THE BIOLOGICAL EFFECTS OF RADIATION: TEN TIMES WORSE THAN ESTIMATED

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ABSTRACT

The latest biological evidence indicates that the estimates of the biological effects of radiation made only five years ago, both for cancer induction and genetic effects, are too low by at least a factor of ten. Among the most critical data is that collected on the workers at the U.S. Hanford facility. This data, to the great surprise of the experts, demonstrates an important and significant increase in the cancer deaths for the workers. All the evidence indicates that workers' exposures should be reduced tenfold. Because of this new information, the nuclear industry should anticipate a number of lawsuits brought by workers who develop cancer.

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1. Introductory Remarks

Radioactivity is a property possessed by some isotopes of the various chemical elements. Tritium, for example, is the radioactive isotope of the element hydrogen. All isotopes of plutonium are radioactive. This property of radioactivity causes the nucleus of the isotope to undergo spontaneous disintegration. In this process, energy is liberated and the disintegration generally results in the formation of a new nuclide (isotope of a different element). The energy is released in the form of radiation, either as electromagnetic waves (X-rays or gamma rays) or as particle radiation (beta or alpha particles). This radiation is called ionizing radiation because as it passes through matter, it is capable of ejecting electrons from atoms and thereby producing ions.

As the radiation is passing through human tissue and producing ions, it is actually transferring its energy to the cells in the tissue. The amount of energy transferred in one of these ionizing reactions is sufficient to bring about chemical changes in the molecules of the cells and thereby produce an abnormal or damaged cell. Such radiation injury to the cells can and does result in the induction of cancer in irradiated tissue. If these chemical changes take place in the germ cells of the gonads, they could result in altered genes or chromosomes which could then be

passed on to and produce genetic defects in future generations. If the radiation dosage is large, so many cells will be damaged that the radiation can be lethal.

The concern over the release of radioactivity to the environment stems from this knowledge that radiation is harmful to man. At high dosages such as could occur following a major accident, the radiation can be lethal. At lower dosages, radiation can induce cancer in the irradiated individuals and induce damage in their genetic material that could be transferred to their offspring and to future generations. Moreover, the range of concern over low dosage effects extends to those encountered in the anticipated day-to-day occupational exposure of employees and releases of radioactivity from the facilities of the nuclear power industry that result in exposure of the public-at-large and the medical uses of X-rays and radiation.

There is no safe level of radiation exposure. The United States National Academy of Sciences, National Research Council, Committee on the Biological Effects of Ionizing Radiation, had this to say about the concept of a safe or threshold dosage of radiation:[1/]

> There is no sufficient theory of radiation carcinogenisis from which the concept can be deduced, and an empirical demonstration has not been made.

Although it did not state it in such explicit terminology, the NAS BIER Committee came to the same conclusions with respect to the genetic effects of radiation.[2/] The International Commission

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1/ NAS BIER Report, Report of the National Academy of Sciences, National Research Council, Committee on the Biological Effects of Ionizing Radiation, "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," Washington, D.C., November, 1972, p. 95.

2/ Ibid, pp. 64-65.

on Radiological Protection has stated:[3]

> It seems unlikely that the dose-response for any kind of genetic effect has any sort of threshold; the underlying mechanism for genetic change is molecular.

Thus, both experimental data and theoretical considerations indicate that any amount of radiation, no matter how small, must be considered as being harmful to man. Consequently, we must be concerned about the smallest radiation exposures and releases of radioactivity from the nuclear power industry.

The BIER Report of the NAS Committee presents the most recent estimates of biological effects of radiation. These estimates are based upon the linear hypothesis. This hypothesis assumes that in the low dose range (dosages below which direct experimental evidence is available), the effects are directly proportional to the dose. This implies that a dose of 1 rem to 1 million people (1,000,000 man-rem) will result in the same quantitative effect such as the number of induced cancers as would a dose of 10 rem given to 100,000 people (this is also 10 X 100,000 or 1,000,000 man-rem). The BIER Report estimates that 1,000,000 man-rem will produce the following effects:

Effects of 1,000,000 Man-Rem	
Induced cancers[4]	100-450
Genetic defects[5]	30-750

3/ ICRP Publication 14, Radiosensitivity and Spatial Distribution of Dose, Reports prepared by Two Task Forces of Committee 1 of the International Commission on Radiological Protection, Pergamon Press, Oxford, 1969, p. 20.

