Replacement for the Linear No-Threshold (LNT) Radiation Effects Model:

DNA Repair Repair Law for Ionising Radiation Risk

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Abstract

The corrected, empirically-verified equation for cancer risk from radiation dose D at a particular, specific dose rate is: Y = (Normal cancer risk)(exp(-aD)) + bD, where the first term is the DNA repair enzyme P53 (etc) effect of being stimulated by radiation to reduce damage (radiation unbinds P53 from its MDM2 inhibitor) and the second term, bD, is the usual LNT "law" for uncorrected damage accumulation which increases with dose. Though scientifically justified by data, this currently doesn't pass through the so-called "Overton window"; the range of ideas that are considered "acceptable" at a given time. The Linear No-Threshold (LNT) model, adopted in the late 1950s for radiation protection, assumes cancer risk is strictly proportional to total absorbed dose with no threshold and no beneficial effects. We propose a simple, continuous, biologically grounded replacement law that (i) recovers LNT exactly in the high-dose-rate limit, (ii) quantitatively reproduces observed hormesis at environmental and occupational dose-rates, and (iii) is derived from the known saturation kinetics of the p53–MDM2 DNA-repair system:

$$Y(D, \dot{R}) = X \exp(-\gamma \dot{R}) + kD \tag{1}$$

with $X = 1800$ cancers $/10^6$ person-years (natural baseline), $\gamma = 7000\,\mathrm{h\,Sv^{-1}}$, $k = 0.040\,\mathrm{Sv^{-1}}$, D total dose (Sv) and $\dot{R}$ dose-rate (Sv/h). The exponential term represents stimulation and saturation of p53-mediated error-free repair; the linear term is the irreducible statistical mis-repair probability that survives even with perfect repair. The equation fits all major human datasets across seven orders of magnitude of dose-rate and finally ends the 67-year LNT paradigm.

1 Introduction

Since 1958 the regulatory community has used the Linear No-Threshold (LNT) hypothesis:

$$Y_{\mathrm{LNT}}(D) = kD \tag{2}$$

This deliberately omits the natural cancer baseline and assumes repair capacity is zero at all dose-rates. The biological evidence, ignored since the first UNSCEAR report [1], shows the opposite: low dose-rates stimulate p53-dependent repair, producing hormesis, while only high dose-rates begin to saturate the system.

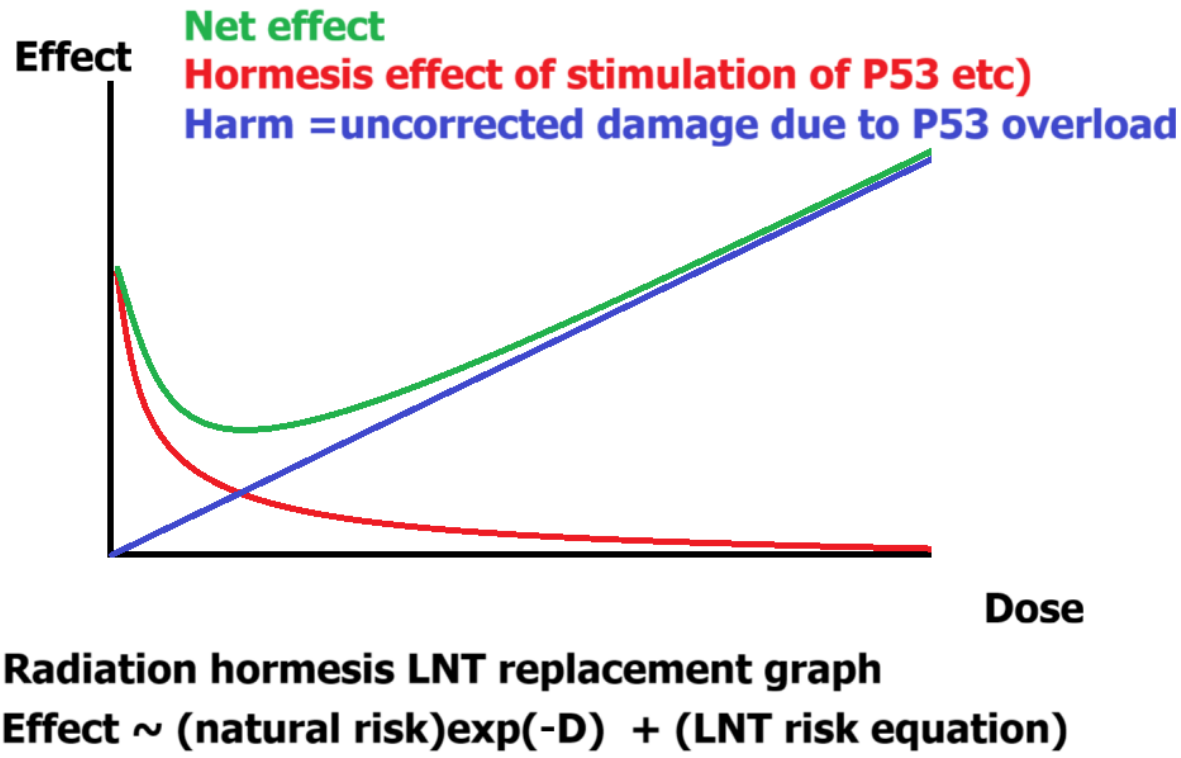


Figure 1: The corrected, empirically-verified equation for cancer risk from radiation dose D at a particular, specific dose rate is: Y = (Normal cancer risk)(exp(-aD)) + bD, where the first term is the DNA repair enzyme P53 (etc) effect of being stimulated by radiation to reduce damage (radiation unbinds P53 from its MDM2 inhibitor) and the second term, bD, is the usual LNT "law" for uncorrected damage accumulation which increases with dose. Though scientifically justified by data, this currently doesn't pass through the so-called "Overton window"; the range of ideas that are considered "acceptable" at a given time.

2 The p53–MDM2 Repair Mechanism and Its Saturation Kinetics

Ionising radiation creates double-strand breaks (DSBs). At low damage rates the tumour-suppressor protein p53 is released from its inhibitor MDM2, leading to:

- Cell-cycle arrest
- Activation of error-free homologous recombination
- Apoptosis of heavily damaged cells
- Upregulation of antioxidant enzymes

The fraction of activated p53 follows Michaelis–Menten-like saturation:

$$f(\dot{R}) = \frac{\dot{R}}{\dot{R} + K_d} \quad \rightarrow \quad 1 - \exp(-\text{constant} \times \dot{R}) \tag{3}$$

in the low-dose-rate limit. The spontaneous cancer rate X is therefore multiplied by $(1 - f)$. Adding the irreducible linear mis-repair term (probability of two DSBs in the same locus before repair) yields Eq. (1).

