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ATOMIC ENERGY ACT—1954

ADMINISTRATIVE REPORT

AR-563-DASA

April 1971

FINAL REPORT
1 October 1970 - 30 April 1971

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employing the concept discussed above. The decoupling would be determined by detonation of a second HE charge in a completely tamped configuration. A proposal of the experiment is being prepared by Whitener describing the concept with recommendations for his preparation of a test plan and participation as Project Officer.

X-RAY VULNERABILITY AND LETHALITY (U)

(U) During the first quarter of 1971 a thermodynamic study of titanium was completed by F. J. Krieger and published: "The Thermodynamics of the Titanium/Titanium Vapor System," DASA 2624 (Appendix XIII). A similar study on beryllium was initiated.

(U) As a result of discussions during the February Predix Meeting at Physics International, San Leandro, California, the following RAND research memoranda were sent to Dr. Robert Kruger, Systems, Science and Software, La Jolla, California, for use in DASA work:

RM-3326-3-PR, "The Thermodynamics of the Graphite, Carbon Vapor System"

RM-3988-PR, "Thermodynamics of the Phenol-Formaldehyde Resin/Carbon-Hydrogen-Oxygen Vapor System"

RM-5876-PR, "The Thermodynamics of the Aluminum Silicate/Aluminum-Silicon-Oxygen Vapor System"

RM-5958-PR, "The Thermodynamics of the Boron Carbide/Boron-Carbon Vapor System"

(U) A total of eighteen RAND research memoranda on the thermodynamics of ablating materials was sent to Lt. John G. Picarelli, AFWL, Kirtland Air Force Base, New Mexico.

FALLOUT AND BIOLOGICAL EFFECTS OF RADIATION (U)

(U) Two controversial issues dealing with low-level radiation damage have been matters of public debate in recent years. The Sternglass analysis which related infant mortalities to nuclear tests, and the no threshold for radiation damage theory of Tamplin and Gofman

have relevancy to the long term consequences of fallout and nuclear warfare.

(U) H. H. Mitchell, M.D. has prepared a brief review of each of these radiation damage issues. The Sternglass controversy comments are summarized in Appendix XIV. A less thorough analysis of the Gofman-Tamplin radiation exposure standards issue is contained in Appendix XV.

WEAPON OUTPUT (U)

(U) Brode has continued as Chairman of the DASA Weapon Output Working Group, which, in recent years, has served to coordinate improvements in codes and help incorporate additional physics treatments of nuclear and x-ray radiation out of weapons and test devices. One significant contribution from this working group is the two-way exchange it promotes between workers in the AEC labs and DASA on matters pertinent to the needs for and applications of weapon disassembly detail. The AEC workers acquire a better appreciation of the weapons information and design detail needed in the analysis of weapon effects, and so can do a better job. The DASA personnel, on the other hand, have come to know enough about the weapon design features and prospects to make more realistic estimates of effects.

(U) The detailed crater coupling work now in progress is also being coordinated with LRL interests in this area.

SYSTEMS VULNERABILITY ASSESSMENTS (U)

(U) The work of Schaefer, Graham, Rogers, Thomas, Gilmore, LeLevier, Brode, Wright, and others have impact on systems vulnerability assessments. Some of these considerations are derived from other specific subjects covered in this report (e.g., TREES, EMP, GROUND SHOCK, HIGH ALTITUDE, etc.), and some work more directly associated with systems survivability assessments (by Schaefer) is covered in Appendices XI and XII.

Appendix XIV

4/16/71

COMMENTS ON THE STERNGLASS CONTROVERSY

H. H. Mitchell, M.D.

What has come to be known as the Sternglass controversy started with a paper presented at the 9th Annual Hanford Biological Symposium in May 1969. Sternglass presented evidence indicating alarming effects due to low level fallout radiation. He stated his conclusions as follows:

> "The earliest evidence for low-level and low dose rate effects was obtained from a detailed analysis of the leukemia increase among children 0 to 10 years old in the Albany-Troy, N.Y. area following the rainout of radioactive debris from a 43-KT nuclear detonation in Nevada in April 1953. The subsequent doubling of the childhood leukemia rate over a period of 8 years was characterized by a 4 to 5 year delay in onset similar to that observed for children who received x-ray radiation in utero or early infancy and a shift in age distribution at onset toward older age similar to that noted by Stewart and Hewitt for intrauterine x-ray irradiation."

> "More recent evidence for the effects of low-dose-rate radiation on the developing fetus, embryo, and young infant comes from a study of fetal, neonatal, and postnatal mortality rates for each state in the United States and for a number of foreign countries. Analysis of the changes in mortality rates following the detonation of specific nuclear weapons shows a geographical distribution that coincides with the known long-range fallout patterns. The changes in mortality rates for

different regions of the United States and the world are found to be closely correlated with the measured amounts of Sr^{90} in the milk and the observed amounts in the bone and teeth of the fetus and newborn."

In an article in Esquire entitled, "The Death of All Children," Sternglass made the following statements:

1. The scientific evidence indicates that already at least one of three children, who died before their first birthday in America in the 1960s, may have died as a result of peacetime nuclear testing.

2. The computer-calculated change in infant mortality was found to have reached close to one excess death in the U.S. per one hundred live births due to the release of only 200 megatons of fission energy by 1963.

3. A release of some 20,000 megatons anywhere in the world, needed in offensive warheads for an effective first strike or in the thousands of defensive ABM warheads required to ensure interception, could lead to essentially no infants surviving to produce another generation.

