Energy Policy Committee
Michigan House of Representatives
Lansing, MI, October 17, 2017

No reason to fear low radiation
It induces beneficial health effects

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**"Nothing in life is to be feared;
it is only to be understood."**

Maria Sklodowska Curie
Two-time Nobel laureate and
discoverer of radium and polonium

Now is the time to use our knowledge and wisdom to understand more, so that we may fear less.

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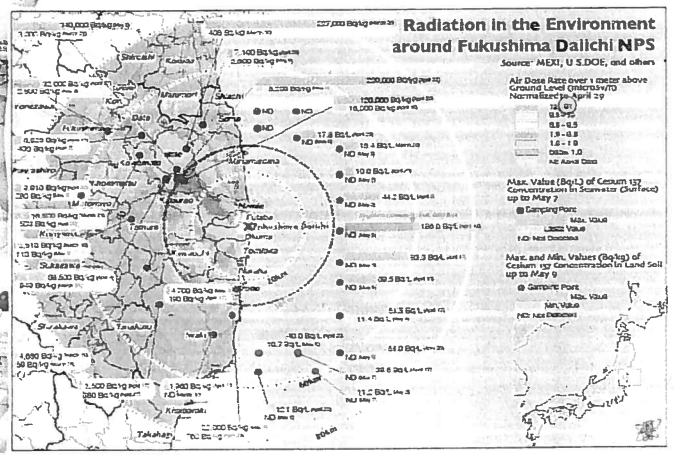
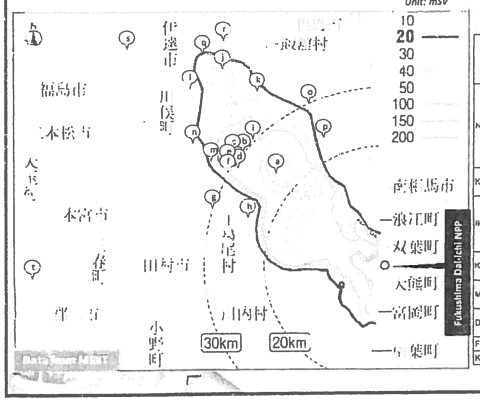
91 μSv/h x 8766 h/y = 798 mSv/y ~ natural HBRAs



Average dose level in City of Ramsar, Iran = 260 mSv/year

Accumulated Radiation Dose around Fukushima Dai-ichi

Distribution Map of Accumulated Radiation Dose up to
(Simulation based on Actual Measurements up to Apr 21 2011)



MEXT has released a map showing how much radiation will be accumulated around Fukushima Dai-ichi Nuclear Power Plant.

Place		Distance from Fukushima Daiichi	Accumulated Radiation (Estimation) unit:mSV
Namie-machi	a	NW 24km	235.4
	b	NW 31km	188.6
	c	NW 31km	110.2
	d	WNW 28km	56.2
	e	WNW 30km	48.2
	f	WNW 30km	24.2
Katsurao-mura	g	WNW 32km	18
	h	NW 25km	11
Iitate-mura	i	NW 33km	61.7
	j	NW 39km	34.8
	k	NW 36km	28.3
	l	NW 44km	10
Kawamata-machi	m	WNW 34km	24.2
	n	WNW 40km	19.6
Minamisoma-shi	o	NNW 30km	15.8
	p	NNW 25km	11.9
Date-shi	q	NW 48km	21.2
	r	NW 46km	16
Fukushima-shi	s	NW 56km	10.6
Koriyama-shi	t	W 60km	10.1

So what happened in Japan in 2011?

- ~ 20,000 killed or missing due to earthquake, tsunami
- ~ 300,000 residents evacuated because of fear of radioactive material from damaged Fukushima NPP
- ~ 1600 older people died prematurely because of the evacuation
- No one harmed by radiation, not even the plant workers

Is it safe to be exposed to low-level radiation?

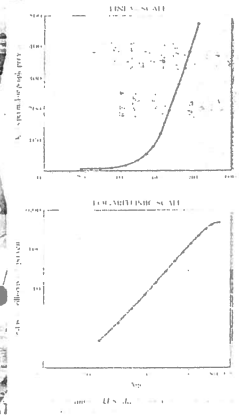
- You can go to 2 different places for the answer on that. If you go to the radiation protection people, whose job it is to protect everyone from any exposure to radiation (human-made) and who don't have any work to do if they are not protecting us, you'll find there's a higher risk of cancer.
- But, if you go out into the real world, who do you know that's ever been harmed by low-level radiation? There's just no evidence of that.

https://www.youtube.com/watch?v=bD4bUFrpFVA

The basic problem of nuclear energy

- People are *afraid* of nuclear plants because we tell them any *radiation* exposure increases the risk of fatal *cancer*
- This is the 1956 antinuclear health scare, and it is *false*.
- *Low* radiation dose or dose-rate *stimulates* adaptive *protection* systems -- more than 150 genes in humans.
- *High* radiation *inhibits* or *damages* these systems.
- For every type of exposure, there is a *threshold*
- A dose below is beneficial; a dose above is harmful.
- Longevity is best measure of health effect, not cancer

Cancer death rate rises exponentially with *age*



Cancer cells from where?

Spontaneous DNA damage: free radicals, reactive oxygen species (ROS), thermal effects

Why the increase?

Protection systems age, i.e., immune system gets weaker

Can we do something?

Low radiation doses stimulate adaptive protection systems

Cancer Immunol Immunother
DOI 10.1007/s00262-017-1992-z

CrossMark

REVIEW

Cancer immunotherapy: how low-level ionizing radiation can play a key role

Marek K. Janiak[1] · Marta Wincenciak[1] · Aneta Cheda[1] · Ewa M. Nowosielska[1] · Edward J. Calabrese[2]

(222 references)

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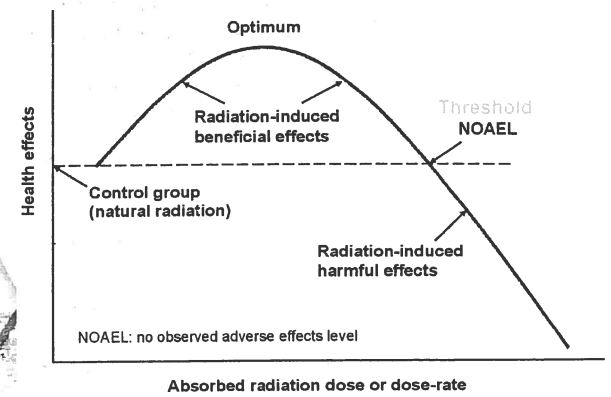
Abstract The cancer immunoediting hypothesis assumes that the immune system guards the host against the incipient cancer, but also "edits" the immunogenicity of surviving neoplastic cells and supports remodeling of tumor microenvironment towards an immunosuppressive and pro-neoplastic state. Local irradiation of tumors during standard radiotherapy, by killing neoplastic cells and generating inflammation, stimulates anti-cancer immunity and/or partially reverses cancer-promoting immunosuppression. These effects are induced by moderate (0.1–2.0 Gy) or high (>2 Gy) doses of ionizing radiation which can also harm normal tissues, impede immune functions, and increase the risk of secondary neoplasms. In contrast, such low-level radiation (LLR) exposures retard the development of neoplasms in humans and experimental animals. Here, we review immunosuppressive mechanisms induced by growing tumors as well as immunomodulatory effects of LLR evidently or likely associated with cancer-inhibiting outcomes of such exposures. We also offer suggestions how LLR may restore and/or stimulate effective anti-tumor immunity during the more advanced stages of carcinogenesis. We postulate that, based on epidemiological and experimental data amassed over the last few decades, whole- or half-body irradiations with LLR should be systematically examined for its potential to be a viable immunotherapeutic treatment option for patients with systemic cancer.

Radiation dose-response model



Beneficial effects of low radiation

Medical practitioners used radiation ~1900 to ~1960, to:

- Eliminate metastases or slow cancer growth
- Accelerate healing of wounds
- Stop infections: gas gangrene, carbuncles and boils, sinus, inner ear, pneumonia
- Treat arthritis and other inflammatory conditions
- Treat swollen lymph glands
- Cure asthma
- New: reverse Alzheimer and Parkinson disease with no apparent increase of cancer incidence

How did Linear No Threshold model happen?