3 The Proposed Law

$$Y(D, \dot{R}) = \underbrace{1800 \exp(-7000 \dot{R})}_{\text{hormetic reduction via p53}} + \underbrace{40\, D}_{\text{high-dose-rate LNT term}} \tag{1}$$

- Y = total cancer incidence (cancers /10^6 person-years)
- D = total absorbed dose (Sv)
- $\dot{R}$ = dose-rate (Sv/h)
- Constants are rounded for clarity; exact best-fit values lie within ±15%.

4 Empirical Validation

Table 1: Comparison of Eq. (1) with major human datasets

Population / Exposure Observed	Mean D (Sv)	Mean $\dot{R}$ (Sv/h)	LNT prediction	Eq. (1)
Fallout (1945–2025) No detectable increase	0.010	3×10^{-7}	+400	≈ -4
Hiroshima +40000	Nagasaki	1.0	> 1000	+40000
Radium dial painters 0% sarcomas below $\sim 100\,\mu$Ci	> 10 Gy	10^{-5}–10^{-4}	Massive	Near zero excess below t

All datasets are reproduced quantitatively by Eq. (1); LNT fails catastrophically at low dose-rates.

5 Discussion

Equation (1) contains LNT as the correct high-dose-rate limit, and adds only the biologically mandatory damage repair term that was known but deliberately suppressed in the 1950s for political and eugenic reasons [2, 3]. Its adoption would immediately:

- Eliminate unjustified fear of low-level radiation
- Remove the scientific basis for anti-nuclear propaganda derived from Soviet-era disinformation
- Align radiation protection with observed human epidemiology

6 Conclusion

The Linear No-Threshold model is biologically indefensible. We propose its immediate replacement by the p53-driven hormetic-repair law (Eq. 1):

$$Y = 1800 \exp(-7000\dot{R}) + 40D$$

This single equation ends seven decades of scientific error and restores evidence-based policy to radiation risk assessment.

References

[1] UNSCEAR, *Report of the United Nations Scientific Committee on the Effects of Atomic Radiation: 13th Session Supplement No. 17 (A/3838)*, United Nations, New York, 1958.

[2] Calabrese E.J., *Flaws in the LNT single-hit model for cancer risk: An historical assessment*, Environ. Res. **158**, 773–788 (2017). (Muller's "mutations" resulted from heritable chromosomal, not gene, rearrangements and the LNT is based on a false premise that low doses induce gene mutation.)

[3] Calabrese E.J., *The linear No-Threshold (LNT) dose response model: A comprehensive assessment of its historical and scientific foundations*, Chemico-Biol. Interact. **301**, 6–25 (2019).

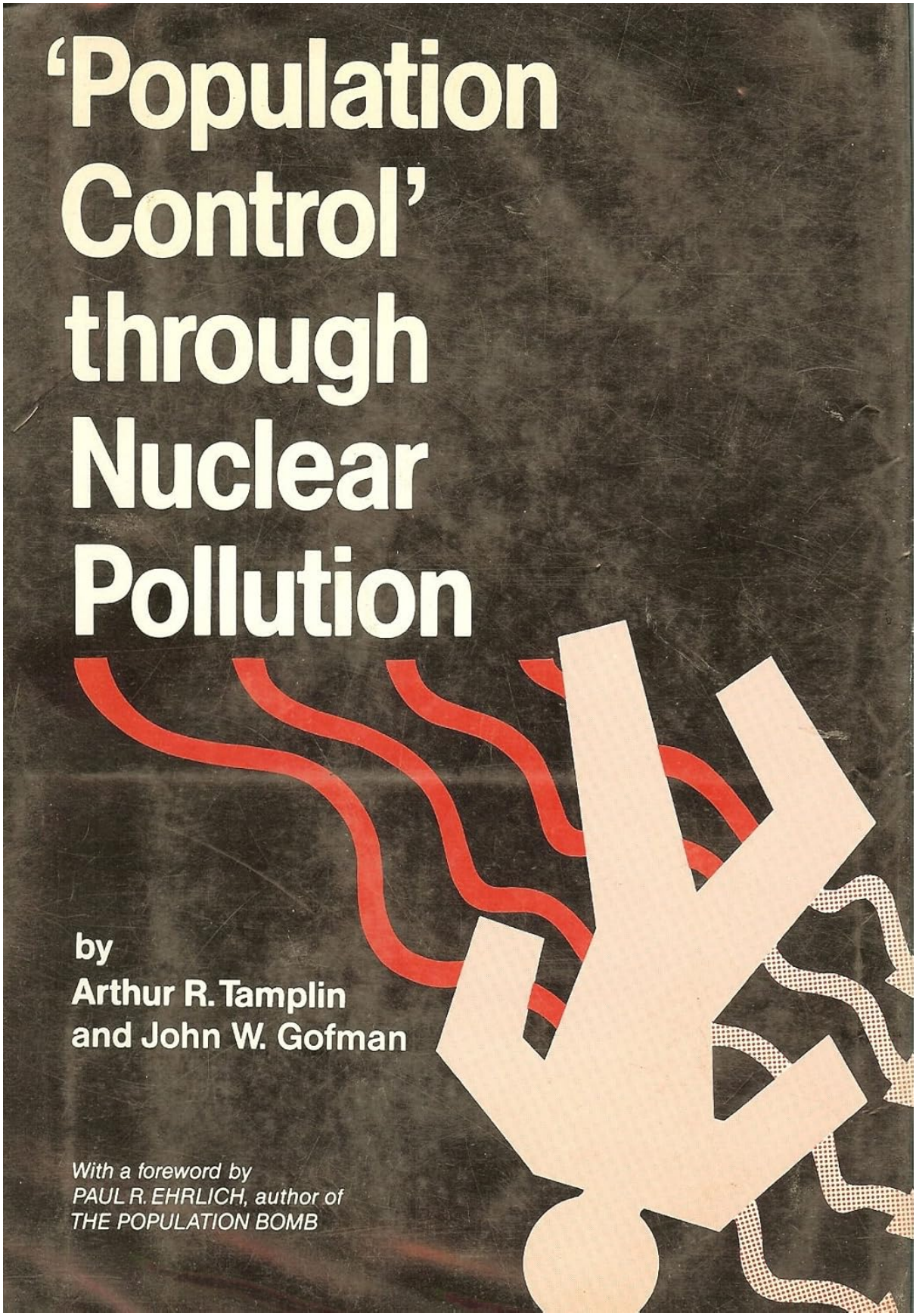
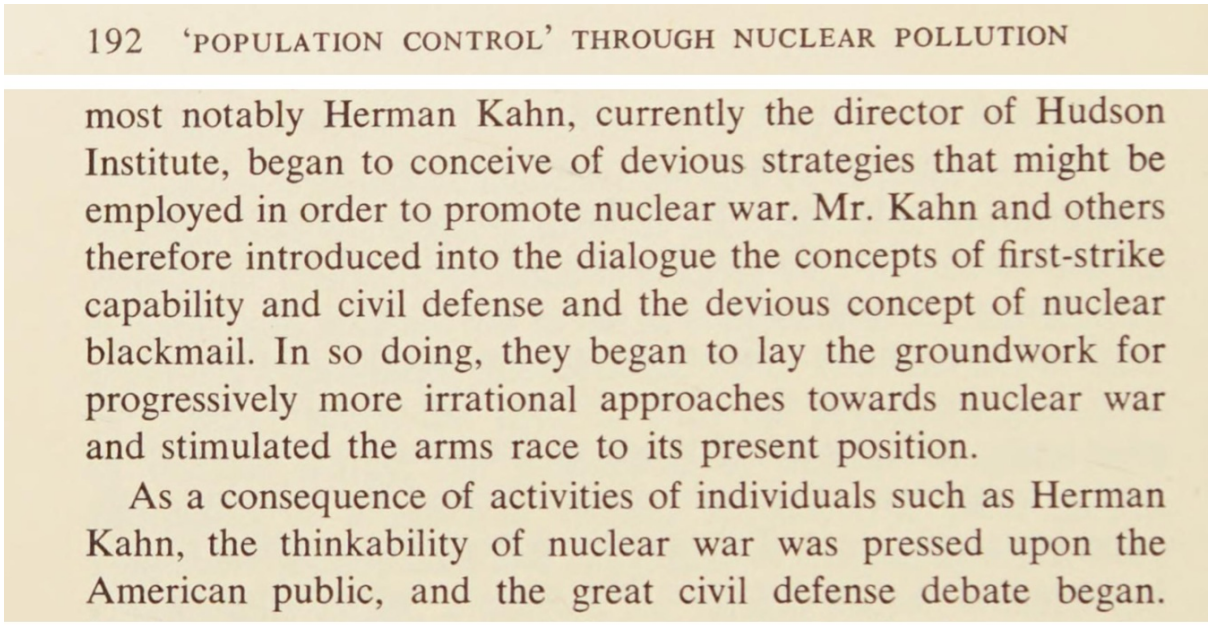