4/ NAS Bier Report, op. cit., p. 90.

5/ Ibid, p. 55.

These estimates apply to a population composed of young and old and male and female. Since only about one-half of the population would be within the reproductive age, the genetic effect estimate given above has been adjusted accordingly. There are several reasons why much more weight should be given to the higher estimates given above. In fact, recent data suggest that even the higher estimates may significantly underestimate the true effects.

2. Cancer Induction

The BEIR Report estimates that one million person-rem will induce 100 to 450 cancers.[6/] The lower estimate is based upon the absolute risk model while the higher is based upon the relative risk model. Both are based upon the linear hypothesis. In the following sections, evidence is presented to demonstrate that even the upper estimate of the BEIR Report is too low, possibly by at least a factor of 10.

2.1 Absolute vs. Relative Risk Model

The BEIR Committee stated:

> No conclusion can be made at this time on the absolute versus relative risk model. 7/

The relative risk model is based upon radiobiological data which indicate that the rate of induction of various cancers per rem is proportional to the spontaneous incidence rate. At younger ages when the spontaneous cancer death per year is low, the rate

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6/ Ibid, p. 90.

7/ Ibid, p. 188.

of induction per year is also low, but at older ages when the spontaneous cancer death rate is 10 times higher, so is the rate of radiation-induced cancers. The absolute risk model tends to average these changing rates into a single value.

If the data base were complete, both models should give the same overall risk estimate. The absolute risk model would, however, obscure the varying radiosensitivity as a function of age. The problem is that the data base is not complete. The follow-up study on irradiated populations extends over a period of some 30 years -- much less than the lifetime of the younger members. The relative risk model represents a biologically reasonable basis for extrapolating the data base throughout the lifespan of the younger members of the study populations. The difference between the two models at this time is a result of this extrapolation for the radiation exposure experience in the early years of life.

Radiological data demonstrate that the young child (0-9 years) and the developing fetus are more sensitive to radiation than adults (>10 years). The higher risk estimate of the BIER Report assumes that the risk associated with radiation exposure of the young child extends throughout the lifespan. On the other hand, the BIER Report assumes that the risk associated with _in utero_ exposure extends only throughout the first 10 years of life. If the risk from _in utero_ exposure were assumed to extend throughout the lifespan, the risk estimate would be doubled and the risk of cancer would become 1,000 cancers per million person-rem. 8/

8/ _Ibid_, p. 188.

There is considerable biological evidence which demonstrates that injury (physiological damage) received during in utero development and in childhood is reflected in decreased physiological competence and increased mortality during adult life.[9/] Thus there is a significant biological basis for assuming that the radiation injury incurred during in utero development and childhood will lead to an enhanced risk of cancer throughout the lifespan. Those who were exposed in utero and 0-9 years by the atom bombs in Japan are now just reaching the age where the important data on this question can be collected. The next few decades will reveal the pattern of excess cancer in adult life. Nevertheless, the present data on those irridiated in utero does conform with the biological expectation that this radiation injury will become manifest in adult life. Hiroo Kato has reported that there is an increased mortality of individuals exposed in utero by the atom bombs after they reached 10 years of age.

2.2 The Linear Hypothesis

The risk estimators in the BEIR Report are based upon the linear hypothesis. This hypothesis assumes that in the low dose range (that below where experimental data is available) the risk is directly proportional to the dose. The BEIR Committee, however,

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9/ See, for example, Tamplin, A.R., et al., "A Criticism of the Stronglass Article on Fetal and Infant Mortality," Lawrence Livermore Laboratory, Livermore, California, UCID-15506, 22 July 1969; and Jones, H.B., "Factors in Longevity," Kaiser Foundation Medical Bulletin, Vol. 4, Nos. 9-10, September-October, 1956.

