For Physicians in a Medical Centre

June 6, 2014

Remedy for Radiation Fear
Beneficial Effects of Low Radiation

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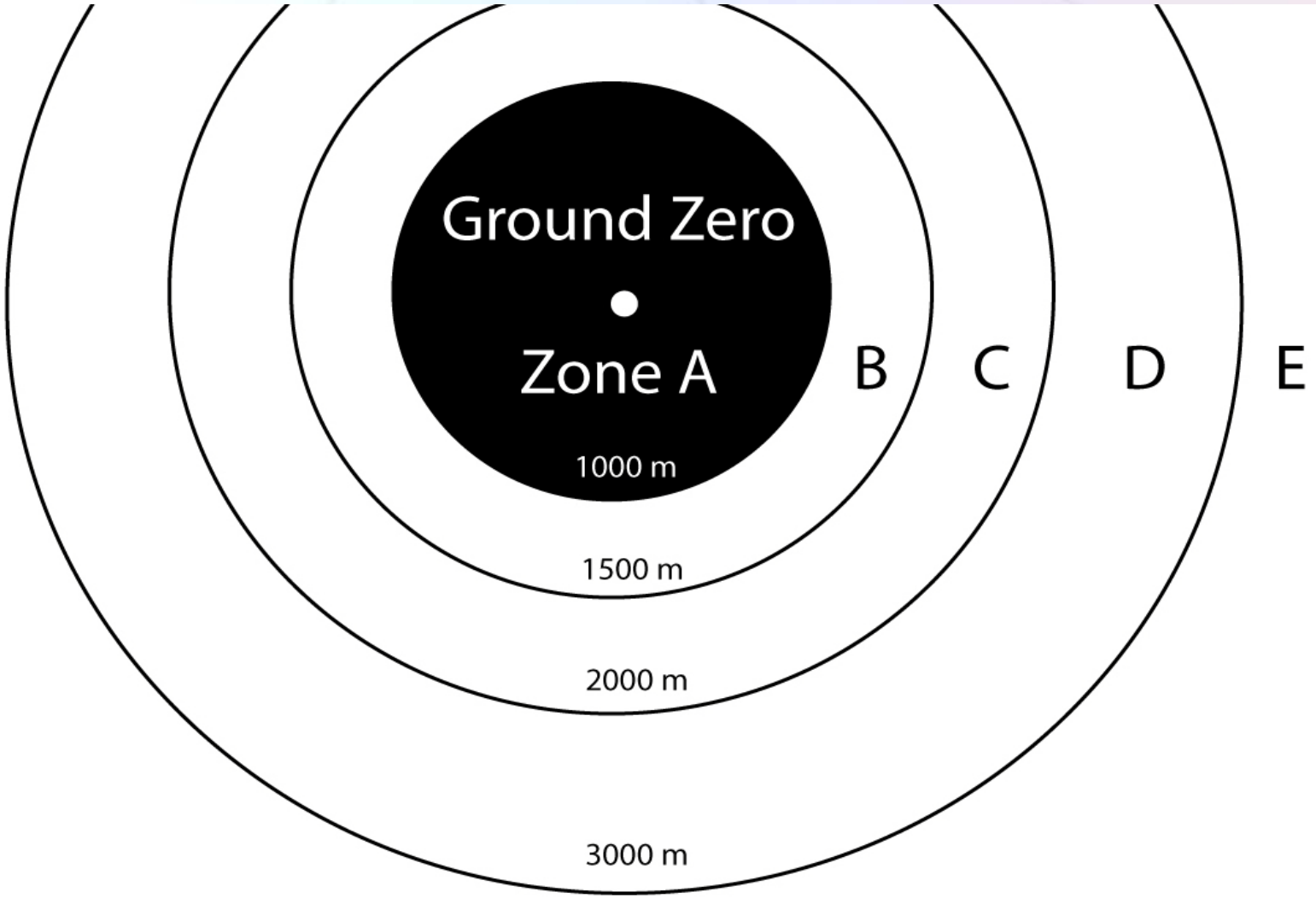
Main Points

- LNT theory is invalid, antinuclear ideology
- Fukushima evacuation 1600 premature deaths
- Precautionary action was not “conservative”
- Hiroshima leukemia incidence at 20 mSv below the controls. Threshold for harm ~500 mSv.
- Chronic radiation is harmful > 700 mGy/year; and beneficial below 700 mGy/year (70 rad/yr)
- Go back to 1934 ICRP standard of ‘tolerance dose’ of 0.2 roentgen/day or ~ 700 mGy/year
- Discard politicized science-based regulations

Hiroshima Atomic Bomb Survivor Zones

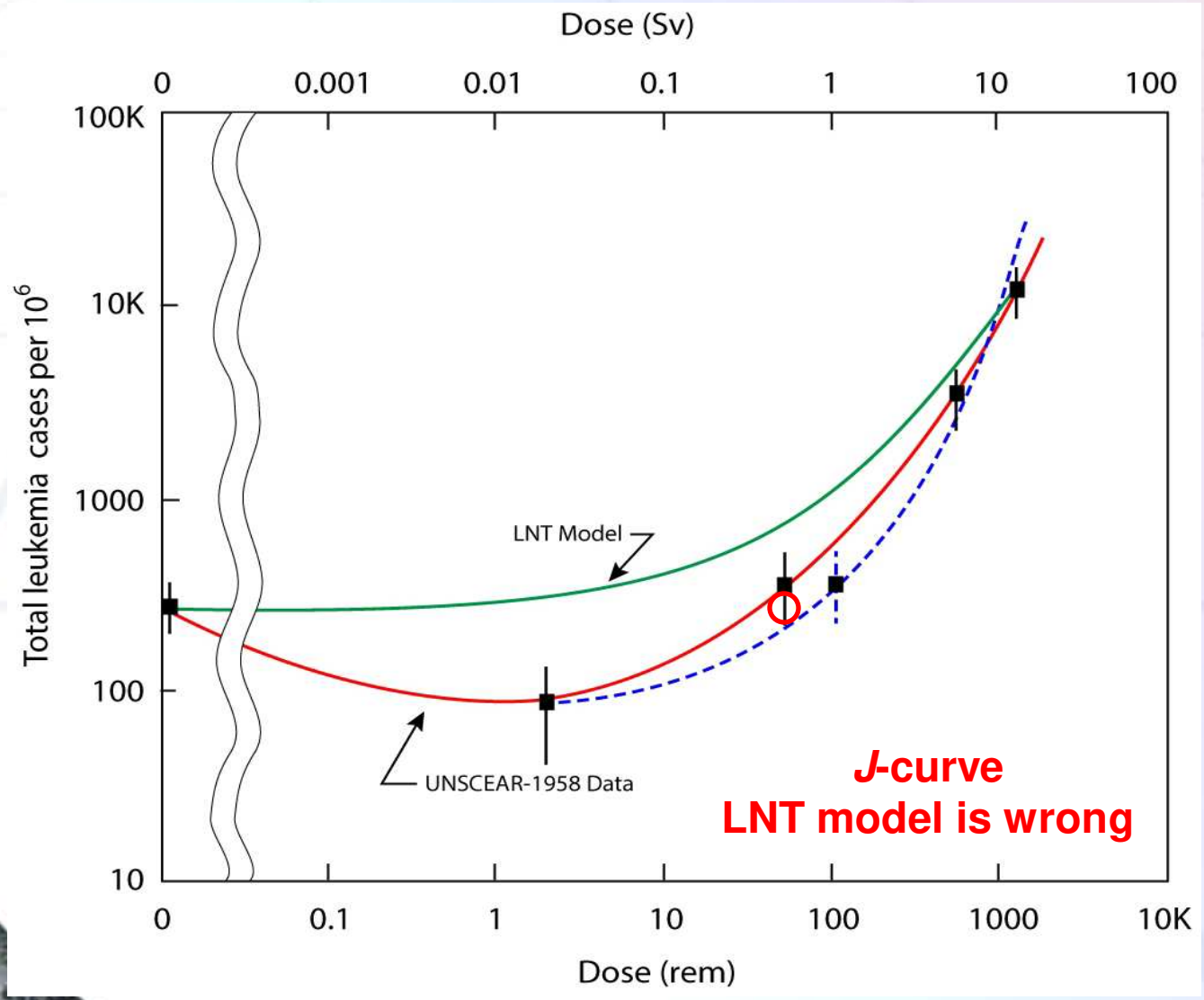


UNSCEAR 1958 Table VII
Leukemia incidence for 1950–57 after exposure at Hiroshima[a]

Zone	Distance from hypocentre (metres)	Dose (rem)	Persons exposed	L (Cases of leukemia)	$\sqrt{L}$	N^b (total cases per 10^6)
A	under 1,000	1,300	1,241	15	3.9	12,087 ± 3,143
B	1,000–1,499	500	8,810	33	5.7	3,746 ± 647
C	1,500–1,999	50[c]	20,113	8	2.8	398 ± 139
D	2,000–2,999	2	32,692	3	1.7	92 ± 52
E	over 3,000	0	32,963	9	3.0	273 ± 91

[c] It has been noted (reference 15, 16) that almost all cases of leukemia in this zone occurred in patients who had severe radiation complaints, indicating that their doses were greater than 50 rem.

Threshold at 50 rem for increased cancer



Arch Toxicol
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REVIEW ARTICLE

How the US National Academy of Sciences misled the world community on cancer risk assessment: new findings challenge historical foundations of the linear dose response

Edward J. Calabrese

Received: 24 April 2013 / Accepted: 11 July 2013

Abstract This paper extends several recent publications indicating that Hermann J. Muller: (1) Made deceptive statements during his Noble Prize Lecture on December 12, 1946, that were intended to promote the acceptance of the linear dose-response model for risk assessment for ionizing radiation and (2) that such actions of Muller were masked by a series of decisions by Muller's long-time colleague and esteemed radiation geneticist Curt Stern, affecting key publications in the mutation literature. Such actions further enhanced acceptance of the linearity dose-response model while preventing Muller's deceptions from being discovered. This paper provides documentation that Muller reinforced such practices within the scientific literature in the early 1950s, by supporting scientifically questionable actions of Stern. Detailed documentation is provided that demonstrates how these actions affected national and international risk assessment policy for ionizing radiation and chemical carcinogens via the recommendations of the National Academy of Sciences Biological Effects of Atomic Radiation committee in 1956, to adopt the linear dose-response model.

Introduction

It was recently discovered that the 1946 Nobel Prize Lecture for Biology and Medicine by Laureate Hermann J. Muller misled the audience on the nature of the dose response in the low-dose zone concerning the effects of ionizing radiation on germ-cell mutagenicity to advance an ideologically motivated risk assessment policy (Calabrese 2011a, b, 2012). Evidence to support this conclusion is found in Muller's own words from letters he sent to Professor Curt Stern of the University of Rochester, an expert in radiation genetics. Stern sent Muller a manuscript by Ernst Caspari and himself on November 6, 1946, for review as Muller was a paid consultant to the project (Calabrese 2011c). This manuscript demonstrated support for a threshold dose response, while challenging the linear dose-response single-hit mutagenicity mechanism model, based on an extensive study of ionizing radiation on mutation in the germ cells of male fruit flies. On November 12, 1946, Muller acknowledged receipt, noting that the findings strongly challenged the linearity dose-response concept and, given their importance, needed to be rep-

Science
AAAS

Artificial Transmutation of the Gene
Author(s): H. J. Muller
Reviewed work(s):
Source: *Science*, New Series, Vol. 66, No. 1699 (Jul. 22, 1927), pp. 84-87
Published by: American Association for the Advancement of Science
Stable URL: http://www.jstor.org/stable/1651551
Accessed: 07/09/2012 13:31

ARTIFICIAL TRANSMUTATION OF THE GENE

MOST modern geneticists will agree that gene mutations form the chief basis of organic evolution, and therefore of most of the complexities of living things. Unfortunately for the geneticists, however, the study of these mutations, and, through them, of the genes themselves, has heretofore been very seriously hampered by the extreme infrequency of their occurrence under ordinary conditions, and by the general unsuccessfulness of attempts to modify decidedly, and in a sure and detectable way, this sluggish "natural" mutation rate. Modification of the innate nature of organisms, for more directly utilitarian purposes, has of course been subject to these same restrictions, and the practical breeder has hence been compelled to remain content with the mere making of recombinations of the material already at hand, providentially supplemented, on rare and isolated occasions, by an unexpected mutational windfall. To these circumstances are due the wide-spread desire on the part of biologists to gain some measure of control over the hereditary changes within the genes.

In conclusion, the attention of those working along classical genetic lines may be drawn to the opportunity, afforded them by the use of X-rays, of creating in their chosen organisms a series of artificial races for use in the study of genetic and "phaenogenetic" phenomena. If, as seems likely on general considerations, the effect is common to most organisms, it should be possible to produce, "to order," enough mutations to furnish respectable genetic maps, in their selected species, and, by the use of the mapped genes, to analyze the aberrant chromosome phenomena simultaneously obtained. Similarly, for the practical breeder, it is hoped that the method will ultimately prove useful. The time is not ripe to discuss here such possibilities with reference to the human species.

The writer takes pleasure in acknowledging his sincere appreciation of the cooperation of Dr. Dalton Richardson, Roentgenologist, of Austin, Texas, in the work of administering the X-ray treatments.

H. J. MULLER

UNIVERSITY OF TEXAS

http://www.genetics.org/content/33/1/75.full.pdf+html?sid=cb861a39-fb63-48c4-bcbe-2433bb5c8d6a

THE INFLUENCE OF CHRONIC IRRADIATION WITH GAMMA-RAYS AT LOW DOSAGES ON THE MUTATION RATE IN *DROSOPHILA MELANOGASTER*[1]

ERNST CASPARI AND CURT STERN[2]
University of Rochester, Rochester, N. Y.

Received November 25, 1947

THE influence of radiation of short wave length on the mutation rate in Drosophila has been measured repeatedly since the pioneer work of MULLER (1927). As a general rule it was found that the mutation rate is directly proportional to the dose of radiation, as expressed in r units. This linear proportionality between radiation dose and mutation rate applies to all dosages of X-rays tested to the present time except for the highest dosages, in which a "saturation effect" comes into play. At the low end of the curve, SPENCER and STERN (1948) found the proportionality maintained down to a dose of 25 r.

SUMMARY

1. The rate of lethal sex-linked mutations in Drosophila exposed to gamma-rays of 2.5 r units per day through 21 days (total 52.5 r) was determined.

2. In a total material of 108,215 chromosomes tested, no significant difference between experimentals and controls was found.

From germ cell mutations to somatic cell mutations to risk of cancer

Science
AAAS

Genetic Effects of Atomic Radiation
Source: *Science*, New Series, Vol. 123, No. 3209 (Jun. 29, 1956), pp. 1157-1164
Published by: American Association for the Advancement of Science
Stable URL: http://www.jstor.org/stable/1749925
Accessed: 04/04/2014 20:33

Genetic Effects of Atomic Radiation

The coming of the atomic age has brought both hopes and fears. The hopes center largely around two aspects: the future availability of vast resources of energy, and the benefits to be gained in biology, medicine, agriculture, and other fields through application of the experimental techniques of atomic physics (isotopes, beams of high-energy particles, and so forth).

Gains in both of these areas can be of great benefit to mankind. Advances in medicine and agriculture are obviously desirable. The wide availability of power can also be of great benefit, if we use this power wisely. For not only should there be enough power to meet the more obvious and mechanical demands, there should be enough to affect society in much more far-reaching and advantageous ways, so as to reduce world tensions by raising the economic standards of areas with more limited resources.

