacuations of such scope would present difficulties bordering on the insurmountable.

Finally, it must again be emphasized that these calculations could well represent a minimum effect of

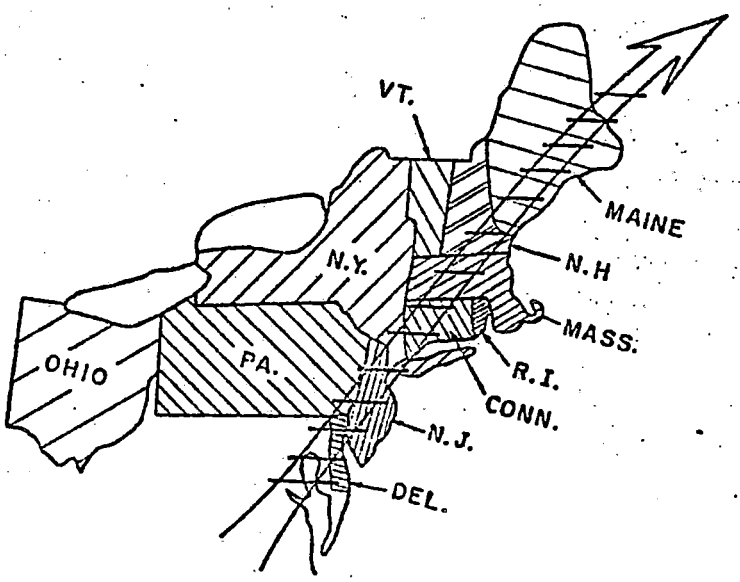
the postulated accident. Only Cs-137 related exposure has been calculated. As can be seen from Table 1, nearly as many curies of Sr-90 are postulated to be released, and duration of exposure would be at least as severe since Sr-90 is incorporated into bone. The I-131 released would not be a major factor over the 30 years, but would be particularly troublesome in the initial period after the accident, when large numbers of persons would presumably be in transit, and exposure would be difficult to monitor and control.

No consideration has been given to the impact on agricultural activities, but it also would be of major proportions. Sr-90 and I-131 are particularly subject to bioaccumulation.

An additional means of exposure which should be quantified is the aquatic pathway. It is anticipated that inventories of radioactivity such as those involved in this postulated accident would soon find their way into marine foodstuffs, possibly being transported along the coast in this manner.

FIGURE 1

PATH OF CHINESE WEAPONS TEST FALLOUT, OCTOBER, 1976



Note that radioactive materials released from the Salem facility, if dispersed in a similar fashion, would expose the most heavily populated regions of the North Atlantic Coast. (From Honecker, 27.)

TABLE I

CHARACTERISTICS OF Sr-90, I-131 and Cs-137 released due to postulated accident.

	Sr-90	I-131	Cs-137
Half-life:	28.1 years	8.05 days	30 years
boiling point 'C:	1,366	183 (sublimes below 114)	690
Tissues affected:	bone	thyroid	muscle, nerve.
Quantities released according to Webb scenario;	40 x 10^6 Ci	9 x 10^6 Ci	50 x 10^6 Ci

TABLE 2

SYMPTOMS AND SIGNS OF ACUTE WHOLE-BODY RADIATION EXPOSURE.*

A. Cerebral syndrome, follows exposures greater than 3000 rads, is invariably fatal. Three phases are recognized:

1. Prodromal nausea and vomiting.
2. Listless and drowsy.
3. Tremors, convulsions, ataxia, death in a few hours.

B. Gastrointestinal syndrome, follows exposures of 400 or more rads:

1. Intractable nausea, vomiting, diarrhea, vascular collapse, death.
2. Toxemia due to necrosis, atrophy of GI mucosa.
3. Hematopoeitic failure within 2 to 3 weeks.

C. Hematopoeitic syndrome (200 to 1000 rads).

1. Anorexia, apathy, nausea and vomiting within 6 to 12 hours.
2. The following 24 to 36 hours are asymptomatic, but well-being declines and lymph nodes, spleen and bone marrow begin to atrophy.
3. Thrombocytopenia becomes prominent is 3 to 4 weeks, leading to massive hemorrhage.

D. Increased susceptibility to infection:

1. Decreased production of leukocytes.
2. Impaired antibody production.
3. Reduced resistance to diffusion.
4. Hemorrhage in skin and bowel yeild to bacteria.

E. Exposures greater than 600 rads are fatal due to B. and C, with the probability of surviving lower exposures being inversely related to dose.

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* From the Merck Manual, pp. 1730-1731.

TABLE 3

DELAYED EFFECTS OF DOSES LESS THAN 450 RADS*

1. Amenorrhea
2. Decreased fertility
3. Decreased female libido
4. Anemia
5. Leukopenia
6. Thrombocytopenia
7. Cataracts
8. Loss of hair
9. Carcinogenesis
10. Et cetera

* From Merck Manual, p. 1731.

TABLE 4

HEIRARCHY OF TISSUES ACCORDING TO RADIATION SENSITIVITY*

Most Sensitive:	1.	Lymphoid cells
	2.	Gonads (testes and ovaries)
	3.	Proliferating cells of bone marrow
	4.	Epithelial cells of bowel
	5.	Epidermis
	6.	Hepatic cells
	7.	Lung and biliary epithelium
	8.	Kidney epithelial cells
	9.	Pleural and peritoneal endothelium
	10.	Nerve cells
	11.	Bone cells
Least Sensitive:	12.	Muscle and connective tissue

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* From Merck Manual, p. 1730.

