I have a PhD in microbiology from UC Davis, and I wrote a book on treatment of radiation exposure[1]. The regulation of this area is wildly out of line with science. The NRC needs to update its procedure and regulations to match reality.
The LNT model was pushed through by Rockefeller in the 1950's when much less was known. The result is virtually everything in regulation is based on false models, and general public understanding is likewise false. In the meantime largely because of the lack of conversion to nuclear, we now have an incredibly dangerous climate problem due to fossil fuels [2]. The situation is dire, and anyone who cares for a human civilization future will end the paper-chase holding up nuclear deployment.

I have cited 7 peer reviewed papers and attached 6 for which I have permission covering 2 categories:

* LNT model of radiation danger from cancer. This model is simply wrong.
  Calabrese 2014 [3], Cardarelli 2018 [4], Sutou 2018 [5], Jaworowski 2010 [6]
* Corrections to the false representations of mutation danger. Mutation is not an problem.
  Neel 1998 [7], Jordan 2016 [8]

1. Hanley, B.P. Radiation – Exposure and its treatment: A modern handbook. **2014** *Amazon Kindle* https://www.amazon.com/Radiation-Exposure-treatment-modern-handbook-ebook/dp/B00D7KLQYY *Permissions*: Permission to re-use for the purpose of comment provided by the author, holder of the copyright. Available on request in pdf form from the author, as it is over the 10 MB limit.

2. Lang, P.A. Nuclear Power Learning and Deployment Rates; Disruption and Global Benefits Forgone. *Energies* **2017**, *10*, 2169. https://doi.org/10.3390/en10122169 *Permissions*: This article is distributed under MDPI Open Access Policy. “All articles published by MDPI are made immediately available worldwide under an open access license. ... everyone is free to re-use the published material if proper accreditation/citation of the original publication is given.” https://www.mdpi.com/openaccess

3. Calabrese, E.J. & O'Connor, M.K. Estimating Risk of Low Radiation Doses – A Critical Review of the BEIR VII Report and its Use of the Linear No-Threshold (LNT) Hypothesis. Radiation Research **2014**, 182(5), 463-474. https://doi.org/10.1667/RR13829.1 *Permissions*: Not provided.

4. Cardarelli J.J. 2nd & Ulsh B.A. It Is Time to Move Beyond the Linear No-Threshold Theory for Low-Dose Radiation Protection. Dose Response **2018,** 16(3):1559325818779651. https://doi.org/10.1177/1559325818779651 *Permissions*: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (http://www.creativecommons.org/licenses/by-nc/4.0/)

5. Sutou, S. Low-dose radiation from A-bombs elongated lifespan and reduced cancer mortality relative to un-irradiated individuals. *Genes and Environ* **2018,** 40(26). https://doi.org/10.1186/s41021-018-0114-3 *Permissions*: This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/)

6. Jaworowski, Z. Observations on the Chernobyl Disaster and LNT. Dose-Response **2010**, 8(2). https://doi.org/10.2203/dose-response.09-029.Jaworowski *Permissions*: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 3.0 License (http://www.creativecommons.org/licenses/by-nc/3.0/)

7. Neel, J.V. Genetic studies at the Atomic Bomb Casualty Commission–Radiation Effects Research Foundation: 1946–1997. PNAS **1998**, 95(10)5432-5436. https://doi.org/10.1073/pnas.95.10.5432

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8. Jordan, B.R. The Hiroshima/Nagasaki Survivor Studies: Discrepancies Between Results and General Perception. *Genetics* 2016, 203(4)1505–1512. https://doi.org/10.1534/genetics.116.191759 by permission of Oxford University Press. *Permissions*: “Our permission is granted without fee to reproduce the material. Use of the OUP Material is restricted to: Inclusion of the full article in support of the forthcoming submission of ‘Comment submitted to the Nuclear Regulatory Commission of the United States on nuclear regulatory policy’ by Brian Hanley to the Nuclear Regulatory Commission (NRC) in September 2022 in electronic format, in the English language.”

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Nuclear Power Learning and Deployment Rates; Disruption and Global Benefits Forgone

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Abstract: This paper presents evidence of the disruption of a transition from fossil fuels to nuclear power, and finds the benefits forgone as a consequence are substantial. Learning rates are presented for nuclear power in seven countries, comprising 58% of all power reactors ever built globally. Learning rates and deployment rates changed in the late-1960s and 1970s from rapidly falling costs and accelerating deployment to rapidly rising costs and stalled deployment. Historical nuclear global capacity, electricity generation and overnight construction costs are compared with the counterfactual that pre-disruption learning and deployment rates had continued to 2015. Had the early rates continued, nuclear power could now be around 10% of its current cost. The additional nuclear power could have substituted for 69,000–186,000 TWh of coal and gas generation, thereby avoiding up to 9.5 million deaths and 174 Gt CO_2 emissions. In 2015 alone, nuclear power could have replaced up to 100% of coal-generated and 76% of gas-generated electricity, thereby avoiding up to 540,000 deaths and 11 Gt CO_2. Rapid progress was achieved in the past and could be again, with appropriate policies. Research is needed to identify impediments to progress, and policy is needed to remove them.

Keywords: nuclear power; construction cost; learning rate; energy transition; disruption; benefits forgone; deaths; CO_2 emissions

1. Introduction

Energy is the lifeblood of modern civilisation. Humans would still be hunter-gatherers if not for our ability to extract and use energy. Major advances in human wellbeing have been driven by transitions to cheaper and more plentiful energy. Examples include: the harnessing of fire, animals, wind and water power, and transitions from wood to coal, and from coal to oil and to gas [1–4]. A transition to cheaper, cleaner electricity globally would improve human wellbeing and reduce the environmental impacts of electricity generation [3,5–8].

People and businesses want cheap, reliable and secure energy. Globally, 1.2 billion people are still living without access to electricity [9]. According to the World Health Organisation (WHO) [8], "around 3 billion people cook and heat their homes using open fires and simple stoves burning biomass (wood, animal dung and crop waste) and coal". WHO [7] estimated that 4.3 million deaths annually are attributable to indoor air pollution and 3.7 million to ambient (outdoor) air pollution. Gohlke et al. [6] found that increased electricity consumption per capita correlates with better health outcomes because of better access to clean water and sanitation, and reduced indoor and outdoor air pollution. They also found that access to a centralised power source is necessary to gain many of the benefits of clean power. Many of the deaths caused by indoor air pollution could be avoided if electricity replaced the burning of biomass and coal in homes, and many of the deaths attributable to outdoor air pollution could be avoided if clean technologies replaced fossil fuel for electricity generation.

Nuclear power produces comparatively little air or water pollution. Substituting nuclear for fossil fuel in electricity generation could prevent most of the deaths attributable to electricity generation.

Cheap electricity increases productivity and economic growth, drives electrification for people without any electricity or with insufficient or unreliable electricity, and thereby more quickly raises living standards and human wellbeing. As the cost of electricity decreases, deployment rate increases. Transition takes place faster and the benefits are delivered sooner.

History is replete with examples of one technology replacing another [2,3]. Large infrastructure transitions have commonly taken around a century [10]. Examples are transitions to canals, railways, highways, oil and gas pipelines, telegraph, and electricity grids. Transitions typically follow an S-curve from 0 to 100% complete, with three phases: accelerating to about 20%, near-linear to about 80%, and decelerating to 100% [3,10]. Electricity grids reached 50% of world population in 1960 and 80% in 2010 [11].

The transition to nuclear power began in 1954 with the first reactor connected to the grid. Until the 1970s, it was envisaged that nuclear would emulate earlier energy transitions. For example, Wilson [12] projected that nuclear power would supply 14 to 21% of world primary energy by 2000. However, the transition to nuclear reached 4% by 1970, then stalled [3]. The deployment rate of nuclear capacity is currently less than in 1972; the transition has been stalled for 44 years.

The rate that technology transitions take place depends, in part, on the technologies being 'fit-for-purpose' and on the learning rates that occur during the transition period. To accelerate the transition to reliable, cheap, clean, safe and comparatively environmentally benign electricity generation, policies need to focus on ways to improve the learning rates and deployment rates of technologies that meet requirements. Historical learning rates provide insight into what rates may be achievable and what could be done to return to rapid rates.

The concept of learning rates, or cost experience curves [I], is widely used to quantify the rate at which costs reduce as experience is gained. Learning rate is the fractional reduction in cost per doubling of cumulative capacity or production. Rubin et al. [13] explain how to calculate learning rates, and summarise learning rates for selected electricity generation technologies. However, their paper has limited information on nuclear power learning rates, and none before 1972 or after 1996.

Lovering et al. [14] [II] provide a comprehensive analysis of nuclear power construction cost experience of early and recent reactors in seven countries; their analysis covers 58% of the reactors ever constructed for electricity generation, between 1954 and 2015. While there have been many studies of the cost escalation of nuclear power plants (e.g., [15,16], and others cited in Lovering et al. [14]), most are for the US and France only, and cover only periods since the 1970s. To the author's knowledge, there are no comprehensive studies, other than Lovering et al. [14], that cover the full period of global commercial nuclear power reactor operation, nor any studies that provide the learning rates over the full period, and that highlight their reversal, which began in the late-1960s.

This study extends the literature by providing learning rates of nuclear power reactors for the seven countries analysed by Lovering et al. for the full period from 1954 through 2015. The aim is to answer two questions. What were the global benefits forgone as a consequence of the reversal of learning rates and the stalled deployment rates? What are the policy implications?

