GT-115-70

16,000 Cancer Deaths from FRC Guideline Radiation (Gofman-Tamplin)

vs

160 Cancer Deaths from FRC Guideline Radiation (Dr. John Storer)

A REFUTATION OF THE STORER ANALYSIS

by

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Supplement to
Testimony presented at Hearings of
The Joint Committee on Atomic Energy
91st Congress of the United States
February 9, 1970

also Submitted to Dr. Paul Tompkins
for the Review of Federal Radiation Council Guidelines
February 9, 1970

This is Document No. 1-B
Previously issued: Nos. 3, 4, 6, 7, 1-A, 8, 9, 2-A, 10, and Summary Testimony (GT-114-70)

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INTRODUCTION

In a recent critique of the Gofman-Tamplin estimate of the 16,000 additional cancers plus leukemias to be expected annually in the USA from radiation exposure at FRC Guidelines, Dr. John Storer has countered with a suggestion that a more appropriate number would be 160 additional cancer plus leukemia deaths. This critique was widely circulated by AEC-DBM and finally was even published in Hearings of the JCAE.(1) This critique is a classic illustration of the kind of "evidence" being presented currently to refute the Gofman-Tamplin work. We are, therefore, appending a copy of Dr. Storer's critique to this report, so that everyone interested in this problem can see that the bulk of the Storer critique rests upon a serious overt misquotation of the Gofman-Tamplin work. (See Exhibit A, Appendix)

THE STORER SUGGESTIONS FOR REDUCTION OF THE 16,000 EXTRA CANCERS + LEUKEMIAS

(a) A minor reduction requested by Dr. Storer because he claims "For low LET radiation, protracted exposure is about 1/5 as effective as single exposures for both genetic and life-shortening effects. This single erroneous assumption makes their estimate (Gofman-Tamplin) of case numbers high by a factor of 5".

We disagree with Dr. Storer in toto. Recently we published a report indicating that the apparent protection by protraction of low LET

radiation is illusory.(2) The experiments on which such apparent protection exist are of the type where the protracted radiation is delivered over a period extending well into the animal's life-span - at which time the sensitivity to radiation-induction of cancer was much reduced. Upton's beautiful experiments on mouse leukemia and ovarian cancer are two classic illustrations.(3) And Upton himself, in a separate paper(4) showed clearly the reduced sensitivity of these mice to radiation-induction of these specific diseases later in life. (corresponding to the late part of the protraction experiments). So, the protraction experiments show nothing about lesser effectiveness of slow delivery of radiation in carcinogenesis. All they show is that radiation later in life produces a lesser effect than radiation early in life, as in the Upton experiments.

We, therefore, deny Dr. Storer this factor of five as totally unjustified.

(b) A major reduction is requested by Dr. Storer because he believes only thyroid cancer and leukemia are radiation-induced. The argument he develops at great length in his critique rests wholly upon an overt misquotation of the Gofman-Tamplin reports. Not only does he misquote Gofman and Tamplin once, but he repeats it (see Exhibit A). Storer states on page 1 of his critique the following assumption - which he attributed to Gofman and Tamplin, although nowhere on earth will he find his absurd assumption in the writings of Gofman and Tamplin. He will find abundant evidence in the Gofman-Tamplin writings of precisely the opposite of his own misquotation.

Storer Quote: "Assumption No. 3 - All human cancers can be radiation-induced in the low dose range. Further, the doubling dose is approximately the same for all cancers as is the latent period".

If Dr. Storer can find a single place in any Gofman-Tamplin report that assumes "the latent period is approximately the same for all cancers", we would indeed be grateful. The entire thrust of much of our argument as to how the radiation-induction of diverse human cancers was missed is because the latent period is less for leukemia than for other cancers.(5)

It is a shame that Dr. Storer went to all the trouble of providing us with an elementary statistics lesson all based upon his overt misquotation of our statements. We might urge Dr. Storer to try reading some of the Gofman-Tamplin reports. Until there is some evidence that he can read our reports and refrain from overt misquotations, there would hardly seem any reason to consider the Storer critique any further.

CONCLUSION:

Dr. Storer's critique rests upon three points:

(a) The minor suggestion dealing with protraction of low LET radiation, which we reject as shown above.