- Early geneticists (Muller) discovered mutations in germ cells of fruit flies by very high x-ray dose, at a high radiation intensity.
- When dose-rate and dose are both very high, then *mutation frequency* is ~ proportional to dose.
- Caspari used *low* dose-rate 2.5 R/day x 21 d (52.5 R), and saw a threshold; exposed flies *same* mutation freq. as unexposed flies
- Muller ignored the Caspari's evidence and proclaimed in his 1946 Nobel prize lecture: *there is no escape from the conclusion that there is no threshold*
- Genetics Panel of NAS BEAR Committee recommended in 1956 the linear no threshold LNT model to assess genetic mutation risk
- Regulators now use LNT model to assess cancer risk in normal somatic cells; but they have *no cancer evidence*

Contents lists available at

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Review article

LNTgate: How scientific misconduct by the U.S. NAS led to governments adopting LNT for cancer risk assessment

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ABSTRACT

This paper provides a detailed rebuttal to the letter of Beyea (2016) which offered a series of alternative interpretations to those offered in my article in Environmental Research (Calabrese, 2015a) concerning the role of the U.S. National Academy of Sciences (NAS) Biological Effects of Atomic Radiation (BEAR) I Committee Genetics Panel in the adoption of the linear dose response model for cancer risk assessment. Significant newly uncovered evidence is presented which supports and extends the findings of Calabrese (2015a), reaffirming the conclusion that the Genetics Panel should be evaluated for scientific misconduct for deliberate misrepresentation of the research record in order to enhance an ideological agenda. This critique documents numerous factual errors along with extensive and deliberate filtering of information in the Beyea letter (2016) that leads to consistently incorrect conclusions and an invalid general perspective.

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1. Introduction

Beyea (2016) offers a series of alternative interpretations to my conclusions (Calabrese, 2015a) on the acceptance of the LNT by the model for genetic risk assessment be switched from a threshold to a linear model in 1956 without discussion or debate based on meeting transcripts. During the second meeting of the Panel (February 5–6, 1956) in Chicago, Illinois panelist Dr. Tracey Son-

Calabrese on NAS Genetics Panel scandal

Commentary

Toxicology Research and Application

http://journals.sagepub.com/doi/pdf/10.1177/2397847317694998

Toxicology Research and Application
1–3
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SAGE

LNTgate: The ideological history of cancer risk assessment

Edward J Calabrese

Abstract

This commentary summarizes a spate of recent papers that provide historical evidence that the 1956 recommendation of the US National Academy of Sciences Biological Effects of Atomic Radiation I Genetics Panel to switch from a threshold to a linear dose–response model for risk assessment was an ideologically motivated decision based on deliberate falsification and fabrication of the research record. The recommendation by the Genetics Panel had far-reaching influence, affecting cancer risk assessment, risk communication strategies, community public health, and numerous medical practices in the United States and worldwide. This commentary argues that the toxicology, risk assessment, and regulatory communities examine this issue, addressing how these new historical evaluations affect the history and educational practices of these fields as well as carcinogen regulation.

Japanese repeat fruit fly study

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

Keiji Ogura, Junji Magae, Yasushi Kawakami and Takao Kosaka

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

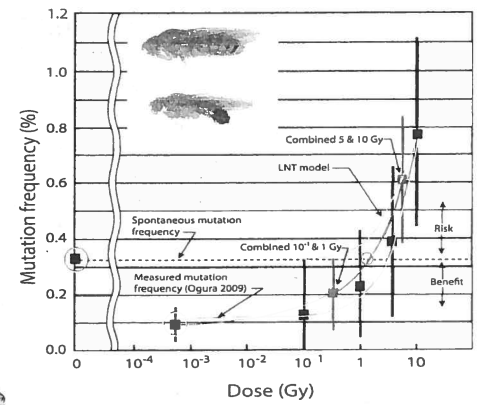
To determine whether the linear no-threshold (LNT) model for stochastic effects of ionizing radiation is applicable to very low-dose radiation at a low dose rate, we irradiated immature male germ cells of the fruit fly, *Drosophila melanogaster*, with several doses of ^{60}Co γ rays at a dose rate of 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 ($P < 0.05$). 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 apoptotic 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. The cancer induced risk was also considered to be proportional to radiation dose at high doses.

Much later, the mutagenic frequency in mouse spermatogonia was found to be dependent not only on the total radiation dose but also on the dose rate. 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 will cause incomplete repair and/or mis-repair, which will consequently result in mutations. Although Russell [illegible] that a low dose rate resulted in a low mutagenicity, the dose–response curve, a threshold dose [illegible] low dose rate dose rate.

In contrast, we reported previously that in the somatic mutation assay using *D. melanogaster*, there was a sex-linked dose at approximately [illegible] Gy and that [illegible] in the DNA repair function decreased the sex-linked recessive [illegible]. The existence of a threshold [illegible] in the sex-linked recessive lethal assay, using [illegible] germ cells (spermatogonia and spermatocytes), [illegible] affected, and it was inferred [illegible]

Germ cell mutation frequency - 22.4 mGy/h



Longevity is best measure of health effects

- The radiation scare: Any dose increases cancer risk
- Cancer is ideal antinuclear scare. Cancer is very complex, many causes, many confounding factors, not well understood, difficult to predict, and we dread it
- Best measure of health effects of radiation is longevity
- Cameron: early radiologists, nuclear shipyard workers
- Calabrese-Baldwin: low γ radiation increases lifespan of average mammals by 10 to 30% vs. unexposed ones
- Radiation stimulates their adaptive protection systems
- They act against the enormous spontaneous rate of cell damage (breathing oxygen) and against damage by all the other causes

Mortality of 1338 British radiologists 1897-1957

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

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.

Nuclear Shipyard Workers Study

John Cameron, APS, Physics and Society, Oct 2001

Table 1

Deaths from All Causes, Person-years and Death Rates[1] for high-dose nuclear workers ($NW_{>0.5\,rem}$); low-dose nuclear workers ($NW_{<0.5\,rem}$); and non-nuclear workers (NNW) (after Matanoski 1991 p. 333)

	> 0.5 rem	< 0.5 rem	Zero dose
Workers in Subset	27,872	10,348	32,510
Person-years	356,091	139,746	425,070
Deaths	2,215	973	3,745
Death Rates Per 1,000[2]	6.4	7.1	9.0
Death Rate (SMR)[3]	0.76	0.81	1.00
95% C.I.[4]	(0.73, 0.79)	(0.76, 0.86)	(0.97, 1.03)

1 Rates calculated per 1000 person-years.
2 Adjusted for deaths excluded from analysis due to unknown date of death.
3 Using age-calendar time specific rates for U.S. white males.
4 C.I. = 95% Confidence intervals.

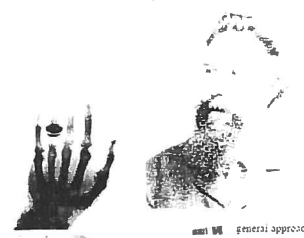
Inkret et al. Los Alamos Science #23, 1995

Radiation and Risk--A Hard Look at the Data

https://fas.org/sgp/othergov/doe/lanl/pubs.00325531.pdf

A Brief History of Radiation

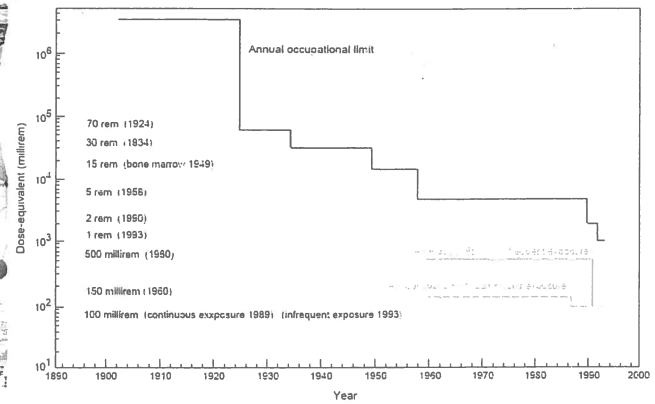
Health physics is concerned with protecting people from the harmful effects of ionizing radiation while allowing its beneficial use in medicine, science, and industry. Since the discovery of radiation and radioactivity 100 years ago, radiation protection standards and the philosophy governing those standards have evolved in somewhat discrete intervals. The changes have been driven by two factors—new information on the effects of radiation on biological systems and changing attitudes toward acceptable risk. The earliest limits were based on preventing the onset of such obvious effects as skin ulcerations that appeared after intense exposure to radiation fields. Later limits were based on preventing delayed effects such as cancer that had been observed in populations of people receiving high doses, particularly from medical exposures and from the atomic-bomb exposures in Hiroshima and Nagasaki.