On the other hand, the atomic age also brings fears. The major fear is that of an unspeakably devastating atomic war. Along with this is another fear, minor as compared with total destruction, but nevertheless with grave implications. When atomic bombs are tested, radioactive material is formed and released into the atmosphere, to be carried by the winds and eventually to settle down at distances which may be very great. Since it does finally settle down it has been aptly named "fallout."

There has been much concern, and a good deal of rather loose public debate, about this fallout and its possible dangers.

Are we harming ourselves; and are there genetic effects which will harm our children, and their descendants, through this radioactive dust that has been settling down on all of us? Are things going to be still worse when presently we have a lot of atomic power plants, more laboratories experimenting with atomic fission and fusion, and perhaps more and bigger weapons testing? Are there similar risks, due to other sources of radiation, but brought to our attention by these atomic risks?

What Complications Are Met in Reaching a Decision?

Now it is a plain fact, which will be explained in some detail later in this report, that radiations [Throughout this report, the word *radiation* is not used in its broadest sense, but refers primarily to gamma rays and/or x-rays and sometimes to other sorts of radiations.] penetrating the bodies of human beings are genetically undesirable. Even very small amounts of radiation unquestionably have the power to injure the hereditary materials. Ought we take steps at once to reduce, or at least to limit, the amount of radiation which people receive?

There are two major difficulties that make it very hard to decide what is

This article is the major portion of the text of the summary report of the Committee on Genetic Effects of Atomic Radiation. It is one of six reports prepared for the Study of the Biological Effects of Atomic Radiation by the National Academy of Sciences. The other five summary reports will be published in subsequent issues of *Science*. The members of the committee are Warren Weaver, Rockefeller Foundation, *chairman*; George W. Beadle, California Institute of Technology; James F. Crow, University of Wisconsin; M. Demerec, Carnegie Institution of Washington; G. Failla, Columbia University; H. Bentley Glass, Johns Hopkins University; Alexander Hollaender, Oak Ridge National Laboratory; Berwind P. Kaufmann, Carnegie Institution of Washington; C. C. Little, Roscoe B. Jackson Memorial Laboratory; H. J. Muller, Indiana University; James V. Neel, University of Michigan; W. L. Russell, Oak Ridge National Laboratory; T. M. Sonneborn, Indiana University; A. H. Sturtevant, California Institute of Technology; Shields Warren, New England Deaconess Hospital; and Sewall Wright, University of Wisconsin. The following changes have been made in the text: The "Foreword," the section entitled "Radioactive material and radiations," and the section entitled "Some basic facts about genetics" have been omitted. References to these sections in the remainder of the text have also been omitted (omissions are marked by ellipsis). A few additions, including a definition of radiation taken from one of the omitted sections and references from one section to another by title instead of number, have been made (additions are marked by square brackets). In addition, all units of measurement have been spelled out. The full texts of the summary reports are available from the National Academy of Sciences, and the texts of the technical reports will be published in monograph form by the NAS.

RADIATION RESEARCH 171, 1–8 (2009)
0033-7587/09 $15.00

Reduction in Mutation Frequency by Very Low-Dose Gamma Irradiation of *Drosophila melanogaster* Germ Cells

Keiji Ogura,[a,b,1] Junji Magae,[a,b] Yasushi Kawakami[b] and Takao Koana[a,2]

[a] *Radiation Safety Research Center, Central Research Institute of Electric Power Industry, Iwado-Kita 2-11-1, Komae, Tokyo 201-8511, Japan; and*
[b] *Biotechnology Department, Institute of Research and Innovation, Takada 1201, Kashiwa, Chiba 277-0861, Japan*

Ogura, K., Magae, J., Kawakami, Y. and Koana, T. Reduction in Mutation Frequency by Very Low-Dose Gamma Irradiation of *Drosophila melanogaster* Germ Cells. *Radiat. Res.* 171, 1–8 (2009).

To determine whether the linear no-threshold (LNT) model for stochastic effects of ionizing radiation is applicable to very low-dose radiation at a low dose rate, we irradiated immature male germ cells of the fruit fly, *Drosophila melanogaster*, with several doses of ^{60}Co γ rays at a dose rate of 22.4 mGy/h. Thereafter, we performed the sex-linked recessive lethal mutation assay by mating the irradiated males with nonirradiated females. The mutation frequency in the group irradiated with 500 μGy was found to be significantly lower than that in the control group ($P < 0.01$), whereas in the group subjected to 10 Gy irradiation, the mutation frequency was significantly higher than that in the control group ($P < 0.03$). A J-shaped dose–response relationship was evident. Molecular experiments using DNA microarray and quantitative reverse transcription PCR indicated that several genes known to be expressed in response to heat or chemical stress and *grim*, a positive regulator of apoptosis, were up-regulated immediately after irradiation with 500 μGy. The involvement of an apoptosis function in the non-linear dose–response relationship was suggested.

for the estimation of cancer risks, because cancer risk was considered to be proportional to mutation rate, and the mutation rate was found to be proportional to radiation dose in high dose ranges. Therefore, cancer risk was considered to be proportional to radiation dose at high doses.

Much later, the mutation frequency in murine spermatogonia was found to be dependent not only on the total radiation dose but also on the dose rate (*3*). It was inferred that the repair function of irradiated cells was sufficient with chronic irradiation and that the cells are able to repair radiation-induced DNA damage without errors. However, doses exceeding the repair capacity would cause incomplete repair and/or misrepair, which would occasionally result in mutations. Although Russell *et al.* (*3*) indicated that a low dose rate resulted in a low inclination of the dose–response curve, a threshold dose was not found at any dose rate.

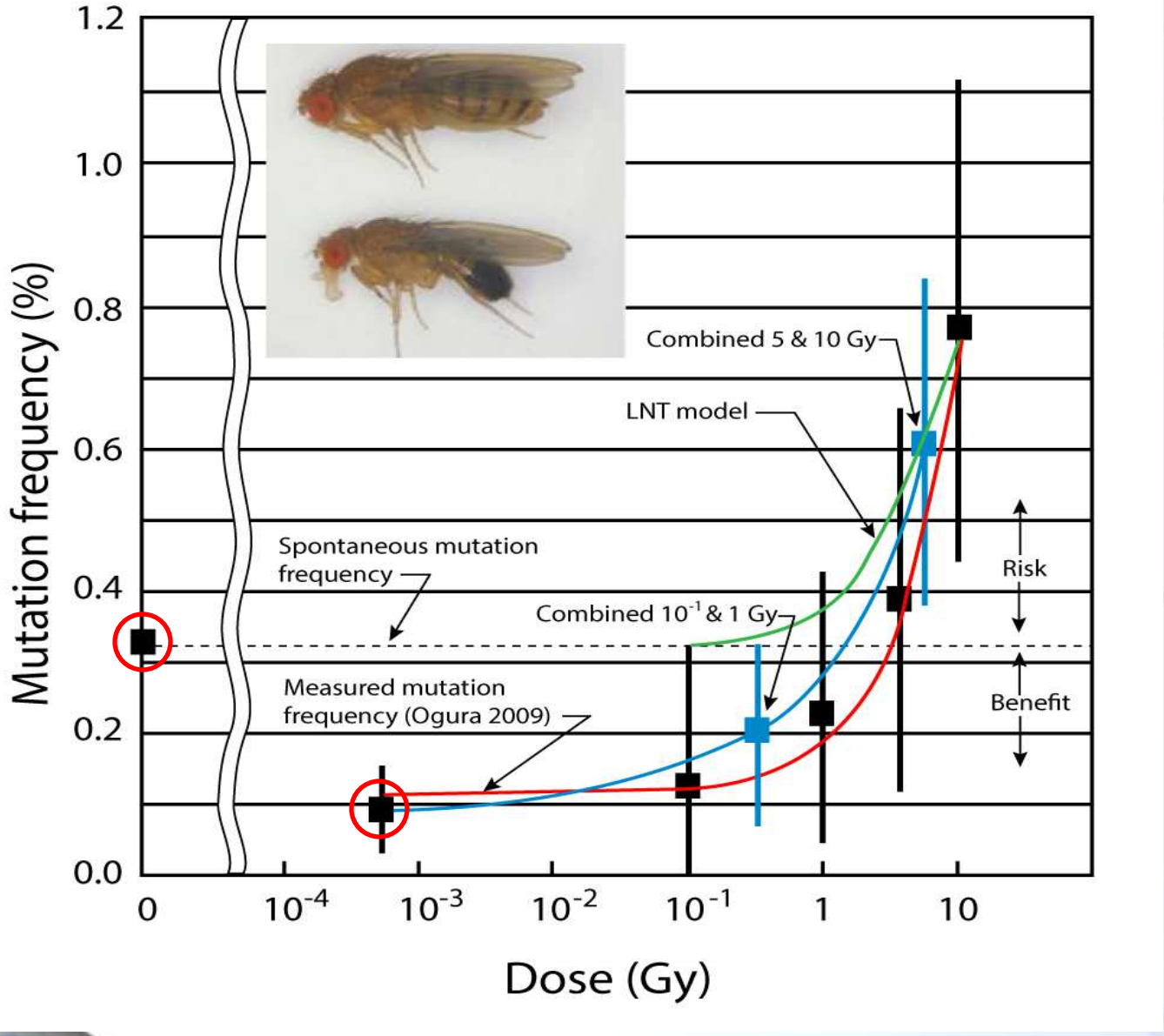
In contrast, we reported previously that in the somatic mutation assay using *Drosophila*, there was a threshold dose at approximately 1 Gy and that a mutation in the DNA repair function decreased the threshold value (*4*). The existence of a threshold, as determined in the sex-linked recessive lethal assay, using repair-proficient immature germ cells (spermatogonia and spermatocytes), was also indicated, and it was inferred that the excision repair function was

Binomial statistics applied to fruit fly mutation data measured by Ogura et al. 2009

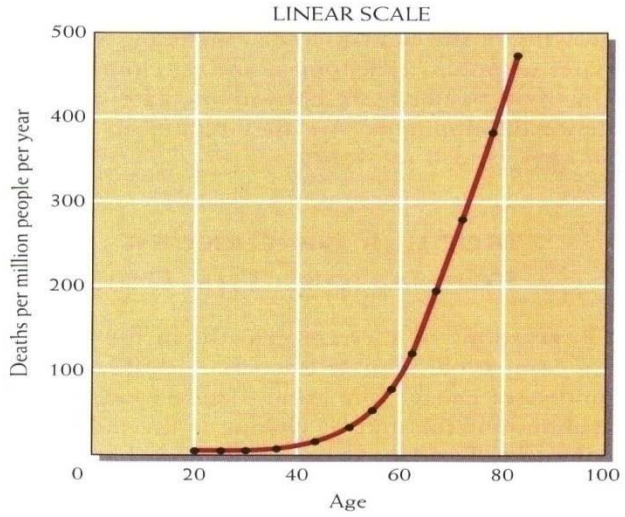
Dose Gy	Number Lethals y	Chromo somes n	Mutat'n Freq. p = y/n	q = 1–p	Var σ^2 n•p•q	Std. dev. σ	2σ/n %	p + 2σ/n %	p – 2σ/n %
0.0005	9	10,500	0.0009	0.9991	9.441	3.07	0.06	0.15	0.03
0.1	2	1507	0.0013	0.9987	1.957	1.399	0.186	0.32	-0.06
1	6	2662	0.0023	0.9977	6.109	2.472	0.186	0.42	0.04
5	8	2055	0.0039	0.9961	7.983	2.825	0.27	0.66	0.12
10	21	2730	0.0077	0.9923	20.86	4.567	0.33	1.10	0.44
0.3	8	4169	0.0019	0.9981	7.906	2.81	0.13	0.32	0.06
7	29	4785	0.0061	0.9939	29.01	5.386	0.225	0.84	0.38

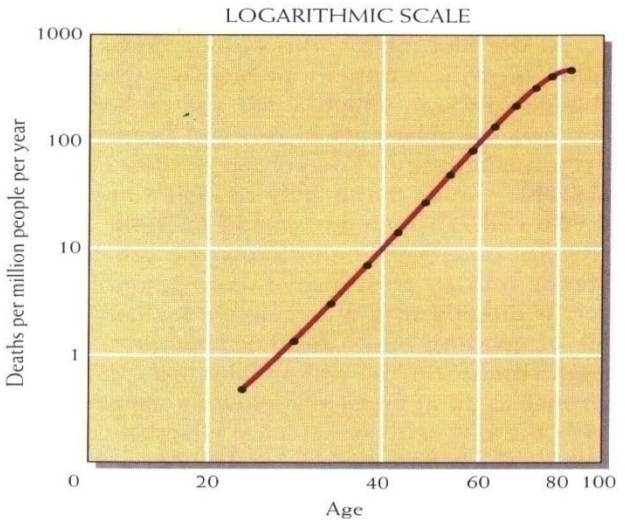
Mutation frequency for controls = 0.0032

Germ cell mutation frequency - fruit flies, 22.4 mGy/h



Cancer death rate rises exponentially with age





Actual annual U.S. death rate from colon cancer in relation to age, 1986.

Main cancer cause is <u>spontaneous</u> DNA damage due to free radicals, reactive oxygen species (ROS), thermal effects

- **Mutations add up**
- **Defences get old**

Mortality of 1338 British Radiologists 1897-1976

Cause of death	Observed (O) and expected (E) numbers of deaths					
	Entry prior to 1921			Entry after 1920		
	O	E	O/E	O	E	O/E
All causes	319	(1) 334.42	0.95	411	541.77	0.76***
		(2) 308.03	1.04		461.14	0.89*
		(3) 327.97	0.97		469.97	0.87**
All neoplasms	62	(1) 49.11	1.26*	72	114.93	0.63***
		(2) 43.07	1.44**		91.07	0.79*
		(3) 35.39	1.75***		68.65	1.05
Other causes	257†	(1) 285.31	0.90*	339†	426.84	0.79***
		(2) 264.96	0.97		370.07	0.92
		(3) 292.58	0.88*		401.32	0.84**

(1) Based on rates for all men in England and Wales.
(2) Based on rates for social class 1.
(3) Based on rates for medical practitioners.
† includes one death with unknown cause.

*P < 0.05
**P < 0.01
***P < 0.001
} One sided in direction of difference.

Smith and Doll 1981, Br J Radiology 54(639) 187-194

From 1920 to 1955
Radiologists used safe "tolerance" dose rate

The level was **0.2 roentgen (R) per day** in 1931, based on applying a factor of 1/100 to the commonly accepted average erythema dose of 600 R, to be spread over one month (30 days).

In September 1924, at a meeting of the American Roentgen Ray Society, Arthur Mutscheller was the first person to recommend this "tolerance" dose rate for radiation workers, a dose rate that could be tolerated indefinitely.

This level is equivalent to **680 mSv/year**.

Lauriston Taylor† in 1980*

- The founder and former president of the NCRPM denounced using the LNT model to calculate annual deaths from x-ray diagnoses:
- **"These are deeply immoral uses of our scientific heritage."**
- **"No one has been identifiably injured by radiation while working within the first numerical standards set by the ICRP in 1934."**

RADIATION RESEARCH 124, 242–245 (1990)

COMMENTARY

Spontaneous DNA Damage and Its Significance for the "Negligible Dose" Controversy in Radiation Protection

DANIEL BILLEN[1]

Oak Ridge Associated Universities, Medical Sciences Division, P.O. Box 117, Oak Ridge, Tennessee 37831-0117

BILLEN, D. Spontaneous DNA Damage and Its Significance for the "Negligible Dose" Controversy in Radiation Protection. *Radiat. Res.* 124, 242–245 (1990).