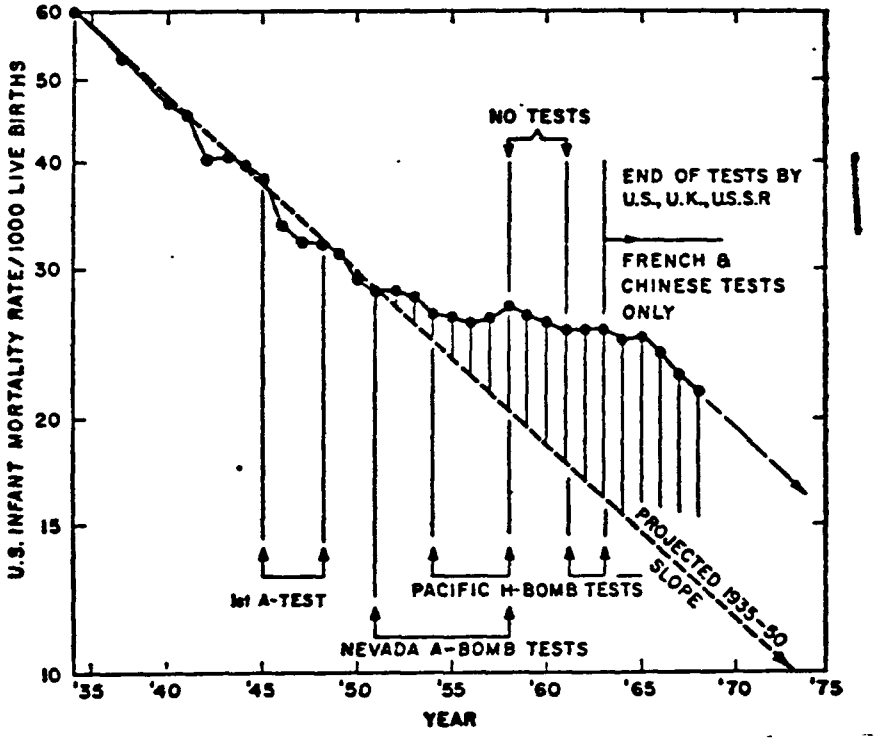
Considering the implication of Sternglass' assertions there has been generated a rather large literature (see references for a representative but incomplete bibliography) examining the data from which the conclusions have been derived.

A. INFANT MORTALITY

Figure 1 represents the data, and the device, used by Sternglass to determine the excess infant mortality rate in the U.S. In essence he has incorporated a "projected 1935-50 slope" on the actual data and has stated that the differential between the expected and the projected values constitutes the excess infant mortality.

FIGURE 1

MORTALITY RATE IN THE U.S. FOR INFANTS 0-1 YR.



In an article entitled, "The Pitfalls of Extrapolation," A. Stewart (New Scientist, July 24, 1969) has stated that "Sternglass has postulated a fetal mortality trend which would eventually produce rates well below the level which--according to his own theory--would result from background radiation." Faulty extrapolation has obviously provided an

implausible, if not impossible, result. It is also true that if one were to extend the projection backward in time the infant mortality would have been 100% at sometime in the past.

Lindop and Rotblat (Nature 224: 1257, 1969) have concisely summarized the various criticism concerning Sternglass' use of the infant mortality data:

> "Infant mortality in a given country at any particular time has several causes, for example, a low standard of hygiene, lack of care, infectious diseases, accidents and genetic effects. The steady increase in standard of living in the developed countries during the past century has resulted in a gradual decrease in the mortality rates. When an important advance is made which eliminates, or greatly reduces, one cause of death, for example, the introduction of sulfonamides and antibiotics, which no doubt markedly reduced infectious diseases which contribute to infant mortality, there is a sudden drop in the mortality curve; the steep slope may continue for some time until this particular cause has been eliminated, and then the curve resumes its smaller slope until a further cause of infant death can be removed. Thus, the mortality curve would be expected to have a general downward trend, but with slopes varying from time to time."

Figure 2 (taken from Sartwell: Preventive Medicine & Public Health, page 656) shows a breakdown by etiology of infant deaths from 1930-1959. It is apparent that the control of pneumonia and influenza as well as enteritis and diarrhea are the chief contributors to the steep slope used by Sternglass for his overall infant mortality rate.