Using counterfactual analysis, Kharecha and Hansen [17] estimated that electricity generated by nuclear power avoided 1.84 million air-pollution-related deaths and 64 Gt of CO_2 emissions between 1971 and 2009. The current analysis also uses a counterfactual approach. Lovering et al. data were re-analysed to calculate the historical learning rates and deployment rates of nuclear power, and to project the early rates to 2015. Evidence of disruption to the learning and deployment rates is presented and some of the benefits forgone are quantified. Estimates are presented for:

- what the Overnight Construction Cost (OCC) [III],[IV] of nuclear power could have been in 2015 if the early learning and deployment rates had continued
- the additional electricity that could have been generated by nuclear power if the early deployment rate trends had continued
- the number of deaths and quantity of CO_2 emissions that could thereby have been avoided.

The analysis finds that the benefits forgone because of the disruption, and the resulting stalled transition from fossil fuels to nuclear power, are substantial.

The purpose of this paper is to publish the evidence and the consequences of the disruption, and to suggest an approach to removing the impediments that are delaying progress. It does not explore the causes of the disruption and cost escalations thereafter; that would require extensive studies beyond the scope of this paper.

To summarise, this study provides learning rates for a full set of reactors in seven countries, covering builds from 1954 through to projects that had been completed by the end of 2015, covering 58% of all power reactors ever built globally. It also provides global deployment rates for that period. It estimates the extra electricity that could have been generated by nuclear power since 1980 and what OCC would have been in 2015 if the learning and deployment rates had not been disrupted. It compares the projections to the actuals to estimate forgone benefits of the disruption. It suggests an approach to removing the impediments that are retarding the transition to nuclear power.

2. Materials and Methods

This section explains the methods and assumptions used to:

- calculate historical OCC learning rates
- estimate the capacity of nuclear power that would have been constructed by 2015 if historical deployment rates had continued
- estimate the OCC of nuclear power in 2015 by applying the pre-1970s learning rates to the capacities estimated from the projected deployment rates
- estimate the quantity of extra electricity that could have been generated by nuclear power if the early deployment rates had continued to 2015; and the deaths and CO_2 emissions that could thereby have been avoided.

Counterfactual analyses require simplifying yet tenable assumptions. As Kharecha and Hansen [17] explain for their counterfactual analysis of deaths and CO_2 avoided by historical nuclear power deployment, "There are of course numerous complications involved in trying to design such a replacement scenario (e.g., evolving technological and socioeconomic conditions), and the . . . energy mix cannot be known with total accuracy and realism; thus, simplifying yet tenable assumptions are necessary and justified." This study assumes, conservatively, that the historical electricity demand did not change (despite the reducing costs) and assumes that the additional electricity generated by nuclear power would have displaced equivalent coal and gas generation.

Costs are in 2010 US dollars as per Lovering et al. [14].

2.1. Learning Rates

The data of Lovering et al. [14] were re-analysed to calculate OCC learning rates. Figure 1 plots OCC ($/kW) against cumulative global capacity (GW) for the nuclear data points in Lovering's Figure 13. There is a marked reversal in the slope of OCC against cumulative global capacity. Before cumulative global capacity reached around 32 GW, which occurred in 1967, OCC was decreasing as cumulative capacity increased (i.e., learning rates were positive). Then an abrupt change occurred; thereafter, OCC was increasing (i.e., learning rates were negative). The trendlines are fitted to the US data points before and after 32 GW to highlight the dramatic reversal.

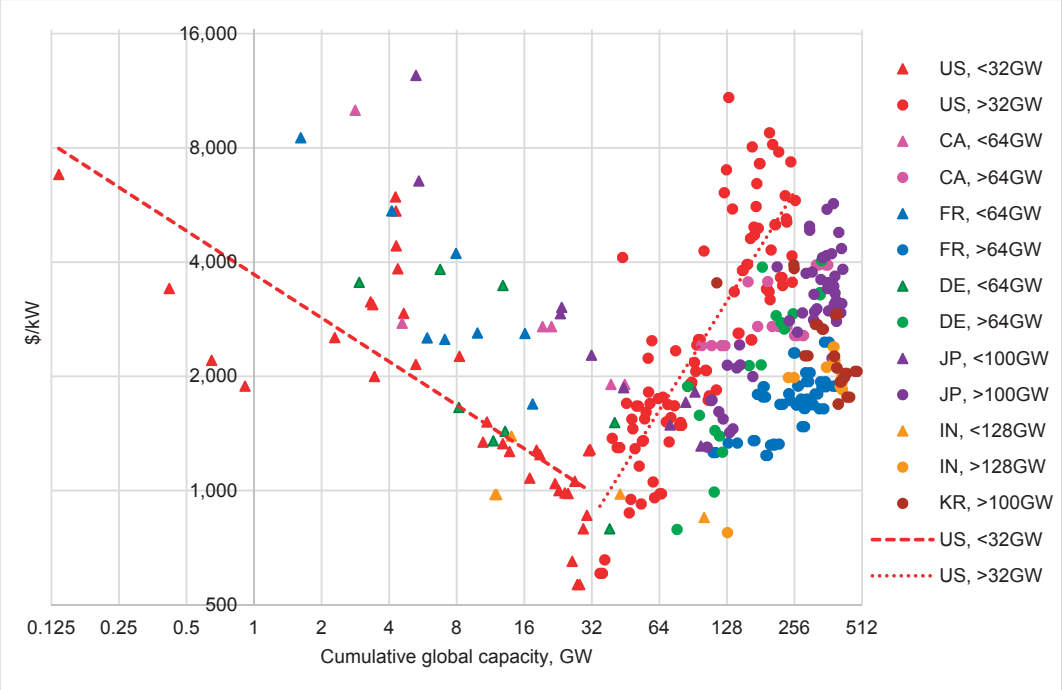

Figure 1. Overnight construction cost (in 2010 US $/kW) plotted against cumulative global capacity (GW), based on construction start dates, of nuclear power reactors for seven countries, including regression lines for US before and after 32 GW cumulative global capacity.

Given this evidence for two phases, learning rates were calculated for two periods, that is, before and after the slope reversal, for each country. The reversal occurred at different times in different countries and regions. It occurred first in the US; there was a lag to Canada and Europe and a further lag to Asia. The reversal points selected were: 32 GW for US; 64 GW for Canada, France and Germany; 100 GW for Japan; and 128 GW for India; there is no reversal point for South Korea because it had no construction starts before 1972, so no pre-reversal data points.

The data points were plotted on log-log axes (base 2), trendlines fitted to the pre- and post-reversal periods for each country, and learning rates calculated for each trendline. Following Rubin et al. [13], learning rates were calculated by regressing OCC against cumulative global capacity using a power function. Learning rate is equal to $1–2^b$ where b is the exponent of the fitted power function.

2.2. Deployment Rate Projections

To calculate the OCC of nuclear power in 2015 requires a projection of what the cumulative global capacity of construction starts would have been. Similarly, to calculate the extra electricity that would have been generated at the higher deployment rates, and the deaths and emissions that could have been avoided, requires a projection of what the global capacity of operating reactors would have been. This projection assumes that, if not for the disruption, the construction period would have been five years [V] and the capacity of power uprates and of reactors permanently shut down each year would have been unchanged from the actual. Three deployment rate scenarios were analysed: the actual historical rate and two projections of early historical rates:

Actual: This is the actual historical deployment from 1954 to 2015. The cumulative global capacity of construction starts was 486 GW in 2013 [14]; 11 GW was added in 2014 and 2015, making the total 497 GW in 2015. The actual global capacity in operation in 2015 was 383 GW [18] [VI].

Linear: The capacity of commercial operation starts peaked at 40 GW in 1985 and averaged 30 GW per year from 1984 to 1986 [18]. The capacity in commercial operation in 1985 was 253 GW [19]. The Linear scenario assumes that commercial operation starts continued at 30 GW per year from

1985 to 2015, and the capacity of power uprates and reactors permanently shutdown each year was as per the historical data. (See Appendix A for further explanation of the calculation method and data sources.)

Accelerating: From 1954 to 1976, the capacity of construction starts was accelerating, then slowed in 1976 (i.e., about 5 to 10 years after the reversal points, which was when OCC started to increase rapidly). If the OCC had continued to reduce at the pre-reversal learning rates, it was assumed the deployment rate also would have continued (all else being equal). A defensible assumption is that the rate continued at that prevailing from 1960 to 1976. A polynomial function was fitted to the data points for 1960 to 1976 and projected to 2015. The cumulative global capacity of commercial operation starts was estimated by subtracting five years (for the assumed average construction duration) from the cumulative global capacity of construction starts and subtracting the actual capacity of reactors permanently shut down (see Appendix A for further explanation).

The Linear and Accelerating scenarios are used to estimate the extra electricity that would have been supplied each year by nuclear power from 1985 to 2015 (for the Linear) and from 1980 to 2015 (for the Accelerating) scenarios.

2.3. Projected Overnight Construction Costs in 2015

OCC in 2015 was calculated for the three deployment rate scenarios by applying the pre-reversal learning rates to the 2015 actual capacity and to the two projections, i.e., by substituting the actual and projected capacity in 2015 in the trendline equations (shown in Figure 2 for each country).

The projected OCC in 2015 were compared with the actuals. IEA [20] published estimates (in 2013 USD) of actual OCC for nuclear power (as well as other technologies) for US, France, Japan and Korea. Here, these were adjusted to 2010 USD for consistency with Lovering et al. using the World Bank GDP deflator [21]. IEA [20] includes OCC for twenty two countries, but not for Canada, Germany and India, so $4000 was assumed for Canada, $5000 for Germany, and $2000 for India. These figures are close to OCC for US, France and Korea respectively, and are approximately consistent with the OCC of the last construction starts for those countries (Figure 2).

