“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.
91 μSv/h x 8766 h/y = 798 mSv/y ~ natural HBRAs
Accumulated Radiation Dose around Fukushima Dai-ichi

Distribution Map of Accumulated Radiation Dose up to Mar 11, 2012
(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.

<table>
<thead>
<tr>
<th>Place</th>
<th>Distance from Fukushima Daiichi</th>
<th>Accumulated Radiation (Estimation) Unit: mSV</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>NW 24km</td>
<td>235.4</td>
</tr>
<tr>
<td>b</td>
<td>NW 31km</td>
<td>188.6</td>
</tr>
<tr>
<td>c</td>
<td>NW 31km</td>
<td>110.2</td>
</tr>
<tr>
<td>d</td>
<td>WNW 29km</td>
<td>56.2</td>
</tr>
<tr>
<td>e</td>
<td>WNW 30km</td>
<td>48.2</td>
</tr>
<tr>
<td>f</td>
<td>WNW 30km</td>
<td>24.2</td>
</tr>
<tr>
<td>g</td>
<td>WNW 32km</td>
<td>18</td>
</tr>
<tr>
<td>h</td>
<td>NW 25km</td>
<td>11</td>
</tr>
<tr>
<td>i</td>
<td>NW 33km</td>
<td>61.7</td>
</tr>
<tr>
<td>j</td>
<td>NW 39km</td>
<td>34.8</td>
</tr>
<tr>
<td>k</td>
<td>NW 36km</td>
<td>26.3</td>
</tr>
<tr>
<td>l</td>
<td>NW 44km</td>
<td>10</td>
</tr>
<tr>
<td>m</td>
<td>WNW 34km</td>
<td>24.2</td>
</tr>
<tr>
<td>n</td>
<td>WNW 40km</td>
<td>19.6</td>
</tr>
<tr>
<td>o</td>
<td>NNW 30km</td>
<td>15.6</td>
</tr>
<tr>
<td>p</td>
<td>NNW 25km</td>
<td>11.9</td>
</tr>
<tr>
<td>q</td>
<td>NW 48km</td>
<td>21.2</td>
</tr>
<tr>
<td>r</td>
<td>NW 46km</td>
<td>16</td>
</tr>
<tr>
<td>s</td>
<td>NW 56km</td>
<td>10.6</td>
</tr>
<tr>
<td>t</td>
<td>W 60km</td>
<td>10.1</td>
</tr>
</tbody>
</table>

Data from MEXT
The basic problem of nuclear energy

• People are afraid of nuclear power plants because we tell everyone that any radiation exposure they receive increases their risk of fatal cancer.
• This is 1950s 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 dose threshold.
• A low dose is beneficial; a high dose (above threshold) is harmful.
• Longevity is best measure of health effect, not cancer.
Radiation dose-response model

Health effects

Absorbed radiation dose or dose-rate

Optimum

Radiation-induced beneficial effects

NOAEL

Control group (natural radiation)

Radiation-induced harmful effects

NOAEL: no observed adverse effects level
How did Linear No Threshold model happen?

- Early geneticists (Muller) saw mutations in germ cells of fruit flies caused by very high x-ray dose at a high rate.
- When the dose-rate and the 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); observed a threshold; exposed flies same as unexposed
- Muller ignored the Caspari’s evidence and proclaimed in his 1946 Nobel prize political lecture that there is: no escape from the conclusion that there is no threshold
- Genetics Panel of NAS BEAR Committee recommended in 1956 the LNT model to assess risk of genetic harm
  Regulators use LNT model to assess risk of cancer in normal somatic cells; they had no cancer evidence
On the origins of the linear no-threshold (LNT) dogma by means of untruths, artful dodges and blind faith

Edward J. Calabrese *

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Mutation
LNT
Ionizing radiation