Fig 2

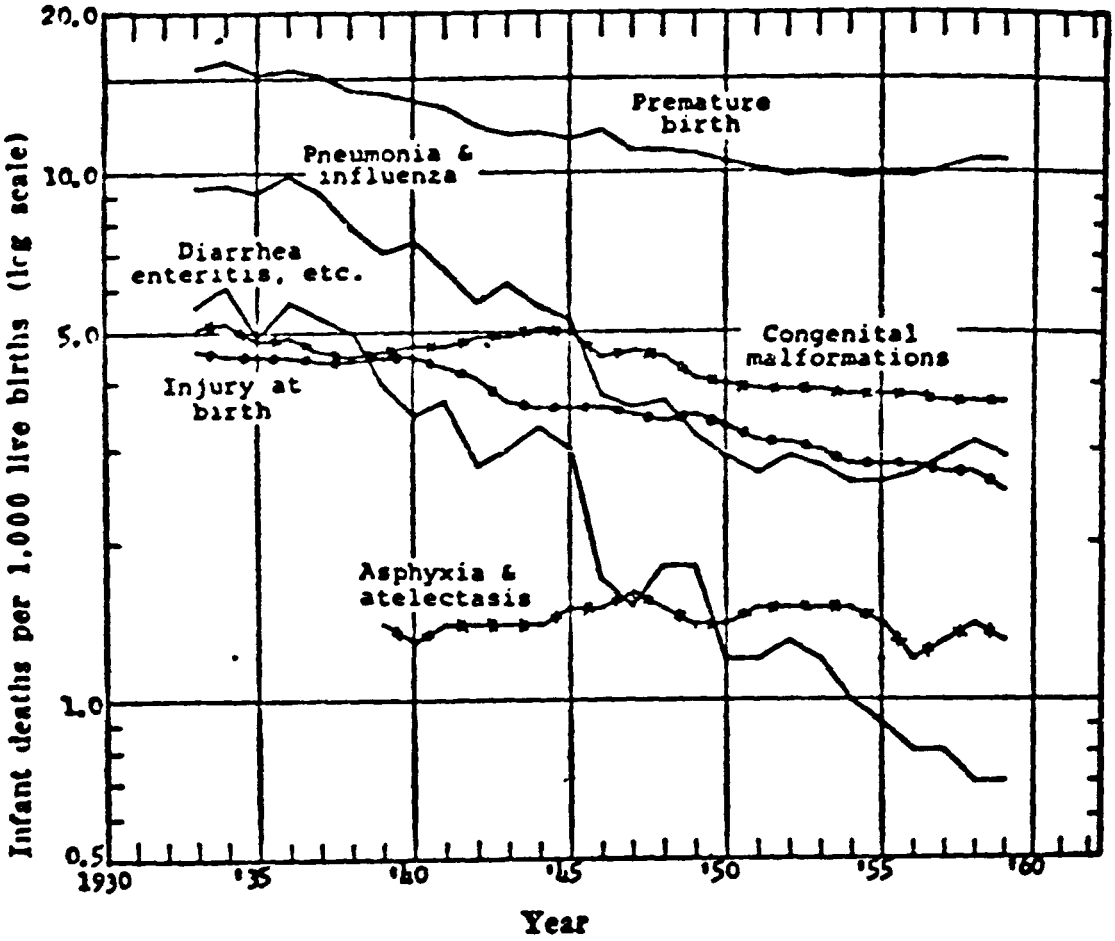


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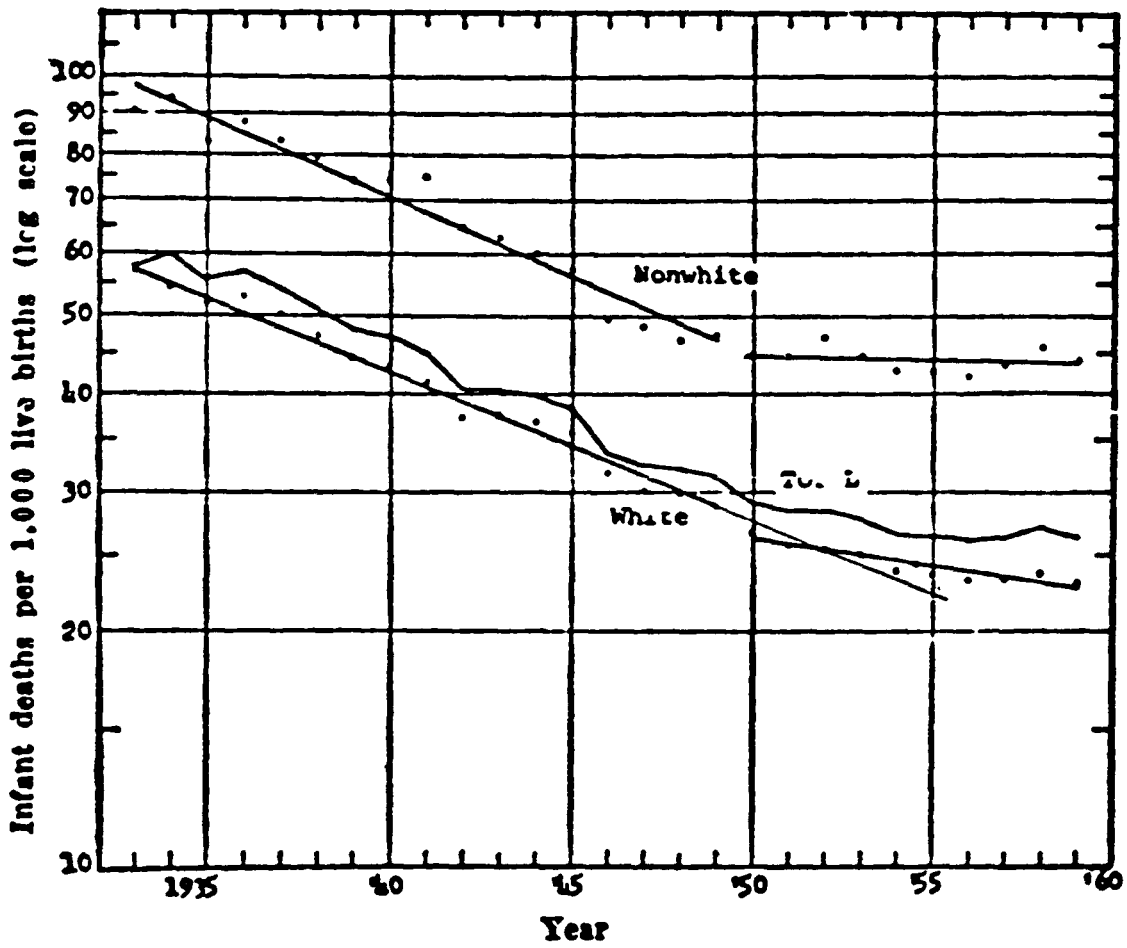
Figure 2 also indicates, if anything, a slightly downward trend in the infant deaths from congenital malformations. This classification would be most likely to rise if deaths due to radiation were introduced into the totality of infant deaths beginning in 1950.

Figure 3 (also from Sartwell, page 656) shows a plot of infant mortality by whites and non-whites. It is evident that the flattening of the total death rate curve is influenced more by the non-white than the white population. One certainly would not expect this kind of result if radiation were the cause of the change in slope of the total death rate curve.

It is also reasonable to assume that the white infant death rate curve would be even less flattened if the white poverty group were removed.

Tompkins and Brown (October, 1969) have questioned Sternglass' comparison of U.S.and Swedish infant mortality data purporting to show that there is no "natural plateau" in the attainable infant mortality rate. The Swedish data level off at about 1957 and by 1961 the slope resumes at the previous rate. Tompkins and Brown further state: "If the 4 years of change in rate of decline in 1957-1960 in the Swedish mortality rate are indeed a reflection of deposition of Sr^{90} from the large tests in the Pacific, there is no explanation as to why the large series of Russian tests in the fall of 1961 and 1962, which led to the world-wide deposition of more Sr^{90} than any other series, is not reflected in the infant mortality in Sweden in 1965 and 1966.

Fig 3



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Rotblat and Lindop also examined the infant mortality curves in several other countries. France, Italy and Japan, with similar amounts of global fallout as the U.S. show considerable variation in their infant mortality curves. The French curve did not flatten until 1962. In Italy the curve was still sloping without a plateau through 1965. In Japan the slope of the curve became steeper after 1958. Although Australia does show a break (similar to the U.S. curve) in 1952 the Sr^{90} deposition is very small (about 10% of northern hemisphere amounts). The Sternglass assumption that this is excess mortality due to Sr^{90} is untenable.