One of the crucial problems in radiation protection is the reality of the negligible dose or *de minimus* concept (*1–4*). This issue of a "practical zero" and its resolution is central to our understanding of the controversy concerning the existence of a "safe" dose in radiological health. However, for very low levels of environmental mutagens and carcinogens including low doses of low-LET radiations (less than 1 cGy or 1 rad), spontaneous or endogenous DNA damage may have an increasing impact on the biological consequences of the induced cellular response. It is this issue that is addressed in this communication.

The following discussion is intentionally limited to a comparison of low-LET radiation since its effects are due primarily to indirect damage in cellular DNA brought about

modification events occur per hour in each mammalian cell due to intrinsic causes.

The current radiation literature will be interpreted to show that ~100 (or fewer) measurable DNA alterations occur per centigray of low-LET radiation per mammalian cell. Therefore every *hour* human and other mammalian cells undergo at least 50–100 times as much spontaneous or natural DNA damage as would result from exposure to 1 cGy of ionizing radiation. Since background radiation is usually less than 100–200 mrem (1–2 mSv)/y, it can be concluded, as discussed by Muller and Mott-Smith (*15*), that spontaneous DNA damage is due primarily to causes other than background radiation.

"INTRINSIC" OR "SPONTANEOUS" DNA DAMAGE

DNA is not as structurally stable as once thought. On the contrary, there appears to be a natural background of chemical and physical lesions introduced into cellular DNA by thermal as well as oxidative insult. In addition, in the

Daniel Billen in Radiation Research 1990

DNA is not as structurally stable as once thought

Natural background of lesions: thermal and oxidative insult

"Cells have mechanisms to bypass or repair these lesions"

Spontaneous rate = 2 x 10^5 DNA alterations/cell/day

Radiation-induced: 10-100 DNA alterations per cell/**cGy**

1 mGy/year radiation < 3 x 10^{-2} DNA alteration/cell/day

This is > **6 million times** lower than spontaneous rate!!!

So radiation is not a significant cause of cancer.

We've known this for more than 20 years!

Therapeutic Nuclear Medicine
Springer 2012 ISBN 978-3-540-36718-5

Hormesis by Low Dose Radiation Effects: Low-Dose Cancer Risk Modeling Must Recognize Up-Regulation of Protection

Ludwig E. Feinendegen, Myron Pollycove, and Ronald D. Neumann

Contents

Abstract

Ionizing radiation primarily perturbs the basic molecular level proportional to dose, with potential damage propagation to higher levels: cells, tissues, organs, and whole body. There are three types of defenses against damage propagation. These operate deterministically and below a certain impact threshold there is no propagation. Physical static defenses precede metabolic-dynamic defenses acting immediately: scavenging of toxins;—molecular repair, especially of DNA;—removal of damaged cells either by apoptosis, necrosis, phagocytosis, cell differentiation-senescence, or by immune responses,—followed by replacement of lost elements. Another metabolic-dynamic defense arises delayed by up-regulating immediately operating defense mechanisms. Some of these adaptive protections may last beyond a year and all create temporary protection against renewed potentially toxic impacts also from nonradiogenic endogenous sources. Adaptive protections have a maximum after single tissue absorbed doses around 100–200 mSv and

Ludwig Feinendegen et al.

- Studies ignore spontaneous (endogenous) DNA damage rate
- Endogenous rate very high compared with radiation-induced rate:
  - Endogenous DNA single-strand breaks > **10^6** x SSBs due to bkgnd radiation
  - Endogenous DNA double-strand breaks > **10^3** x DSBs from bkgnd radiation
- Low-dose radiation up-regulates adaptive protection systems
- Static defences act immediately to remove toxins, repair molecules (DNA), remove/replace damaged cells and tissue
- Followed by dynamic defence of up-regulated adaptive systems that may last more than a year and protect against renewed toxic impacts from radiation and non-radiation, endogenous sources
- Adaptive protections have a maximum after 150 mGy acute dose
  - Chronic or repetitive radiation initiates protection at lower level
  - Adaptive protections reduce risks = less cancer, life extension

Beneficial Effects of Low Radiation

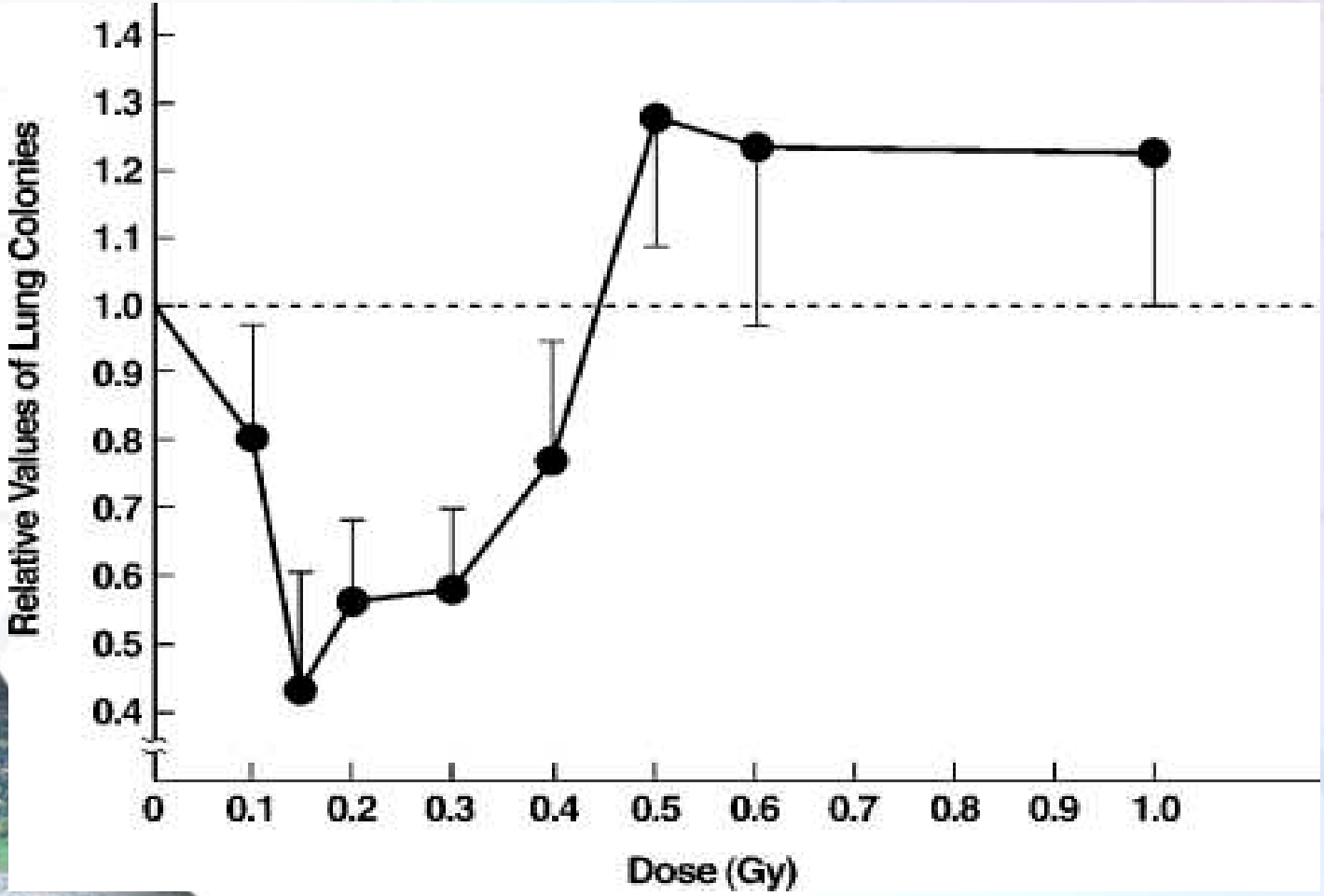
Medical practitioners used radiation for decades to up-regulate adaptive protection systems:

- Eliminate tumors or slow tumor growth
- Accelerate healing of wounds
- Stop infections: gas gangrene, carbuncles and furuncles (boils), sinus, inner ear, etc.
- Treat arthritis, other inflammatory conditions
- Treat swollen lymph glands
- Cure pneumonia, and

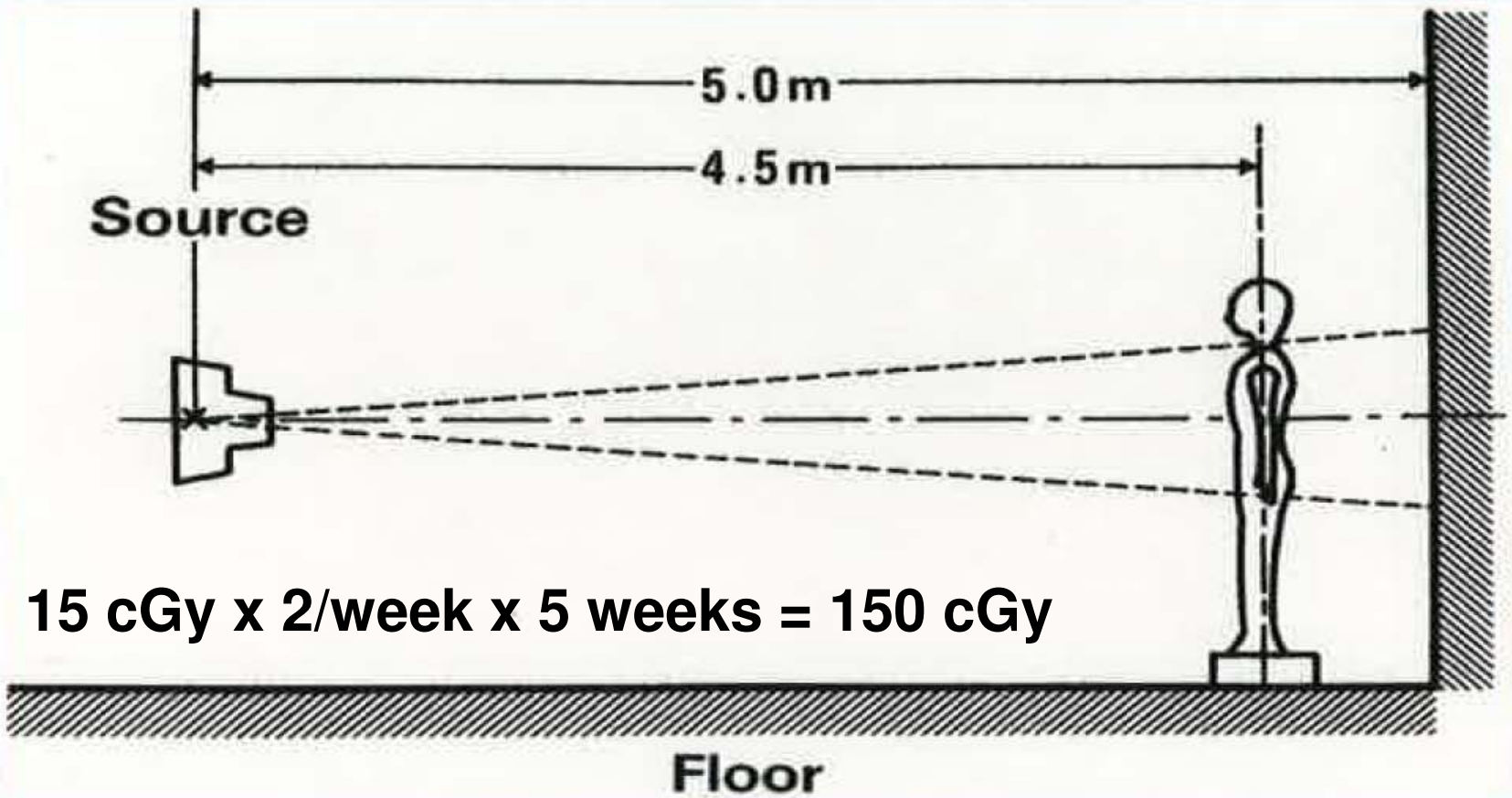
  no apparent increases of cancer incidence

Results of one of Sakamoto's studies:

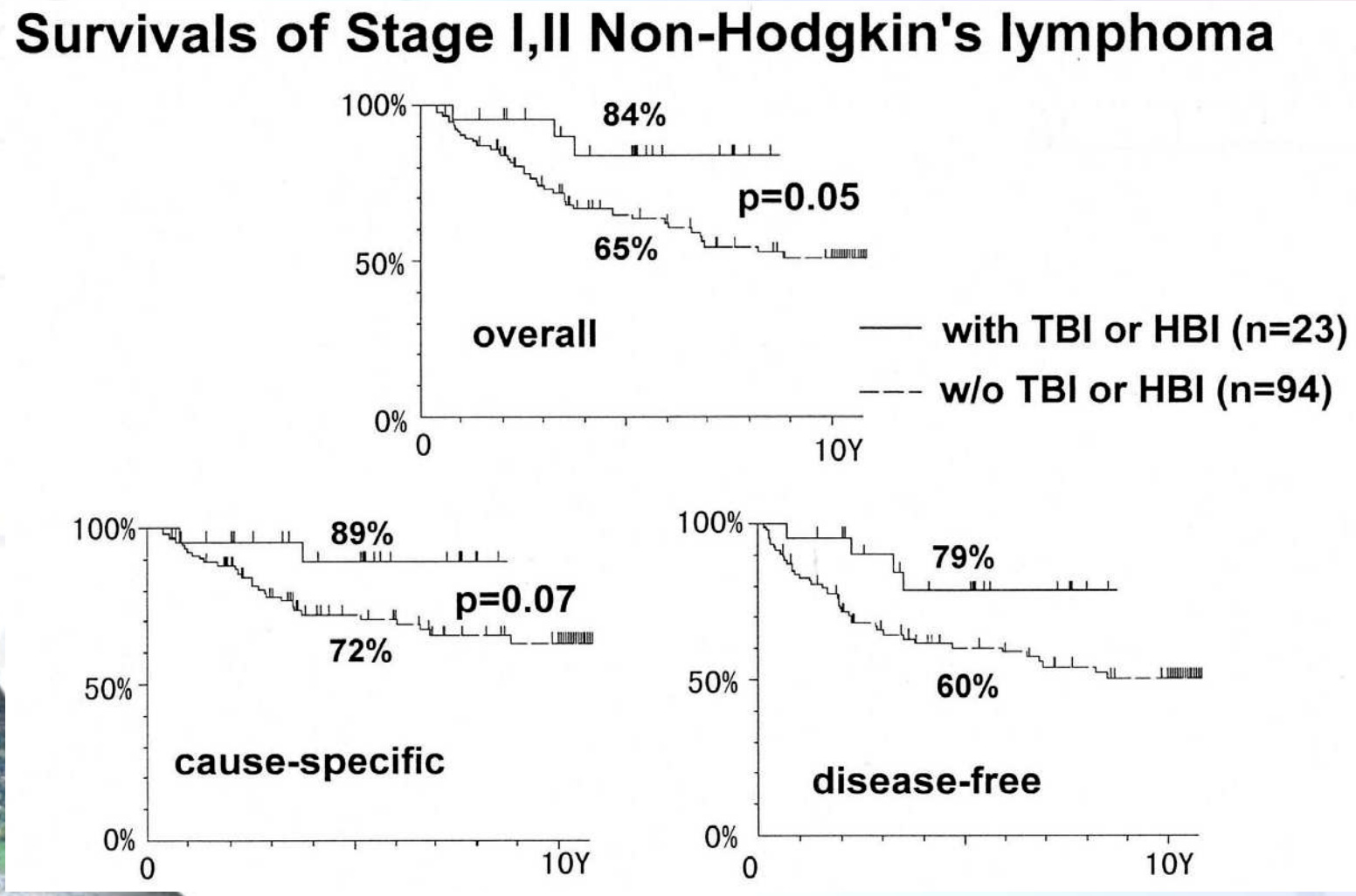
Spontaneous Lung Metastasis vs. TB Dose



Source – Patient Schema for HB LDR



HBI or TBI for Non-Hodgkin's Lymphoma

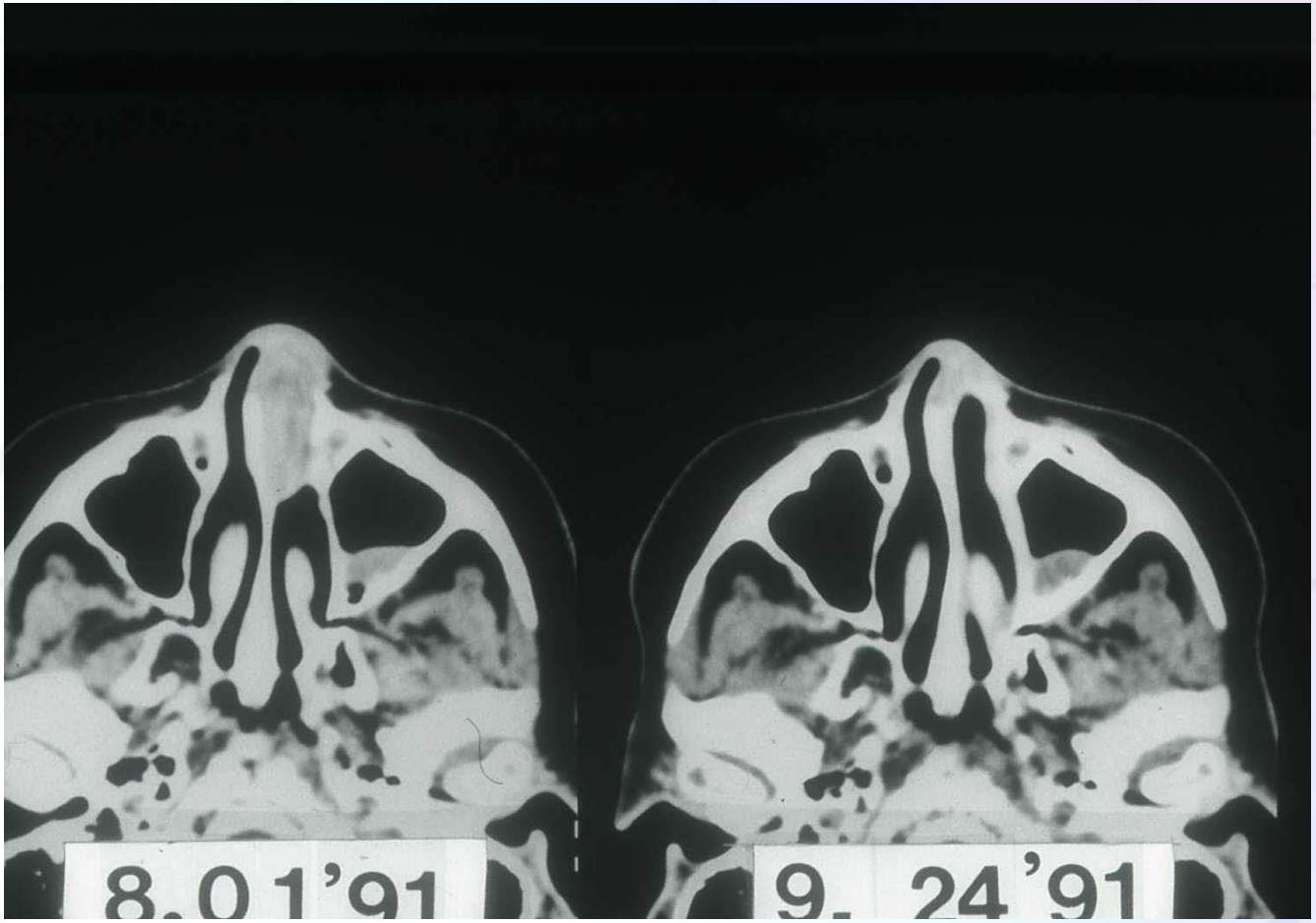


LDR Therapy for Hurthle Cell Carcinoma

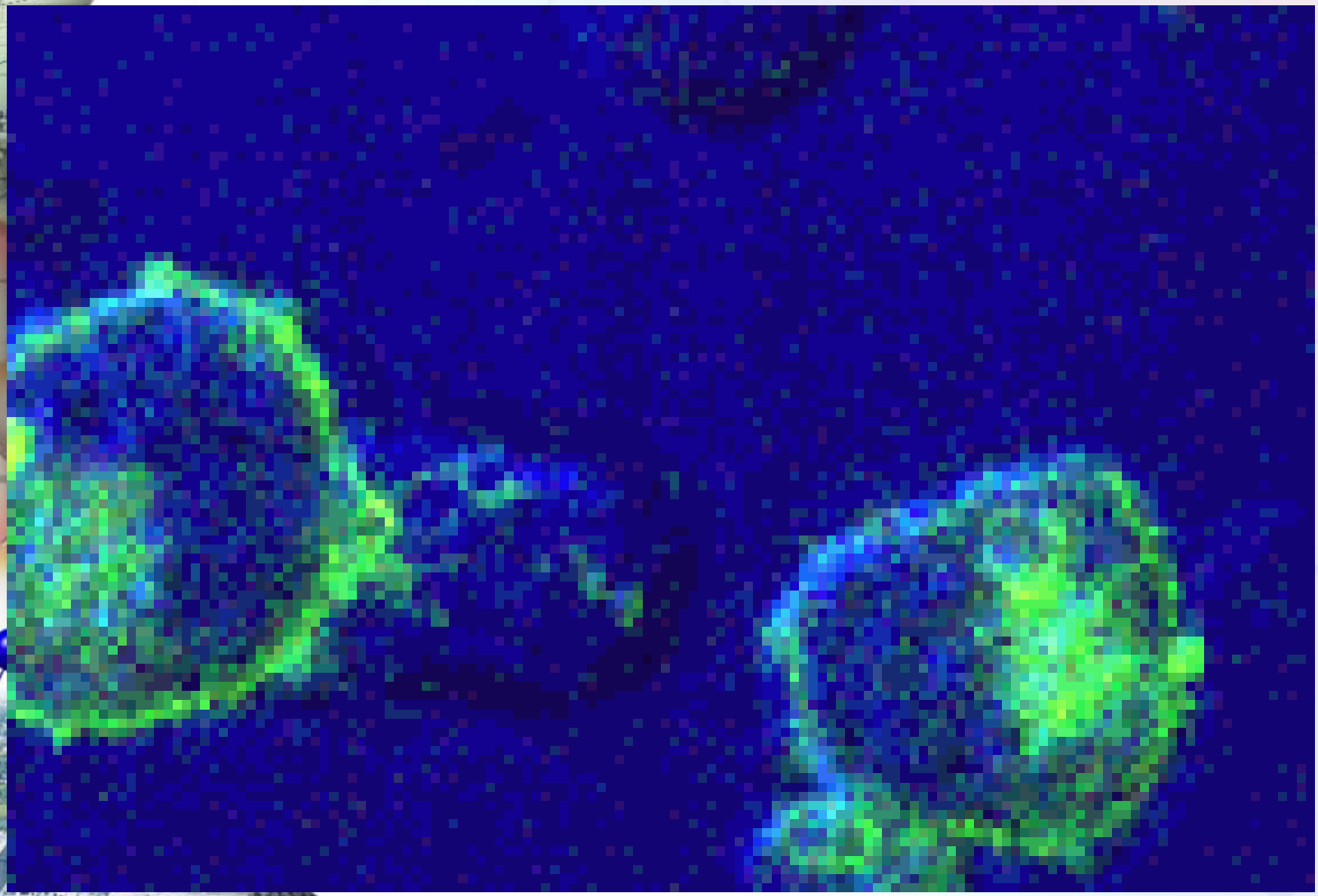
HB-LDI Therapy 1500 mGy; prophylaxis against cancer

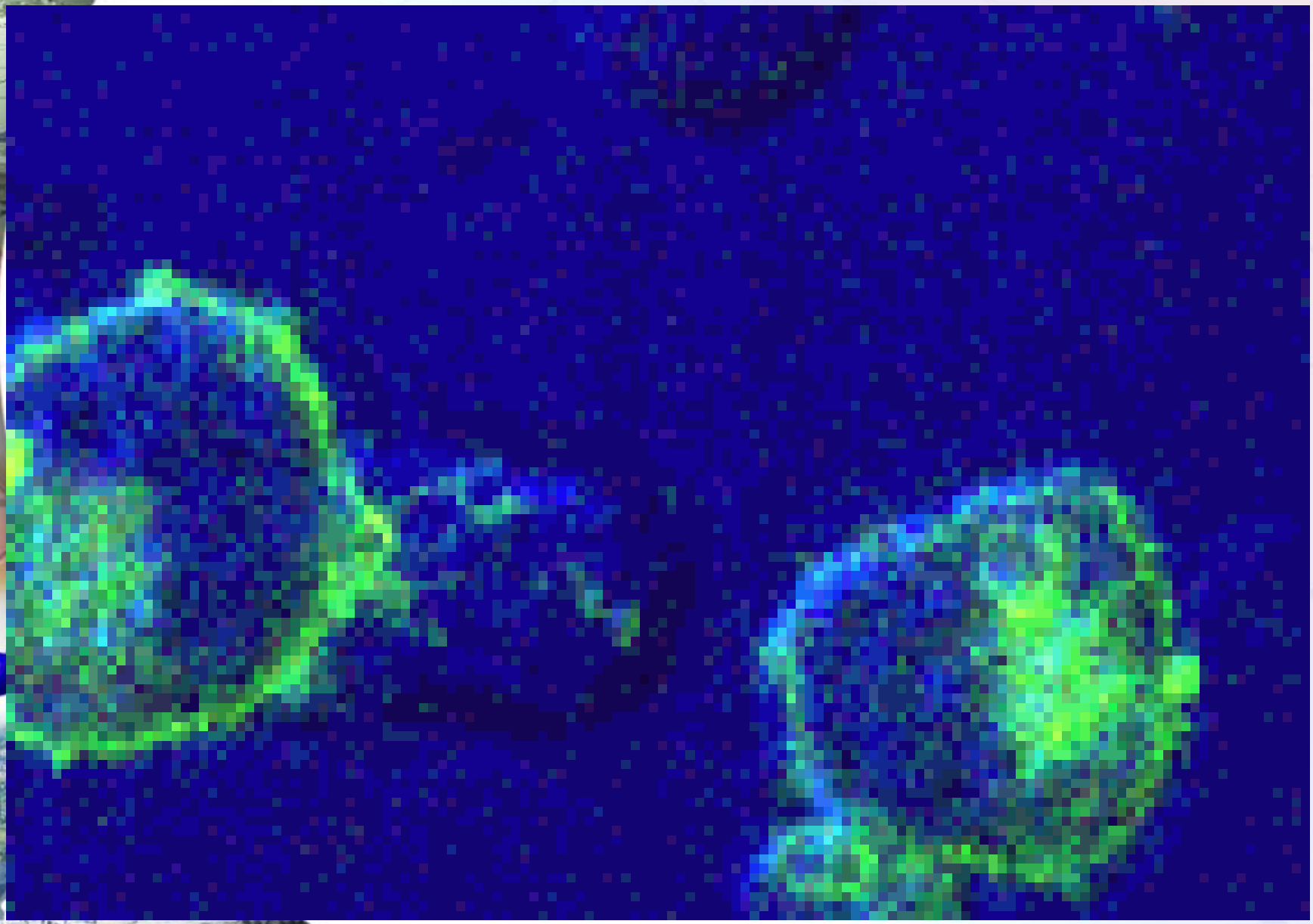
150 mGy x twice/week x 5 weeks = 1500 mGy