ABSTRACT

This paper is an historical assessment of how prominent radiation geneticists in the United States during the 1940s and 1950s successfully worked to build acceptance for the linear no-threshold (LNT) dose–response model in risk assessment, significantly impacting environmental, occupational and medical exposure standards and practices to the present time. Detailed documentation indicates that actions taken in support of this policy revolution were ideologically driven and deliberately and deceptively misleading; that scientific records were artfully misrepresented; and that people and organizations in positions of public trust failed to perform the duties expected of them. Key activities are described and the roles of specific individuals are documented. These actions culminated in a 1956 report by a Genetics Panel of the U.S. National Academy of Sciences (NAS) on Biological Effects of Atomic Radiation (BEAR). In this report the Genetics Panel recommended that a linear dose response model be adopted for the purpose of risk assessment, a recommendation that was rapidly and widely promulgated. The paper argues that current international cancer risk assessment policies are based on fraudulent actions of the U.S. NAS BEAR I Committee, Genetics Panel and on the uncritical, unquestioning and blind-faith acceptance by regulatory agencies and the scientific community.
Review article

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

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Keywords:
Linear dose response
Cancer risk assessment
Dose response
Hormesis
Mutation
Threshold dose response

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-
Who is more rational? public or nuclear industry

Industry says: “Radioactive waste is not very dangerous, but we are going to bury it 600 metres underground.” To reassure people?

Public’s rational response: This is most dangerous material humans ever produced. We don’t bury anything else 600 metres underground.

“Safety is the top priority.”

Public’s rational response: If safety really is more important than generating electricity or cost, then why not just stop doing it?

“We spent a fortune on a monitoring system that can detect radioactivity many thousands of times below danger levels.”

Public’s rational response: This cannot be true.

Either they wilfully wasted a vast amount of my money, or they are misleading us about the dangers involved.

NOBODY spends a fortune to detect something that does no harm.
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 and Takao Koana a,2

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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 60Co 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.
# Binomial statistics applied to fruit fly mutation data measured by Ogura et al. 2009

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

Mutation frequency for controls = **0.0032**
Germ cell mutation frequency - 22.4 mGy/h
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, etc.
- Treat arthritis and other inflammatory conditions
- Treat swollen lymph glands
- Cure pneumonia
- Cure asthma
- Cure or prevent Alzheimer disease (dementia)

with no apparent increase of cancer incidence
Longevity is best measure of health effects

- The radiation scare: Higher cancer risk with more dose
- Cancer is ideal antinuclear scare. It is very complex, many causes, confounding factors, uncertainties, 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: gamma radiation increases median lifespan of low-dose group by 10 to 30% over “controls”
- Radiation stimulates the adaptive protection systems
- They act against the enormous spontaneous rate of cell damage 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

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Observed (O) and expected (E) numbers of deaths</th>
<th>Entry prior to 1921</th>
<th>Entry after 1920</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>O</td>
<td>E</td>
</tr>
<tr>
<td>All causes</td>
<td></td>
<td>(1)</td>
<td>334.42</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2)</td>
<td>308.03</td>
</tr>
<tr>
<td>All neoplasms</td>
<td></td>
<td>(1)</td>
<td>49.11</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2)</td>
<td>43.07</td>
</tr>
<tr>
<td>Other causes</td>
<td></td>
<td>(1)</td>
<td>285.31</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2)</td>
<td>264.96</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(3)</td>
<td>292.58</td>
</tr>
</tbody>
</table>

(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.

One sided in direction of difference.

*P < 0.05  
**P < 0.01  
***P < 0.001
### 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)

<table>
<thead>
<tr>
<th></th>
<th>High dose</th>
<th>Low dose</th>
<th>Zero dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Workers in Subset</td>
<td>27,872</td>
<td>10,348</td>
<td>32,510</td>
</tr>
<tr>
<td>Person-years</td>
<td>356,091</td>
<td>139,746</td>
<td>425,070</td>
</tr>
<tr>
<td>Deaths</td>
<td>2,215</td>
<td>973</td>
<td>3,745</td>
</tr>
<tr>
<td>Death Rates Per 1,000(^2)</td>
<td>6.4</td>
<td>7.1</td>
<td>9.0</td>
</tr>
<tr>
<td>Death Rate (SMR)(^3)</td>
<td><strong>0.76</strong></td>
<td>0.81</td>
<td><strong>1.00</strong></td>
</tr>
<tr>
<td>95% C.I.(^4)</td>
<td>(0.73, 0.79)</td>
<td>(0.76, 0.86)</td>
<td>(0.97, 1.03)</td>
</tr>
</tbody>
</table>