indicated that the linear hypothesis may either overestimate or underestimate the risk when applied in low dose/low dose rate situations.[10/] Recent evidence indicates that the linear hypothesis may underestimate the effect of low dose/low rate irradiation. Much of this latest information has been summarized by Dr. Karl Z. Morgan, who concluded:

> Frequently in the literature it is stated that the linear hypothesis is a very conservative assumption. During the past years, however, many studies have indicated that this probably is not true in general and that at low doses and dose rates somatic damage per rad (and especially that from α-irradiation) probably is usually greater than would be assumed on the linear hypothesis. 11/

Dr. J. Martin Brown also reviewed the pertinent data on cancer induction and concluded:

> Contrary to popular belief there are quite good data on cancer induction in humans by low doses and/or low dose rates of low LET radiation. Risk estimates at these low doses were found not to be less than comparable risk estimates made from high doses. If anything, in fact, the data suggest the opposite. 12/

Dr. Irwin D. J. Bross has shown that there are some individuals in the population who are affected by radiation orders of magnitude greater than other individuals.[13/] Thus, in interpreting the

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10/ NAS BEIR Report, op. cit., p. 90.

11/ Morgan, Karl Z., Suggested Reduction of Permissible Exposure to Plutonium and Other Transuranium Elements, Journal of American Industrial Hygiene, August, 1975.

12/ Brown, J. Martin, Linearity vs. Non-Linearity of Dose Response for Radiation Carginogenesis, Health Physics 31, September, 1976, p. 231.

13/ Bross, Irwin D.J., Leukemia from Low-Level Radiation, New England J. of Med., Vol. 287, No. 3, 20 July 1972, pp. 107-110.

effects of radiation one must be cognizant of these highly susceptible individuals. Dr. John W. Baum has shown this population heterogeneity with respect to radiation sensitivity causes the linear hypothesis to underestimate the risk at low doses.[14/] He concludes:

> It is concluded from this analyst that linear extrapolation from high doses to predict effects at low doses is not always conservative. This seems particularly so for radiation induced cancer in a heterogeneous population such as man. The need for much more experimental and epidemicological data at low doses and for low dose-rates is apparent. [15/]

2.3 The Mancuso Report

On October 13, 1976, Drs. Thomas Mancuso, Alice Stewart and Mr. George Kneale presented a paper (hereinafter "Mancuso Report") at the Health Physics Society Symposium in Saratoga Springs, New York.[16/] This report presents an analysis of a 29-year follow-up of the workers at the Hanford installation. It relates their cause of death to their occupational radiation exposure. It is, therefore, a study of the effects of low level radiation. The significant finding of this report is that for a total exposure of 2756 person-rem[17/] there

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14/ Baum, J.W., Population Heterogeneity Hypothesis on Radiation Induced Cancer, Health Physics 25, August 1973, pp. 97-104.

15/ Ibid, p. 103.

16/ Mancuso Report, Mancuso, T.F., A. Stewart and G. Kneale, Radiation Exposure of Hanford Workers Dying From Various Causes, presented at the Tenth Midyear Symposium of the Health Physics Society, Saratoga Springs, New York, 13 October 1976, Revised Draft, March 1977.

17/ Ibid, Table 2.

were 29 (27 to 31) radiogenic cancer cases.[18] It indicates that the linear hypothesis underestimates the risk for low dose/low dose rate irradiation by a significant factor.

A criticism of the Mancuso Report was presented at the same symposium by Marks and Gilbert.[19] It is significant to note that in their criticism Marks and Gilbert failed to mention an early report by Gilbert and Buschbom that came to conclusions similar to the Mancuso Report.[20] This report confirmed the findings of an earlier report by Dr. Samuel Milham, Jr., who concluded that the occupation exposure at Hanford resulted in a higher incidence of cancer. Gilbert and Buschbom concluded:

> We have established that there is a relationship between cancer as a cause of death and the total external dose received. [21]
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> * * *
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> For the time being radiation must be placed high on the list of suspects in considering possible explanations for the observed relationship. [22]

18/ Ibid, p. 24.

19/ Marks, S., and E. Gilbert, comments on the paper by Mancuso, Stewart and Kneale presented at the Tenth Midyear Symposium of the Health Physics Society for presentation of the Mancuso Report.