It would appear from the foregoing arguments that the character of the infant death rate curve is made up of many elements. These do not fit the Sternglass assumption that low-level radiation is responsible via a genetic effect for the flattening of the slope beginning in 1950.

B. THE TRINITY SHOT

Sternglass has also contended that the fallout from the Trinity shot (July 1945) crossed the southeastern portion of the U.S..and claims to show that subsequent to this there was an increase in infant mortality in these states. There is a summary refutation of this Sternglass presentation in the AEC "evaluation" paper (see bibliography):

1) R. Engelman (a meteorologist with the AEC) has evaluated the weather data and concluded that Trinity fallout did not cross the Mississippi river into the eastern states south of the Iowa, Illinois area.

2) Harley (Director of AEC Health and Safety Laboratory): "The trajectory of the 1945 Alamagordo test has been plotted for winds at the 10,000, 20,000, and 30,000 foot levels. These would indicate that the majority of the fallout would have been deposited in New Mexico and that the cloud would have crossed the states towards the northeast, going into Kansas and Nebraska. This would agree with the findings of Webb of the Eastman Kodak Co. that paperboard produced in this area following the Trinity test was radioactive. The portion of the cloud at 30,000 ft. did start toward the southeast but turned back into Texas without crossing the Mississippi."

Since it is well established that the fallout from Trinity did not occur where Sternglass said it did, his discussion of infant mortality rates related to Sr^{90} from this shot can have no significance.

C. FETAL MORTALITY AND CHILDHOOD LEUKEMIA

The Sternglass arguments with regard to fetal mortality and childhood leukemia being caused by Sr^{90} derived from fallout are even weaker than the case made for infant mortality. The refutations by his critics are included in the bibliography but it seems to be an unnecessary exercise to attempt to summarize these.

D. THE STERNGLASS HYPOTHESIS ON THE MECHANISM OF THE SR^{90} EFFECT

In the Esquire article, "The Death of all Children," Sternglass has stated "The causation puzzle now appears to be solved. In 1963, K. G. Luning and his co-workers in Sweden published their discovery that small amounts of strontium 90, injected into male mice three or four weeks prior to mating, produced an increase in fetal deaths among their offspring. No such increase appeared when corresponding amounts of chemically different radioactive cesium 137 were injected.

Rotblat and Lindop refute this causative explanation in their paper on "Strontium 90 and Infant Mortality."

> "The claim that evidence from experimental data with animals supports the Sternglass effect falls to the ground when a quantitative analysis is made. Professor Luning of Stockholm, whose work is quoted by Sternglass, has made a public statement in which he repudiates the use of his data as a basis for the Sternglass effect. Luning and his colleagues injected Sr^{90} intraperitoneally into male mice, mated them with unirradiated females, and studied the foetal mortality in the offspring. The effect observed was so small that

they had to use a very large number of animals. The final results have not yet been published, but in a personal communication Luning gave us some preliminary data: 18,000 controls and over 20,000 irradiated mice were used in the experiment and the foetal mortality rate was 7.5% in the controls and 8.7% in the irradiated mice. This small difference is probably significant, but it should be noted that Luning's mice received 18 micro-curies of Sr^{90} which is an enormous dose compared with the strontium from fallout. Assuming that only 1% of this Sr^{90} was retained in the mice, the concentration of Sr^{90} per gramme of body weight was 100,000 times greater than that in the human adult from all the tests of nuclear weapons."

Sagan in the October 1969 Bulletin of the Atomic Scientists commented on the genetic damage argument of Sternglass:

"A possible effect of radiation in induction of peri-natal mortality through genetic damage to the parents has not been overlooked as a possibility by investigators. Studies of the outcome of pregnancy among atom bomb survivors have failed to show either a decrease in birth weight or increased mortality among offspring. Nor was such an effect seen among children of radiologists or women who received therapeutic radiation. Animal studies carried out with swine fed strontium 90 at Hanford have shown no effects among offspring even at very high doses."

Crissman and Kirk in their paper "Strontium Metabolism with Special Consideration of Genetic Effects" stated:

" No doubt the dangers of Sr^{90} are real; however, no low or comparable dose rate studies of the existing problem have been done. To extrapolate from studies of mice given large doses of Sr^{90} to the highly complex statistical evaluation of infant mortality figures is simply not realistic.... The effects of Sr^{90} on the reproductive system and on alterations in genetic material have not been quantitated. The role of these possible alterations and their contribution to infant mortality appear to be quite small at the body burdens of Sr^{90} existing today."

E. CONCLUSIONS

1. The reported demonstration by Sternglass of excess deaths (childhood leukemia, fetal mortality, infant mortality) appear to be due to improper use of epidemiological data.
2. His proposed Sr^{90} genetic damage mechanism has been refuted by proper interpretation of the same experimental data which he used to support his claim.
3. No serious scientific defense of the Sternglass work has been published by anyone not connected with Sternglass himself.

BIBLIOGRAPHY

1. Sternglass, E. J., "Evidence for Low Level Radiation Effects in the Human Embryo and Fetus," in Radiation Biology of the Fetal and Juvenile Mammal, 9th Annual Hanford Symposium, May 5-8, 1969, December 1969.

2. Sternglass, E.J., "Strontium-90: Evidence for a Possible Genetic Effect in Man," presented at the 14th Annual Meeting of the Health Physics Society, Pittsburgh, Pennsylvania, June 8-12, 1969.

3. Sternglass, E.J., "The Death of All Children," Esquire, September 1969.

4. Sternglass, E.J., "Infant Mortality and Nuclear Tests," Bulletin of Atomic Scientists, Vol. 25, No. 18, 1969.

5. Sternglass, E.J., "Can the Infants Survive?", Bulletin of Atomic Scientists, Vol. 25, No. 6, 1969.

6. AEC Staff, Evaluation and Critique of the Arguments presented by E. J. Sternglass (personal communication).

7. Aranoff, . and Boylan, E. S., "Flaws in the Sternglass Thesis," Hudson Institute, HI-1248-DP, August 5, 1969.

8. Crissman, J. D. and Kirk, J. H., "Strontium Metabolism with Special Consideration of Genetic Effects," Review 1-70-USAF School of Aerospace Medicine, Brooks Air Force Base, Texas, January 1970.