---

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.
Blood system very sensitive

HEMPOIETIC 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 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.
Blood system response to chronic radiation

- 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 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
Continuous Co-60 irradiation of dogs

0.3 cGy/d = 1100 mGy/year = 110 rad/year

Blood counts of 0.3 cGy/d same as 0 cGy/d

Fatal tumors of 0.3 cGy/d same as 0 cGy/d
<table>
<thead>
<tr>
<th>Dose Rate (cGy/day)</th>
<th>Dose Rate (mGy/year)</th>
<th>Lifespan - days (50% mortality)</th>
<th>Lifespan (normalized)</th>
</tr>
</thead>
<tbody>
<tr>
<td>background</td>
<td>$2.4 \times 10^0$</td>
<td>4300</td>
<td>1.00</td>
</tr>
<tr>
<td>0.3</td>
<td>$1.1 \times 10^3$</td>
<td>4100</td>
<td>0.95</td>
</tr>
<tr>
<td>0.75</td>
<td>$2.7 \times 10^3$</td>
<td>3300</td>
<td>0.77</td>
</tr>
<tr>
<td>1.88</td>
<td>$6.9 \times 10^3$</td>
<td>3000</td>
<td>0.70</td>
</tr>
<tr>
<td>3.75</td>
<td>$1.4 \times 10^4$</td>
<td>1900</td>
<td>0.44</td>
</tr>
<tr>
<td>7.5</td>
<td>$2.7 \times 10^4$</td>
<td>410</td>
<td>0.095</td>
</tr>
<tr>
<td>12.75</td>
<td>$4.7 \times 10^4$</td>
<td>160</td>
<td>0.037</td>
</tr>
<tr>
<td>26.25</td>
<td>$9.6 \times 10^4$</td>
<td>52</td>
<td>0.012</td>
</tr>
<tr>
<td>37.5</td>
<td>$1.4 \times 10^5$</td>
<td>32</td>
<td>0.0074</td>
</tr>
<tr>
<td>54</td>
<td>$2.0 \times 10^5$</td>
<td>24</td>
<td>0.0056</td>
</tr>
</tbody>
</table>
Median lifespan versus Co-60 radiation level

Threshold for shorter lifespan $\sim 700$ mGy/year

![Graph showing the relationship between normalized lifespan and dose rate. The graph indicates a threshold for shorter lifespan at around 700 mGy/year.]
Radiotoxicity of Inhaled $^{239}$PuO$_2$ in Dogs

Bruce A. Muggenburg, a Raymond A. Guilmette, a Fletcher F. Hahn, a Joseph H. Diel, a Joe L. Mauderly, a
Steven K. Seilkop b and Bruce B. Boecker a,1

a Lovelace Respiratory Research Institute, Albuquerque, New Mexico 87108; and b SKS Consulting Services, Siler City, North Carolina 27344


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 (LRRRI) to study the life-span health effects of different degrees of $\alpha$-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 $\alpha$-particle dose being 5.9 Gy. The primary carcinogenic effects also occurred almost exclusively in the lung because of the short range of the $\alpha$-particle emissions. The earliest lung cancer was