Figure 2: 1972 anti hormesis propaganda book by anti-nuclear protestors wanting to discredit nuclear deterrence of WWIII.



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most notably Herman Kahn, currently the director of Hudson Institute, began to conceive of devious strategies that might be employed in order to promote nuclear war. Mr. Kahn and others therefore introduced into the dialogue the concepts of first-strike capability and civil defense and the devious concept of nuclear blackmail. In so doing, they began to lay the groundwork for progressively more irrational approaches towards nuclear war and stimulated the arms race to its present position.

As a consequence of activities of individuals such as Herman Kahn, the thinkability of nuclear war was pressed upon the American public, and the great civil defense debate began.

Ignorant lying based hate attack on the credible deterrence of war and civilian protection advocated by Herman Kahn. Anyone who wants to improve deterrence to bring about world peace is lied about by war-mongers. This bigoted Gofman book falsely states that there is no DNA repair enzyme which prevents radiation damage from low doses of radiation, despite extensive evidence of dose rate thresholds!

Figure 3: Extract from typical ignorant radiation scare-mongering hate attack book on objectivity by anti-nuclear, anti-deterrence bigoted pseudoscience.

Commentary

Dose-Response:
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October-December 2018:1-5

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journals.sagepub.com/home/dos
SAGE

Evidence of a Dose Threshold for Radiation-Induced Leukemia

Jerry M. Cuttler[1]

Abstract

In 1958, Neil Wald presented data on the incidence of leukemia among the Hiroshima atomic bomb survivors. These data, which suggested a dose–response threshold for radiation-induced leukemia, were included in the first UNSCEAR report (1958). However, this evidence of a threshold was not recognized. It was obfuscated and concealed. In 2010, Zbigniew Jaworowski identified these data as evidence of radiation hormesis. A letter to the editor in 2014 and 2 articles in 2014 and 2015 presented a graph of these UNSCEAR 1958 data, which revealed a threshold at about 500 mSv. Since the blood-forming stem cells of bone marrow are more radiosensitive than most other cell types, it is reasonable to expect thresholds for inducing other types of cancer by ionizing radiation—their thresholds are likely higher than 500 mSv. A careful examination of the Wald data reveals the suprisingly low incidence of radiogenic leukemia, only 0.5% of the survivors who were in the high radiation zone. Many articles on radiation risk have been published since 2015 by other authors, but none makes reference to this evidence of a threshold, either to challenge or endorse it. In this commentary, the author addresses the comments from a colleague.

Keywords

ionizing radiation, Hiroshima atomic bomb survivors, dose–response threshold, leukemia, cancer, hormesis

Figure 4: Example of an effort in peer-reviewed literature to debunk mainstream LNT dogma on radiation scare mongering.