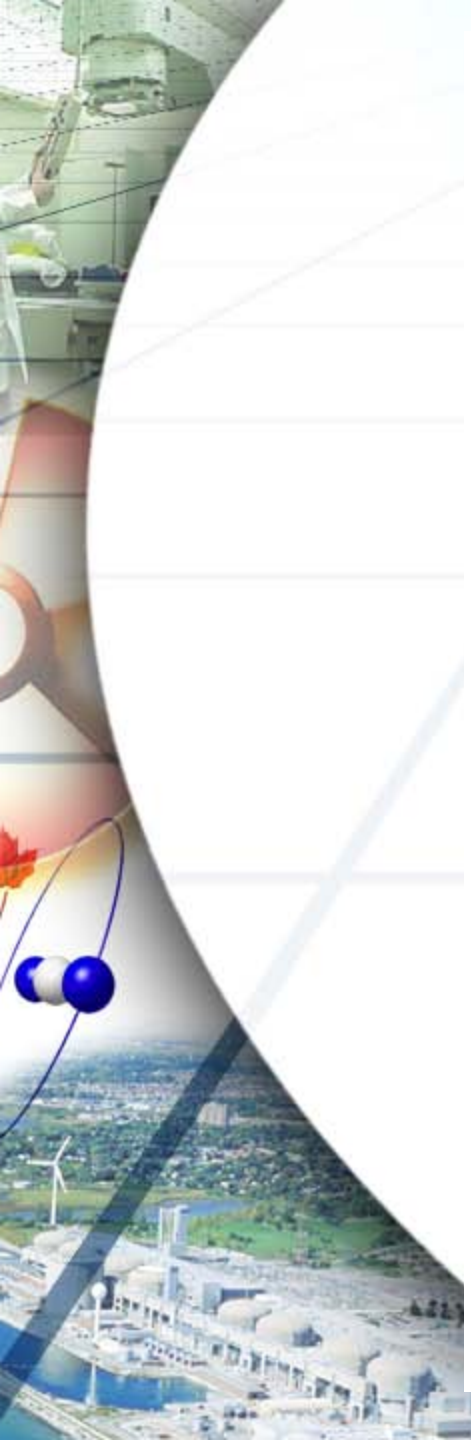
Abscopal Effect



Shu-Zheng Liu and Jerry Cuttler at CVH







Lauriston S. Taylor Lectures in Radiation Protection and Measurements

Lecture No. 16

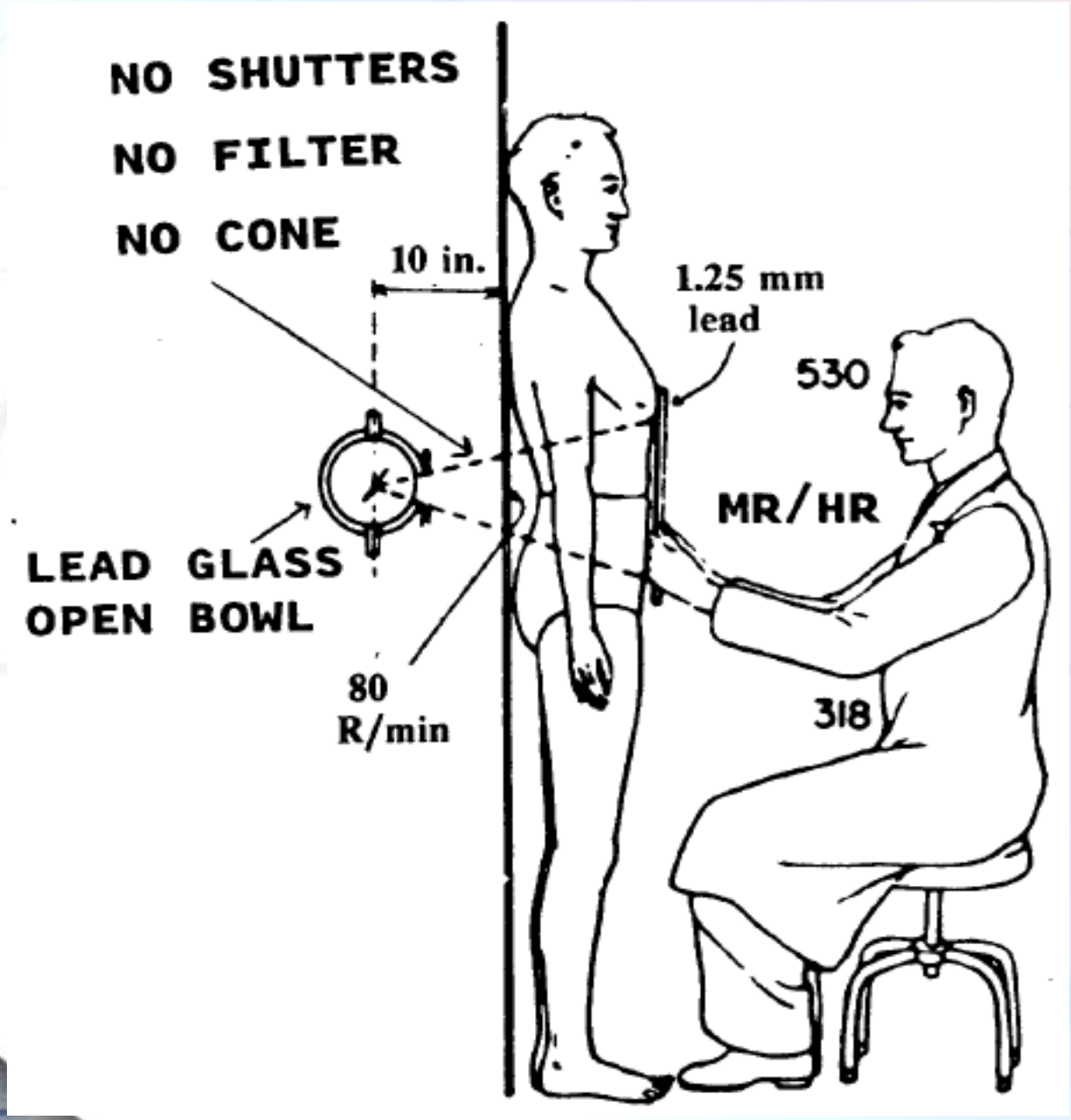
Dose and Risk in Diagnostic Radiology: How Big? How Little?

by Edward W. Webster

Presented April 1, 1992
Issued September 1, 1992

National Council on Radiation Protection and Measurements
7910 WOODMONT AVENUE/BETHESDA, MD. 20814

Fluoroscopy

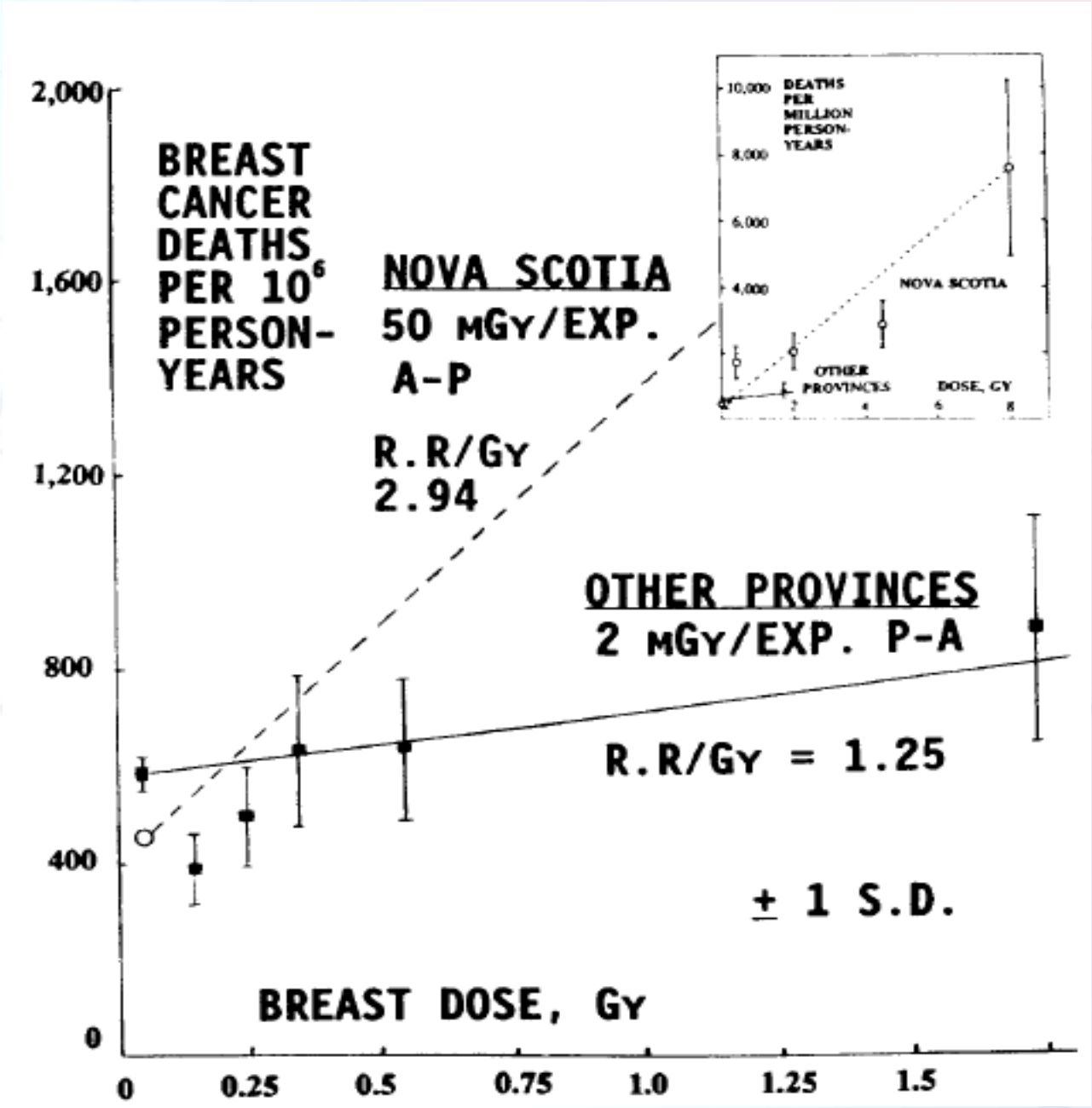


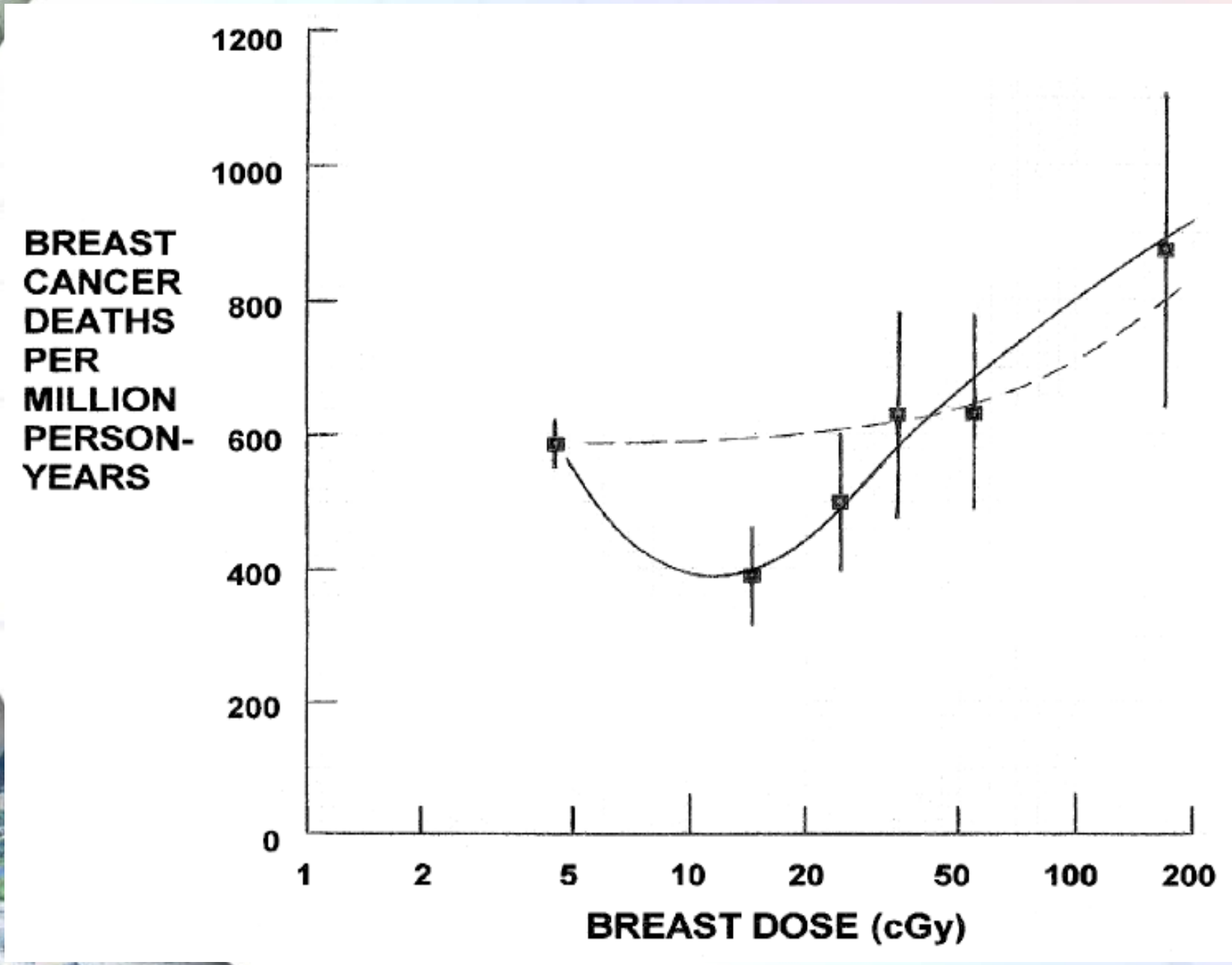
Canadian Breast Cancer Study

Table 1. Observed Rates of Death from Breast Cancer, According to the Dose of Radiation Received.

Dose (Gy)	Standardized Rate per 10^6 Person-Years*		
	Nova Scotia	Other Provinces	All Provinces
0–0.09	455.6 (13)	585.8 (288)	578.6 (301)
0.10–0.19		389.0 (29)	421.8 (32)
0.20–0.29		497.8 (24)	560.7 (26)
0.30–0.39	1709 (11)	630.5 (17)	650.8 (18)
0.40–0.69		632.1 (19)	610.0 (19)
0.70–0.99			1362 (13)
1.00–2.99	2060 (14)		1382 (17)
3.00–5.99	2811 (13)	873.1 (14)	2334 (14)
6.00–10.00	7582 (8)		8000 (9)
≥10.00	21,810 (12)		20,620 (13)

*The number of deaths is shown in parentheses. The calculations exclude the values for 10 years after the first exposure and have been standardized according to age at first exposure (10 to 14, 15 to 24, 25 to 34, and ≥35 years) and time since first exposure (10 to 14, 15 to 24, 25 to 34, and ≥35 years) to the distribution for the entire cohort.


BREAST CANCER DEATHS PER 10^6 PERSON-YEARS
2,000
1,600
1,200
800
400
0
NOVA SCOTIA
50 mGy/EXP.
A-P
R.R/Gy
2.94
OTHER PROVINCES
2 mGy/EXP. P-A
R.R/Gy = 1.25
± 1 S.D.
BREAST DOSE, Gy
0
0.25
0.5
0.75
1.0
1.25
1.5
DEATHS PER MILLION PERSON-YEARS
10,000
8,000
6,000
4,000
NOVA SCOTIA
OTHER PROVINCES
DOSE, Gy


BREAST CANCER DEATHS PER MILLION PERSON-YEARS
1200
1000
800
600
400
200
0
1
2
5
10
20
50
100
200
BREAST DOSE (cGy)

4133 Identified Radium Dial Painters in USA

Bone cancer threshold at 10 Gy or 1000 rad of radium alpha radiation

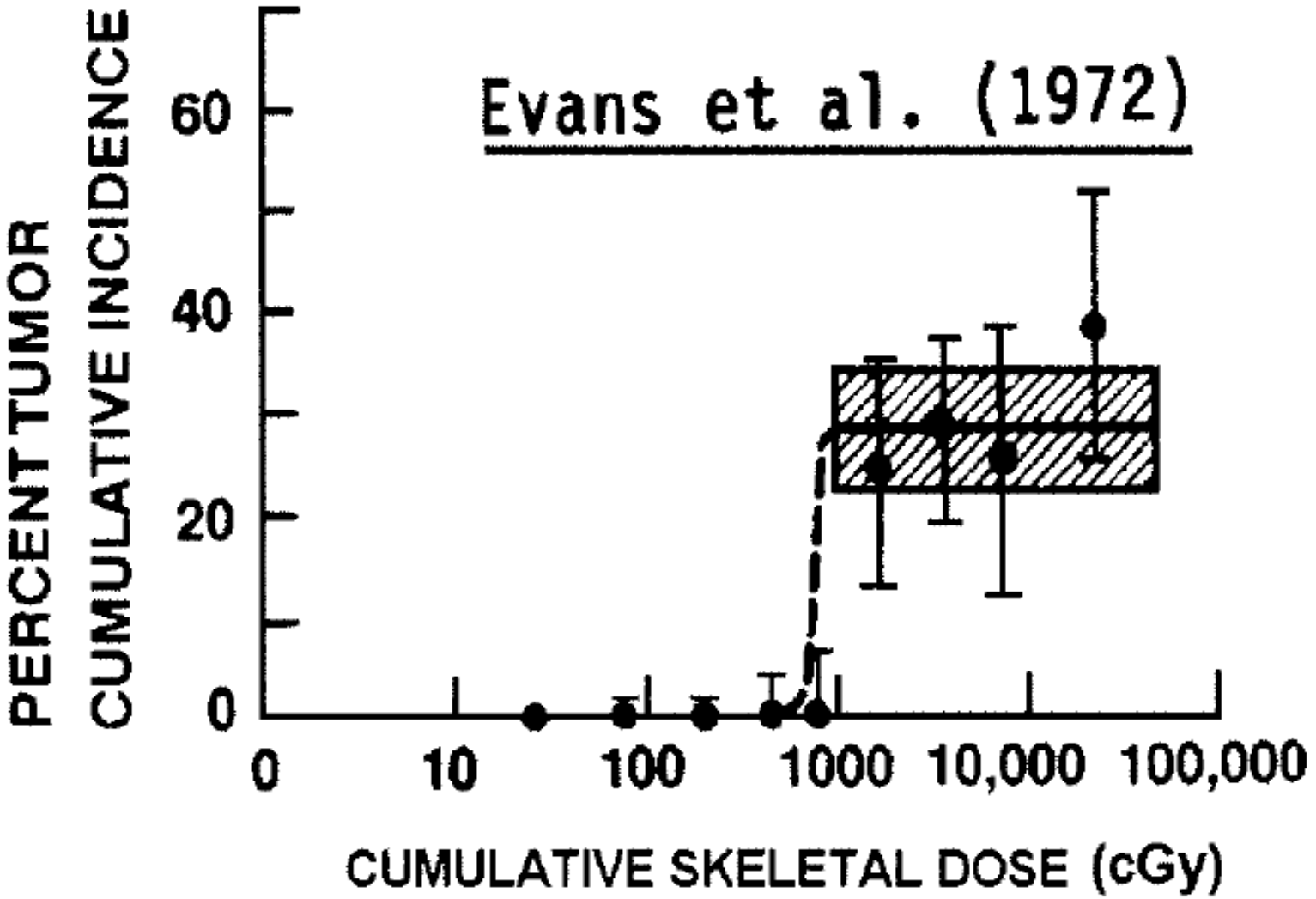
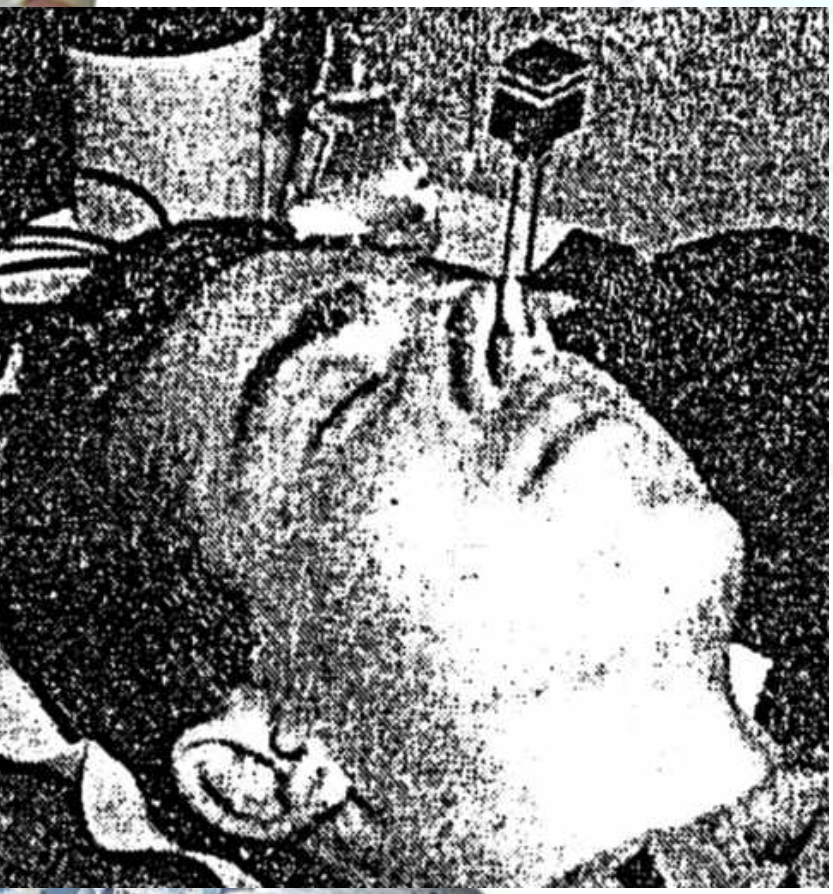