operations, 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. $^{239}$Pu has a radioactive half-life of about 24,000 years and decays primarily by $\alpha$-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 its discovery more than 60
Radiotoxicity of inhaled $^{239}$PuO$_2$ in beagle dogs
<table>
<thead>
<tr>
<th>Exposure Level</th>
<th>Initial Lung Burden kBq/kg</th>
<th>Lung Dose to Death cGy</th>
<th>Age to Death days</th>
<th>Normalized Lifespan 50% mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>0</td>
<td>0</td>
<td>5150</td>
<td>1.00</td>
</tr>
<tr>
<td>1</td>
<td>0.16</td>
<td>160</td>
<td>5316</td>
<td>1.03</td>
</tr>
<tr>
<td>2</td>
<td>0.63</td>
<td>620</td>
<td>4526</td>
<td>0.88</td>
</tr>
<tr>
<td>3</td>
<td>1.6</td>
<td>1300</td>
<td>3482</td>
<td>0.68</td>
</tr>
<tr>
<td>4</td>
<td>3.7</td>
<td>2400</td>
<td>2421</td>
<td>0.47</td>
</tr>
<tr>
<td>5</td>
<td>6.4</td>
<td>3500</td>
<td>1842</td>
<td>0.36</td>
</tr>
<tr>
<td>6</td>
<td>14</td>
<td>4500</td>
<td>1122</td>
<td>0.22</td>
</tr>
<tr>
<td>7</td>
<td>29</td>
<td>5900</td>
<td>807</td>
<td>0.16</td>
</tr>
</tbody>
</table>
Median lifespan versus $^{239}\text{PuO}_2$ lung burden
Carcinogenesis from inhaled $^{239}\text{PuO}_2$ 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 1980’s, Pacific Northwest National Laboratory conducted life-span studies in beagle dogs on the biological effects of inhaled plutonium ($^{238}\text{PuO}_2$, $^{239}\text{PuO}_2$, and $^{239}\text{Pu}[\text{NO}_3]_4$) 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 $^{239}\text{PuO}_2$ dogs compared to controls—with particular focus on the dose-response at relatively low lung doses. A $^{239}\text{PuO}_2$ 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 $130 \pm 48$ Bq (3.5 ± 1.3 nCi), corresponding to 1 Bq g$^{-1}$ lung tissue ($0.029 \pm 0.001$ 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) $^{239}\text{PuO}_2$, 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 $^{239}\text{PuO}_2$. If a threshold for lung cancer incidence exists, it will be observed in the range 15 to 40 cGy.

Health Phys. 99(3):357–362; 2010

Key words: alpha particles; analysis, risk; dogs; $^{239}\text{Pu}$

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
Inhaled PuO₂ in dogs, dose on log scale

Fisher and Weller (2010) data

LNT model

Controls, natural incidence of lung tumors

NOAEL ~ 60 cGy
Threshold-NOAEL for radon-induced cancer

- Raabe (2011): The average dose rate determines the cancer risk
- Dose rate of inhaled $^{239}\text{PuO}_2$ NOAEL = $60 \text{ cGy} \div 12 \text{ year} = 5.0 \text{ cGy/y}$
- ICRP-115 (2010) gives 17 mSv/year as effective dose for 300 Bq/m$^3$ of radon in homes with 0.4 equilibrium factor and 80% occupancy factor
- Absorbed dose $D_{T,R} = E/(w_R \times w_T)$; 17 mSv/y ÷ (20 x 0.12) = 7.1 mGy/y
- Radon level of $300 \times 5.0 \div 0.71 = 2100 \text{ Bq/m}^3$ or 57 pCi/L is the radon NOAEL that corresponds to 5.0 cGy/year NOAEL of inhaled $^{239}\text{PuO}_2$
- EPA action level is 150 Bq/m$^3$, which is 14 times below 2100 Bq/m$^3$
- Recommend radon limit of $1000 \text{ Bq/m}^3$, which gives optimum benefit
Threshold for Radon-Induced Lung Cancer From Inhaled Plutonium Data

Jerry M. Cuttler¹ and Charles L. Sanders²

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/m³. The US Environmental Protection Agency should consider raising its radon action level from 150 to at least 1000 Bq/m³.