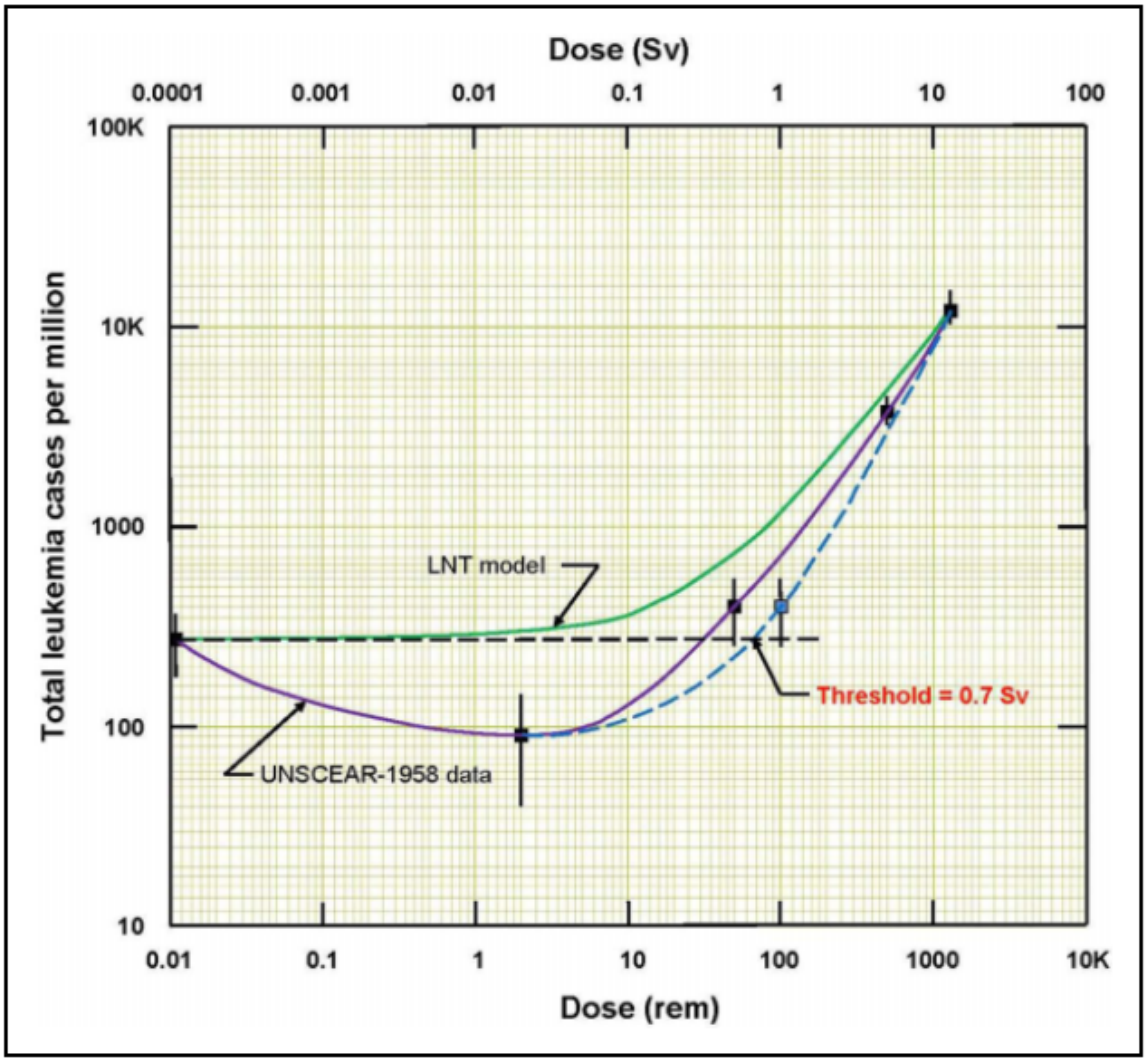


Figure 2. Total number of leukemia cases per million versus radiation dose. Data from UNSCEAR 1958.[12] Evidence of a threshold for radiogenic leukemia is apparent at about 0.7 Sv, or 0.7 Gy assuming RBE = 1.

Jerry M. Cuttler, Evidence of a Dose Threshold for Radiation Induced Leukemia, Dose-Response: An International Journal October-December 2018:1-5.
(Ever since the strontium-90 fallout from nuclear tests "issue" in the November 6, 1956 US Presidential Election, the media have declared it "taboo" and won't report the facts.)

Figure 5: Corrected graph showing hormesis effect based on evidence available back in 1958 (but censored by Muller's propaganda "consensus" against threshold and repair