During the evolution of standards, the general approach has been to rely on risk estimates that have

Tolerance dose rate in 1924 = 70 rem/year

1/10 erythema dose = 60 rem, with factor of safety of 10, per month



Original Article

Evidence That Lifelong Low Dose Rates of Ionizing Radiation Increase Lifespan in Long- and Short-Lived Dogs

Dose-Response
An International Journal
2017 1-5
The Author(s) 2017
DOI: 10.1177/1559325817692903
journals.sagepub.com/home/dos
SAGE

Jerry M. Cuttler[1], Ludwig E. Feinendegen[2], and Yehoshua Socol[3]

Abstract

After the 1956 radiation scare to stop weapons testing, studies focused on cancer induction by low-level radiation. Concern has shifted to protecting "radiation-sensitive individuals." Since longevity is a measure of health impact, this analysis reexamined data to compare the effect of dose rate on the lifespans of short-lived (5% and 10% mortality) dogs and on the lifespans of dogs at 50% mortality. The data came from 2 large-scale studies. One exposed 10 groups to different γ dose rates; the other exposed 8 groups to different lung burdens of plutonium. Reexamination indicated that normalized lifespans increased more for short-lived dogs than for average dogs, when radiation was moderately above background. This was apparent by interpolating between the lifespans of nonirradiated dogs and exposed dogs. The optimum lifespan increase appeared at 50 mGy/y. The threshold for harm (decreased lifespan) was 700 mGy/y for 50% mortality dogs and 1100 mGy/y for short-lived dogs. For inhaled α-emitting particulates, longevity was remarkably increased for short-lived dogs below the threshold for harm. Short-lived dogs seem more radiosensitive than average dogs and they benefit more from low radiation. If dogs model humans, this evidence would support a change to radiation protection policy. Maintaining exposures "as low as reasonably achievable" appears questionable.

Keywords

ionizing radiation, beagle dogs, individual sensitivity, longevity benefit, harmful thresholds, adaptive protection

International Dose-Response Society

Blood system very sensitive

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;

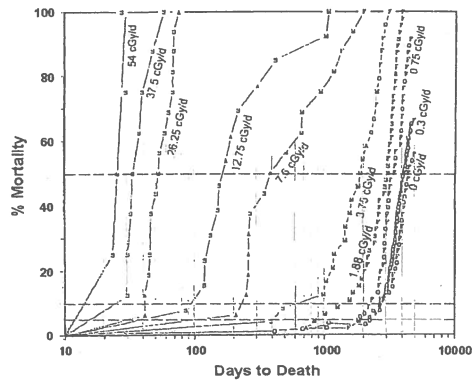
Ludwig E. Feinendegen — Heinrich-Heine-Universität Düsseldorf, Germany, and Brookhaven National Laboratory, Upton, NY, USA

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 10 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 patients, and 3) such findings in dogs chronically exposed in large lifespan 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 lines of functional cells that maintain homeostasis of hemopoiesis throughout life. Further studies particularly of selected hemopoietic stem cells may unravel the molecular-biology mechanisms underlying such apparent tolerance and adaptation.

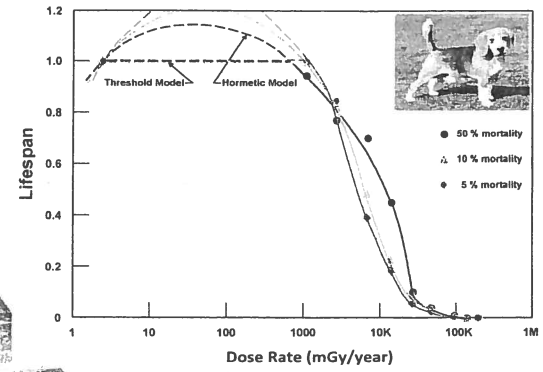
Blood system response to chronic radiation

- Reviewed histories of *humans* in 10 radiation accidents and studies on rats and dogs
- Radiation effect is a function of dose-rate *and* total dose
- Blood stem cells are usually very radiosensitive, but they tolerate and adapt to *chronic* radiation --- adapt better at *lower* dose rate.
- Get clones of functioning cells; maintain a lifetime of service
- Beagle dogs at 0.3 rad/day had *same* cancer rate as control dogs
- 1934 ICRP standard: tolerance dose of 0.2 r/day or 50 rad/y is okay
- Present ICRP recommendations (LNT & ALARA) not justified

Mortality curves for different Co-60 dose rates



Lifespan vs. cobalt-60 dose rate



RADIATION RESEARCH 170, 736–757 (2008)
0033-7587/08/$15.00/0
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Radiotoxicity of Inhaled $^{239}PuO_2$ in Dogs

Bruce A. Muggenburg, Raymond A. Guilmette, Fletcher F. Hahn, Joseph H. Diel, Jon L. Mauderly, Steven K. Seilkop* and Bruce B. Boecker*

*Lovelace Respiratory Research Institute, Albuquerque, New Mexico 87108; and *Battelle, 505 King Avenue, Columbus, Ohio 43201

Muggenburg, B. A., Guilmette, R. A., Hahn, F. F., Diel, J. H., Mauderly, J. L., Seilkop, S. K. and Boecker, B. B. Radiotoxicity of Inhaled $^{239}PuO_2$ in Dogs. *Radiat. Res.* 170, 736–757 (2008).

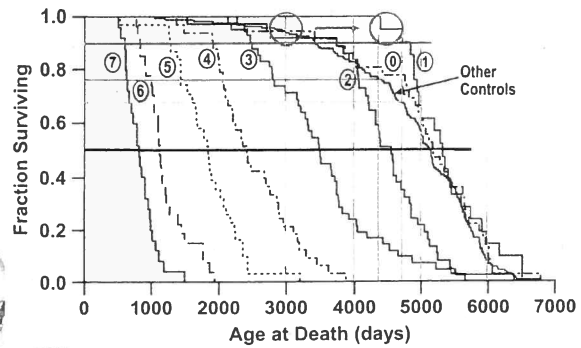
Beagle dogs inhaled graded exposure levels of insoluble plutonium dioxide ($^{239}PuO_2$) aerosols in one of three monodisperse particle sizes at the Lovelace Respiratory Research Institute (LRRI) to study the life-span health effects of different degrees of α-particle dose non-uniformity in the lung. The primary noncarcinogenic effects seen were lymphopenia, atrophy and fibrosis of the thoracic lymph nodes, and radiation pneumonitis and pulmonary fibrosis. Radiation pneumonitis/pulmonary fibrosis occurred from 105 days to more than 11 years after exposure, with the lowest associated α-particle dose being 5.9 Gy. The primary carcinogenic effects also occurred almost exclusively in the lung because of the short range of the α-particle emissions. The earliest lung cancer was

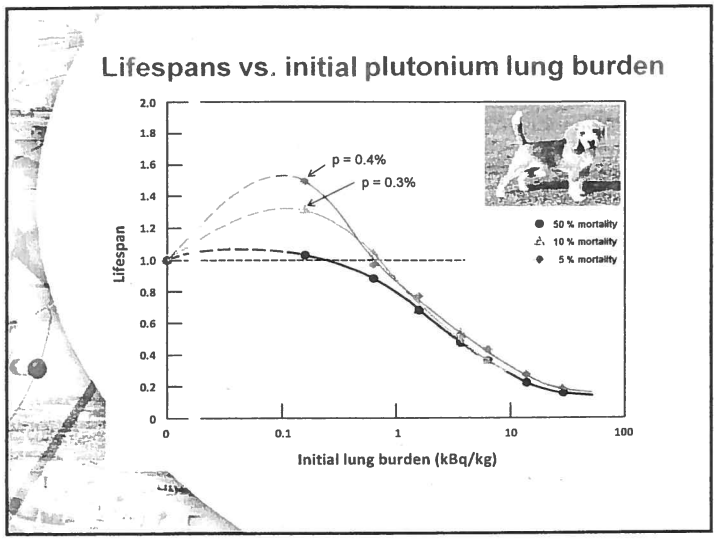
erations the possibility of plutonium environmental exposure exists through a severe reactor accident such as that at Chernobyl, various nuclear weapons testing activities, and waste disposal practices at various nuclear sites. Of increasing concern is the possible use by terrorists of ^{239}Pu in an improvised nuclear device (IND) or in a radiological dispersal device (RDD). The inventories of ^{239}Pu that exist around the world are mainly in the metallic or dioxide form.

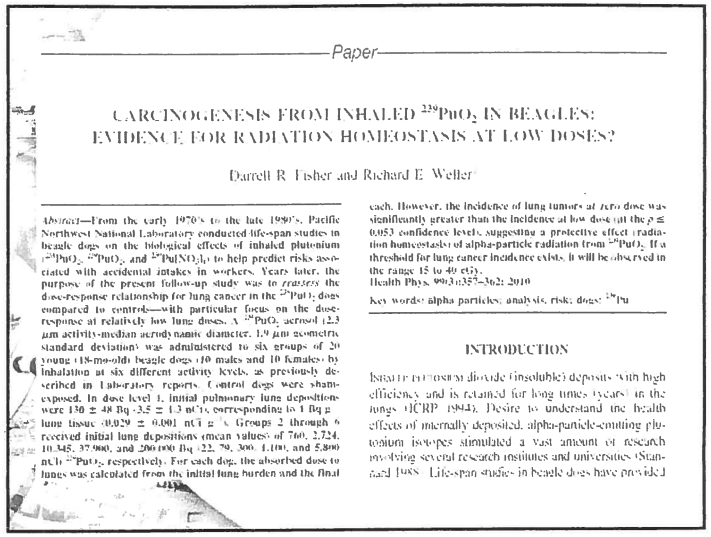
Pu has a radioactive half-life of about 24,000 years and decays primarily by α-particle emissions. Due to its abundance and long half-life, accidental and intentional human exposures continue to be important concerns.