20/ Gilbert, E.S., and R.L. Buschbom, An Evaluation of Milham's Analysis of Hanford Deaths, Draft, Battelle Northwest, July 1975.

21/ Ibid, p. 28.

22/ Ibid, p. 29.

In truth, the Mancuso Report only confirms these earlier studies. Gilbert had published a subsequent paper which was also not mentioned in the Marks and Gilbert criticism.[23/] In this paper, a different statistical approach still confirms the Milham (and hence Mancuso) findings. In this paper Gilbert concludes:

> Although the use of Milham cutpoints does suggest a slight dose-cancer relationship, it does not approach statistical significance and is not nearly as strong a relationship as found with the Milham data. [24/]

The analysis upon which these conclusions are based is summarized in Table A-1 of the report. This table is reproduced on the following page of this submission. Although no one of these 20 figures in this table attains statistical significance, a simple Spearman rank-correlation test (of the SMR and the % dying of cancer in the exposure groups) yields a correlation of 1.0 in all five groups which is significant at the 5% level.

One of the criticisms of the Mancuso Report was that it included all workers and not just longer term employees. The Gilbert paper considered, as a special group, only craftsmen and operators with 5 or more years at Hanford and found that the cancer excess was significant at the 0.01 level.[25/] The craftsmen and operators had the highest exposures.[26/]

Clearly, these studies of the Hanford workers show a relationship between low dose occupational exposure and cancer. They

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23/ Gilbert, E.S., Proportional Mortality Analysis of Hanford Deaths, Draft Progress Report, Battelle Northwest, July 1976.

24/ Ibid, p. 16.

25/ Ibid, p. 5.

26/ Ibid, pp. 2-3.

TABLE A-1

The Relationship of Cancer as a Cause of Death
and the Total Exposure Groups Used in the Milham Study

Dose in Rems	0.00-	1.00-	2.24-	6.60-	TOTAL
A) All Deaths					
SMR	1.06	1.08	1.19	1.29	
Number in Group	1835	318	185	94	2472
B) Ages 45-74, Year of Death 1960-1971					
Percent Dying from Cancer	21.1	21.4	23.5	26.6	21.8
Number in Group	959	234	119	77	1369
C) Milham Study Deaths Ages 45-74, Year of Death 1950-1973					
Percent Dying from Cancer	20.0	23.6	22.1	39.6	23.7
Number in Group	200	161	95	71	527
D) Milham Study Deaths Ages 45-74, Year of Death 1950-1971					
Percent Dying from Cancer	19.5	25.6	22.7	40.0	24.3
Number in Group	169	121	75	50	415
E) All Local Deaths Ages 45-74, Year of Death 1960-1971					
Percent Dying from Cancer	22.4	23.8	25.9	38.9	24.8
Number in Group	294	147	85	54	580

suggest a relationship that is 15 times larger than the upper estimate of the BIER Report (6500 cancers per million person rem rather than 450).

3. Genetic Effects

The latest biological evidence also indicates that the BIER Committee estimate of the genetic effects of radiation was too low. This should not come as a surprise because even the BIER Committee cautioned that its estimate of genetic effects may be too low:

> A major concern of the Subcommittee is the possible existence of a class of radiation-induced genetic damage that has been left out of the estimates. By relying so heavily on experimental data in the mouse we may have overlooked important effects that are not readily detected in mice, or the mouse may not be a proper laboratory model for the study of man. 27/

As if to reemphasize this, the Committee concluded this section by stating:

> We remind all who may use our estimates as a basis for policy decisions that these estimates are an attempt to take into account only known tangible effects of radiation, and that there may well be intangible effects in addition whose cumulative impact may be appreciable, although not novel. 28/

Present data indicate that its estimates were too low for two reasons. In the experiments of Dr. William L. Russell at the Oak Ridge National Laboratory, it was observed that the induced mutation

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27/ NAS BIER Report, op cit, p. 57.