9. Friedlander, M. W. and Klarmann, J., "How Many Children?", Environment, Vol. 11, No. 4, 1969.

10. Graham, S. and Thro, E., "The Sternglass Phenomenon," Nuclear News, Vol. 12, No. 37, 1969.

11. Greenwald, P. and Kinch, "Relation of Radioactive Fallout to Leukemia and Fetal Mortality; A Reconsideration," U. S. Atomic Energy Commission, EXEP-690501, December 1969.

12. Harley, J. H., "Comments on Fallout Correlations Made by Dr. Ernest Sternglass," HASL-214, Quarterly Report, October 1, 1969.

13. Lindop, P. J. and Rotblat, J., "Strontium-90 and Infant Mortality," Nature, Vol. 224, No. 1257, 1969.

14. Nilsson, A. and Walinder, G., "Strontium-90 Dosages and Infant Mortality," Bulletin Atomic Scientists, Vol. 26, No. 40, 1970.

15. Rotblat, J. and Lindop, P. J., "Strontium-90 and Infant Mortality," 19th Pugwash Conference on Science and World Affairs, October 1969.

16. Sartwell, P.E., Preventive Medicine and Public Health, 9th Ed. Appleton-Century-Crofts, 1965.

17. Tamplin, A. R., "Fetal and Infant Morality and the Environment," Bulletin of Atomic Scientists, December 1969.

18. Tompkins, E. and Brown, M. L., "Evaluation of a Possible Causal Relationship between Fallout Deposition of Strontium 90 and Infant and Fetal Mortality Trends," DBE69-2, U.S. Department of Health, Education and Welfare (Public Health Services, Bureau of Radiological Health), Rockville, Maryland.

19. Sagan, L. A., "Infant Mortality Controversy," Bulletin of Atomic Scientists, October 1969.

20. Sagan, L., "A Reply to Sternglass," New Scientist, Vol. 44, No. 16, 1969.

21. Stewart, A., "The Pitfalls of Extrapolation," New Scientist, July 1969, page 181.

Appendix XV

The Gofman and Tamplin Argument Over the Federal Radiation Standard of 170 mr Per Year for Population Exposure

Harold H. Mitchell, M.D.

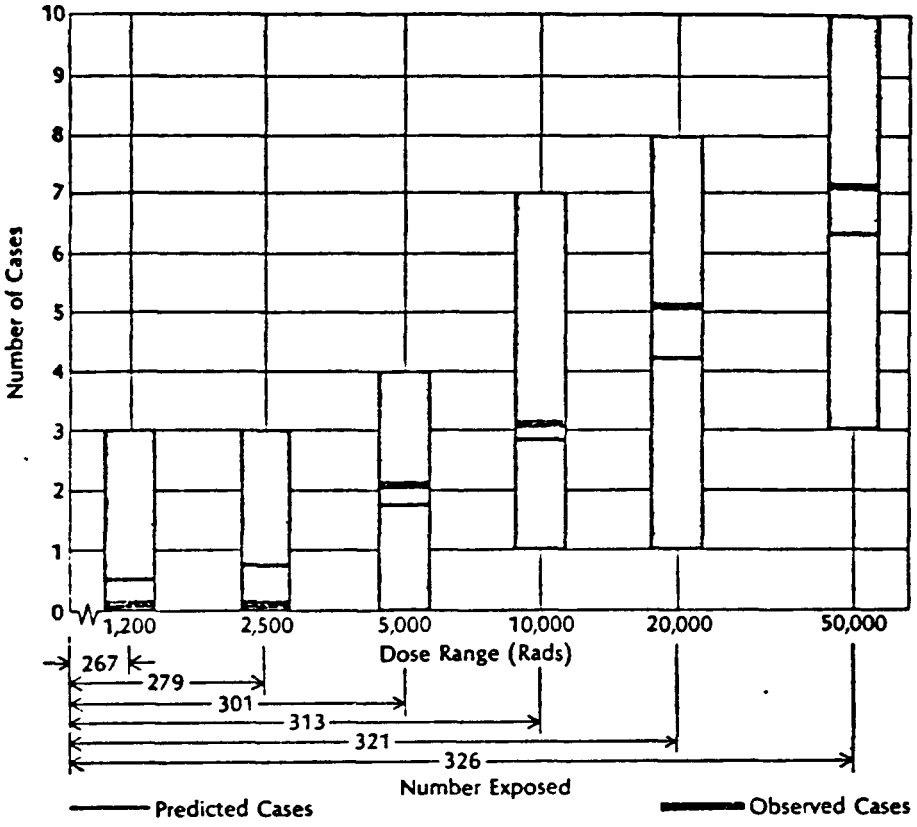
John W. Gofman and Arthur R. Tamplin have raised the issue of the wisdom and safety embodied in the federal standards for radiation exposure. They have argued that the population radiation exposure standard of 170 millirem per year (5 rem in 30 years) is too high, by a factor of ten, as a guide for the peacetime activities of the use of atomic energy.

These workers have examined the following data:

1) Radium poisoning cases.
2) Hiroshima and Nagasaki exposure.
3) Breast cancer study on 900 Canadian women following fluoroscopic irradiation (tuberculosis cases) as well as lung cancer study on 500,000 Israelis (tuberculous versus non-tuberculosis cases).
4) About 6000 uranium and other hard rock miners (lung cancer).
5) About 14,000 ankylosing spondylitis cases who received spinal irradiation.
6) Several thousand U.S. individuals irradiated for status thymicolymphaticus (thyroid and other cancers).
7) Numerous animal experimental studies.

Figure 1 illustrates their interpretation of the radium ingestion cases and purports to show consistency with the concept of no threshold for cancer causation and the linear concept of carcinogenesis.