In the early years after plutonium was discovered, data on the possible long-term health effects in humans were absent. Therefore, numerous studies of the dosimetry and health effects of internally deposited ^{239}Pu were conducted in laboratory animals since the discovery more than 60

Fraction surviving different Pu lung burdens







Paper

CARCINOGENESIS FROM INHALED $^{239}PuO_2$ IN BEAGLES: EVIDENCE FOR RADIATION HOMEOSTASIS AT LOW DOSES?

Darrell R. Fisher and Richard E. Weller

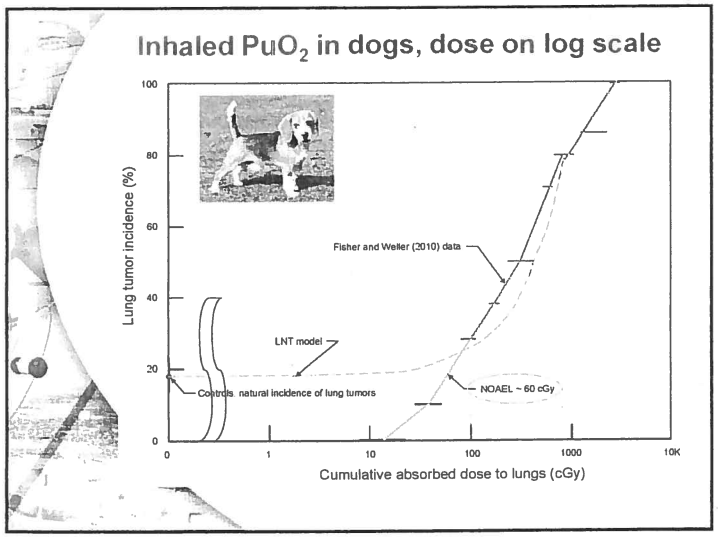
Abstract—From the early 1970's to the late 1990's, Pacific Northwest National Laboratory conducted life-span studies in beagle dogs on the biological effects of inhaled plutonium ($^{239}PuO_2$, $^{238}PuO_2$, and $^{239}Pu(NO_3)_4$) to help predict risks associated with accidental intakes in workers. Years later, the purpose of the present follow-up study was to *reassess* the dose-response relationship for lung cancer in the $^{239}PuO_2$ dose compared to controls—with particular focus on the dose-response at relatively low lung doses. A $^{239}PuO_2$ aerosol (2.3 µm activity-median aerodynamic diameter, 1.9 µm geometric standard deviation) was administered to six groups of 20 young (18-mo-old) beagle dogs (10 males and 10 females) by inhalation at six different activity levels, as previously described in Laboratory reports. Control dogs were sham-exposed. In dose level 1, initial pulmonary lung depositions were 136 ± 48 Bq (3.5 ± 1.3 nCi), corresponding to 1 Bq g^{-1} lung tissue (0.029 ± 0.001 nCi g^{-1}). Groups 2 through 6 received initial lung depositions (mean values) of 760, 2,724, 10,345, 37,900, and 200,000 Bq (22, 79, 300, 1,100, and 5,800 nCi) $^{239}PuO_2$, respectively. For each dog, the absorbed dose to lungs was calculated from the initial lung burden and the final each. However, the incidence of lung tumors at zero dose was significantly greater than the incidence at low dose (at the $p \leq 0.053$ confidence level), suggesting a protective effect (radiation homeostasis) of alpha-particle radiation from $^{239}PuO_2$. If a threshold for lung cancer incidence exists, it will be observed in the range 15 to 40 cGy.

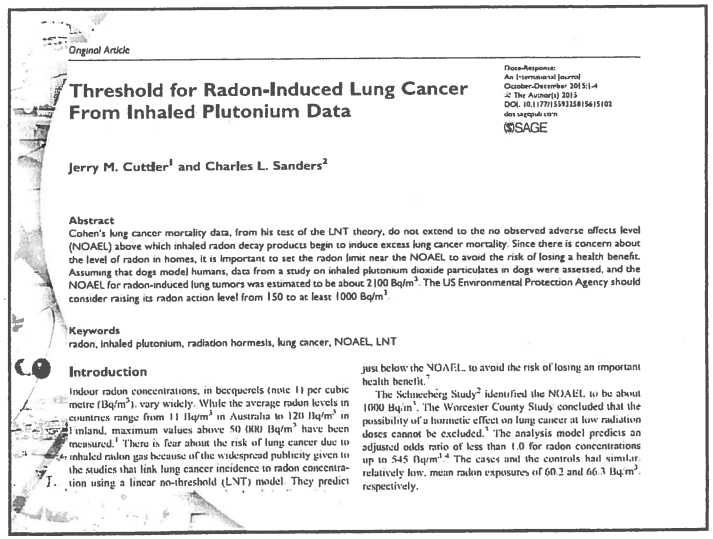
Health Phys. 98(3):357–362; 2010

Key words: alpha particles; analysis; risk; dose; ^{239}Pu

INTRODUCTION

Inhaled plutonium dioxide (insoluble) deposits with high efficiency and is retained for long times (years) in the lungs (ICRP 1994). Desire to understand the health effects of internally deposited, alpha-particle-emitting plutonium isotopes stimulated a vast amount of research involving several research institutes and universities (Stannard 1988). Life-span studies in beagle dogs have provided





Original Article

Threshold for Radon-Induced Lung Cancer From Inhaled Plutonium Data

Dose-Response: An International Journal
October-December 2015:1-4
© The Author(s) 2015
DOI: 10.1177/1559325815615102
dose-response.com
SAGE

Jerry M. Cuttler[1] and Charles L. Sanders[2]

Abstract
Cohen's lung cancer mortality data, from his test of the LNT theory, do not extend to the no observed adverse effects level (NOAEL) above which inhaled radon decay products begin to induce excess lung cancer mortality. Since there is concern about the level of radon in homes, it is important to set the radon limit near the NOAEL to avoid the risk of losing a health benefit. Assuming that dogs model humans, data from a study on inhaled plutonium dioxide particulates in dogs were assessed, and the NOAEL for radon-induced lung tumors was estimated to be about 2100 Bq/m^3. The US Environmental Protection Agency should consider raising its radon action level from 150 to at least 1000 Bq/m^3.

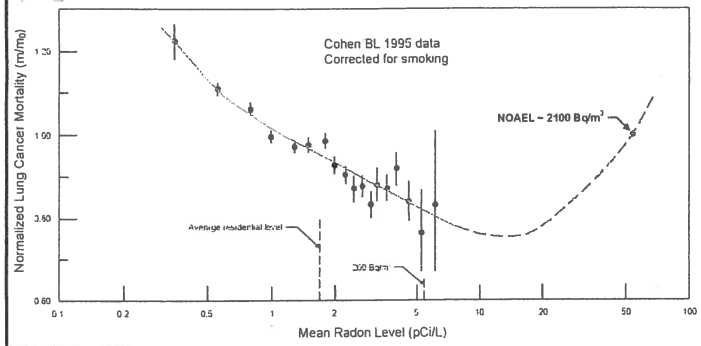
Keywords
radon, inhaled plutonium, radiation hormesis, lung cancer, NOAEL, LNT

Introduction

Indoor radon concentrations, in becquerels (note 1) per cubic metre (Bq/m^3), vary widely. While the average radon levels in countries range from 11 Bq/m^3 in Australia to 120 Bq/m^3 in Finland, maximum values above 50 000 Bq/m^3 have been measured.[1] There is fear about the risk of lung cancer due to inhaled radon gas because of the widespread publicity given to the studies that link lung cancer incidence to radon concentration using a linear no-threshold (LNT) model. They predict just below the NOAEL, to avoid the risk of losing an important health benefit.[7]

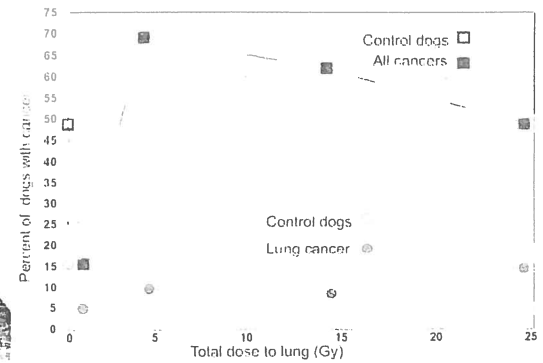
The Schneeberg Study[2] identified the NOAEL to be about 1000 Bq/m^3. The Worcester County Study concluded that the possibility of a hormetic effect on lung cancer at low radiation doses cannot be excluded.[3] The analysis model predicts an adjusted odds ratio of less than 1.0 for radon concentrations up to 545 Bq/m^3.[4] The cases and the controls had similar, relatively low, mean radon exposures of 60.2 and 66.3 Bq/m^3, respectively.