28/ Ibid.

frequency at low dose rates was about 1/3 that observed at high dose rates. The factor of 1/3 was used by the BIER Committee. However, Dr. Mary F. Lyon, et al., have analyzed the Russell data along with additional data from experiments at low dose rates.[29] Their analysis shows that as the dose rate drops below some 0.01 r./min., the induced mutation frequency begins to increase. They conclude:

> In future estimates of the genetic hazards of environmental radiation, therefore, it would be prudent to increase this last figure to a value above that seen in mice at 0.01 r./min., for which the maximum likelihood estimate given by the data considered here is 10×10^{-8}.[30]

The value adopted in the BIER Report was 2.5×10^{-8} or a factor of 4 lower. This would lead to a factor of 4 underestimate in the genetic effects. These conclusions by Lyons, et al., are confirmed by the analysis of Abrahamson and Wolff, who also conclude that the value should be 4 times higher.[31]

Most, if not all, human disease has a genetic component. This includes such disease as heart disease, epilepsy, schizophrenia, asthma, diabetes and other degenerative diseases. These diseases are inherited in a complex fashion, i.e., they are the result of a single gene mutation. They are called multifactorial diseases.

29/ Lyon, Mary F., D.G. Papworth and Rita J.S. Phillips, "Dose-rate and Mutation Frequency after Irradiation of Mouse Spermatogonia," Nature New Biology, Vol. 238, July 26, 1972, pp. 101-104.

30/ Ibid, p. 104.

31/ Abrahamson, S.E. and S. Wolff, "Reanalysis of Radiation-Induced Specific Locus Mutations in the Mouse," Nature, 264, p. 715, 1976.

Recent studies have shown that these diseases are twice as frequent as that estimated in the BIER Report.[32/] These diseases also represent the bulk of the genetically-determined diseases. When this is combined with the factor of 4 above, it indicates that the BIER Report underestimated the genetic effects by a factor of 8.

4. Radiation Protection Standards

The radiation protection standards in use throughout the world closely follow the recommendations of the International Commission on Radiological Protection (ICRP). These protection standards are not intended to represent safe levels. Rather, they are intended to be levels where the associated risk is acceptably low.

It is important to note that the history of these standards is that as new biological evidence has accumulated, the standards have been made more restrictive. For example, in 1934 the ICRP whole body occupational exposure standard was 0.2 rem/day. This would correspond to some 50/rem yr. The present standard is 10 times lower, 5 rem/yr. The latest biological evidence cited in the above section indicates that, in order to keep the risk acceptably low, this standard should be reduced by at least another factor of ten to 0.5 rem/yr.

4.1 Plutonium Body Burden Standard

Dr. Karl Z. Morgan has proposed that the present approach

32/ Trimble, B.K. and J.H. Doughty, "The Amount of Hereditary Disease in Human Populations," Ann. Human Genetics, 38, 1974.

used to calculate the Pu-239 bone dose underestimates the dose in rem by a factor of 60 to 240.[33/] In effect, he suggests that the quality factor used is too low by this amount. The U.S. Nuclear Regulatory Commission Staff, accepting some and rejecting other of Morgan's arguments, acknowledges that present approach underestimates the bone dose by a factor of 10.[34/]

As a consequence, the present maximum permissible body burden for plutonium is at least 10 times too high. An individual who has 0.5 the present permitted burden is actually being exposed to at least 5 times the appropriate burden. In other words, this risk associated with the present permissible burden is at least 10 times larger than was estimated when the present standard was established.

4.2 Worker Lawsuits

Because this latest data demonstrates that the biological effects of radiation are at least 10 times worse than previous estimates, the nuclear power industry has a number of lawsuits from employees who develop cancer. The first of these will occur in England in November. I anticipate that the data from the Hanford workers will cause the lawsuits to be decided in favor of the workers.

33/ Morgan, Karl Z., Suggested Reduction of Permissible Exposure to Plutonium and Other Transuranium Elements, American Industrial Hygiene Journal, August 1975, pp. 567-575.

34/ U.S. Nuclear Regulatory Commission, hearing In the Matter of Generic Environmental Statement on Mixed Oxide Fuel (GESMO), Docket No. RM-50-5, Transcript, p. 3217, January 26, 1977.