2.4. Extra Nuclear Electricity, Avoided Deaths and CO_2

The number of deaths and the quantity of CO_2 emissions that could have been avoided with the Linear and Accelerating scenarios were estimated. The extra nuclear electricity generated was estimated by factoring up the historical global annual nuclear net generation [22] in proportion to projected capacity divided by actual capacity [19]. (See Appendix A.)

To calculate the deaths and CO_2 emissions that could have been avoided, it was assumed the extra electricity generated by nuclear substituted for coal generation in the Linear scenario and for coal and gas generation in the Accelerating scenario, with conversion factors [VII]:

- Deaths: Coal = 60/TWh [VIII], Gas = 4/TWh (Wang [23]).
- CO_2 emissions: Coal = 1 Mt/TWh, Gas = 0.6 Mt/TWh (Kharecha and Hansen [17])

This study conservatively adopts the historical demand profile (despite the declining cost). Appendix A explains the calculations and data sources.

3. Results and Discussion

3.1. Learning Rates

Figure 2 has a chart for each of the seven countries and one for all seven combined; trendlines were fitted to the data points before and after the trend reversal points. The equation for each trendline is shown on the charts.

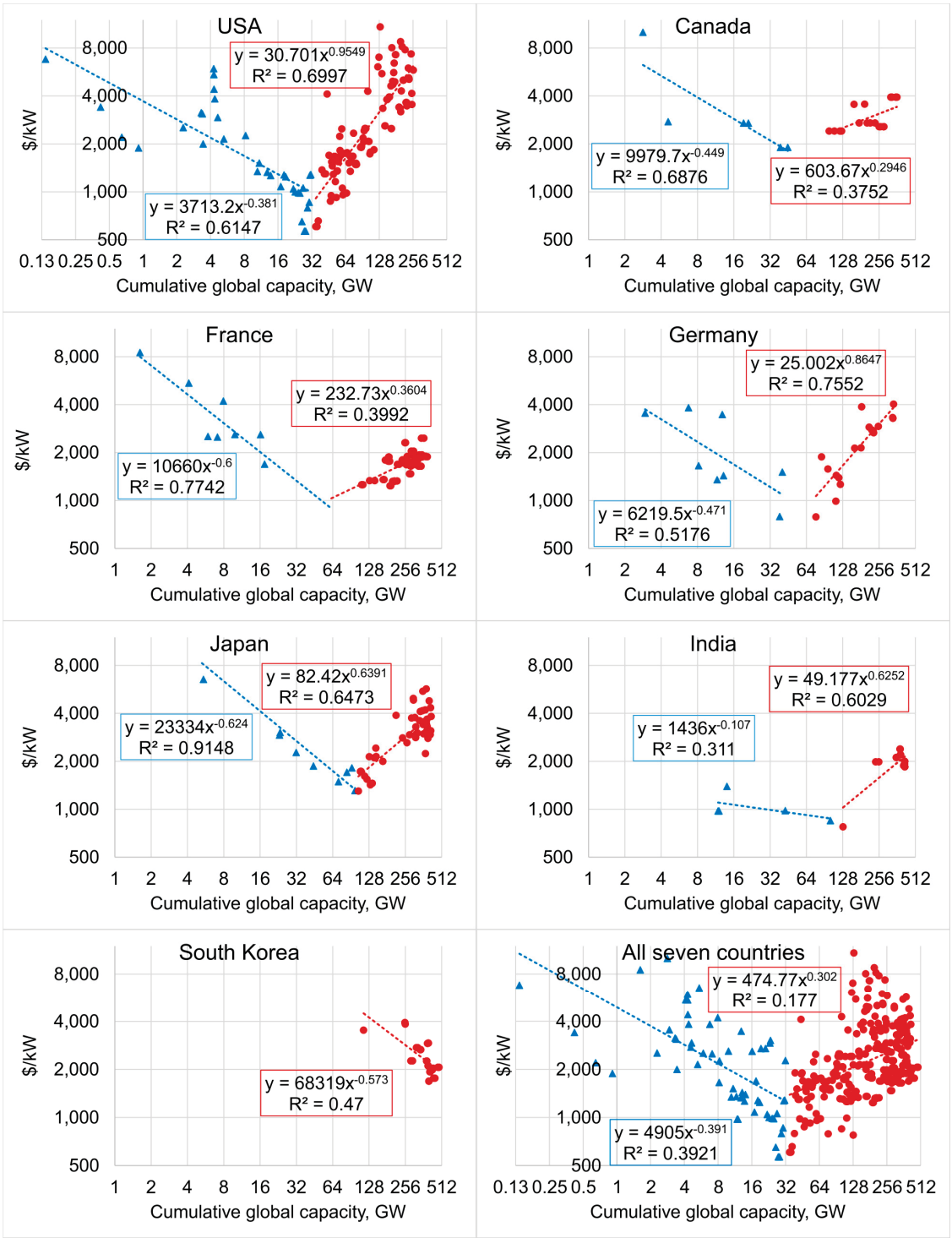

Figure 2. OCC (2010 US $/kW) plotted against cumulative global capacity (GW) of nuclear power reactors, based on construction start dates; regression lines fitted to points before and after trend reversals.

To compare trends for the seven countries, Figure 3 shows all the regression lines. Japan and France had the fastest pre-reversal learning rate; South Korea had a similar rate since it started building reactors in 1972, although it started from a high OCC after the reversal and initial rapid cost escalation in the other countries.

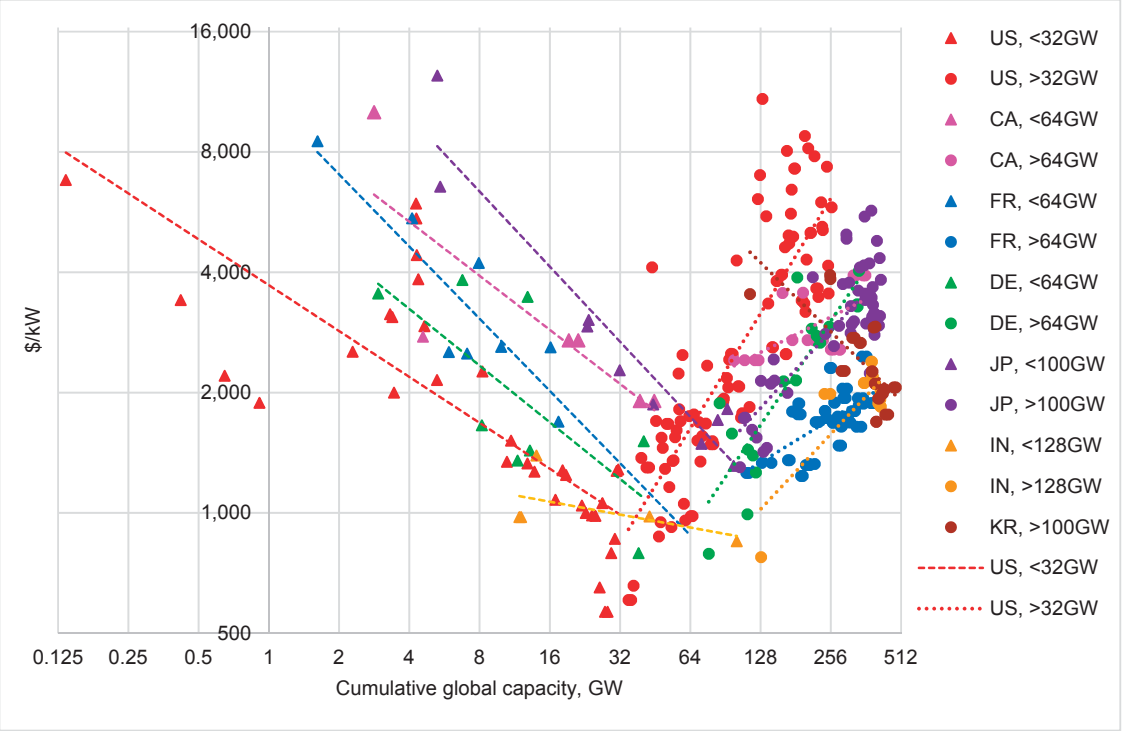

Figure 3. Regression lines for seven countries: OCC plotted against cumulative global capacity of construction starts.

Table 1 lists the learning rates for both periods in each country for both the cumulative global and the cumulative country capacity. The sixth and seventh columns are the selected reversal point (cumulative global capacity of construction starts, and approximate year it occurred) for each country. The last column is the projected OCC at 497 GW cumulative global capacity if the pre-reversal learning rates had continued.

Table 1. Learning rates for pre-reversal and post-reversal, selected reversal point and projected overnight construction cost at 497 GW cumulative global capacity of construction starts.

Country	Pre-Reversal		Post-Reversal		Reversal Point		Projected OCC
	Global	Country	Global	Country	GW	Year	at 497 GW
US	23%	24%	−94%	−102%	32	1967	$349
CA	27%	19%	−23%	−20%	64	1968	$614
FR	34%	28%	−28%	−10%	64	1968	$257
DE	28%	16%	−82%	−62%	64	1968	$334
JP	35%	23%	−56%	−35%	100	1970	$485
IN	7%	2%	−54%	−8%	128	1972	$739
KR	N/A	N/A	33%	12%	N/A	N/A	N/A
All	24%	N/A	−23%	N/A	32	1967	$433

Learning rates are affected by the growth of cumulative capacity both globally and locally. Following Lovering et al., cumulative global capacity was used as the reference. Figure 4 plots the learning rates against the time span of the construction starts for each period in each country.