Fig. 11. Cumulative bone sarcoma incidence in people exposed to ^{226}Ra as a function of cumulative dose to the skeleton as reported by Evans et al. (1972).

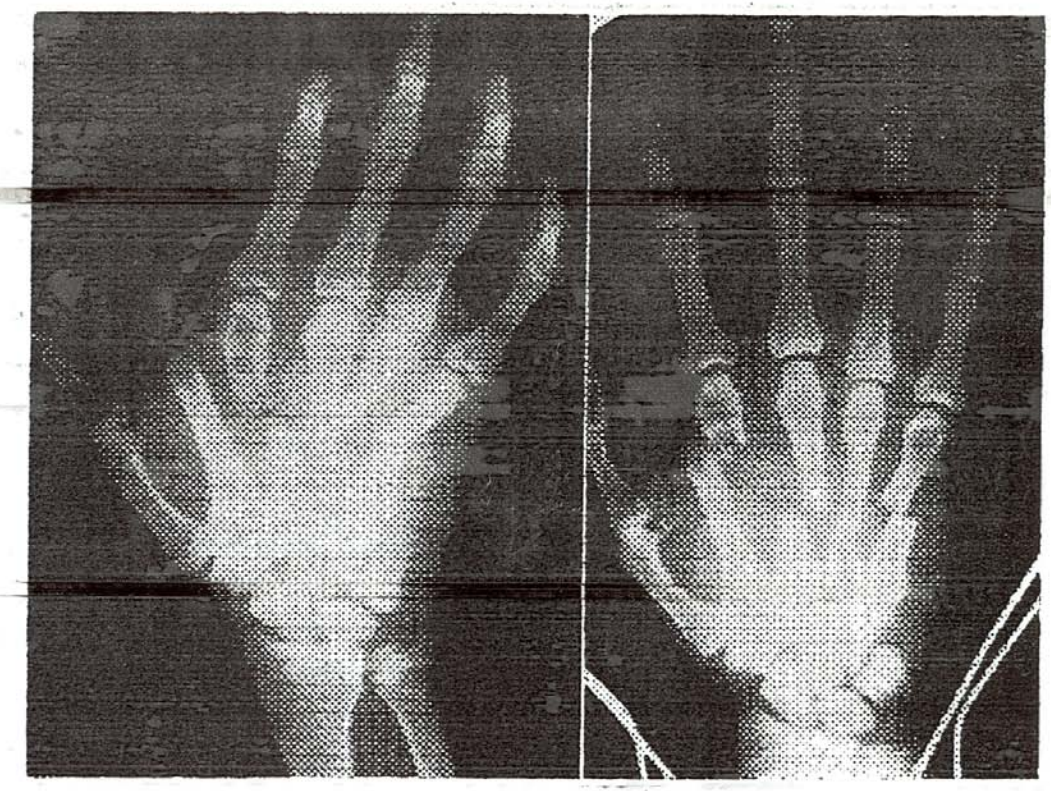
Nasal Radium Irradiation

US CDC estimate: up to 2,600,000 children received NRI from 1945-1961 as a standard medical practice to shrink adenoids. Typical Navy protocol: four 10 minute irradiations 2-4 weeks apart. **Contact** gamma dose = 2000 rad (20 Gy); **1 cm depth** dose = 206 rad (2 Gy) Beta dose 68 rad (0.7 Gy) from each applicator. Excess lymphoid tissue at Eustachian tube openings tended to prevent pressure equalization, aggravation middle ear problems.



Position of the child patient during treatment

Anesthesia with cocaine precedes introduction of the applicator which is then left in place for twelve minutes on each side
(From Proctor, D.F., *"The Tonsils and Adenoids in Childhood"*, p. 17, Charles C. Thomas, Publisher, 1960)



Figs. 7–8. Case 1: Severe hand injury, with multiple compound fractures and some gas in tissues (left). Fig. 8 (right) shows same hand a few days after prophylactic x-ray irradiation: no gas in the tissues, no infection, hand on way to complete recovery.

TABLE V: CASES WHICH RECEIVED PROPHYLACTIC IRRADIATION AND HAVE BEEN REPORTED IN THE LITERATURE

Cases Which

those which do not appear until three or four days have elapsed. It is evident from Figure 6 that the second, third, and

Henry Kaplan was the first one to use a linear accelerator at Stanford Hospital in San Francisco in 1957. The patient was a boy (Gordon Isaacs) that was suffering from a tumor in his eye (retinoblastoma). The treatment saved the child's sight and he lived the rest of his life with his vision intact.

Below is a picture taken during the treatment.

CNS Bulletin Article – March 2014

Personal Memoir

On Radium and Radiation

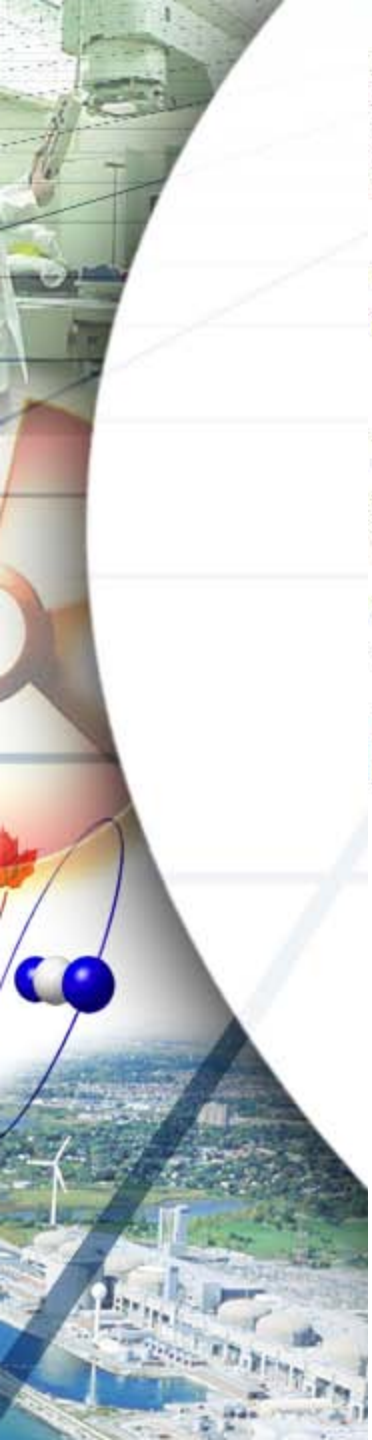
by DON WILES, Almonte, Ontario

On graduating with an Honours degree in Chemistry in 1947, I was offered a job as radium chemist in Port Hope, Ontario, in what was then called Eldorado Mining and Refining Co. While their main function was the production of Uranium oxide, or "x-metal"as it was called, Radium was an important by-product, to be exported to many European countries. As late as the1950s, artificial radioactive isotopes (technetium, cobalt and others) were not yet available for medical uses. Instead, Radium was used exactly as Marie Curie had done decades earlier. This meant that Radium had to be extracted from the Uranium ore and purified for use. Ultimately the radium, in platinum 'needles' about 5 mm diameter by about 3 - 4 cm long, was surgically inserted into cancerous locations, with the idea that the radiation would destroy the cancer.

I was to be employed in the radium finishing lab, which meant purifying radium for subsequent packaging in platinum needles for ultimate use in treating cancers. I was to be number three in a small group in which the group leader had been temporarily given leave because of a serious radiation burn on his abdomen. It seems that someone had put about 2 - 300

of this solution, partial crystallization followed. It was known that radium bromide is less soluble than barium bromide, so fractional crystallization would concentrate the radium in the crystals. The crystals, now slightly richer in radium, were separated and transferred to a second evaporator. (Marie Curie usually used radium chloride in this same way.) After several such evaporations, the crystals were moved to another section of the lab and crystallized several more times, now in quartz bowls about 18 inches in diameter.

The crystals were then taken to the 'flask lab' where a feeble old guy performed yet more crystallization to produce crystals that were by then much more concentrated in radium. Finally, this crystalline mixture of barium and radium bromides was transferred to me in the purification lab. Each month I was given about 8 Curies of radium, along with a lot of barium. It was then my task to crystallize this mixture many more times – first in beakers of about 100 mL, and later in quartz crucibles of perhaps 20 mL capacity. This was all done in a ventilated fume hood with a thick lead shield protecting some parts of my body. The final crystals were deemed 'pure' if they were measured to be over 80% radium bromide.



Dose-Response, 10:644-663, 2012
Formerly Nonlinearity in Biology, Toxicology, and Medicine

ISSN: 1559-3258
DOI: 10.2203/dose-response.12-014.Feinendegen

International Dose-Response Society

HEMOPOIETIC RESPONSE TO LOW DOSE-RATES OF IONIZING RADIATION SHOWS STEM CELL TOLERANCE AND ADAPTATION

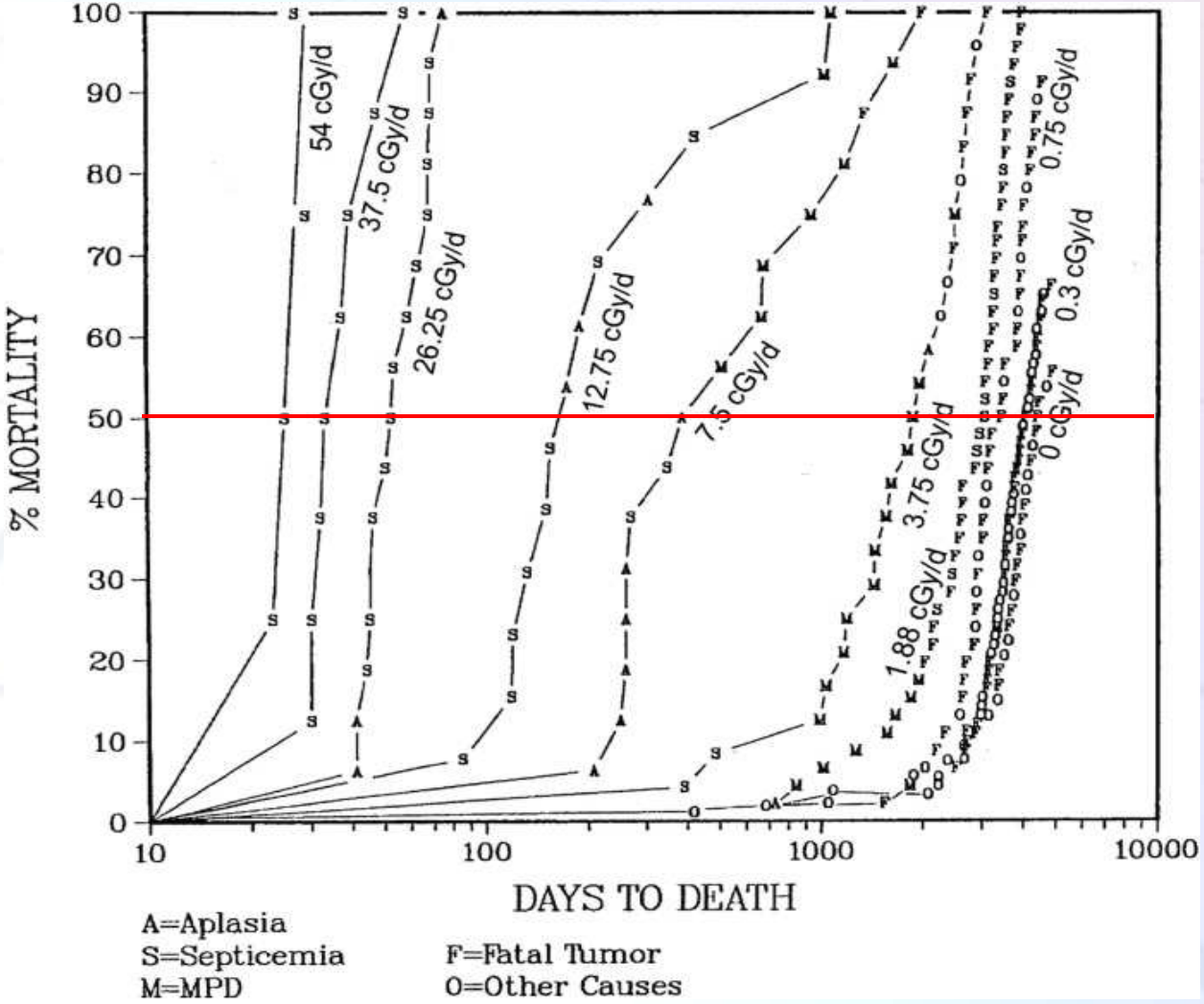
Theodor M. Fliedner, Dieter H. Graessle □ Radiation Medicine Research Group and WHO Liaison Institute for Radiation Accident Management, Ulm University, Germany

Viktor Meineke □ Bundeswehr Institute of Radiobiology Affiliated to the University of Ulm, Germany;

Ludwig E. Feinendegen □ Heinrich-Heine-Universtät Düsseldorf, Germany, and Brookhaven National Laboratory, Upton, NY, USA

□ Chronic exposure of mammals to low dose-rates of ionizing radiation affects proliferating cell systems as a function of both dose-rate and the total dose accumulated. The lower the dose-rate the higher needs to be the total dose for a deterministic effect, i.e., tissue reaction to appear. Stem cells provide for proliferating, maturing and functional cells. Stem cells usually are particularly radiosensitive and damage to them may propagate to cause failure of functional cells. The paper revisits 1) medical histories with emphasis on the hemopoietic system of the victims of ten accidental chronic radiation exposures, 2) published hematological findings of long-term chronically gamma-irradiated rodents, and 3) such findings in dogs chronically exposed in large life-span studies. The data are consistent with the hypothesis that hemopoietic stem and early progenitor cells have the capacity to tolerate and adapt to being repetitively hit by energy deposition events. The data are compatible with the "injured stem cell hypothesis", stating that radiation–injured stem cells, depending on dose-rate, may continue to deliver clones of functional cells that maintain homeostasis of hemopoiesis throughout life. Further studies perhaps on separated hemopoietic stem cells may unravel the molecular-biology mechanisms causing radiation tolerance and adaptation.