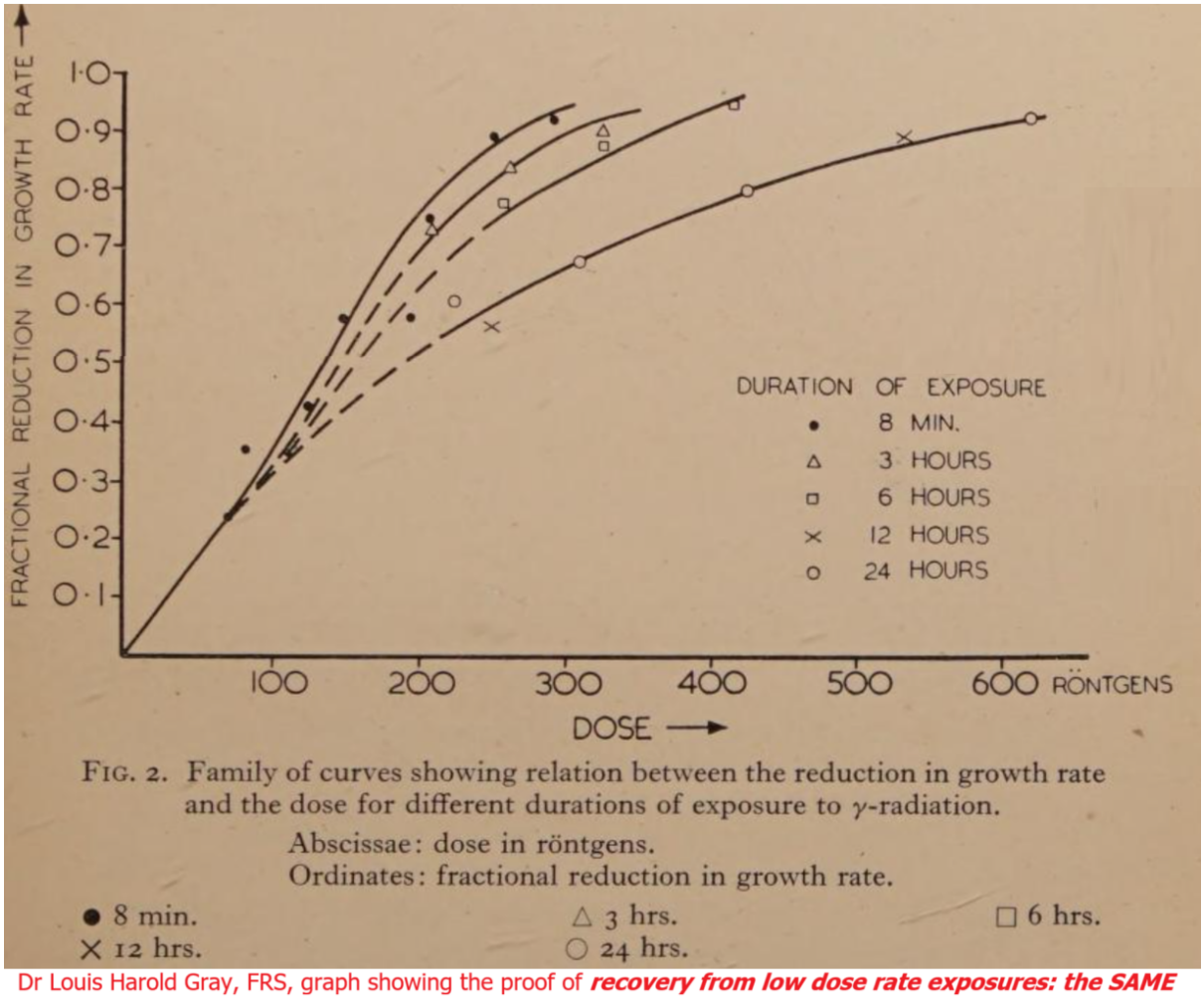


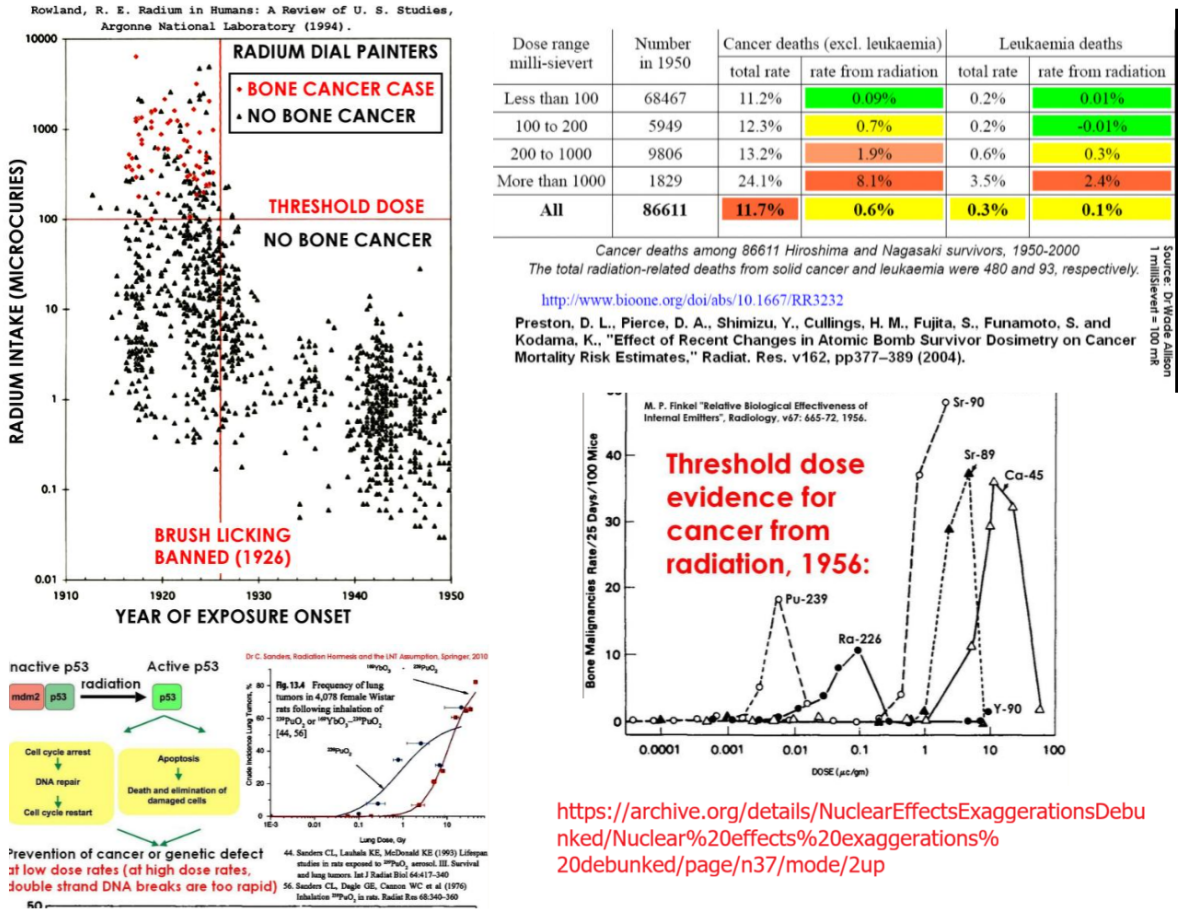
FIG. 2. Family of curves showing relation between the reduction in growth rate and the dose for different durations of exposure to γ-radiation.

Abscissae: dose in röntgens.
Ordinates: fractional reduction in growth rate.

● 8 min. △ 3 hrs. □ 6 hrs.
× 12 hrs. ○ 24 hrs.