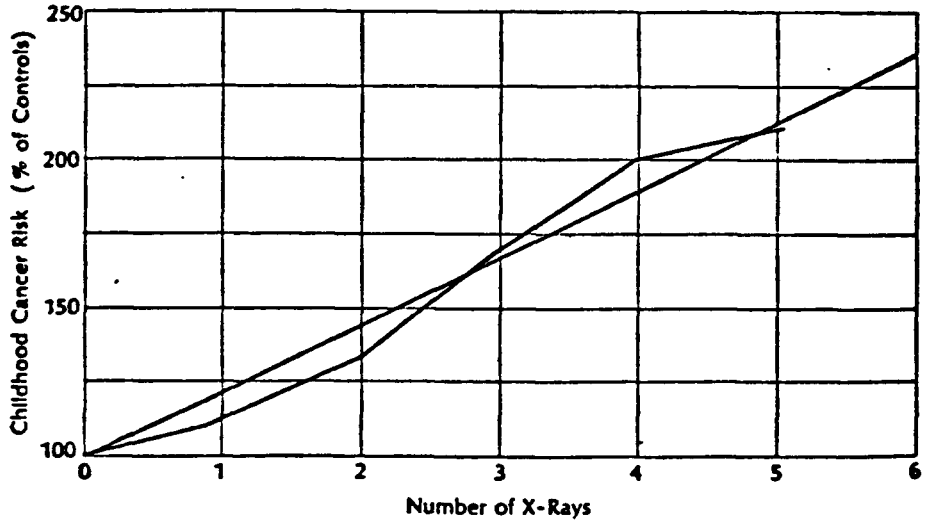
Figure 1



Linear concept of carcinogenesis, holding that cancer incidence is directly proportional to radiation dose, is supported by data on watch painters who ingested radium. ? ber of observed cases falls well within range predicted by linear assumptions.

Figure 2 is a plot of the Stewart and Kneale data on children irradiated in utero and also approximates a linear relationship between radiation dose and cancer risk. There is an indication of increased cancer risk at a radiation dose equivalent to 0.5 rad.

Figure 2



Data by Stewart and Kneale (Lancet 1:1185, June 6, 1970) on children irradiated in ... relationship. In the radiation-sensitive fetus,

Table I is the Gofman-Tamplin summary on the radiogenic lung cancer among the Uranium miners. The figures which follow (3, 4, 5, 6) are graphic representations of the data in the tables.

Table I

Radiogenic Lung Cancer Among Uranium Miners

Dose Range	*A Expected Spontaneous Cases*	*B Observed Cases*	*C Total Number in Group*	*D "Excess" Cases (Presumed Radiogenic) [B − A]*	*E "Excess" Cases per 1,000* $\left[\frac{D \times 10^3}{C}\right]$	*F Number of Doubling Doses* $\left[\frac{D}{A}\right]$	*G Mean Dose (WLM)*	*H Calculated Doubling Dose (WLM)* $\left[\frac{G}{F}\right]$	*I "Excess" Cases per Million per WLM* $\left[\frac{E \times 10^3}{G}\right]$
<120 − ≧3720	5.68	45	1981	39.32	19.7	6.93	973.9	140.5	20.2
<120 − 3719	5.50	34	1918	28.50	14.9	5.18	808.8	156.1	18.4
<120 − 1799	4.71	25	1700	20.29	11.9	4.31	558.6	129.6	21.3
<120 − 839	3.52	14	1300	10.48	8.1	2.98	324.3	108.8	24.9
<120 − 359	2.24	9	804	6.76	8.4	3.02	154.3	51.1	54.4
<120	1.02	3	383	1.98	5.2	1.94	60.0	30.3	86.7

Figure 3 is a graph of the raw data and merely shows the crude relationship between dosage and number of "excess" cases.

Figure 3

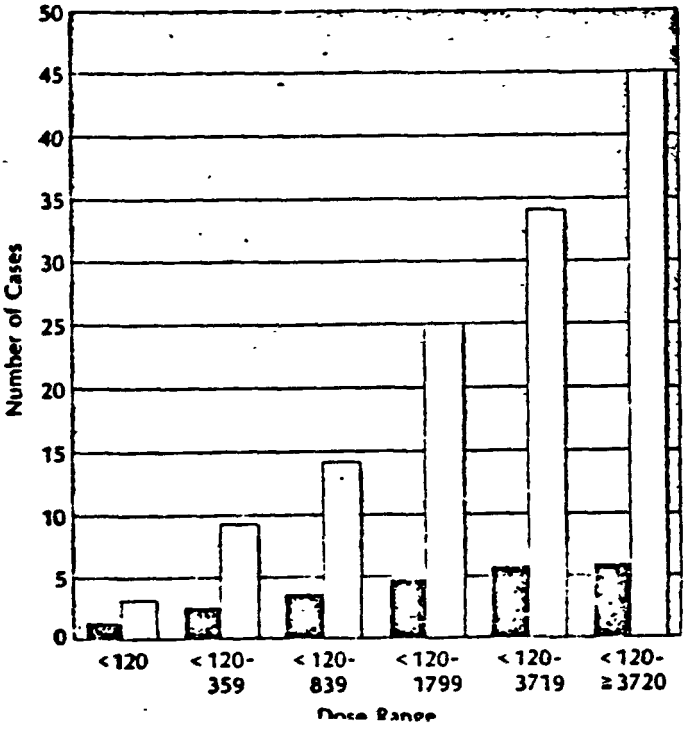


Figure 4 is the calculated case rate per 1000 plotted against mean dose. It approximates the expected linear relationship. The curve is uncertain at lowest dose due to small number of cases.