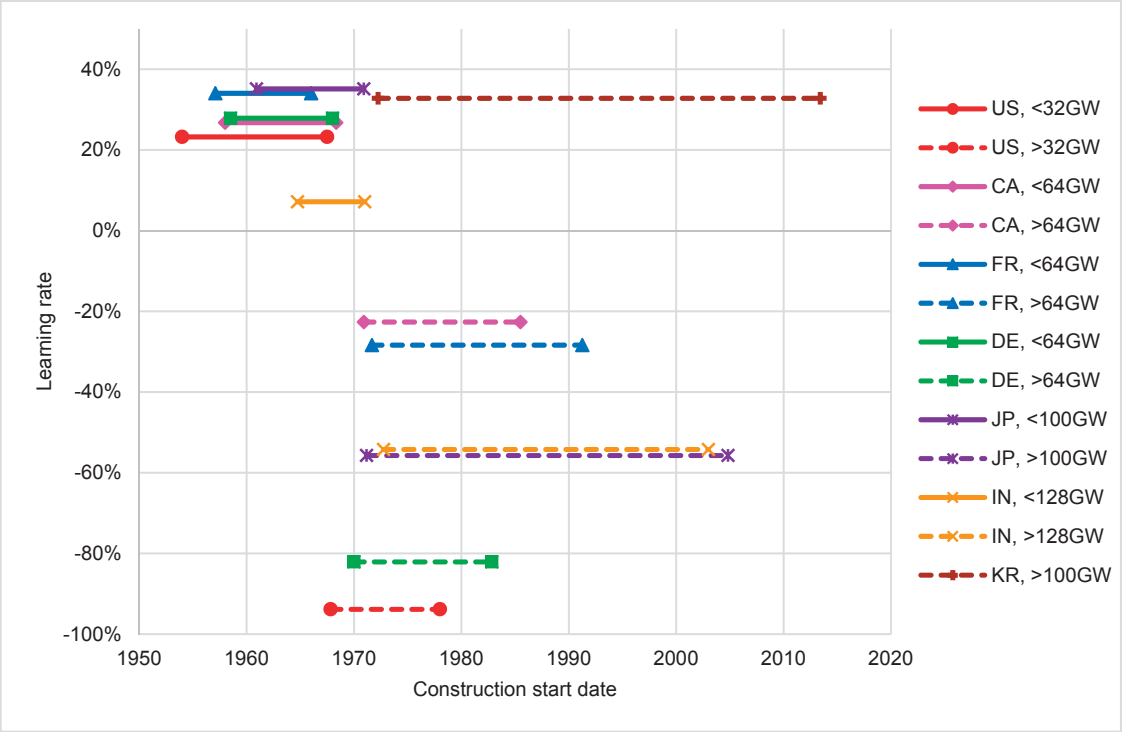

Figure 4. Learning rates pre- and post-reversal points vs. time span of construction starts.

Table 1 and Figure 4 show that, before the reversal, OCC learning rates were 23% in the US, 27% to 35% in the other countries except India (where it was 7%), and 24% for all countries combined. At the reversal, learning rates changed abruptly and became negative (−94% in the US, −82% in Germany, −23 to −56% in the other countries, except in South Korea, and −23% for all seven countries); South Korea started building nuclear power plants after the initial rapid cost-escalation period, achieving a 33% learning rate since 1972. The fact that fast learning rates existed up to about 1970, and in South Korea since, suggests they could be achieved again [IX].

The US's post-reversal learning rate was the worst of the seven countries. The reversal occurred one to five years later in the other countries and the real cost increase was not as severe as in the US. This suggests the US may have negatively influenced the development of nuclear power in all seven countries (and probably all countries). It also shows that technology learning and transition rates can change quickly and disrupt progress, in this case delaying progress for about half a century so far.

3.2. Deployment Rates and Projections to 2015

Figure 5 shows the annual global capacity of construction starts [X] and commercial operation starts from 1954 to 2015 [18]. The capacity of construction starts was accelerating until about 1970, peaked in 1976, then stalled. The annual capacity of commercial operation starts peaked in 1985, averaged 30 GW per year from 1984 to 1986, then declined rapidly and has not recovered. IAEA [24] shows grid connections peaked at 31 GW per year in 1984 and 1985 and declined rapidly thereafter.

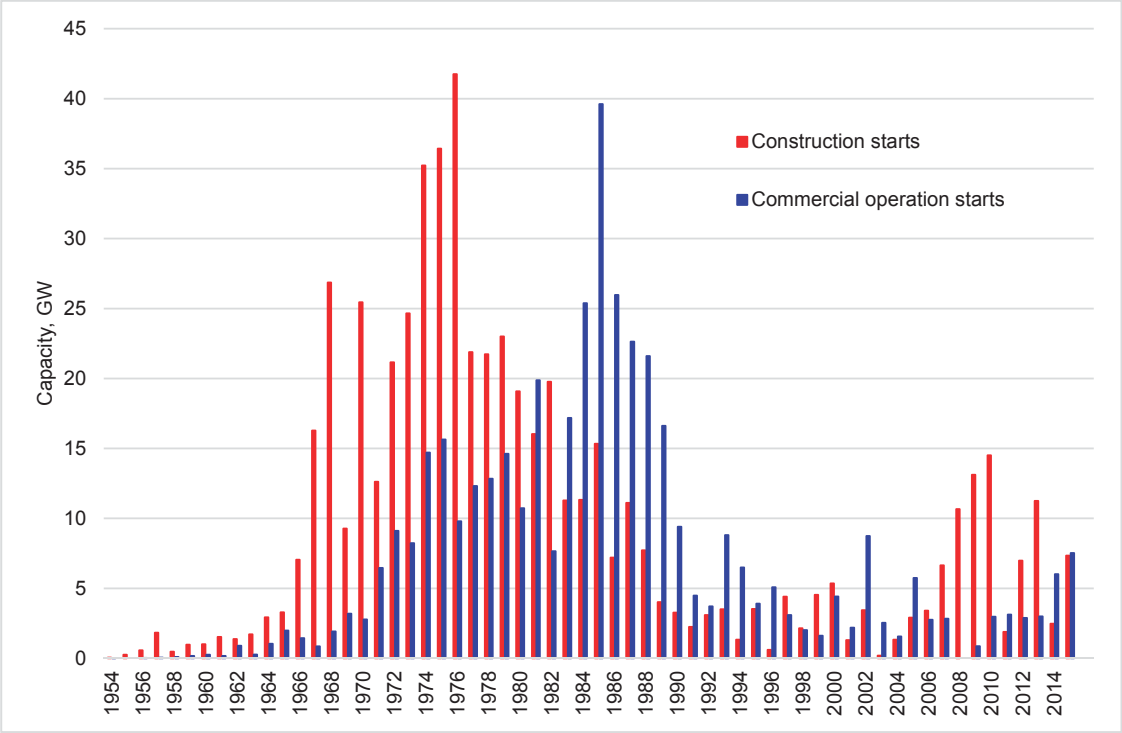

Figure 5. Annual global capacity of construction starts and commercial operation starts, 1954–2015.

Figure 6 shows cumulative global capacity of construction starts and commercial operations starts plotted against time (top panel), and projections of what they would have been in 2015 if the early deployment rates had continued (bottom panel).