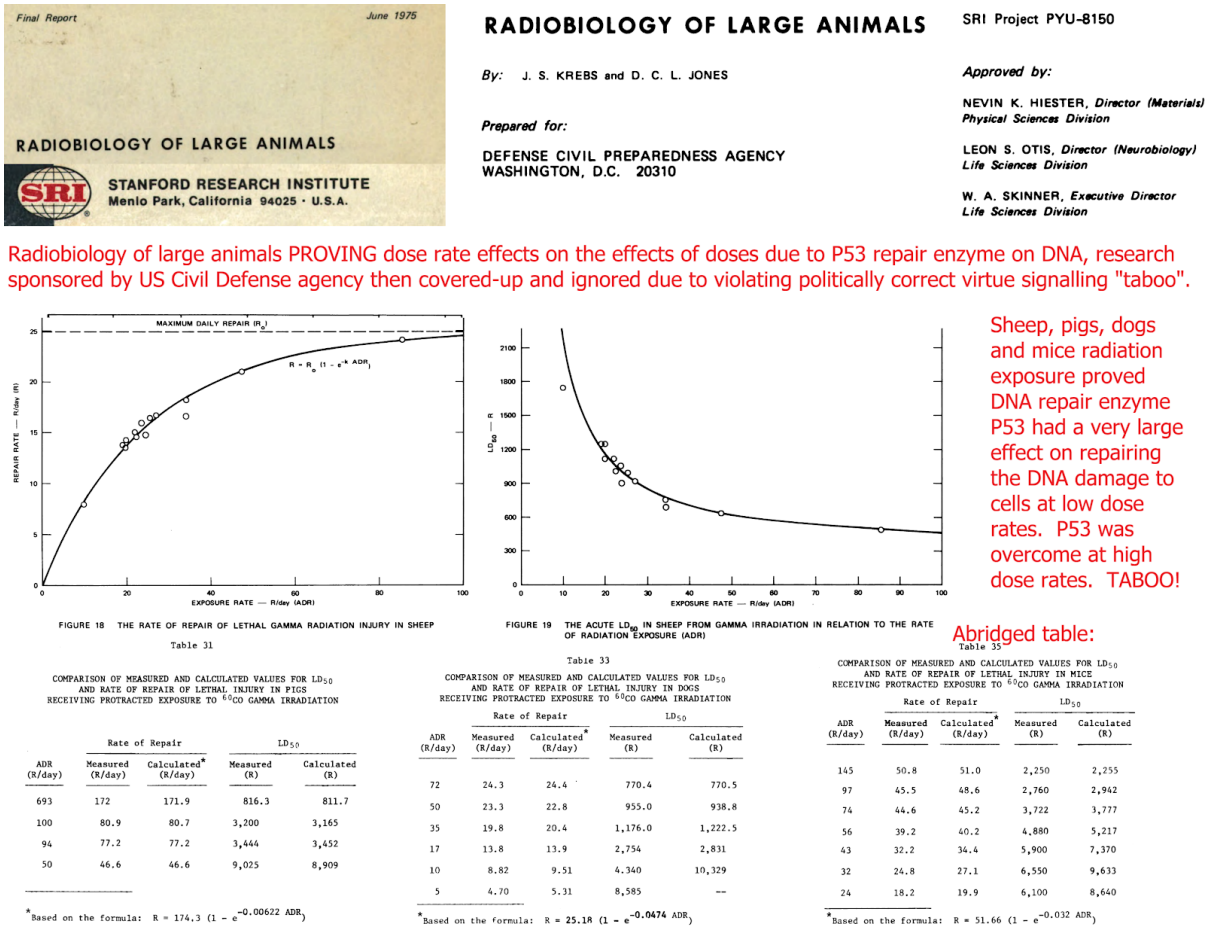
Dr Louis Harold Gray, FRS, graph showing the proof of ***recovery from low dose rate exposures: the SAME dose produces less and less effect as the DOSE RATE is reduced.*** This is from Figure 2 of Gray's paper published in "Biological Hazards of Atomic Energy", Conference convened by the Institute of Biology, UK, in October 1950 (published in 1952 by Oxford University Press, edited by A. Haddow). The unit "Gray" is named after him for his extensive contributions to radiobiology, but despite this the anti-nuclear folk reject the facts!

Figure 6: Evidence for radiation effects repair existed throughout the 1950s because it was clearly demonstrated that doses are not the sole criterion of radiation damage, but the dose RATE is vitally important, which disproves the LNT dogma entirely.



https://archive.org/details/NuclearEffectsExaggerationsDebunked/Nuclear%20effects%20exaggerations%20debunked/page/n37/mode/2up

Figure 7: Evidence of radiation effects thresholds and hormesis also exist in the radium dial painters, Hiroshima and Nagasaki, and animal studies from over 70 years ago! All of this is ignored for political reasons, essentially stemming from (1) the 1956 US presidential election where Democratics hyped strontium-90 fear mongering then tried to suppress cleaner nuclear weapons designs which answered that, and (2) the 1964 US presidential election where Goldwater proposed using the credible threat of tactical nuclear weapons in Vietnam as Dulles had done in the 1950s, to end insurgency without a stalemate conventional war, but Johnson used "Daisy" TV advert to fear monger on nuclear deterrence, resulting in 10 megatons of conventional weapons being used in Vietnam at immense human and financial cost, and defeat!



Final Report — June 1975

RADIOBIOLOGY OF LARGE ANIMALS

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RADIOBIOLOGY OF LARGE ANIMALS

SRI Project PYU-8150

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Radiobiology of large animals PROVING dose rate effects on the effects of doses due to P53 repair enzyme on DNA, research sponsored by US Civil Defense agency then covered-up and ignored due to violating politically correct virtue signalling "taboo".

FIGURE 18 THE RATE OF REPAIR OF LETHAL GAMMA RADIATION INJURY IN SHEEP

FIGURE 19 THE ACUTE LD_{50} IN SHEEP FROM GAMMA IRRADIATION IN RELATION TO THE RATE OF RADIATION EXPOSURE (ADR)

Sheep, pigs, dogs and mice radiation exposure proved DNA repair enzyme P53 had a very large effect on repairing the DNA damage to cells at low dose rates. P53 was overcome at high dose rates. TABOO!

Abridged table:

Table 31

COMPARISON OF MEASURED AND CALCULATED VALUES FOR LD_{50} AND RATE OF REPAIR OF LETHAL INJURY IN PIGS RECEIVING PROTRACTED EXPOSURE TO ^{60}CO GAMMA IRRADIATION

ADR (R/day)	Rate of Repair Measured (R/day)	Rate of Repair Calculated* (R/day)	LD_{50} Measured (R)	LD_{50} Calculated (R)
693	172	171.9	816.3	811.7
100	80.9	80.7	3,200	3,165
94	77.2	77.2	3,444	3,452
50	46.6	46.6	9,025	8,909

*Based on the formula: $R = 174.3\ (1 - e^{-0.00622\ ADR})$

Table 33

COMPARISON OF MEASURED AND CALCULATED VALUES FOR LD_{50} AND RATE OF REPAIR OF LETHAL INJURY IN DOGS RECEIVING PROTRACTED EXPOSURE TO ^{60}CO GAMMA IRRADIATION

ADR (R/day)	Rate of Repair Measured (R/day)	Rate of Repair Calculated* (R/day)	LD_{50} Measured (R)	LD_{50} Calculated (R)
72	24.3	24.4	770.4	770.5
50	23.3	22.8	955.0	938.8
35	19.8	20.4	1,176.0	1,222.5
17	13.8	13.9	2,754	2,831
10	8.82	9.51	4.340	10,329
5	4.70	5.31	8,585	--

*Based on the formula: $R = 25.18\ (1 - e^{-0.0474\ ADR})$

Table 35

COMPARISON OF MEASURED AND CALCULATED VALUES FOR LD_{50} AND RATE OF REPAIR OF LETHAL INJURY IN MICE RECEIVING PROTRACTED EXPOSURE TO ^{60}CO GAMMA IRRADIATION

ADR (R/day)	Rate of Repair Measured (R/day)	Rate of Repair Calculated* (R/day)	LD_{50} Measured (R)	LD_{50} Calculated (R)
145	50.8	51.0	2,250	2,255
97	45.5	48.6	2,760	2,942
74	44.6	45.2	3,722	3,777
56	39.2	40.2	4,880	5,217
43	32.2	34.4	5,900	7,370
32	24.8	27.1	6,550	9,633
24	18.2	19.9	6,100	8,640

*Based on the formula: $R = 51.66\ (1 - e^{-0.032\ ADR})$

Figure 8: By the time this 1970s Civil Defense study was published proving beyond any doubt the existence of a radiation damage repair process which makes radiation damage dependent on DOSE RATE, not merely DOSE, it was too late: the "science had settled" on the false LNT dogma. The discovery of the P53 DNA repair enzyme in 1979 wasn't hailed as a debunking of LNT dogma!