(b) His (Storer's) undying belief in a "safe threshold" of radiation, which we have abundantly rejected elsewhere.(6)(7)

(c) The major suggestion of Storer resting totally upon an overt misquotation by him.

In view of the non-evidence provided by Storer, we see no reason whatever to assign any merit whatever to his suggestion that we lower our estimate of 16,000 additional deaths per year in the USA for FRC Guideline exposure (170 millirads).

REFERENCES

1. Storer, J. "Comments on manuscript "Low Dose Radiation, Chromosomes, and Cancer" by J. W. Gofman and A. R. Tamplin in "Environmental Effects of Producing Electric Power", Hearings of the Joint Committee on Atomic Energy. Part I. 91st Congress, October 28-November 7, 1969, pp 653-654. (Reproduced here as Exhibit A)

2. Gofman, J.W. and Tamplin, A.R. "The Mechanism of Radiation Carcinogenesis". Testimony presented at Hearings of the Joint Committee on Atomic Energy, 91st Congress USA, January 28, 1970. (GT-109-70) To be published in "Environmental Effects of Producing Electric Power" Part II.

3. Upton, A.C. "Comparative Observations on Radiation Carcinogenesis in Man and Animal" in "Carcinogenesis, a Broad Critique" in Twentieth Annual Symposium on Fundamental Cancer Research 1966, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas. Williams and Wilkins Co., Baltimore, 1967, pp 631-675.

4. Upton, A.C., Odell, T.T.,Jr., and Sniffen, E.P. "Influence of Age at time of Irradiation on Induction of Leukemia and Ovarian Tumors in R F Mice". Proc. Soc. Exp. Biol. and Med. 104, 769-772, 1960.

5. Gofman, J.W. and Tamplin, A.R. "Federal Radiation Council Guidelines for Radiation Exposure of the Population-at-Large -- Protection or Disaster?" Testimony presented before the Subcommittee on Air and Water Pollution, Committee on Public Works, United States Senate, 91st Congress, November 18, 1969. (GT-102-69) (Reprinted in Reference 1, pp 655-684).

6. Gofman, J.W. and Tamplin, A.R. "Studies of Radium-Exposed Humans: The Fallacy Underlying a Major Foundation of NCRP, ICRP, AND AEC Guidelines for Radiation Exposure to the Population-at-Large". Reference 5. (Reprinted in Reference 1, pp 695-706. (Also GT-103-69)

7. Gofman, J.W. and Tamplin, A.R. "Studies of Radium-Exposed Humans II: Further Refutation of the R.D. Evans Claim that 'The Linear, Non-Threshold Model of Human Radiation Carcinogenesis is Incorrect' ". Reference 2. To be published in "Environmental Effects of Producing Electric Power" Part II. (also GT-105-69)

APPENDIX (Exhibit A)

The overt misquotations by Dr. Storer of the Gofman-Tamplin work are indicated by arrow in the attached document of Dr. Storer taken from JCAE Hearings, "Environmental Effects of Producing Electric Power", Part I, pp 653-654.

p.1

OAK RIDGE NATIONAL LABORATORY,
Oak Ridge Tenn., November 10, 1969.

To: Dr. John Totter, Director, Division of Biology Medicine, AEC.

From: Dr. John Storer, Scientific Director for Pathology and Immunology, Biology Division.

Subject: Comments on manuscript "Low Dose Radiation, Chromosomes, and Cancer" by J. W. Gofman and A. R. Tamplin.

In order to provide this critique quickly, I have not included specific references to document my statements. Documentation can be provided if desired. The conclusions of Drs. Gofman and Tamplin depend upon a number of assumptions. The validity of the assumptions, therefore, should be examined.

Assumption No. 1—The dose-response curve for radiation injury is linear and goes through the zero intercept.—Historically this assumption is usually made in setting radiation standards in order to be extremely conservative. In attempting a real assessment and assigning numbers to expected case of injury, a more realistic relationship should be employed. For example, in the case of induction of bone tumors in the radium dial painters the conclusion should be inescapable that there is an effective threshold dose below which tumors do not appear. The same comment applies to dogs exposed to internal emitters. The most likely explanation is that favored by R. D. Evans, namely that the induction period at small doses exceeds the lifespan. The dose-response curve for leukemia in Nagasaki (which is the relevant experience because of the low neutron component in the radiation) appears curvilinear. In general, except for cases of neutron exposure, where adequate information is available for estimating cancer induction versus dose in the low to medium dose range, there appears to be either a threshold or a curvilinear relationship.