Inhaled radon in homes

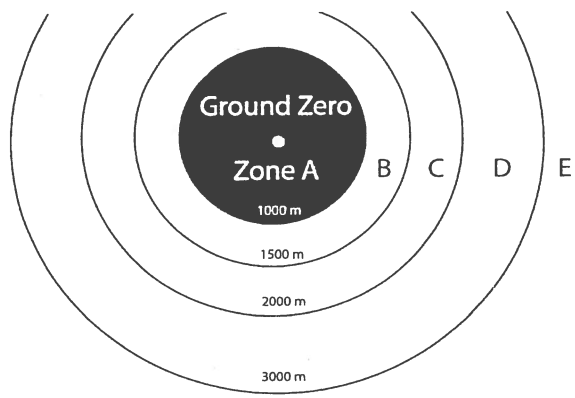


Brooks-2009: Summary of cancer frequency

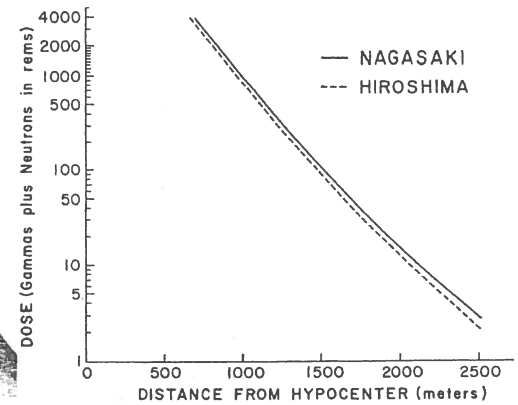
for inhaled beta-gamma emitting ^{90}Sr, ^{144}Ce, ^{91}Y and ^{90}Y



Hiroshima atomic bomb survivor zones



Radiation dose vs. distance from ground zero



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.

17 May 1957, Volume 125, Number 3255 SCIENCE

Leukemia and Ionizing Radiation

E. B. Lewis

Quantitative estimates of the genetic effects of ionizing radiation on human beings have been carried out by a number of investigators (1–3). Estimates of this kind involve extrapolating from induced mutation rates in such organisms as

radiostrontium is outlined. Certain properties of the disease, relevant to the radiation studies, are presented first.

Description of the Disease

The male and female crude death rates in that population were 79 and 58 per million per year, respectively, in 1954 (10). The observed increase in death rate from this disease may be partly due to improvements in diagnosis. Other factors may also be responsible, such as the increased exposure of the population to ionizing radiations employed in medicine and dentistry, as was recently discussed by Dameshek and Gunz (12).

MacMahon and Clark (13) have recently studied the spontaneous incidence of the common forms of leukemia. They have attempted to determine the total number of valid cases diagnosed among residents of the borough of Brooklyn from 1943 to 1952, inclusive. In this study the over-all ratio of acute to chronic forms among the white population was nearly 1/1 (726/732), but there were marked differences in the incidence

Epidemiological trick to "average the data"

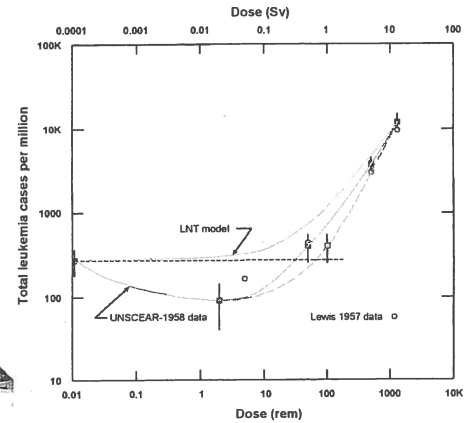
E.B. Lewis stated in his paper:

"Since the majority of the population in zone D (from 2000 meters on) was beyond 2500 meters, the average dose is under 5 rem and is thus so low that zone D can be treated as if it were a "control" zone."

His averaging of the data in the zones D and E concealed the evidence of a dose threshold.

Threshold level at ~ 50 rem (500 mSv)

J-curve, not LNT model



https://www.esciencecentral.org/journals/leukemia-and-ionizing-radiation-revisited-2329-6917-1000202.php?aid=65327

Leukemia

Cuttler and Welsh, J Leuk 2015, 3 –

Brief Report Open Access

Leukemia and Ionizing Radiation Revisited

Jerry M. Cuttler* and James S. Welsh

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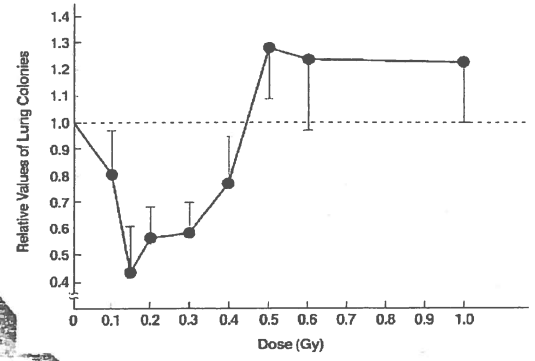
Abstract

A world-wide radiation health scare was created in the late 1950s to stop the testing of atomic bombs and block the development of nuclear energy. In spite of the large amount of evidence that contradicts the cancer predictions this fear continues. It impairs the use of low radiation doses in medical diagnostic imaging and radiation therapy. This brief article revisits the second of two key studies, which revolutionized radiation protection, and identifies a serious error that was missed. This error in analyzing the leukemia incidence among the 195 000 survivors, in the combined exposed populations of Hiroshima and Nagasaki, invalidates use of the LNT model for assessing the risk of cancer from ionizing radiation. The threshold acute dose for radiation-induced leukemia, based on about 96 800 humans, is identified to be about 50 rem, or 0.5 Sv. It is reasonable to expect that the thresholds for other cancer types are higher than this level. No predictions or hints of excess cancer risk (or any other health risk) should be made for an acute exposure below this value until there is scientific evidence to support the LNT hypothesis.

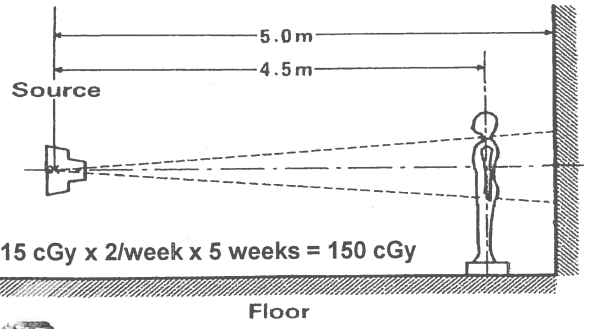
Results of one Sakamoto study

Spontaneous lung metastasis vs. total-body dose

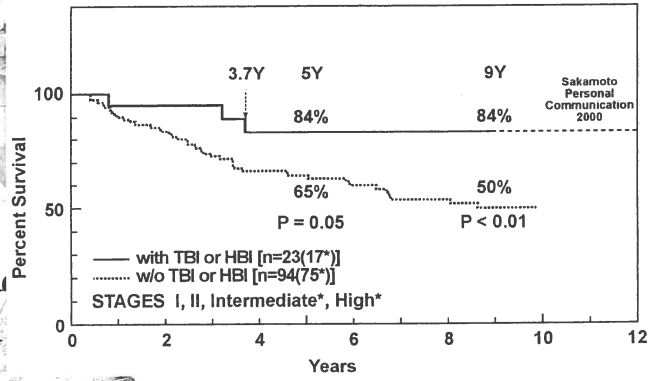


Source – patient schema for half-body LDR

"Observed the total removal of tumors in all regions of the body of a patient with advanced ovarian cancer."