TABLE 5

DOSE AND CARCINOGENIC EFFECTS OF Cs-137 RELEASED FROM SALEM SPENT FUEL ACCIDENT IN SELECTED METROPOLITAN AREAS.

Metropolitan Area:	Wilmington	Philadelphia	New York City
Population (Rand McNally, 1966):	318,700	4,200,000	15,400,000
Distance in miles from Salem Plant:	15	39	122
Dose dinimution factor relative to the dose at 1 mile ($1/R^2$):	1/225	1/1520	1/14,900
Estimated 30 year dose/person due to Cs-137 in rems:	195 to 8,440	30 to 1,300	3 to 130
Total person-rems exposure in 30 yrs:	6.2×10^7 to 2.7×10^9	1.3×10^8 to 5.6×10^9	4.6×10^7 to 2.0×10^9
Total cancers induced in 30 years:			
UNSCEAR risk factor of $450/10^6$ person-rems:	2.8×10^3 to 1.2×10^6	5.8×10^3 to 2.5×10^6	2.1×10^3 to 0.9×10^6
Gofman risk factor of $7,200/10^6$ person-rems:	4.5×10^5 to 1.9×10^8	9.4×10^5 to 4.1×10^7	3.3×10^5 to 1.4×10^8

TABLE 6

GENETIC EFFECTS OF Cs-137 RELEASED FROM SALEM SPENT FUEL ACCIDENT ON SELECTED METROPOLITAN AREAS.

	Metropolitan Area:	Wilmington	Philadelphia	New York City
2	Predicted live-births over 30 years:	1.7×10^5	2.2×10^6	8.1×10^6
3	Dose to each parent: in rems (from Table 6)	195 to 8,440	30 to 1,300	3 to 130
4	Total induced mutations in 30 yrs: (rems x live births x 10^{-6} x 8,000)	2.7×10^5 to 1.2×10^8	5.3×10^5 to 2.3×10^8	1.9×10^5 to 8.2×10^7
5	Deaths in first generation due to induced mutations: (2.5% of line 4.)	6.7×10^3 to 3.0×10^6	1.3×10^4 to 5.8×10^6	4.8×10^3 to 1.2×10^6

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QUALIFICATIONS OF DAVID B. FANKHAUSER, Ph.D.

David B. Fankhauser was born November 22, 1941 and graduated from Olney Friends School, Barnesville, Ohio, 1959.

In 1963, he graduated from Earlham College, Richmond, Indiana with a B.A. in Chemistry.

From 1963 to 1965 he worked as a medical research technician at the University of Cincinnati Medical School under the direction of Dr. Michael Carsiotis.

In 1965 he entered graduated school at John Hopkins University, Baltimore, Md., in the Department of Biology.

During the summer of 1967, he participated in the Bacterial Viruses course at the Cold Spring Harbor Laboratory for Quantitative Biology on Long Island, N.Y.

In the summer of 1969, he conducted research in mutagenesis in the laboratory of Dr. Bruce Ames, Dept. of Biochemistry, University of California, Berkeley. (Dr. Ames has received global recognition for the development of a bacterial test which detects mutagens/carcinogens with extreme sensitivity.)

During the school years of 1968-1970, he taught laboratory courses at John Hopkins in first year Biology and Genetcis.

In 1971, he received his Ph.D. from Johns Hopkins. His thesis, "The Promotor-Operator Region of the His Operon in Salmonella Typhimurium" was researched and written under the advisorship of Dr. Philip E. Hartman. It concerns the effects and locations of mutations which alter the regulation of a set of genes responsible for the biosynthesis of the amino acide histidine.

From 1973 to the present he has taught Biology at Clermont College, University of Cincinnati, Batavia, Ohio. He has developed a laboratory program for first year Biology students entitled "Appropriate Biology" which incorporates many of the cottage craft skills gained in his lifestyle research as well as lab techniques which assay the quality of the student's environment. Student response has been highly favorable.

From 1972 he has been involved in the Atomic Energy Commission's and now the Nuclear Regulatory Commission's hearings regarding the Zimmer Nuclear Power Plant, Moscow, Ohio. He has been granted intervenor status in those proceedings. Issues raised in 1972, such as waste disposal, epidemiology of low level exposure, evacuation and monitoring have recently become subjects of national interest. He maintains an active speaking schedule on these subjects, appearing at 10 to 20 engagements a year.

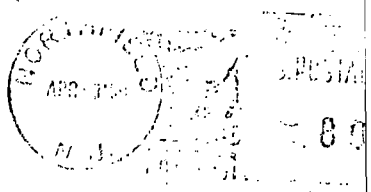
Dr. Fankhauser also has been invited several times by members of the Ohio Legislature to testify on health related aspects of nuclear power.

In 1979, he served on review committees for the United States Environmental Protection Agency. The purpose of these consultations was to set limits on environmental concentrations of toxic susbstances to preclude adverse health effects.

In 1980 he accepted a position at Northern Kentucky University, Highland Heights, Ky., as lecturer in Epidemiology.

He has published papers in the following journals and publications:

Genetics
Journal of Bacteriology
Neurospora Newsletter
Health Forum
The Earlhamite



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