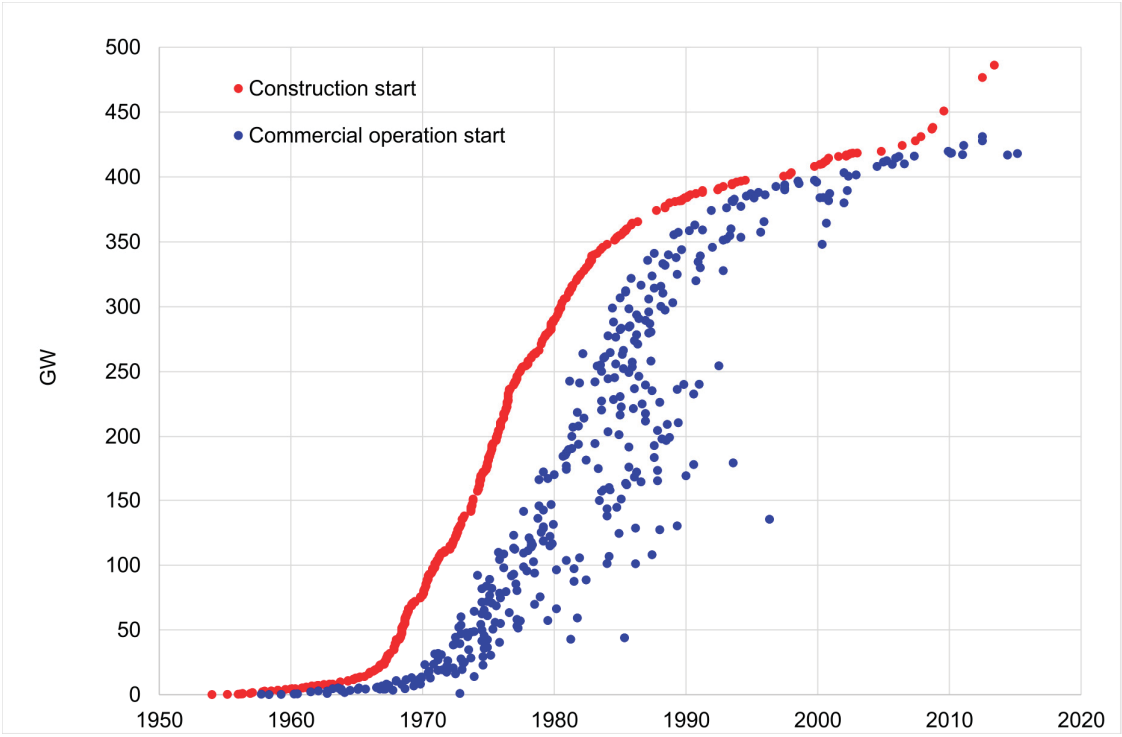

Figure 6. *Cont.*

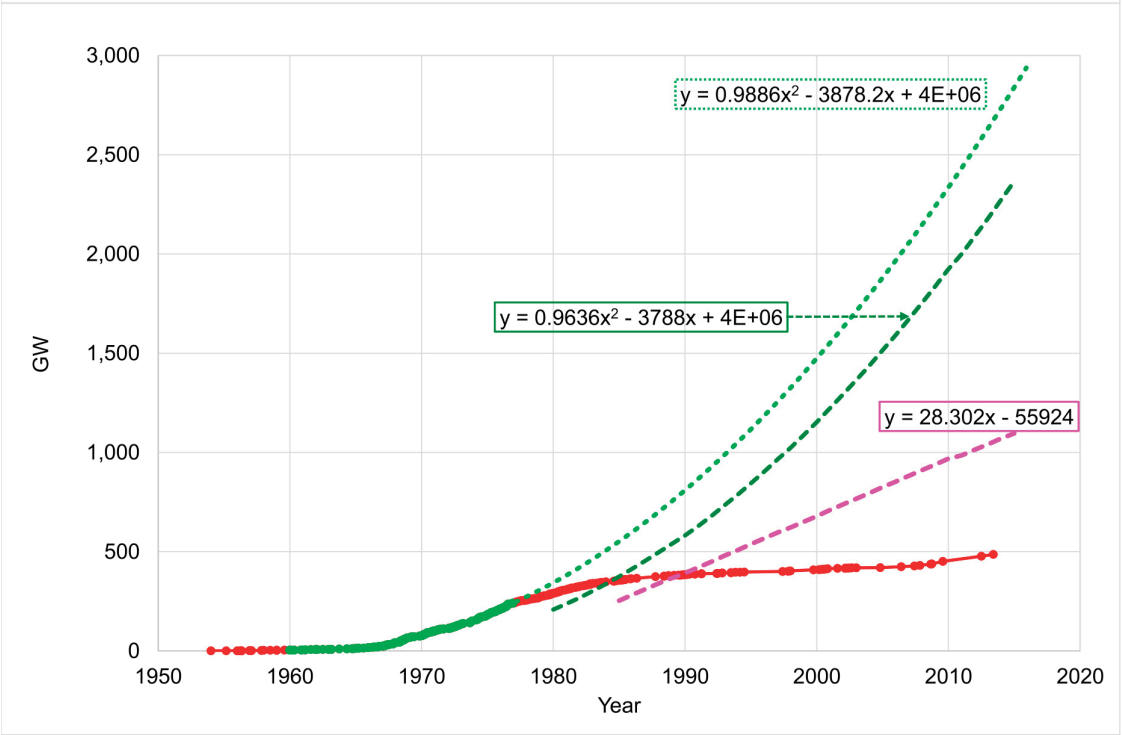

Figure 6. (**Top**) Cumulative global capacity of construction starts and of commercial operation starts (sorted by construction start date); (**Bottom**) Cumulative global capacity of construction starts (red and green data points); accelerating projection of 1960–1976 data points (dotted green line); Linear and Accelerating projections of capacity in commercial operation (dashed pink and green lines).

Table 2 summarises the cumulative global capacity of actual and projected construction starts and the capacity in commercial operation at the end of 2015 for each scenario.

Table 2. Actual and projected cumulative global capacity of construction starts and global capacity in commercial operation in 2015 for the three scenarios.

Deployment Rate Scenario	Construction Starts (GW)	Commercial Operation (GW)
Actual	497	383
Linear	1246	1096
Accelerating	2941	2366

The Linear and Accelerating projections of cumulative global capacity by 2015 in Table 2 represent scenarios calculated on the basis of the stated deployment rate assumptions. The increases in projected cumulative global capacity by 2015 compared with Actual are large. It is useful to compare these scenarios with projections made in the 1970s. For example, the Accelerating deployment rate projects a global nuclear capacity of 1152 GW by 2000. The Workshop on Alternative Energy Strategies (WAES) [12], projected global nuclear capacity in 2000 at between 913 GW and 1722 GW [XI]. So the present projection is quite consistent with the outlook of 40 years ago.

3.3. Projected Overnight Construction Costs in 2015

Table 3 lists the projected OCC in 2015 and the percentage reduction from the actual OCC [20] for the six countries that were constructing reactors before the learning rate reversals. Actual OCC for Canada, Germany and India are approximate, as noted in Section 2.3.

Table 3. Projected 2015 OCC by country for the three deployment rate scenarios at the projected pre-reversal learning rates. Actual OCC [20] for comparison. Percentage change of projected OCC compared with actual OCC.

	US	CA	FR	DE	JP	IN	All
Learning rate for projections	23%	27%	34%	28%	35%	7%	24%
	Overnight Capital Cost (2010 US$)						
Deployment rate scenarios							
Actual	349	614	257	334	485	739	433
Linear	246	407	148	217	273	670	302
Accelerating	177	277	89	145	160	611	216
Actual OCC	3881	4000	4797	5000	3676	2000	4022
	OCC Change from 2015 Actual						
Deployment rate scenarios							
Actual	9%	15%	5%	7%	13%	37%	11%
Linear	6%	10%	3%	4%	7%	33%	8%
Accelerating	5%	7%	2%	3%	4%	31%	5%

Shaded cells are approximate (see text).

If the pre-reversal learning rates had continued, with the Actual deployment unchanged, until 2015 when cumulative global capacity of construction starts was 497 GW, the OCC of nuclear power would be 5 to 15% of what it was in 2015 (except in India); for example, the OCC would be $349/kW in the US, $257/kW in France, and $484/kW in Japan (Table 3). These are much lower than the OCC of fossil fuel and other alternative electricity generation technologies [20].

If the pre-reversal learning rates and the Linear and Accelerating deployment rates had continued, the OCC would be approximately 2% to 10% of what it was in 2015 (except in India where it would be 31 to 33%) (Table 3).

These are striking cost reductions that, to be achieved, would have required pre-reversal learning rates and deployment rates to continue. If the rapid learning and deployment rates that prevailed pre-reversal could be achieved again, nuclear power would become much cheaper than fossil fuel technologies in the future. Some may regard this as too optimistic. However, there is no apparent physical or technical reason why they could not have continued and cannot prevail again. They have prevailed in South Korea over the past 40 years (Figure 4), and there are examples in other complex technologies and industries of cost reductions at similar rates that persisted over the past 50 years at the same time as the OCC of nuclear power was increasing rapidly [XII].

3.4. Extra Nuclear Electricity, Avoided Deaths and CO_2

Figure 7 shows the annual electricity generated by fuel type for the three deployment rate scenarios: Actual, Linear and Accelerating.

Table 4 shows that, at the Linear deployment rate, the extra nuclear generation from 1985 to 2015 could have substituted for 69,000 TWh of mostly coal-generated electricity globally and avoided approximately 4.5 million deaths (from outdoor air pollution and all other causes in the respective energy chains, but not including deaths that could have been avoided by increasing access to clean water and sanitation services) and 69 Gt CO_2 emissions.