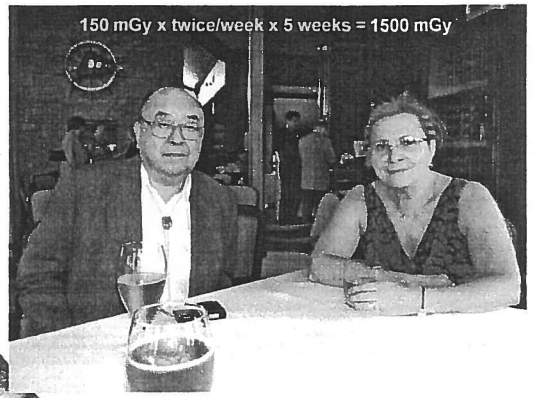


HBI or TBI for non-Hodgkin's lymphoma



LDR therapy for Hurthle cell carcinoma

HB-LDI therapy; prophylaxis against cancer



Original Article

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DOI: 10.1177/1559325817697531
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Treatment of Cancer and Inflammation With Low-Dose Ionizing Radiation: Three Case Reports

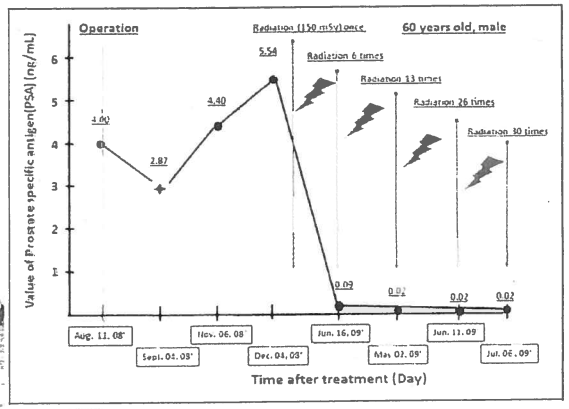
http://journals.sagepub.com/doi/pdf/10.1177/1559325817697531

Shuji Kojima[1], Mitsutoshi Tsukimoto[1], Noriko Shimura[2], Hironobu Koga[3], Akishisa Murata[3], and Tsuyoshi Takara[4]

Abstract

There is considerable evidence from experimental studies in animals, as well as from clinical reports, that low-dose radiation hormesis is effective for the treatment of cancer and ulcerative colitis. In this study, we present 3 case reports that support the clinical efficacy of low-dose radiation hormesis in patients with these diseases. First, a patient with prostate cancer who had undergone surgical resection showed a subsequent increase in prostate-specific antigen (PSA). His PSA value started decreasing immediately after the start of repeated low-dose X-ray irradiation treatment and remained low thereafter. Second, a patient with prostate cancer with bone metastasis was treated with repeated low-dose X-ray irradiation. His PSA level decreased to nearly normal within 3 months after starting the treatment and remained at the low level after the end of hormesis treatment. His bone metastasis almost completely disappeared. Third, a patient with ulcerative colitis showed a slow initial response to repeated low-dose irradiation treatment using various modalities, including drinking radon-containing water, but within 8 months his swelling and bleeding had completely disappeared. After 1 year, the number of bowel movements had become normal. Interest in the use of radiation hormesis in clinical practice is increasing, and we hope that these case reports will encourage further clinical investigations.

Recurrent Prostate Cancer



Treatment of Prostate Cancer with LD Radiation

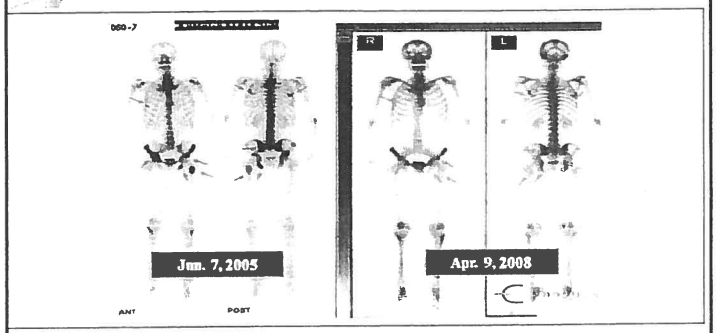
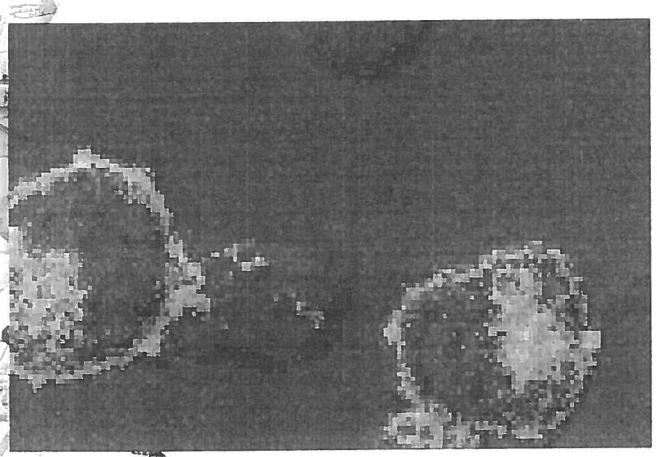
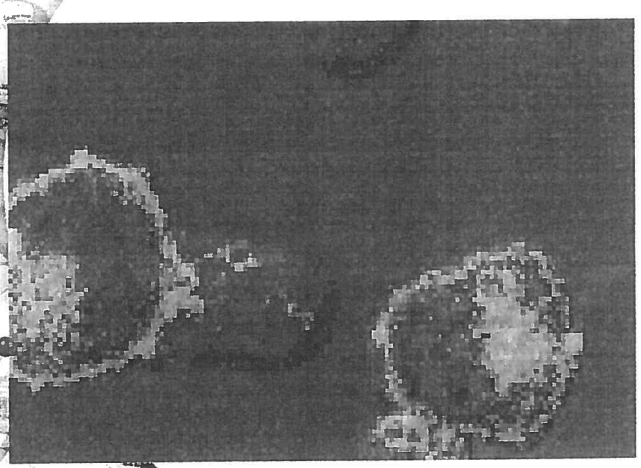


Figure 4. Bone scintigraphy with 99mTc-hydroxymethylene diphosphonate (^{99m}Tc-HMDP) of the patient with prostate cancer with bone metastasis before and after radiation hormesis treatment.

Shu-Zheng Liu and Jerry Cuttler in Mississauga





Journal of Cancer Clinical Trials

Cuttler et al., J Cancer Clin Trials 2016, 1:2

Review Open Access

Adjuvant Therapy for Resected Exocrine Pancreatic Cancer by Half-Body Low-Dose Irradiation

Jerry M. Cuttler[1*], Philippe Garzon[2], Ron E. J. Mitchel[3], Ludwig E. Feinendegen[4], Kiyohiko Sakamoto[5] and James S. Welsh[6]

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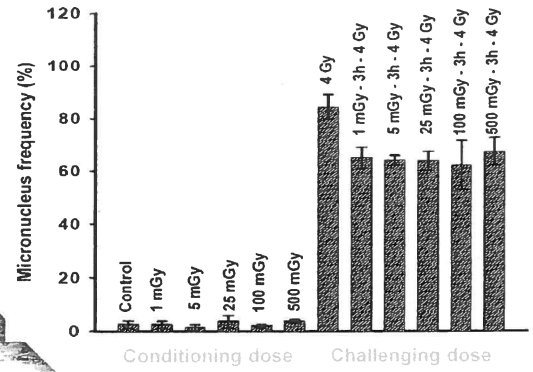
Abstract

After surgery, pancreatic cancer has an extremely high rate of systemic recurrence and a very high rate of local recurrence, more than 90 percent and 20 percent, respectively. Conventional adjuvant therapy prolongs median survival, 28 versus 15 months, by eliminating many of the metastases before they grow into new tumours. In an effort to improve outcomes, the authors recommend evaluating half-body low-dose irradiation (HB LDI) therapy because limited clinical studies have shown HB LDI to be successful as an adjuvant treatment for different types of cancer. Each dose fraction in LDI therapy is 15 cGy, about 13 times below the 200 cGy dose fraction employed in

Adaptive response

Low radiation dose up-regulates cell repair capability

Decreases risk of 4 Gy challenging dose



Original Article

Treatment of Alzheimer Disease With CT Scans: A Case Report

Dose-Response
An International Journal
April-June 2016 1-7
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DOI: 10.1177/1559325816640073
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SAGE

Jerry M. Cuttler[1], Eugene R. Moore[2], Victor D. Hosfeld[3], and David L. Nadolski[4]

Abstract

Alzheimer disease (AD) primarily affects older adults. This neurodegenerative disorder is the most common cause of dementia and is a leading source of their morbidity and mortality. Patient care costs in the United States are about 200 billion dollars and will more than double by 2040. This case report describes the remarkable improvement in a patient with advanced AD in hospice who received 5 computed tomography scans of the brain, about 40 mGy each, over a period of 3 months. The mechanism appears to be radiation-induced upregulation of the patient's adaptive protection systems against AD, which partially restored cognition, memory, speech, movement, and appetite.