Figure 4

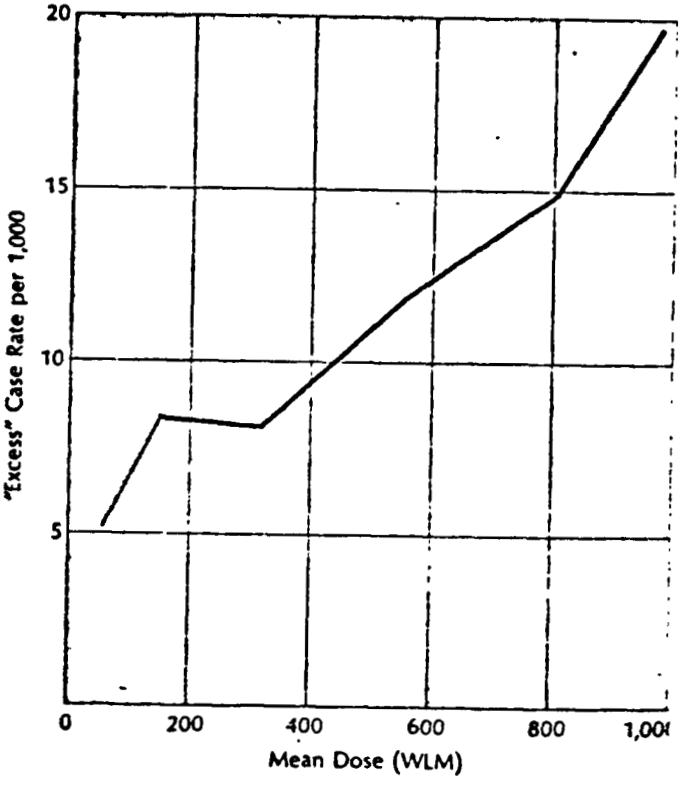
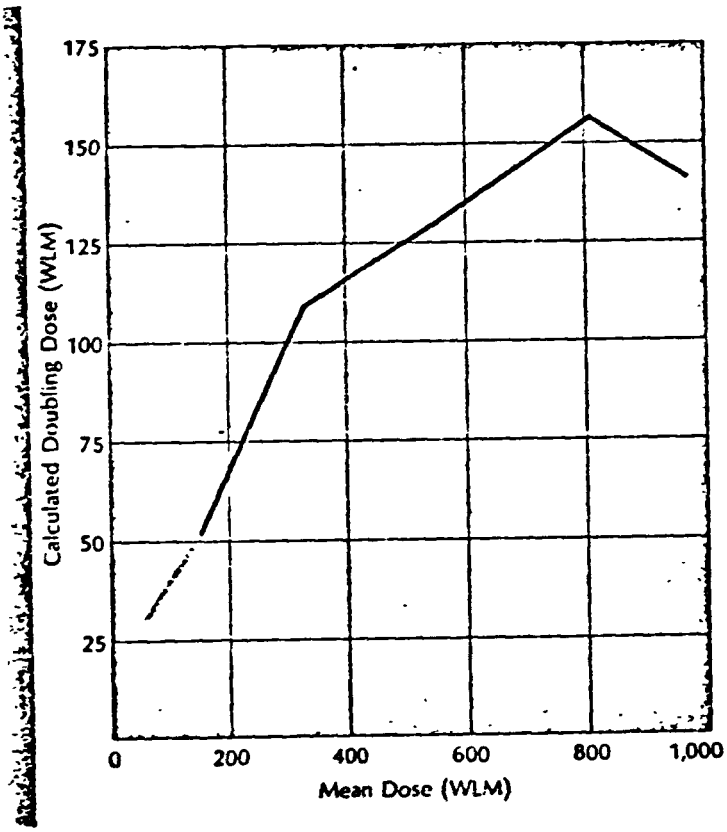


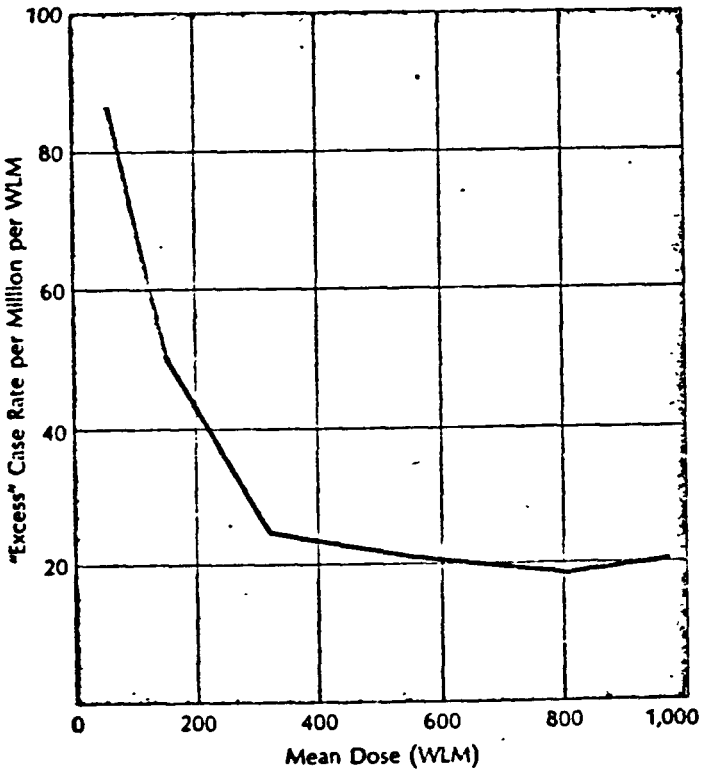
Figure 5 shows that the calculated doubling dose drops off as the mean dose falls. If there were a threshold effect at low dosage, this curve would be expected to rise rather than fall.

Figure 5



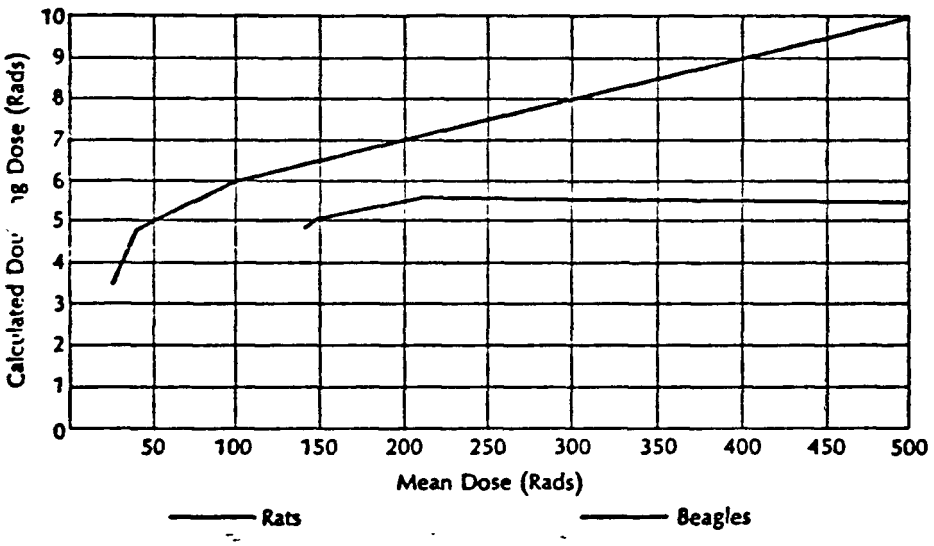
Finally, Figure 6 is the "clearest expression of differential impact with varying dose and shows that the effect per WLM (working level months) is higher at lowest dosages."