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

Introduction
Indoor radon concentrations, in becquerels (note 1) per cubic metre (Bq/m³), vary widely. While the average radon levels in countries range from 11 Bq/m³ in Australia to 120 Bq/m³ in Finland, maximum values above 50 000 Bq/m³ 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 lung cancer incidence just below the NOAEL, to avoid the risk of losing an important health benefit.⁷

The Schneberg Study² identified the NOAEL to be about 1000 Bq/m³. 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/m³.⁴ The cases and the controls had similar, relatively low, mean radon exposures of 60.2 and 66.3 Bq/m³, respectively.
Inhaled radon in homes

Cohen BL 1995 data
Corrected for smoking

NOAEL ~ 2100 Bq/m³

Average residential level

Mean Radon Level (pCi/L)

Normalized Lung Cancer Mortality (m/m₀)
Brooks-2009: Summary of cancer frequency for inhaled beta-gamma emitting $^{90}\text{Sr}$, $^{144}\text{Ce}$, $^{91}\text{Y}$ and $^{90}\text{Y}$
Hiroshima atomic bomb survivor zones

Ground Zero

Zone A

B

C

D

E

1000 m

1500 m

2000 m

3000 m
Radiation dose vs. distance from ground zero

Graph showing the relationship between dose (in rems) and distance from the hypocenter (in meters) for Nagasaki and Hiroshima.
UNSCEAR 1958 Table VII
Leukemia incidence for 1950–57 after exposure at Hiroshima

<table>
<thead>
<tr>
<th>Zone</th>
<th>Distance from hypocentre (metres)</th>
<th>Dose (rem)</th>
<th>Persons exposed</th>
<th>$L$ (Cases of leukemia)</th>
<th>$\sqrt{L}$</th>
<th>$N^b$ (total cases per 10$^6$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>under 1,000</td>
<td>1,300</td>
<td>1,241</td>
<td>15</td>
<td>3.9</td>
<td>12,087 ± 3,1±3</td>
</tr>
<tr>
<td>B</td>
<td>1,000–1,499</td>
<td>500</td>
<td>8,810</td>
<td>33</td>
<td>5.7</td>
<td>3,746 ± 6±7</td>
</tr>
<tr>
<td>C</td>
<td>1,500–1,999</td>
<td>500</td>
<td>20,113</td>
<td>8</td>
<td>2.8</td>
<td>398 ± 139</td>
</tr>
<tr>
<td>D</td>
<td>2,000–2,999</td>
<td>2</td>
<td>32,692</td>
<td>3</td>
<td>1.7</td>
<td>92 ± 52</td>
</tr>
<tr>
<td>E</td>
<td>over 3,000</td>
<td>0</td>
<td>32,963</td>
<td>9</td>
<td>3.0</td>
<td>273 ± 91</td>
</tr>
</tbody>
</table>

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.
rem. 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.

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
Threshold level at ~ 50 rem (500 mSv)

*J*-curve, not LNT model
Leukemia and Ionizing Radiation Revisited

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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.”

15 cGy x 2/week x 5 weeks = 150 cGy
HBI or TBI for non-Hodgkin’s lymphoma

![Graph showing survival rates](graph.png)

- 3.7 years: 84%
- 5 years: 84%
- 9 years: 50%

P = 0.05

Sakamoto Personal Communication 2000

*STAGES I, II, Intermediate*, *High*
Shu-Zheng Liu and Jerry Cuttler in Mississauga
LDR therapy for Hurthle cell carcinoma
HB-LDI therapy; prophylaxis against cancer

150 mGy \times \text{twice/week} \times 5 \text{ weeks} = 1500 \text{ mGy}
Adjuvant Therapy for Resected Exocrine Pancreatic Cancer by Half-Body Low-Dose Irradiation

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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 80 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
Treatment of Alzheimer Disease With CT Scans: A Case Report

Jerry M. Cuttler¹, Eugene R. Moore², Victor D. Hosfeld³, and David L. Nadolski⁴

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 the senior population. The symptoms include memory loss,

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.
AD patient, spouse and author on Dec 4, 2015
Cancer death rate rises exponentially with age

Cancer cells from where?
**Spontaneous** DNA damage: free radicals, reactive oxygen species, 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
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

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6 Three Categories of Physiological Defenses of Complex Biological Systems ..............................................
7 Low-Dose Induced Adaptive Protections ..............................................
8 Physiological Defenses Against Cancer ..............................................
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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 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 $\sim 10^6$, DSB $\sim 10^{-1}$
  - Radiation-induced (1 mGy/yr, $\gamma$ background): total $\sim 10^{-2}$, DSB $\sim 10^{-4}$
- Ratio of DNA alterations (endogenous/rad’n): total $\sim 10^8$, DSB $\sim 10^3$

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

- No shutters
- No filter
- No cone

Lead glass open bowl

10 in.