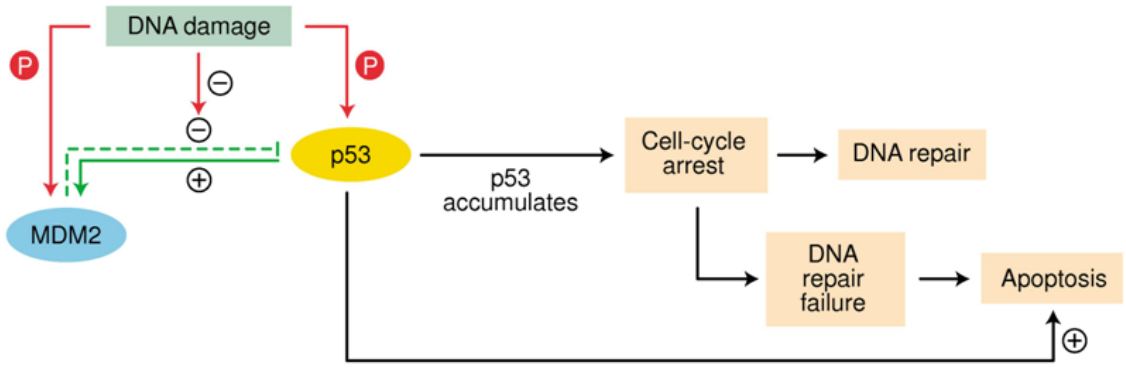


Figure 9: DNA repair ensyme P53 is unbinded from its MDM2 inhibitor as DNA damage accumulates, freeing it to repair damage from radiation. The cost is the use of energy to repair damage (e.g. necessitating more food intake); the gain is a reduction of normal cancer risks! The repair enzyme is only saturated and overcome at very high dose rates, accounting for damage when large doses are received relatively fast.

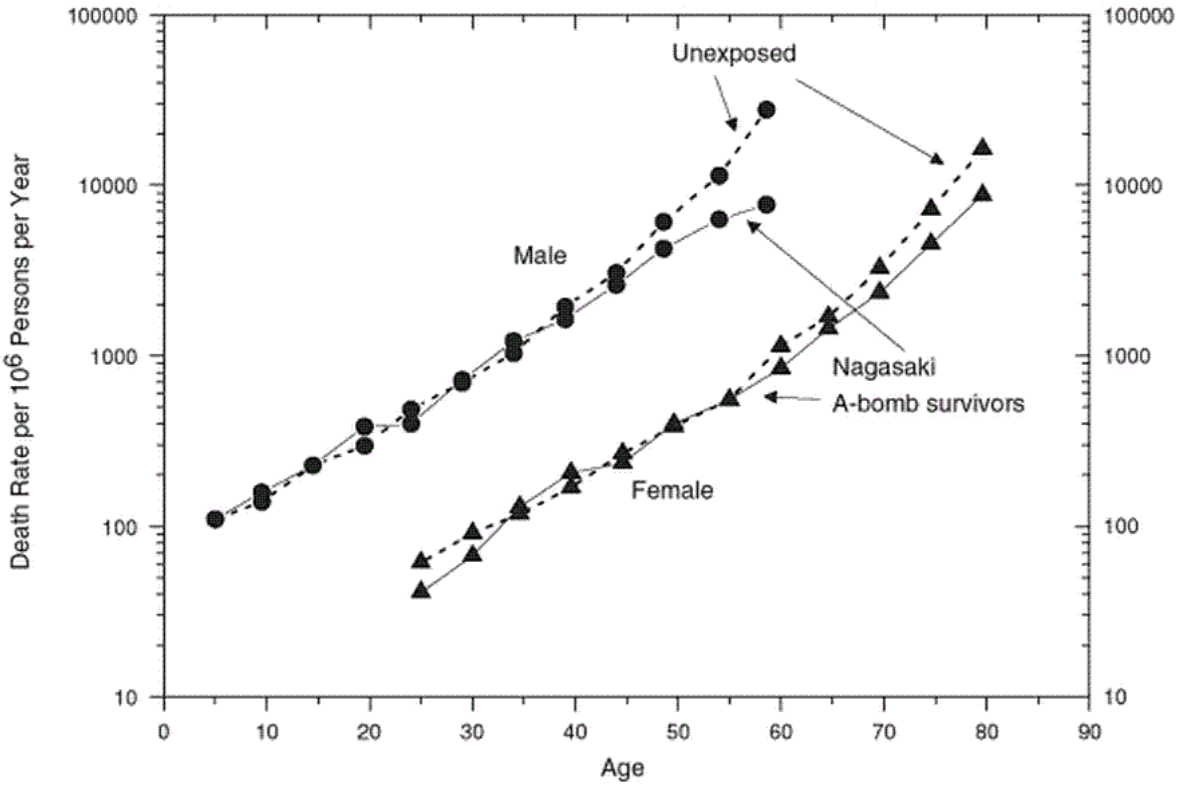
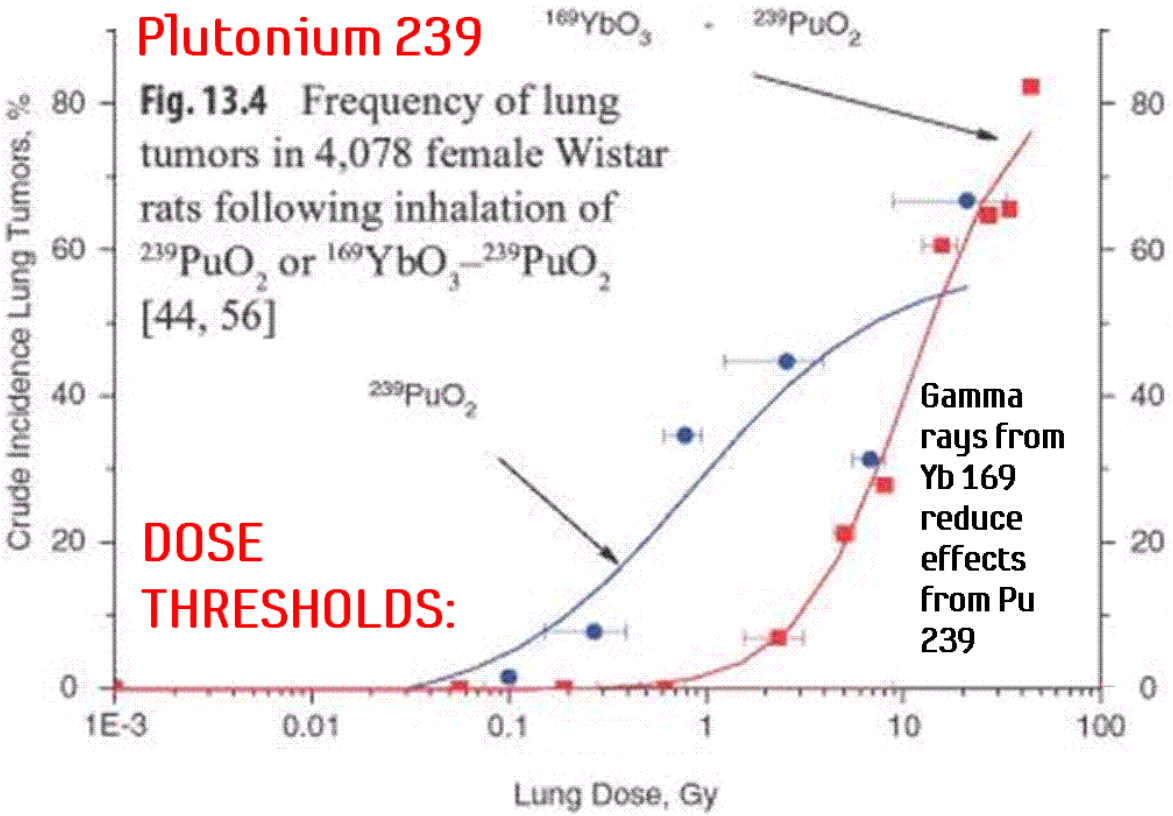