Figure 6



Gofman and Tamplin have summarized (Figure 7) two experiments, using rats and beagles, to show that as the total dose falls there is either a constant doubling dose calculated or a falling off of the calculated doubling dose.

Figure 7



After examining all the data available on radiation induced cancer in humans, Gofman and Tamplin summarized their findings as follows:

Table 2

Best Estimates of Doubling Dose of Radiation for Human Cancers and the Increase in Incidence Rate per Rad of Exposure

Organ Site	Approximate Doubling Dose	% Increase in Incidence Rate per Rad
Leukemia	30-60 Rads	1.6-3.3%
Thyroid Cancer		
(adults)	100 Rads	1%
(young persons)	(5-10 Rads)	(10-20%)
Lung Cancer	175 Rads	0.6%
Breast Cancer	100 Rads	1%
Stomach Cancer	230 Rads	0.4%
Pancreas Cancer	125 Rads	0.8%
Bone Cancer	40 Rads	2.5%
Lymphatic + other Hematopoietic organs	70 Rads	1.4%
Carcinomatosis of miscellaneous origin	60 Rads	1.7%

This data has led them to make a startling and highly controversial generalization: "that a given quantity of radiation can be expected to increase all forms of neoplasia in a given population in direct proportion to their spontaneous incidence in that population. In other words, the carcinogenic effects of radiation are not additive...but multiplicative--i.e. synergistic with other carcinogenic processes."

In addition to the data presented in Table 2 the effects of radiation on the fetus in utero are summarized. 2 to 3 rads is the estimated amount of radiation delivered. From the Stewart and Kneale data and the MacMahon data we have:

Table 3

Type of Cancer	Radiation Induced Increase
Leukemia	50% increase over spontaneous incidence
Lymphosarcoma	50% " " " "
Cerebral Tumors	50% " " " "
Neuroblastoma	50% " " " "
Wilms' Tumor	60% " " " "
Other cancers	50% " " " "
From the MacMahon data, we have the following highly similar estimates:	
Leukemia	50% increase over spontaneous incidence
Central Nervous System Tumors	60% " " " "
Other cancers	40% " " " "

On the basis of all their studies, Gofman and Tamplin have concluded that the following estimates are reasonable:

For adults:
a) 100 rads is the estimated doubling dose for all cancers.
b) 1% increase in incident rate per year follows each increase of one rad of exposure.

For those under 20 yrs of age:
a) Between 5 and 100 rads is the estimated doubling dose.
b) Between 1 and 20% increase in incidence rate per year per rad of exposure.

For infants in-utero:
a) 6 rads is the doubling dose.
b) 17% increase in incidence rate per year per rad of exposure.

Using the estimates cited above they calculated that the U.S. population would experience 16,000 additional cases of cancer and leukemia on the basis of the 170 mr standard for 30 years (5 r cumulative). Any increase in population would increase the cancer calculation proportionately. Prenatal exposure effects are not included in this calculation.

With regard to genetic effects of radiation, they have used some recent data and estimated that the minimum consequences (from population exposure to current standard of 170 mr/yr) after several generations is a 5% increase in total morbidity and mortality. This would be at least 150,000 additional deaths per year, according to their calculations.

* * *

As expected there is developing a considerable rebuttal to the Gofman-Tamplin arguments.

A. Dr. John Storer (personal communication to Dr. John Totter):

1. The linearity of the dose-respons curve for radiation injury with passage through the zero intercept:

This assumption is usually made in setting radiation standards in order to be conservative.

In the radium dial painters there is a threshold dose below which tumors do not appear. This also applies to the dog experiments with internal emitters.

The dose response curve for leukemia in Nagasaki is not linear but rather curvilinear.

Generally, a curvilinear relationship or threshold appears to be the best interpretation of cancer induction in the low to medium dose range.

2. The equal doubling dose concept for all cancers is seriously questioned. If this concept were true then the ABCC studies should already show significant increases for all cancers with a greater normal frequency thanthat of leukemia and thyroid cancer. It is

easier to detect a doubling of a relatively frequently occurring event than a doubling of a rare event.

"According to Segi and Kurakura 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."

The increased incidence of leukemia among American radiologists should have shown a significant increase of all cancers that normally occur with a greater frequency than leukemia if Gofman and Tamplin are correct. This has not apparently happened.

3. The Gofman-Tamplin assertion that protracted exposure is equally effective as single brief exposures is untenable. This erroneous assumption alone makes their estimate of cases high by a factor of 5.

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

B. AEC Staff Comments:

"...the author's presentation appears to rely on a single assumption: namely, that available data on the incidence of radiation-induced cancer should be interpreted in terms of doubling dose and that all forms of cancer show closely similar doubling doses and closely similar increases in incidence per rad." They conclude that the concept of a doubling dose as applied to carcinogenesis by Gofman and Tamplin is not valid.

C. Comments of Alexander Grendon (Donner Laboratory).

"Gofman and Tamplin have been selective in the data they have chosen to use in deriving their multiplying factor. The various studies of human radiation exposure reported in the scientific literature show equivocal results, some of which are contrary to those selected. Moreover, the very studies they cite contain elements which they disregard that tend to contradict their hypothesis.

"The scientific aspects of the issues raised by Gofman and Tamplin do not, then, appear to support their thesis. The human data are at least equivocal, thus undermining the basis of their conclusion, which is the asserted consistency of effects. The animal data, not considered by these authors, show many instances of effects quite contradictory of their hypothesis. In sum, there seems to be no sound reason to depart from the studied conclusion that an annual average radiation dose of 170 mr presents no serious threat to the health of the individual exposed.

D. The controversy is a continuing one and more reports are expected.