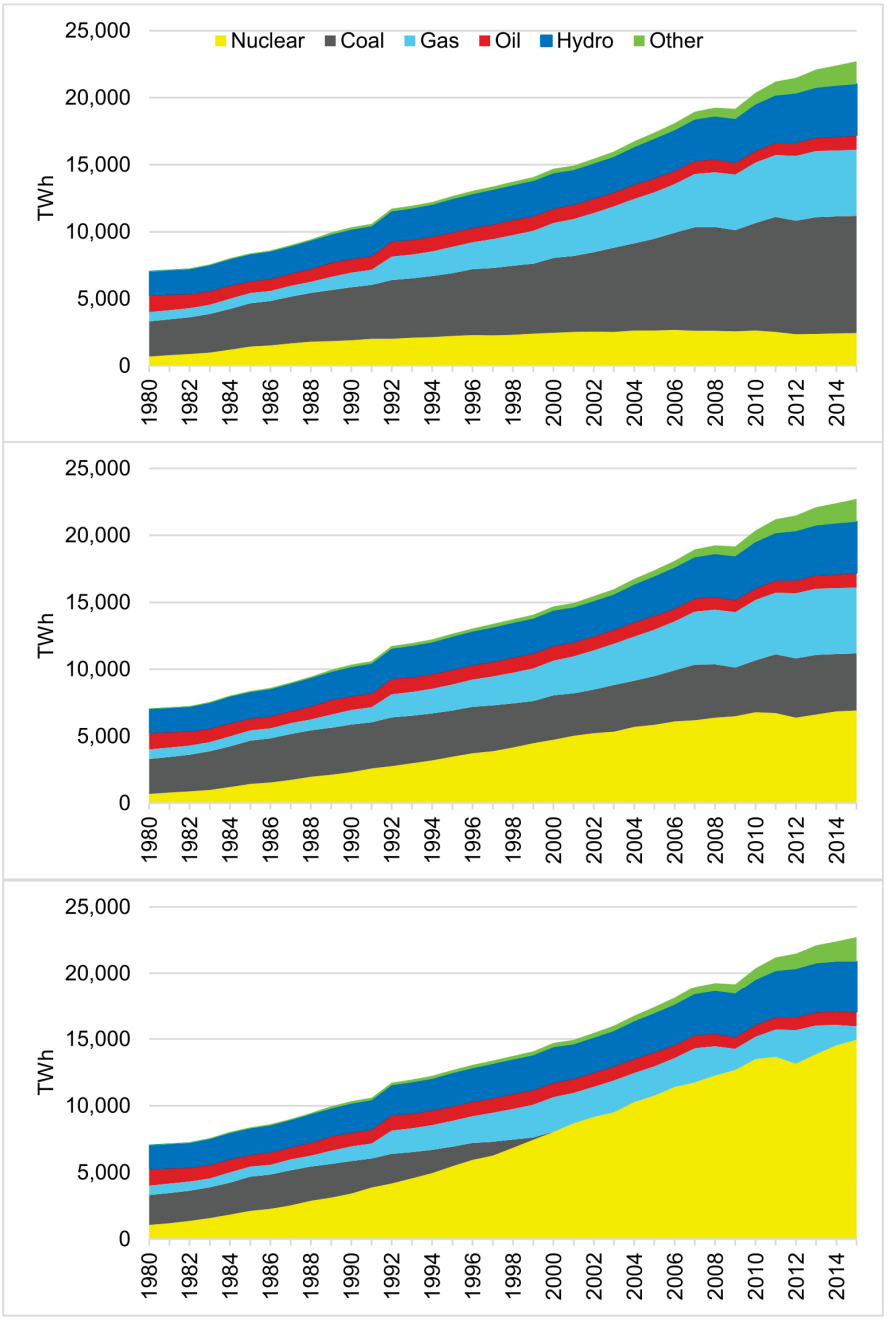

Figure 7. Electricity generated by fuel type by the Actual (**top**); and by the projected capacity in Linear (**middle**) and Accelerating (**bottom**) deployment scenarios (TWh).

Table 4. The extra electricity that could have been generated by nuclear power, and the consequent deaths and CO_2 emissions that could have been avoided with the Linear and Accelerating deployment rate scenarios [XIII].

Benefits Forgone	Units	Linear (1985–2015)	Accelerating (1976–2015)
Extra electricity supplied	TWh	69,315	186,067
Premature deaths avoided	million	4.2	9.5
CO_2 emissions avoided	Gt	69	174

At the Accelerating rate, the extra nuclear generation could have exceeded the actual generation from coal by year 2000 (assuming electricity demand did not change). If the extra nuclear generated electricity had substituted for coal and gas generation, about 9.5 million deaths and 174 Gt CO_2 may have been avoided.

In 2015 alone, if the extra nuclear generation had replaced coal and gas generation, and electricity demand was unchanged, nuclear could have:

- substituted for 49% of coal-generated electricity, thus avoiding 273,000 deaths and 4.5 Gt CO_2 emissions (Linear scenario)
- substituted for 100% of coal- and 76% of gas-generated electricity, thus avoiding 540,000 deaths and 11 Gt CO_2 emissions (Accelerating scenario) [XIV].

3.5. Other Benefits Forgone

If the pre-reversal learning rates had continued, OCC, and consequently the cost of electricity, would undoubtedly have declined. Arguably this would have led to other benefits not estimated in this analysis, such as increased productivity, faster economic growth, improved standard of living, and better health and education outcomes.

The declining cost of electricity would probably have caused increasing demand and consumption. Substitution of electricity for fossil fuels for heat and transport may have proceeded faster. With declining costs and increasing demand, electricity grids may have expanded faster with more people being connected. Alstone et al. [11] charts the world population and the number of people who were connected to an electricity grid for the period 1830 to 2013. If grid connections had continued to accelerate at the rate that prevailed between 1950 and 1975, many of the 1.2 billion people who were not grid-connected in 2015 could have been.

With increased consumption, electricity could have substituted for some combustion of fuels by the 3 billion people who cook and heat their homes using open fires and simple stoves burning wood, animal dung, crop waste and coal, thereby reducing the 4.3 million deaths per year attributable to indoor air pollution [7]. And clean water and sanitation systems could have been provided to more people, reducing deaths from contaminated water [6].

The benefits forgone may have been substantially greater than estimated in the present counterfactual analyses. World energy consumption slowed in the 1970s [3] and GDP growth rate slowed too [25]. If the transition from fossil fuels to nuclear power had not been disrupted, world GDP growth may not have slowed as much. The global economy could have been significantly different from what it is now.

3.6. Policy Implications

Policies that increase the real cost of energy would be damaging economically, and are unlikely to be politically sustainable and, therefore, unlikely to succeed in the long term. To reduce the emissions that are detrimental to health and the environment, countries will need access to low-emissions technologies that are cheaper than high-emissions technologies. In this case, carbon pricing and command-and-control policies, such as incentives for low emissions and penalties for high emissions technologies, would not be required.

Cheap electricity increases productivity and economic growth, drives faster electrification for the people without access to electricity or with insufficient and/or unreliable electricity, and thus more quickly lifts the world's population to higher living standards. As electricity costs decrease, the deployment rate increases and capacity doublings occur faster. Consequently, costs reduce faster; i.e., we progress more quickly down the learning curve [XV] [26]. Technology transition takes place faster and the benefits are delivered sooner.

These benefits could be achieved in the future if the impediments that disrupted the transition to nuclear power are removed. While this paper does not attempt to discuss the causes of the disruption and cost escalations thereafter, many others have (e.g., Cohen [15], Grubler [16], and Lovering et al. [14]

cites a number of studies). A likely root-cause of many of the causes discussed in the literature was the growing concern about the safety of nuclear power, fanned by the anti-nuclear protest movement, which began in the mid-1960s (Daubert and Moran [27]; Wyatt [28]), and the ongoing political, legislative and regulatory responses to the concerns.

The fact that rapid learning and deployment rates prevailed in the past suggests they could be achieved again. To achieve them, it is suggested four steps are needed:

- First, recognise that the disruption to the transition occurred and the impediments to progress continue to this day.
- Second, recognise the consequences of the disruption for the global economy, human wellbeing and the environment, and the ongoing delays to progress.
- Third, identify the root causes of the disruption and cost escalations since, and the solution options.
- Fourth, implement policies to remove impediments that are retarding the transition.

The benefits forgone cannot be recovered, but future benefits can be increased by amending the policies that caused the cost increases and slowed the deployment of nuclear power. Human wellbeing could improve faster if the impediments that are slowing the development and deployment of nuclear power are removed.

4. Conclusions

From 1954 to the late-1960s, learning rates of nuclear power OCC were positive (i.e., OCC decreased as capacity increased). In the late-1960s, learning rates turned negative (i.e., OCC increased as capacity increased) and have remained negative ever since in all the seven countries analysed, except South Korea.

The disruption to learning rates was followed by stalled deployment rates.

If the pre-1970s learning rates had continued, and assuming the actual deployment did not change, OCC of nuclear power in 2015 could have been around 5 to 15% of what it actually was.

If both the pre-1970s learning rates and the Linear or Accelerating deployment rates had continued, OCC in 2015 could have been around 2 to 10% of actual.

If deployment had continued to add 30 GW to global capacity per year since 1985, 69,000 TWh of extra nuclear electricity could have been generated. Assuming this replaced coal-fired electricity generation, 4.2 million deaths and 69 Gt CO_2 may have been avoided.

If deployment had continued from 1976 at the Accelerating rate that prevailed from 1960 to 1976, 186,000 TWh of extra nuclear electricity could have been generated. Assuming this extra nuclear generation replaced coal- and gas-powered electricity generation, 9.5 million deaths and 174 Gt CO_2 may have been avoided.

In 2015 alone, assuming electricity demand was unchanged, nuclear could have replaced between 49% of coal-powered generation (at the Linear deployment rate) and 100% of coal-powered plus 76% of gas-powered generation (at the Accelerating deployment rate), thereby avoiding between 273,000 and 540,000 deaths and between 4.5 and 11 Gt CO_2.

The policy implications are substantial. Benefits would be available in the future by returning to the pre-disruption learning and deployment rates. To achieve this requires a recognition of the disruption and its consequences, identification of its causes, and amelioration of the impediments that are slowing progress.

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Abbreviations

CO_2	Carbon dioxide
OCC	Overnight Construction Cost
EIA	Energy Information Administration
GDP	Gross Domestic Product
GEA	Global Energy Assessment
IAEA	International Atomic Energy Agency
IEA	International Energy Agency
WAES	Workshop on Alternative Energy Strategies
WHO	World Health Organisation
Gt	gigatonne
GW	gigawatt
GWh	gigawatt hour
MWh	megawatt hour
TWh	terawatt hour

Appendix A. Calculation of Extra Nuclear Electricity Generated, Deaths Avoided, CO_2 Avoided

Appendix A.1. Calculate Extra Nuclear Electricity Generated

The methods and data sources used are described below:

Historical actual nuclear electricity generated (TWh) per year, 1980–2015. Data source: EIA [22].

Historical actual global capacity (GW) of nuclear power plants in commercial operation each year, 1980–2015. Data source: EIA [19].

Projected global capacity (GW) of nuclear power plants in operation each year, 1980–2015. (The data sources and methods are different for the Linear and Accelerating projections of nuclear capacity.)