1.25 mm lead

530 MR/HR

80 R/min

318
Table 1. Observed Rates of Death from Breast Cancer, According to the Dose of Radiation Received.

<table>
<thead>
<tr>
<th>Dose (Gy)</th>
<th>Nova Scotia</th>
<th>Other Provinces</th>
<th>All Provinces</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.09</td>
<td>455.6</td>
<td>585.8</td>
<td>578.6</td>
</tr>
<tr>
<td></td>
<td>(13)</td>
<td>(288)</td>
<td>(301)</td>
</tr>
<tr>
<td>0.10–0.19</td>
<td></td>
<td>389.0</td>
<td>421.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(29)</td>
<td>(32)</td>
</tr>
<tr>
<td>0.20–0.29</td>
<td></td>
<td>497.8</td>
<td>560.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(24)</td>
<td>(26)</td>
</tr>
<tr>
<td>0.30–0.39</td>
<td>1709</td>
<td>630.5</td>
<td>650.8</td>
</tr>
<tr>
<td></td>
<td>(11)</td>
<td>(17)</td>
<td>(18)</td>
</tr>
<tr>
<td>0.40–0.69</td>
<td></td>
<td>632.1</td>
<td>610.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(19)</td>
<td>(19)</td>
</tr>
<tr>
<td>0.70–0.99</td>
<td></td>
<td></td>
<td>1362</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(13)</td>
</tr>
<tr>
<td>1.00–2.99</td>
<td>2060</td>
<td></td>
<td>1382</td>
</tr>
<tr>
<td></td>
<td>(14)</td>
<td></td>
<td>(17)</td>
</tr>
<tr>
<td>3.00–5.99</td>
<td>2811</td>
<td>873.1</td>
<td>2334</td>
</tr>
<tr>
<td></td>
<td>(13)</td>
<td>(14)</td>
<td>(14)</td>
</tr>
<tr>
<td>6.00–10.00</td>
<td>7582</td>
<td></td>
<td>8000</td>
</tr>
<tr>
<td></td>
<td>(8)</td>
<td></td>
<td>(9)</td>
</tr>
<tr>
<td>≥10.00</td>
<td>21,810</td>
<td></td>
<td>20,620</td>
</tr>
<tr>
<td></td>
<td>(12)</td>
<td></td>
<td>(13)</td>
</tr>
</tbody>
</table>

*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
Adaptive response

Low radiation dose up-regulates cell repair capability
Decreases risk of 4 Gy challenging dose
4133 identified radium dial painters in USA

Bone cancer *threshold* at 10 Gy (1000 rad) radium alpha radiation
1000cGy threshold radium-induced bone cancer

**Evans et al. (1972)**

**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)
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 8,000 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.
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

Case which 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

Irradiated Group

Control Group
Tubiana: 5000 survivors of childhood cancer

Incidence of second malignant neoplasms

Average radiation dose to the volume (Gy)
Residents ingested Mayak radioactive discharges into Techa River, in early 1950s. UNSCEAR recognized this as opportunity to estimate dose–effect of long-term irradiation.

Mortality incidences from leukemia and cancer of CRS people did not exceed cancer incidences for exposed people without CRS and for Russia as a whole.

Threshold for CRS is an annual dose of 700 to 1000 mGy.
RADIOBIOLOGY SPECIAL FEATURE: COMMENTARY

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

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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 dose from these lower dose examinations is comparatively unimportant from a public health perspective.
Conclusions

• Social concern about nuclear energy safety is caused by policy that links human-made radiation to risk of cancer
• Radiation scare of 1950s (to stop atom-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 500 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)_