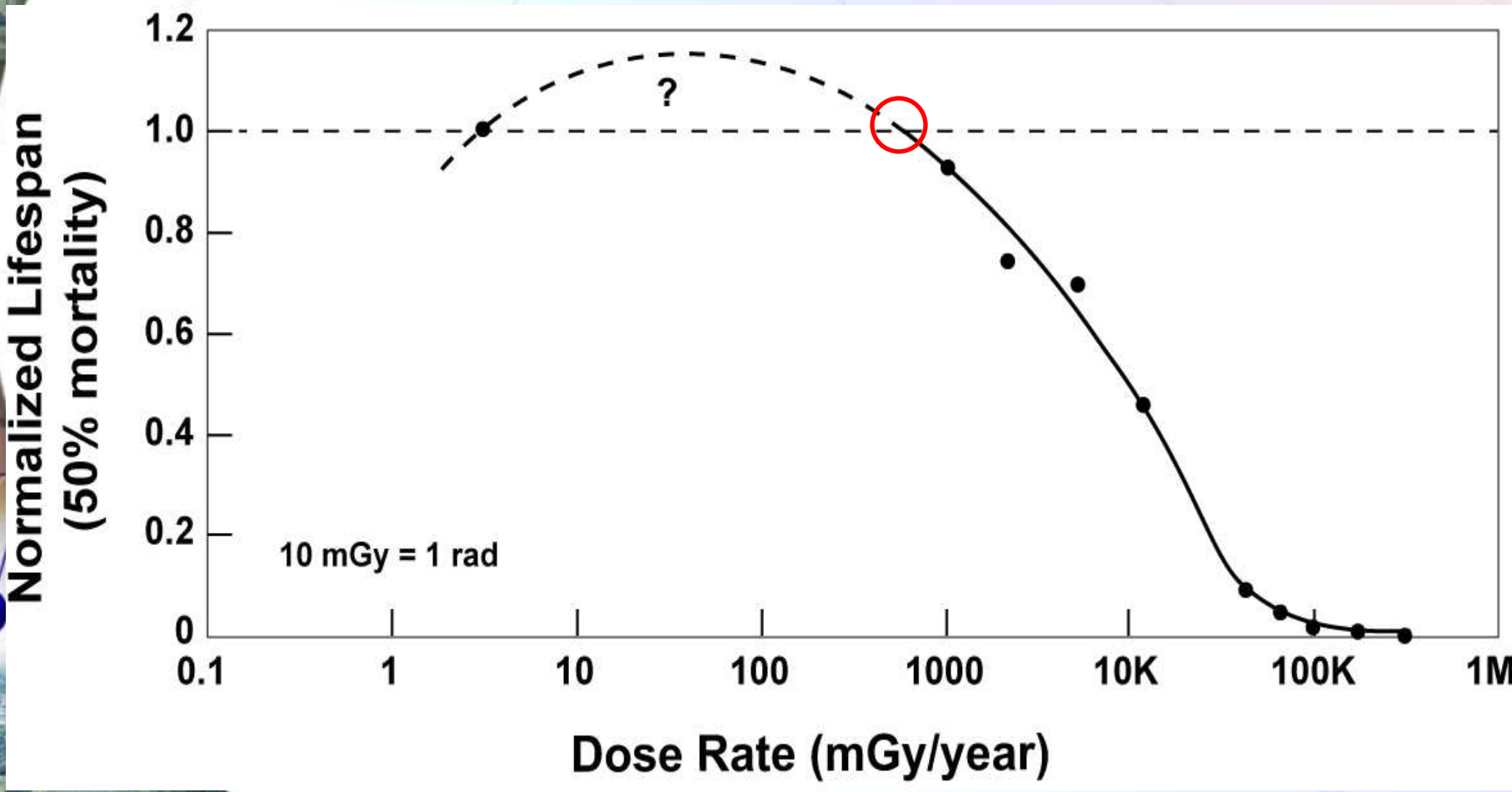
Continuous Co-60 Irradiation of Dogs



0.3 cGy/d = 1100 mSv/year = 110 rad/year
No significant changes in blood counts
No apparent increase in tumor incidence

Dose Rate (cGy/day)	Dose Rate (mGy/year)	Lifespan - days (50% mortality)	Lifespan (normalized)
backgnd	2.4×10^{0}	4300	1.00
0.3	1.1×10^{3}	4100	0.95
0.75	2.7×10^{3}	3300	0.77
1.88	6.9×10^{3}	3000	0.70
3.75	1.4×10^{4}	1900	0.44
7.5	2.7×10^{4}	410	0.095
12.75	4.7×10^{4}	160	0.037
26.25	9.6×10^{4}	52	0.012
37.5	1.4×10^{5}	32	0.0074
54	2.0×10^{5}	24	0.0056

Lifespan versus Radiation Level



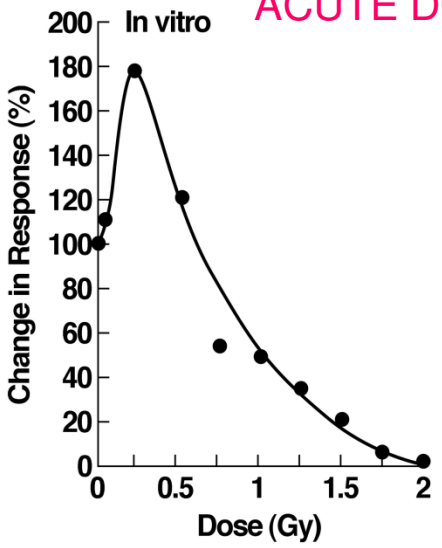
Threshold at ~ 700 mGy per year

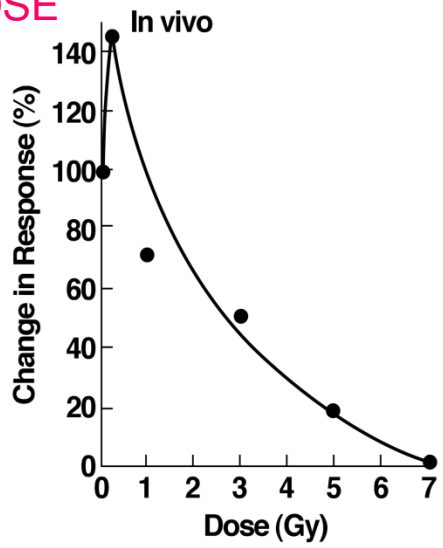
Fliedner: blood cell response to chronic radiation

- Review paper in Dose-Response Journal, Dec 2012
- He reviewed histories of humans in 10 radiation accidents (including 28,000 in Techa and 1,800 in Mayak) and studies on rats and dogs
- Radiation effect on mammals is function of dose-rate and total dose
- Blood stem cells are usually very radiosensitive; however, they can tolerate and adapt to chronic radiation---adapt better at lower rate.
- Deliver clones of functioning cells that maintain a lifetime of service
- Beagle dogs at 0.3 rad/day ~ same cancer rate as control dogs
- ICRP standard early 1930s: a tolerance dose of 0.2 r/day or 70 rad/y
- Present-day ICRP recommendations (LNT and ALARA) unjustified

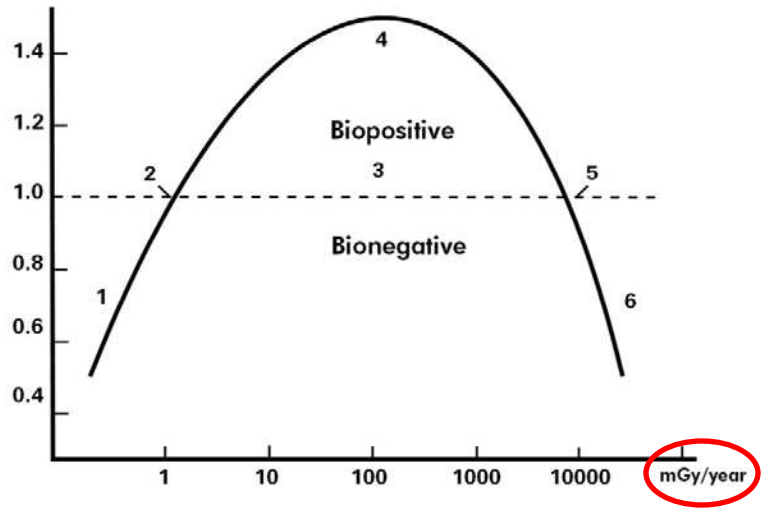
Radiation Hormesis

ACUTE DOSE





CONTINUOUS DOSE



Organisms are stressed: physical, chemical, biological, radiation

Organisms adapt to stress

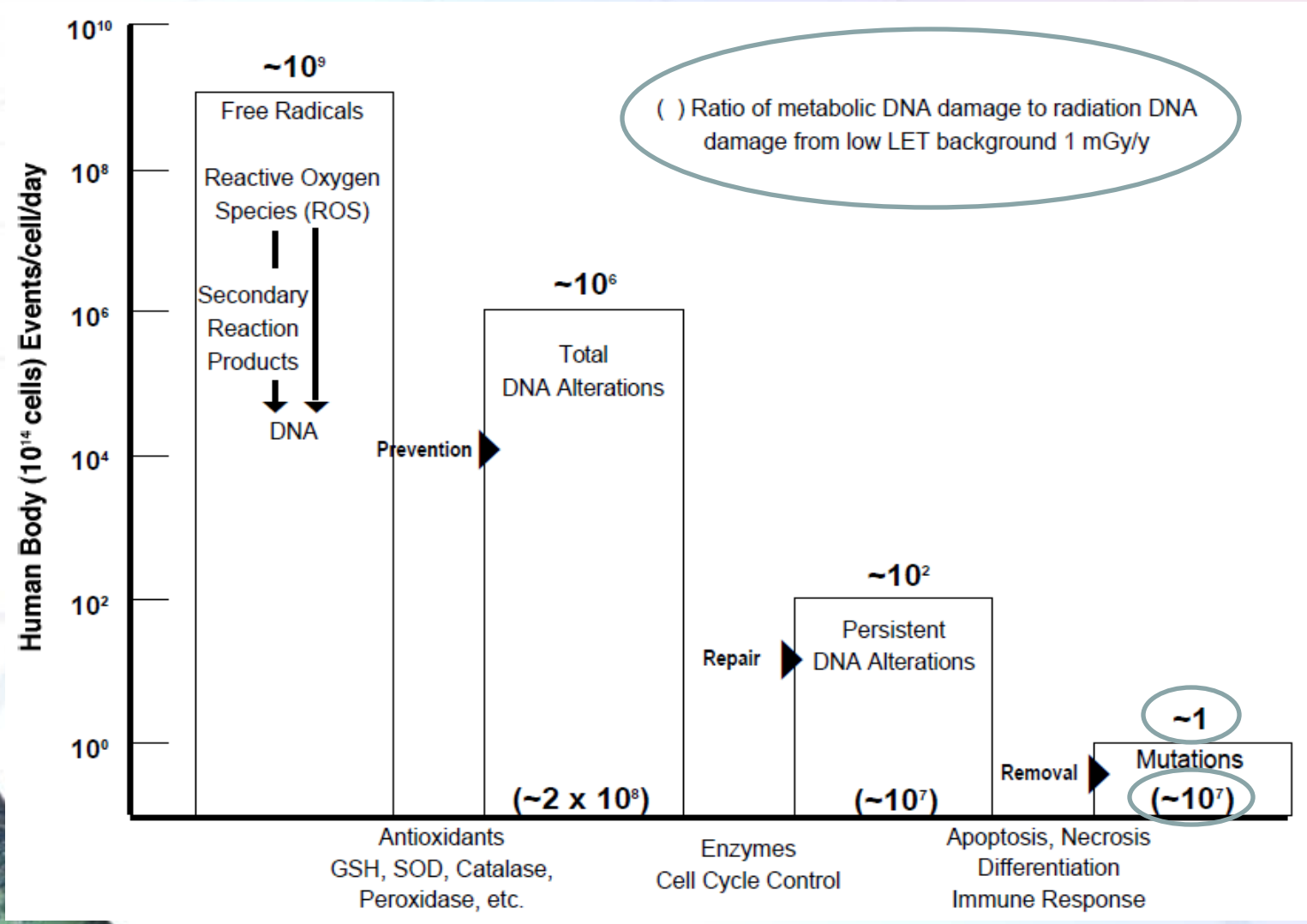
Radiation modulates organism's protection systems

Low radiation dose/dose-rate reduces cancer incidence because it stimulates:

- prevention of DNA damage
- repair of DNA damage
- removal of damaged cells and removal of cancer cells