- Linear projection: Add 30 GW per year from 1985 to 2015 to the 253 GW global capacity in operation in 1985 [19] and subtract the capacity of reactors permanently shut down since 1985 [29].
- Accelerating projection: The cumulative global capacity of commercial operation starts is assumed to be equal to the cumulative global capacity of construction starts five years prior minus the cumulative global capacity of reactors permanently shut down. Therefore, the cumulative global capacity of commercial operation starts for a given year is calculated by subtracting five years from construction start date in the equation for the Accelerating projection and subtracting the total capacity of permanent shutdowns to date; that is:

$$0.9886 \times (\mathrm{CS} - 5)^2 - 3878.2 \times (\mathrm{CS} - 5) + 3{,}803{,}469 - \mathrm{PS}$$

where CS means construction start date (at the end of the year, e.g., for 2015, enter 2015.99), and PS means total capacity of reactors permanently shut down to date.

Projected nuclear electricity generated per year (TWh):

- TWh (projected) = TWh (actual) × [GW (projected) ÷ GW (actual)]
- (This assumes the average capacity factor of the additional plants would have been the same as for the existing plants in each year.)

Extra nuclear electricity generated per year (TWh):

- Extra TWh = TWh (projected) − TWh (actual)

Appendix A.2. Calculate Deaths Avoided and CO_2 Avoided

The methods and data sources are described below:

Linear: Assumes extra nuclear generation substituted for electricity generated by coal. To calculate deaths avoided, multiply extra nuclear generation (TWh) by 60/TWh (Wang [23]). To calculate Mt CO_2 avoided, multiply extra nuclear generation (TWh) by 1 Mt/TWh (Kharecha and Hansen [17]).

Accelerating: For the Accelerating deployment rate scenario, the extra nuclear generation exceeded the total electricity generated by coal by the year 2000. For simplicity, it is assumed that extra nuclear substituted for coal until all coal was replaced, then the remaining extra nuclear substituted for gas generation.

Under these assumptions, in 2015, extra nuclear generation would have substituted for 100% of electricity generated by coal and 76% of electricity generated by gas [XVI].

To calculate deaths avoided, multiply extra nuclear generation (TWh) substituting for coal by 60/TWh and substituting for gas by 4/TWh (Wang [23]).

To calculate Mt CO_2 avoided, multiply extra nuclear generation (TWh) substituting for coal by 1 Mt/TWh and substituting for gas by 0.6 Mt/TWh (Kharecha and Hansen [17]).

The electricity generated by coal and gas each year from 1980 to 2014 was sourced from The Shift Project Data Portal, Historical Electricity Generation Statistics [30]. Data for 2015 is not yet published; the trend from 2013 to 2014 was projected to 2015.

Appendix B. Notes

[I] Learning curve' and 'learning rate' are used throughout this paper because they are more widely used and recognised than the arguably more appropriate term 'experience curve'.

[II] Lovering's analysis has been critiqued by Koomey et al. [31] and Gilbert et al. [32]. Lovering et al. [33] responded with clarifications and additional information that are relevant for this paper.

[III] Lovering et al. [14] define Overnight Construction Cost (OCC) as: "The metric OCC includes the costs of the direct engineering, procurement, and construction (EPC) services that the vendors and the architect-engineer team are contracted to provide, as well as the indirect owner's costs, which include land, site preparation, project management, training, contingencies, and commissioning costs. The OCC excludes financing charges known as Interest During Construction."

[IV] Lovering et al. [14], explained why they used construction start dates rather than completion dates:

> "In contrast to other studies that assess historical cost trends by the reactor's date of commercial operation (Koomey and Hultman [34] and Grubler [16]), this study uses reactor construction start dates from the IAEA PRIS database, defined as the first foundation concrete pour. Because construction durations have been exceptionally long, up to 10–20 years at the extremes, the state of technology and the reactor designs are not representative of the date of eventual completion, but rather, more representative of the date of the start of construction. Using construction start dates to analyze the nuclear power experience allows for a focus on the cost characteristics of the "best available technology" at the time of deployment, consistent with the technological learning literature."

[V] The average construction duration of the early nuclear power reactors built globally (i.e., all countries) was: 3.5 years for the first three, 4.0 years for the first ten, 4.4 years for the first twenty, 5 years for the first thirty, and 5.4 years for the first eighty [18]. The first completed US power reactor was constructed and sending power to the grid in 1.8 years [29,35]. That was 60 years ago.

It's useful to compare how construction duration decreased in other large, complex systems as more were built. Fifty Casablanca Class aircraft carriers were built and commissioned for the US Navy between November 1942 and July 1944. The duration was reduced from a maximum of 277 days to 101 days [36]. This represents a learning rate for build duration of 22% for all fifty, and 34% for the last thirty-eight.

[VI] The 114 GW difference between cumulative global capacity of construction starts and of operating reactors is because 67 GW were under construction, with the remainder a combination of power uprates and permanent shutdowns.

[VII] These factors may be underestimates. Assuming the cost of nuclear plants was declining at the pre-reversal learning rates, and no changes to electricity demand profiles, few new coal plants would have been built; therefore, the coal plants that would have been displaced by nuclear would have been older plants of mostly 1950s, 1960s and early 1970s designs. These plants, comprising both black and brown coal, would have had relatively low thermal efficiencies, high emissions intensities of about 0.9 to 1.5 t CO_2/MWh and higher levels of pollution harmful to health. Furthermore, the proportion of nuclear replacing fossil fuels in non-OECD countries would have been accelerating, so the global averages for CO_2 emissions-intensity, pollution and deaths per TWh would have been higher than the figures quoted above, which are based mostly on the recent periods. The deaths avoided may be underestimated because the accelerating rate of deployment would imply more people would have gained access to electricity; this could have substantially reduced deaths as a result of greater access to clean water and sanitation services and less indoor pollution from burning biofuels and coal for heating and cooking.

[VIII] This note explains why the factor 60 Deaths/TWh, sourced from Wang, 2012 [23], was used for the counterfactual analyses of deaths avoided.

60 Deaths/TWh = "coal electricity—world average" (60) minus nuclear (0.09) (Wang, 2012) [23]. Brook et al. [37], use factors sourced from Wang (2011) [38] and modified (Brook, [39]). Conca and Wright [40] quote global average factors in deaths/TWh of 161 for coal, 4 for gas, and 0.04 for nuclear, sourced from Wang (2008) [41]. Kharecha and Hansen [17] use Markandya and Wilkinson [42] factors for the EU average, not the global average; they include an estimated mortality rate for China of 77/TWh, but do not give a global average. Cropper et al. [43] estimate the mortality rate for India at 99/TWh for three pollutants only (PM2.5, SO_2, NO_x) but do not include life cycle analysis, such as accident fatalities. Hirschberg et al. [44] do not present results for global average deaths/TWh. Following Wang [23], this analysis uses 60 deaths/TWh global average. However, this is an estimate for recent years. The rates have reduced significantly over the period 1985–2015. Therefore, the 60 deaths/TWh rate may be too low for the global average over the period, in which case the estimated number of deaths that could have been avoided may be an underestimate.

[IX] Discussion of the causes of disruption and the cost escalations thereafter is beyond the scope of this paper. However, one cause that has been recognised is real cost increases that applied generally, for example, add-on environmental requirements and materials and labour cost increases (McNerney et al. [45]). However, these are not the root causes. The root causes are what caused the add-on environmental controls, and the materials and labour cost increases.

Lovering et al. [14] explain that other electricity generation technologies, such as coal, also experienced increasing costs and negative learning rates since the 1970s, and suggest some possible causes. McNerney et al. [45] shows the learning rate for coal in the US was 12% from 1902 to 2006 (Learning rate = 1 − PR (Progress Ratio)). However, the learning rate from 1968 to 2006 was negative, coinciding with the period of negative learning rates of nuclear in the US (c.f. Lovering et al. [14], Figure 14). The cost of US coal plants increased by a factor of less than 2 during this period, whereas, the cost of US nuclear power reactors increased by a factor of around 7 for construction starts between 1968 and 1978 (the last construction start that went into commercial operation before the end of 2015). Arguably, the cost increases for environmental controls were justifiable for coal but not for nuclear. The nuclear learning rates have not been adjusted to remove the factors that also apply to other technologies.

[X] The IAEA data plotted in Figure 5 include all power reactors that started construction (584 GW), whereas Lovering et al. data (total 497 GW to 2015, including 11 GW added in 2014–2015) exclude those that did not enter commercial operation and demonstration reactor types that did not become commercial.

[XI] WAES [12] said: "Uncertainties surround all our estimates of demand and supply to 2000. Because different countries may choose different nuclear policies, the range of uncertainty in our nuclear projection is greater than for other fuels. On the other hand, extended delays on nuclear programs in various countries could hold nuclear power to the levels projected for 1985, which are based on commitments and construction already under way in most cases. On the other hand, a new awareness of the imminence of a deeper and continuing energy shortfall arising from reduced oil supplies might lead to a public re-appraisal of the risks and benefits of nuclear energy and a decision to accept the risks. All that we can do in this report is to show the scale of the contribution nuclear could make in 2000 and describe the issues in the public debate which will influence each country's political decision on nuclear risks."