Assumption No. 2—Protracted exposure is equally effective as single brief exposures.—This assumption is not tenable even for genetic effects in mammalian systems. Life shortening effects which effectively summate all the deleterious effects of radiation including cancerogenesis have been the most thoroughly studied. For low LET radiation, protracted exposure is about 1/5 as effective as single exposures for both genetic and life shortening effects. This single erroneous assumption makes their estimate of case numbers high by a factor of 5.

Misquote No. 1 →

Asumption No. 3—All human cancers can be radiation-induced in the low dose range. Further, the doubling dose is approximately the same for all cancers as is the latent period.—This assumption cannot be true. If it were, then all cancers which occur with a normal frequency greater than that of thyroid cancer or leukemia would have been shown by now to be significantly increased in the irradiated survivors in the ABCC studies. The reason for this is that statistically it is much easier to detect a doubling of a relatively frequently occurring event than it is to detect a doubling of a rarely occurring event.

According to Segi and Kurahara (Cancer Mortality for Selected Sites in 24 Countries No. 4 1962-63), cancer of the thyroid is rare in Japan and leukemia is also relatively rare. For example, cancers of the esophagus, stomach, large intestine, rectum, lung, breast and uterus are all more frequent in occurrence. Yet of these, only for lung and breast is there even a suggestion of an increased incidence in irradiated survivors. (See the excellent report by R. W. Miller in Science, Oct. 31, 1969).

The assertion that it is easier to detect a doubling of a reasonably frequently occurring event than it is to detect a doubling of a rarely occurring event can be easily supported by a simple example. Since most testing of significance of increases of this type is done by use of some modification of a simple 2 x 2 Chi-square, I will use such a test in my example.

Suppose we have an event occurring 5% of the time in a control sample of 100 people and we observe it 10% of the time in a treated sample of the same size. Then:

Group	Number positive	Number negative	Total
Control	5	95	100
Treated	10	90	100
Total	15	185	200

p.2

In this case, Chi-square=1.80 P<.70.

Now lets take another event that occurs twice as frequently in a control sample of the same size and calculate Chi-square if the event is twice as frequent in the treated. We have:

Group	Number positive	Number negative	Total
Control	10	90	100
Treated	20	80	100
Total	30	170	200

Chi-square=3.92 P<.05.

Finally, consider an event which is four times as frequent as in our first case.

Group	Number positive	Number negative	Total
Control	20	80	100
Treated	40	60	100
Total	60	140	200

Chi-square=9.52 P<.01.

Misquote No. 2 →

On statistical grounds, then, Gofman and Tamplin's third assumption cannot be correct. Incidentally, the same argument applies to the studies of American radiologists. Leukemia was significantly increased. If all cancers have the same doubling dose and induction period, then the radiologists should have shown a significant increase in all cancers that normally occur with a frequency greater than leukemia.

From animal experimentation we know that the incidence of a number of tumors cannot be increased by moderate to large (but sublethal) doses of radiation. Some tumors can be induced by massive local doses of radiation (for example, skin tumors) but are not seen at lower doses. Even in man, the cancers of the lung of varieties other than the undifferentiated or small cell type do not appear to be induced even by massive doses of high LET radiation.

MISCELLANEOUS COMMENTS

Gofman and Tamplin, using the assumptions outlined above, have calculated a theoretical number of expected excess cancer deaths if the entire U.S. population were exposed continuously to maximum permissible dose levels. They indicate that a reduction of MPD's by a factor of 10 would produce a result acceptable to them.

Their calculated values can be immediately reduced by a factor of 5 because of the well-documented lower level of effectiveness of protracted radiation of low LET (the type relevant to the argument).

Even giving them linearity of the dose-response curve and a zero intercept, their estimate can further be reduced by a factor of 20 because only leukemia and thyroid tumors have been unequivocally shown to increase with increasing doses of radiation in the low to moderate dose range. (These two tumors account for 5% of the cancer incidence in the U.S.)

I would conclude conservatively that they have overestimated the expected increase in cancer at the MPD by at least a factor of 100.