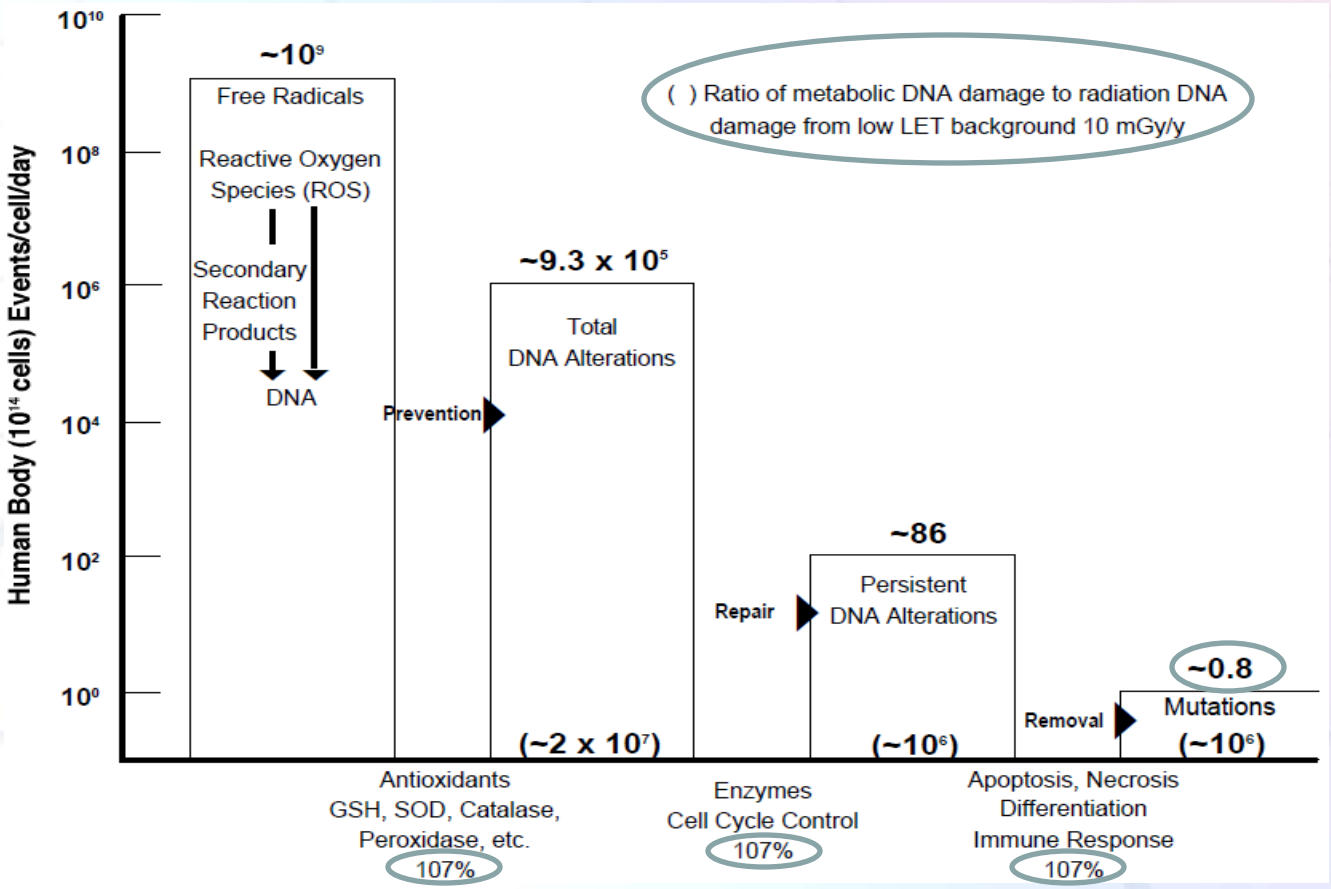
High radiation dose/level has opposite effects

Model for Spontaneous DNA mutations



Pollycove-Feinendegen, BELLE, Feb 2003, pg 2-21
Spontaneous rate: 1 in 10 cells has DSB per day
Bkgnd rad level: 1 in 10,000 cells has DSB per day

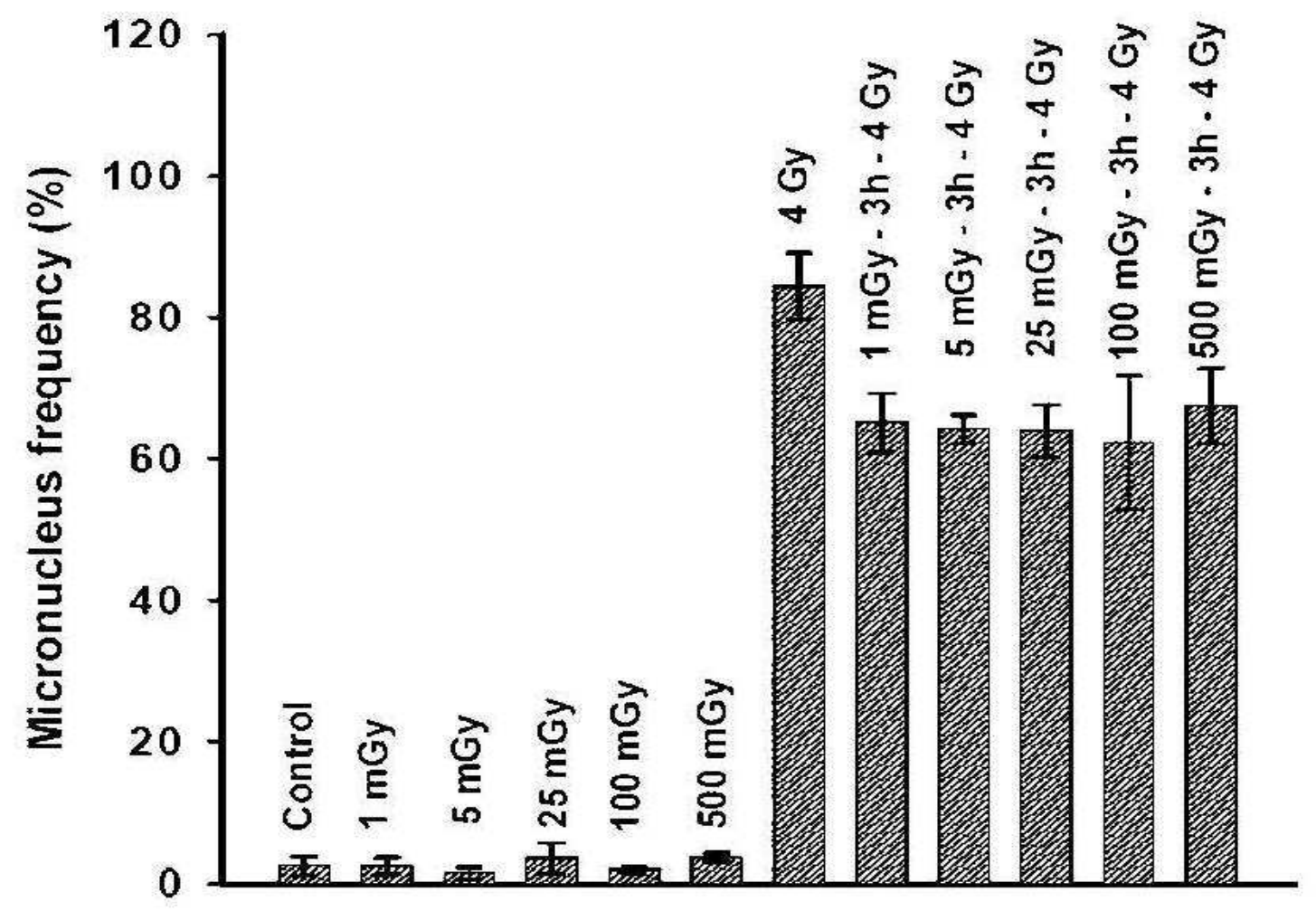
Radiation Hormesis - Stimulation of Defences



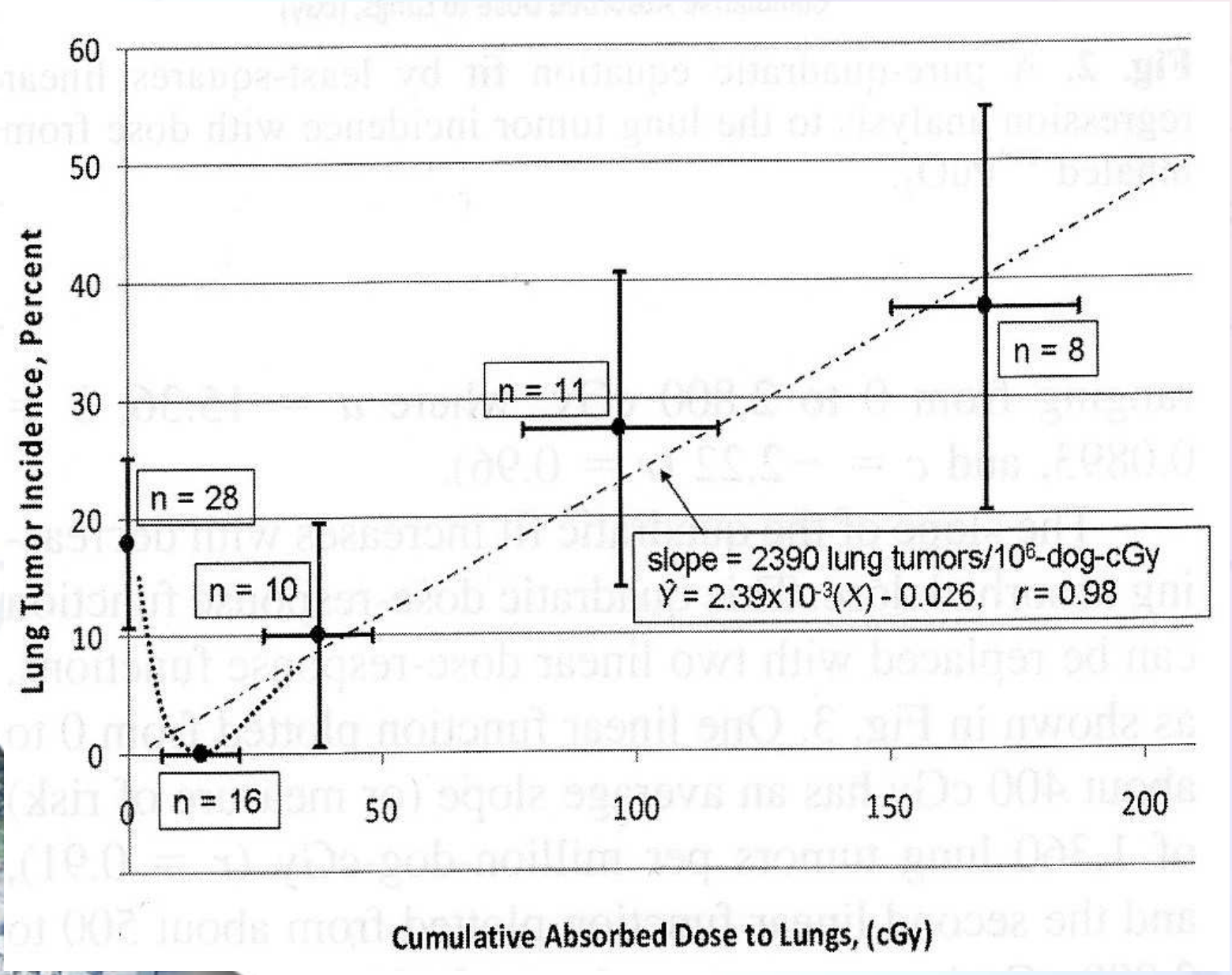
Low dose stimulates defences:
to prevent, repair & remove spontaneous DNA alterations due to thermal and oxidative processes (leakage of ROS)

Spontaneous DNA damage rate is ~10 million times greater than bkgnd rad'n DNA damage rate
x10 increase background radiation gives ~20% lower mutation rate

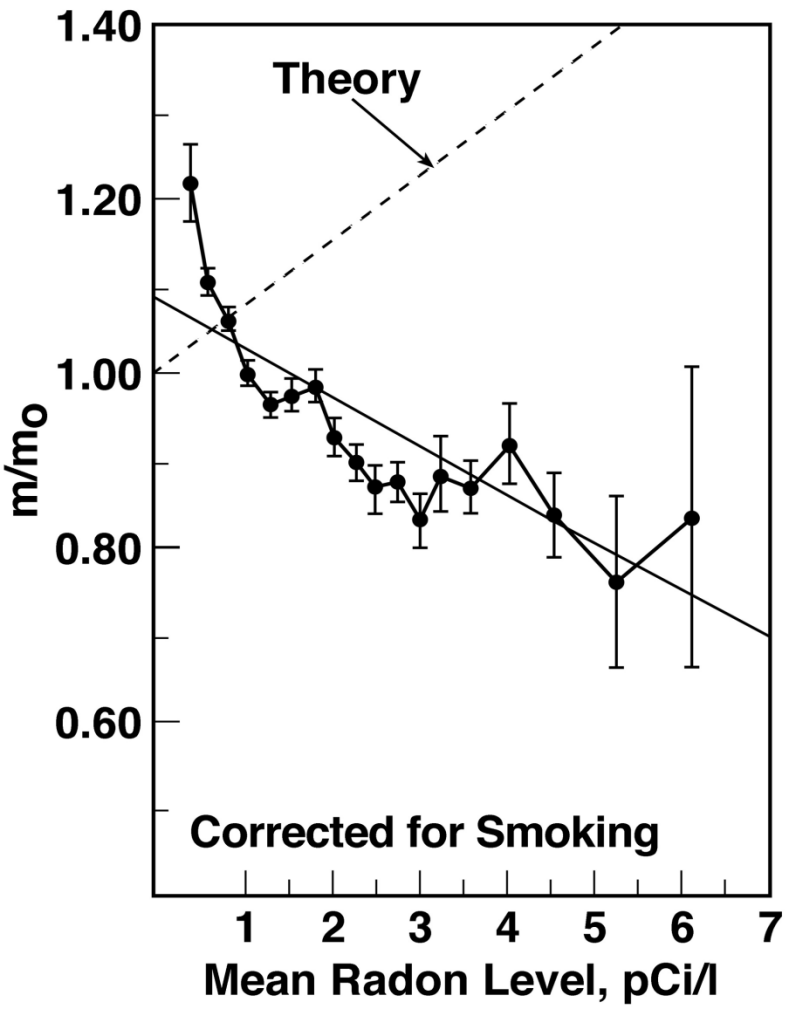
Low radiation dose up-regulates cell repair



PuO_2 in Beagle Dog Lungs



Radon Exposure Study Disproves the LNT Hypothesis



Greatest natural radiation exposure is radon gas from uranium activity

Cohen tested the LNT model, as used, and clearly disproved it; lung cancer mortality *lower* where radon *higher*

Lung cancer *higher* where radon is *lower* than the average of 1.7 pCi/L

Instead of discarding LNT assumption, objection raised (ecological study). This is not relevant to testing model

Authorities still accept LNT assumption

Appearance of db/db mice at 90th week of age

Irradiated Group

Control Group

Is low radiation a cancer risk? No!

- Spontaneous (natural) DNA damage[1] occurs at very high rate > 1000 x background radiation DNA damage[1] rate
- Organisms have very powerful protection systems against all cell and tissue damage (internal and external)
- Low rad'n up-regulates protections → less damage/cancer
  High radiation impairs protection → more damage, harm

---

[1] double-strand breaks

Repeat these points over and over and over again

Discussion

- Many use LNT model to predict cancer risk from small radiation dose
- Straight line to 0 from high-dose cancer data of atom bomb survivors
- This procedure can only give a risk of cancer from any radiation dose
- Epidemiological studies assume harm, so are designed to measure radiation-induced cancer risk. They do not observe beneficial effects
- Cancer data are fitted to the LNT model, they presume LNT is valid
- Scott et al list 7 approaches that make it difficult to see positive effects
- Scott (2008) describes 3 epidemiological “tricks” to obtain LNT curve
- They use *relative risk* or *odds ratio* instead of cancer incidence data
- Misrepresentation of data and deceptions are used to fit LNT model

Conclusions

- Social concern about nuclear safety caused by ideological link of human-made radiation to a risk of cancer, via LNT
- Radiation scare in 1950s to stop atomic-bombs continues in spite of extensive evidence of low-dose beneficial effects
- Refusal to revert to threshold model for radiation protection blocks social acceptance of nuclear energy and radiation diagnostics. Remedy is to discard politicized science.
- Nuclear regulations are overprotective and very costly

Chernobyl and Fukushima victims suffered **not** from cancer but **"a psychosis of fear"**

Recommendations

- Scientific societies should organize events to discuss radiation benefits and health risks
- Regulatory bodies and health organizations should examine the scientific evidence
- Change to science-based (threshold) regulations
- Stop calculating nuclear safety cancer risk with LNT
- Stop regulating harmless radiation sources (radon)
- Develop public communication programs
- **Raise radiation level threshold for evacuation from 20 to 700 mSv/year (2 to 70 rem/year)**