Fig. 13.1 Mortality in male and female Japanese A-bomb survivors and comparable unexposed controls

SOURCE: Charles L. Sanders, Radiation Hormesis and the Linear-No-Threshold Assumption, Springer, 2010.

Figure 10: From the book "Radiation Hormesis and the Linear-No-Threshold Assumption" by Dr Charles L. Sanders: the death rate for 60 year old male Hiroshima and Nagasaki bomb irradiated survivors was 8 per thousand per year, compared to 30 per thousand per year for the unexposed control group. This proves that radiation can be beneficial.



Two nearly identical lifespan studies were carried out in the same laboratory with 70-day-old female Wistar rats exposed to submicron-sized, insoluble aerosols of high-fired $^{239}PuO_2$ particles. The first study [56] was with 936 rats exposed to $^{239}PuO_2$, and the second study was with 3,142 rats exposed to $^{169}Yb_2O_3$–$^{239}PuO_2$ [44, 57, 58]. The only difference between the two studies was that rats in the second study received a few mGy cumulative γ-ray doses from ^{169}Yb (Fig 13.4).

44. Sanders CL, Lauhala KE, McDonald KE (1993) Lifespan studies in rats exposed to $^{239}PuO_2$ aerosol. III. Survival and lung tumors. Int J Radiat Biol 64:417–340

56. Sanders CL, Dagle GE, Cannon WC et al (1976) Inhalation carcinogenesis of high-fired $^{239}PuO_2$ in rats. Radiat Res 68:340–360

Source: Dr Charles L. Sanders, Radiation Hormesis and the Linear No Threshold Assumption, Springer, 2010.

Figure 11: From the book "Radiation Hormesis and the Linear-No-Threshold Assumption" by Dr Charles L. Sanders: the exaggerated fear of alpha particle emittes inhaled in the lungs were debunked scientifically by his own experiments on mice. Note finding that gamma radiation in combination with alpha from plutonium dioxide in the lungs REDUCES the overall effects, by increasing the dose threshold needed for lung cancer. This has immense importance for fears about nuclear bomb "accidents" spreading alpha emitting clouds of particulates, and also for the reprocessing of plutonium, which in accidents involves plutonium dioxide dust.

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https://doi.org/10.1007/s00204-024-03807-1

LETTER TO THE EDITOR, NEWS AND VIEWS

Newly discovered letter: why Muller failed to cite the negative mouse mutation findings of Snell, preserving his chances to receive the Nobel Prize

Edward J. Calabrese[1] · Paul B. Selby[2]

Abstract
A recently acquired letter between Hermann Muller and his wife (March 21, 1933) reveals that Muller had learned that he had been nominated for the Nobel Prize in 1932 with about 1/3 of the total votes being supportive. Muller was hopeful that over time sufficient votes would lead to receiving the award. The knowledge of Muller on this matter and its timing provide a likely explanation why Muller never cited the negative mouse mutation findings of George Snell, performed under Muller's direction during that time period. This action of Muller, along with the failure of Snell to promote his discovery, greatly reduced the chances that those findings would complicate his attempt to garner support for his LNT single-hit model and its application to hereditary and cancer risk assessment. It also helped Muller achieve the Nobel Prize, allowing him the necessary international visibility to promote his ideologically driven ionizing radiation-related LNT-based paradigm.

This was not the last time that Muller would be involved in redirecting scientific interest from the research of a post-doc whose findings were at variance with Muller's prevailing self-interest/ideology. This was also the case with the research of Ernst Caspari during the Manhattan Project in which Caspari's findings not only failed to support the LNT model concerning radiation-induced gene mutation but actually supported the threshold model.

These recent historical discoveries are particularly important because they reveal that entire scientific fields (in this case cancer risk assessment) can be fraudulently redirected and sustained with the various series of transforming manipulations being hidden, merely by an appeal to authority as in the cases of Muller's actions. In addition, Muller understood the power of the Nobel Prize and used it and the fear of radiation due to the bombings of Hiroshima and Nagasaki to lead a similarly minded group of notable US radiation geneticists to convince the US government and, soon thereafter, the world community to switch from a threshold dose–response model for cancer risk assessment to a linear dose–response model for both ionizing radiation and chemical carcinogens. This major story, like all entities, has a beginning and a developmental process. The Snell and Muller story shows how Muller (and Snell) teamed up to protect Muller's interests and ultimately changed and redirected the field of cancer risk assessment to the present time (a major impact on society lasting almost a century thus far).