Keywords

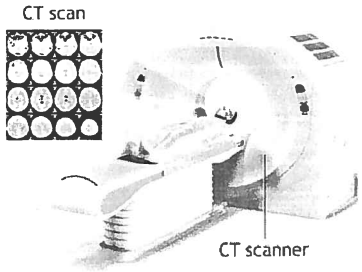
Alzheimer disease, CT scan, adaptive protection systems, ionizing radiation

Introduction

Alzheimer disease (AD) is a neurodegenerative disorder of uncertain cause and pathogenesis which primarily affects older adults. It accounts for more than 50% of the cases of dementia and is one of the leading sources of morbidity and mortality in

In patients with the typical form of the illness, deficits in other cognitive domains may appear with or after the development of memory impairment. Executive dysfunction and visuospatial impairment are often present relatively early, while deficits in language and behavioral symptoms often manifest later. These deficits develop and progress insidiously.

CT scanner



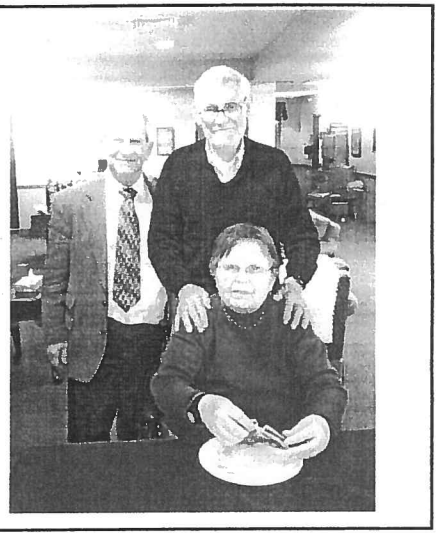
A.D.A.M.

Update on a Patient With Alzheimer Disease Treated With CT Scans

SAGE

Jerry M. Cutler[1], Eugene R. Moore[2], Victor D. Hosfeld[3], and David L. Nadolski[4]

Figure 1. Preservation of appetite of patient with Alzheimer disease (AD).



AD patient, spouse and author on Dec 4, 2015

Date	Interval (days)	Dose (mGy)
07/23/2015		82**
08/06/2015	14	39
08/20/2015	14	47
10/01/2015	42	39
02/24/2016	146	40
06/22/2016	119	40
10/27/2016	127	40
12/13/2016	47	40
01/24/2017	41	80**

** two CT scans

Treating Parkinson disease with CT scans

- On discovering the efficacy of CT scans for AD, the spouse requested a scan to alleviate his Parkinson disease
- After a scan Oct 6, 2015, he noticed the complete absence of tremors while sleeping (and waking at about 4 am)
- Decreased medication, Carbidopa/Levodopa 25/100 mg, from 6 to 2-3 pills/day
- Jun 13, 2016, received in-depth neuropsychological exam
- Based on experience receives booster scan every 4-weeks

Date and x-ray dose ($CTDI_{vol}$) of PD treatments

Date	Interval (days)	Dose (mGy)
10/06/2015		40
06/16/2016	253	40
07/13/2016	28	40
09/29/2016	51	40
11/21/2016	80	40
12/21/2016	30	40

doi:10.1259/bjr/20130629

RADIOBIOLOGY SPECIAL FEATURE: COMMENTARY

What we know and what we don't know about cancer risks associated with radiation doses from radiological imaging

D J BRENNER, PhD, DSc

Professor David J. Brenner

ABSTRACT

Quantifying radiation-induced cancer risks associated with radiological examinations is not easy, which has resulted in much controversy. We can clarify the situation by distinguishing between higher dose examinations, such as CT, positron emission tomography–CT or fluoroscopically guided interventions, and lower dose "conventional" X-ray examinations. For higher dose examinations, the epidemiological data, from atomic bomb survivors exposed to low doses and from direct epidemiological studies of paediatric CT, are reasonably consistent, suggesting that we do have a reasonable quantitative understanding of the individual risks: in summary, very small but unlikely to be zero. For lower dose examinations, we have very little data, and the situation is much less certain, however, the collective risk from these lower dose examinations is comparatively unimportant from a public health perspective.

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

1 Introduction
2 The Meaning of Absorbed Dose in the Low Dose Region
3 Primary Biological Interactions
4 Damage to DNA and its Repair
5 Hierarchy Level Responses in Biological Systems
6 Three Categories of Physiological Defenses of Complex Biological Systems
7 Low Dose Induced Adaptive Protections
8 Physiological Defense Against Cancer
9 Damage and Protection at the "Dose Probability Model" of Cancer Risk
10 Chronic Irradiation
11 Conclusions
References

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 non-radiogenic endogenous sources. Adaptive protections have a maximum after single tissue absorbed doses around 100–200 mGy and

Ludwig Feinendegen et al.

- Studies ignore spontaneous (endogenous) DNA *damage* rate
- Endogenous rate is very high compared with radiation-induced rate
- Average number of DNA alterations per average cell, per day

  Endogenous (mainly due to metabolic ROS): total ~ 10^6, DSB ~ 10^{-1}

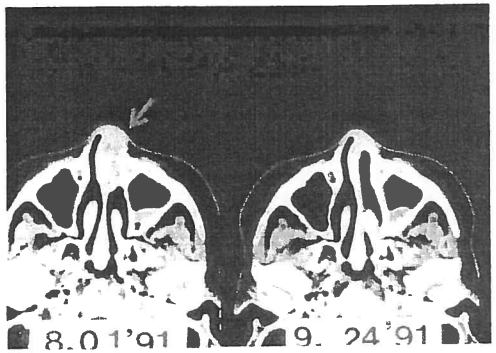
  Radiation-induced (1 mGy/yr, γ background): total ~ 10^{-2}, DSB ~ 10^{-4}
- Ratio of DNA alterations (endogenous/rad'n): total ~ 10^8, DSB ~ 10^3

Adapted from Pollycove and Feinendegen 2003

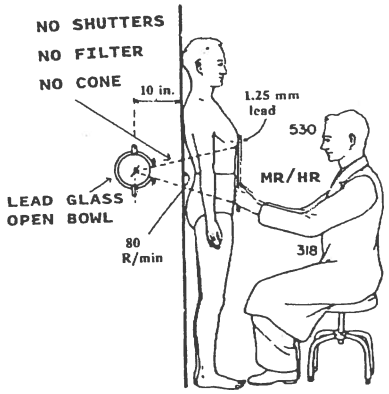
Ludwig Feinendegen et al. #2

- Low doses of radiation up-regulate adaptive protection systems
- *Fast defences* act immediately to remove toxins, repair molecules (DNA), remove/replace damaged cells and tissue, followed by ...
- *Delayed defences* of up-regulated adaptive systems (> 150 genes) that may last more than a year and protect against renewed toxic impacts from *both* radiation sources and non-radiation sources
- Adaptive protections are highly stimulated by 150 mGy acute dose
- Chronic or repetitive radiation initiates protection at lower level
- Adaptive protections reduce risks ⇨ less cancer extends life span

Abscopal effect 54 days after HB LDI



Fluoroscopy circa. 1930



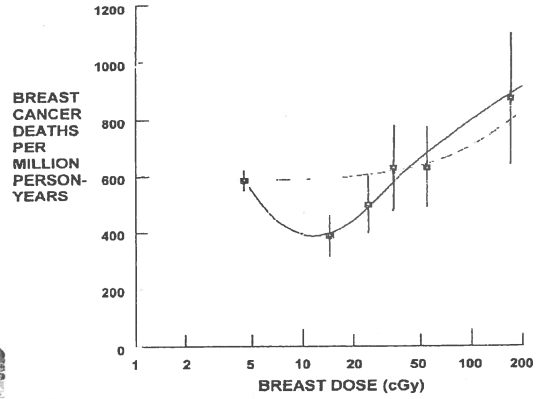
Canadian Breast Cancer Study

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)

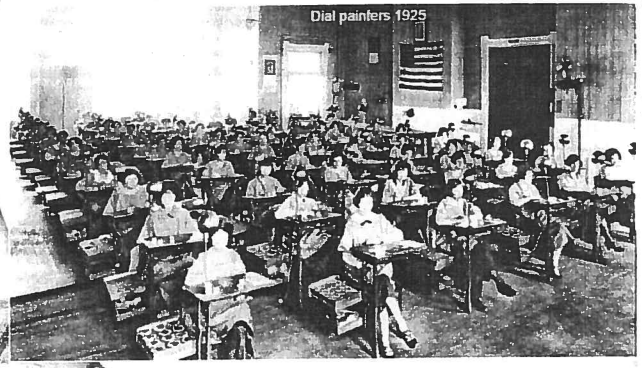
*The number of deaths is shown in parentheses. The calculations exclude the values for 10 years after the first exposure and have been standardized according to age at first exposure (10 to 14, 15 to 24, 25 to 34, and ≥35 years) and time since first exposure (10 to 14, 15 to 24, 25 to 34, and ≥35 years)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

Breast cancer mortality of TB patients



4133 identified radium dial painters in USA

Bone cancer *threshold* at 10 Gy (1000 rad) radium alpha radiation



1000cGy threshold radium-induced bone cancer

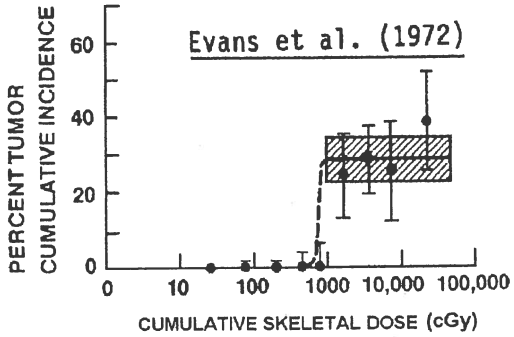


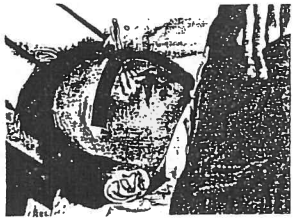
Fig. 11. Cumulative bone sarcoma incidence in people exposed to ^{226}Ra as a function of cumulative dose to the skeleton as reported by Evans et al. (1972).

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)



http://www.cancer.gov/cancertopics/factsheet/Risk/nasopharyngeal-radium

National Cancer Institute

No link to any disease

Reviewed: January 10, 2003

Nasopharyngeal Radium Irradiation (NRI) and Cancer: Fact Sheet

Key Points

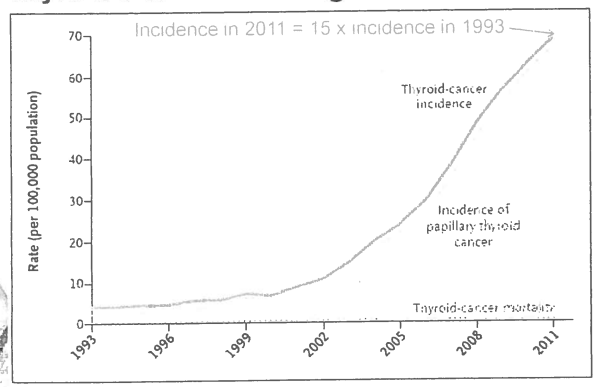
- Nasopharyngeal radium irradiation (NRI) was widely used from 1940 through 1970 to treat ear dysfunctions in children and military personnel. Use of NRI was stopped when concern arose about possible adverse effects, including cancer.
- The purpose of NRI was to shrink swollen tissue in the nasopharyngeal cavity—the opening behind the nose and mouth. The treatment involved inserting a radioactive compound through the nostril into the nasopharyngeal opening for short periods of time. Some radiation exposure to the salivary, thyroid, and pituitary glands, and to brain tissue also occurred during this process.
- NRI was used in several European countries, Canada, and the United States. In the United States, it is estimated that between 0.5 million and 2.5 million children and at least 5,000 military personnel were treated with NRI.
- Children are considered to be the most vulnerable to radiation-related cancers.
- At this time, worldwide studies have not confirmed a definite link between NRI exposure and any disease.

Lubin et al. 2017 Thyroid cancer after childhood LDR

This study has serious deficiencies:

- Genetic differences in children in cohort not corrected for
- Confounding factors affecting cancer not controlled
- Incidence naturally-occurring occult thyroid cancer not corrected
- Screening results in enormous over-diagnosis
- Used LNT model, which has been discredited for past 8 years
- Cell mutation rate due to radiation is negligible vs. natural rate
- LDR stimulates protection systems; it lowers mutation frequency
- Immune system destroys cancer cells, LDR stimulates immunity
- No cases of childhood thyroid cancer for x-ray dose < 200 rad
- Threshold for leukemia ~ 50 rem. Thyroid cancer threshold is higher
- Hyperthyroidism treated with I-131 for 70 years. No excess cancers

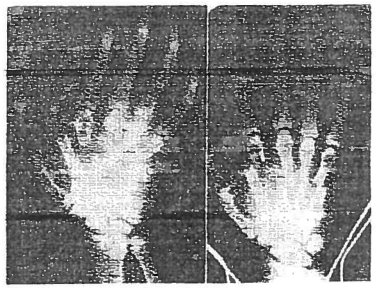
Thyroid cancer screening and overdiagnosis



Thyroid-Cancer Incidence and Related Mortality in South Korea, 1993–2011.

Ahn et al. N Engl J Med. 2014; 371(19):1765-7

LDR cures gas gangrene infections



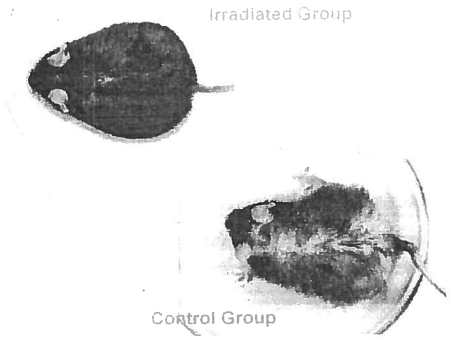
Figs. 7–8. Case 1: Severe hand injury, with multiple compound fractures and some gas in tissues (left). Fig. 8 (right) shows same hand a few days after prophylactic x-ray irradiation: no gas in the tissues, no infection, hand on way to complete recovery.

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

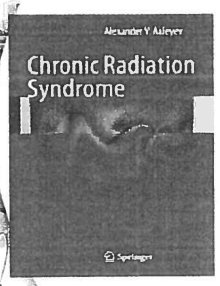
those which do not appear until three or four days have elapsed. It is evident from Figure 6 that the second, third, and

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

Irradiated diabetic mice are healthier and live longer



Effects of Mayak releases on residents



Residents ingested Mayak radioactive discharges (into Techa River in early 1950s) UNSCEAR used this as opportunity to study dose-effect of long-term irradiation.

Incidence of mortality from leukemia and cancer of CRS people did not exceed cancer incidence for exposed people without CRS and for Russia as a whole

Threshold for chronic radiation syndrome is a dose of 700-1000 mGy per year

Is it safe to store used nuclear fuel on site?

Ted Rockwell said it best
https://www.youtube.com/watch?v=bD4bUFrpFVA

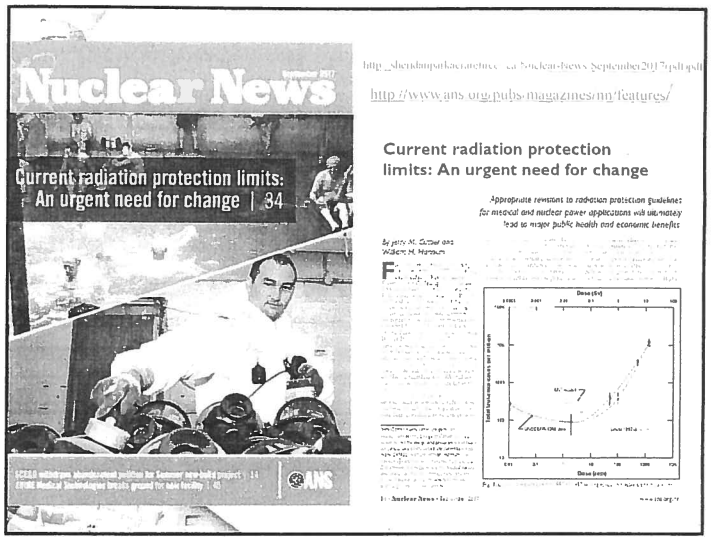
"You can go to two different places for the answer on that. If you go to the scientists, whose job it is to come up with problems and who don't have any work to do if they can't find problems, you'll find there are a lot of problems.

But, if you go out into the real world, who do you know that's ever been hurt by nuclear waste? ..."

Conclusions

- Social concern about nuclear energy safety is caused by policy that links *human-made* radiation to risk of cancer
- Radiation scare of 1956 (to stop atomic bombs) continues
- Authorities are ignoring beneficial effects of low doses
- Need policy change for acceptance of nuclear energy and medical radiation diagnostics: x rays, CT scans
- The 1934 ICRP "tolerance dose" of 700 mGy/year is still okay for radiation protection

Recommendations

- Scientific societies to discuss radiation health benefits
- Regulatory bodies and health organizations should examine the data and use The Scientific Method
- Stop calculating low radiation-induced cancer risk
- Develop/implement public communication programs
- Learn 3 lessons from Chernobyl and Fukushima:
  - Severe accidents result in *low* radiation dose-rate levels
  - Long-term evacuations are not appropriate when no risk
  - Emergency precautionary actions cause stress and deaths

Raise radiation level threshold for evacuation from 20 to 700 mGy/year (2 to 70 rad/year)