[XII] Some readers may question the credibility of the projections of OCC in 2015. This is a counterfactual analysis of what the consequences would have been if the pre-disruption learning and deployment rates had continued. There is no apparent physical or technical reason why these rates could not have persisted. Actual learning rates may have been faster or slower than the pre-disruption rates depending on various socio-economic factors. It is beyond the scope of this paper to speculate on what global economic conditions, electricity demand, public opinion, politics, policy, regulatory responses and a multitude of other influencing factors may or may not have occurred over the past half century if the root causes of the disruption had not occurred. However, consider the following. A defensible assumption is that if the high level of public support for nuclear power that existed in the 1950s and early 1960s [12,27,28] had continued, the early learning rates may have continued and, therefore, the accelerating global deployment rate may have continued. With cheaper electricity, global electricity consumption may have been higher, thus causing faster development and deployment. In that case, we could have greatly improved designs by now—small, flexible and more advanced than anything we might envisage, with better safety, performance and cost effectiveness.

Rapid learning rates persisted since the 1960s for other technologies and industries, where public support remained high. The aviation industry provides an example of technology and safety improvements, and cost reductions, achieved over the same period in another complex system with high public concern about safety. From 1960 to 2013, US aviation passenger-miles increased by a factor of 19 [46], while aviation passenger safety (reduction in fatalities per passenger-mile) increased by a factor of 1051 [47], a learning rate of 87% for passenger safety. The learning rate for the cost of US commercial airline passenger travel during this period was 27% [46,48]. Similarly, the learning rate for solar PV (with persistent strong public support, favourable regulatory environments and high financial incentives) has remained high at 10 to 47% according to Rubin et al. (Figure 8) [13]. Cherp et al. [49] compare energy transitions of wind, solar and nuclear power in Germany and Japan since the 1970s and find their progress depends on the level of public support, political goals and policies of each country.

[XIII] This figure does not include the deaths that could have been avoided by increasing access to clean water and sanitation services and by reducing indoor air pollution as the declining cost and accelerating deployment of nuclear power enabled electricity to substitute for coal, oil and biofuels (wood, dung and crop residues) used for cooking, heating and lighting.

[XIV] With the Accelerating scenario, nuclear would have generated 66% of global electricity in 2015 (a lesser proportion if global electricity demand had grown faster). Is this a plausible scenario? France provides an example of what was achieved over the period despite the disruption to

learning rates. Nuclear was generating 75% of France's electricity by 1989 and generated 77% of its electricity between 1989 and 2015 [30].

[XV] "A focus on learning rates suggests two general categories of policy options. The first includes policies to speed progress down the learning curve, i.e., to speed the rate at which experience is accumulated in order that costs drop more quickly. The second category includes policies to steepen the learning curve by increasing the learning rate." (Rogner et al. [26]).

[XVI] Replacing 100% of coal- and 76% of gas-generation globally between 1975 and 2015 is recognised as an unlikely scenario. More likely is that, if the pre-reversal learning rates had continued so costs reduced as projected, demand for electricity would have increased. Electrification could have increased, including to some of the 1.2 billion people who are currently without it. Electricity could have substituted for other fuels, such as for some gas for heat and some oil for transport. Consequently, as demand increased, the extra nuclear generation would have replaced a lesser proportion of coal and gas generation. Therefore, less CO_2 would have been avoided. However, perhaps more deaths may have been avoided because of the reduction in deaths from indoor air pollution as electrification expanded into lower-income regions and the reduction of mortality and morbidity as supplies of clean water and sanitation services expanded to people without them.

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Original Article

It Is Time to Move Beyond the Linear No-Threshold Theory for Low-Dose Radiation Protection

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Abstract

The US Environmental Protection Agency (USEPA) is the primary federal agency responsible for promulgating regulations and policies to protect people and the environment from ionizing radiation. Currently, the USEPA uses the linear no-threshold (LNT) model to estimate cancer risks and determine cleanup levels in radiologically contaminated environments. The LNT model implies that there is no safe dose of ionizing radiation; however, adverse effects from low dose, low-dose rate (LDDR) exposures are not detectable. This article (1) provides the scientific basis for discontinuing use of the LNT model in LDDR radiation environments, (2) shows that there is no scientific consensus for using the LNT model, (3) identifies USEPA reliance on outdated scientific information, and (4) identifies regulatory reliance on incomplete evaluations of recent data contradicting the LNT. It is the time to reconsider the use of the LNT model in LDDR radiation environments. Incorporating the latest science into the regulatory process for risk assessment will (1) ensure science remains the foundation for decision making, (2) reduce unnecessary burdens of costly cleanups, (3) educate the public on the real effects of LDDR radiation exposures, and (4) harmonize government policies with the rest of the radiation scientific community.

Keywords

LNT, risk assessment, threshold, radiation, dose–response, hormesis

Introduction

The US Environmental Protection Agency (USEPA) was established in 1970 and gained authority to promulgate environmental standards to limit man-made radioactive materials in the environment and develop national radiation protection guidance for Federal and State agencies.[1] Congress enacted several statutes providing USEPA the authority to regulate hazardous materials (eg, Clean Air Act, Safe Drinking Water Act, and the Comprehensive Environmental Response Compensation and Liability Act), including both chemical and radiological hazards.[2] Among many federal programs whose regulatory authorities were transferred to the USEPA, the Public Health Service Act (PHSA) authorities are of particular interest in this article. The PHSA authorities give the USEPA the ability to conduct monitoring of environmental radiation, perform research on the environmental and human health effects of exposure to radiation, and provide technical assistance to states and other federal agencies. These authorities are consistent with the mission of the USEPA to protect human health and the environment.

This article examines the radiation protection framework and policies of the USEPA as they are applied to low-dose, low-dose rate (LDDR) radiation exposures. It focuses on current scientific literature, policy implications, public health impacts, and future directions for developing a radiation protection framework based on sound scientific principles.

In this article, we refer to dose in Gy (or mGy), unless citing a direct quote that uses other units. Low-dose throughout this report is arbitrarily defined as a dose of 100 mGy (10 rad) above natural background. Low-dose rate is defined as <0.01 mGy/min (1 mrad/min) above natural

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background. The definitions for LDDRs have varied over time but generally fall below 200 mGy for low-dose and <0.05 mGy/min for low-dose rate.[3]

The USEPA relies on the linear no-threshold (LNT) dose–response model developed in the US National Academy of Sciences (NAS) biological effects of ionizing radiation VII report[4] to (1) set regulatory standards to protect human health,[5] (2) project risks of LDDR radiation exposure among the US population, and (3) develop tools to help establish cleanup levels.[6] We critically review the latest scientific literature and present alternative risk assessment models (eg, threshold or hormesis) for determining radiological cleanup levels in environments containing low-level residual radioactivity. Throughout this article, we note USEPA's public policy positions for radiation protection and suggest alternative risk assessment approaches that are consistent with the latest science, protective of human health and the environment, and reduce unnecessary public health and financial burdens to society affected by low-level residual contamination from man-made or natural radioactive materials.

Two recent petitions to US regulators have drawn increased attention to this issue. In 2015, several members of the group, Scientists for Accurate Information (SARI), submitted petitions[7,8] to the US Nuclear Regulatory Commission (NRC), requesting "... that the NRC greatly simplify and change Part 20 to eliminate the use of the LNT paradigm and take radiation hormesis into account." This petition cited 36 references in support of the petitioners' request. The bases of the petition were also presented in a peer-reviewed scientific article.[9] The USEPA submitted comments opposing the petition[10]; however, the USEPA's comments declined to address all but 2 references cited by the petitioners. The SARI also recently submitted a letter to the current administrator of the USEPA,[11] requesting that USEPA cease the application of the LNT for LDDR environments. The USEPA's response[12] cited its comments on the NRC petition.

Another recent event relevant to this topic is the issuance of Executive Order 13777[13] by the President of the United States. This Executive Order established a policy to eliminate unnecessary regulatory burdens. As a result, the USEPA formed a Regulatory Reform Task Force to evaluate existing regulations and identify regulations that should be repealed, replaced, or modified. The USEPA administrator advised the Office of Air and Radiation (OAR) to provide recommendations regarding specific rules that could be repealed, replaced, or modified to make them less burdensome by May 15, 2017. The OAR hosted a public meeting on April 24, 2017, to solicit proposals. The Health Physics Society (HPS) gave verbal comments during the meeting urging USEPA to reconsider their adherence to LNT and to improve several documents (eg,[6,14-17]) by better addressing uncertainties in LDDR environments. The HPS also stated that reliance on the LNT model "... tends to foment the public's fear of all types of radiation." The HPS followed up with written comments, which stated,

> As a scientific organization of professionals who specialize in radiation safety, the HPS believes the EPA's reliance on the LNT model, especially at very low doses and dose rates, is inappropriate and can exaggerate the risk. Of most concern to the HPS is the EPA's extrapolation of the LNT model to calculate collective dose and the use of collective dose as a metric for risk.[18,19]

This article is divided into sections addressing several questions regarding the continued use of the LNT model for LDDR radiation environments:

I. Introduction
II. What is the scientific basis for using the LNT in LDDR radiation environments?
III. Is the USEPA using the concept of collective dose appropriately?
IV. Is there scientific consensus for using the LNT model to estimate risk in LDDR environments?
V. Should the BEIR VII report continue to be used to justify the use of the LNT model for LDDR radiation environments?
VI. What other information is available in the scientific literature and does it support the continued use of the LNT model for LDDR environments?
VII. Is it appropriate to regulate ionizing radiation in the same manner as toxic chemicals?
VIII. Should the current USEPA regulatory radiation policies be reconsidered and harmonized with the radiation protection philosophy given the lessons learned from Fukushima?
IX. Discussion
X. Conclusion

What is the Scientific Basis for Using the LNT in LDDR Radiation Environments?

Studies to understand health effects on people exposed to LDDR are especially important, since they most closely reflect the environment following a radiological cleanup effort. They also serve to help regulatory agencies determine whether the cleanup policies are adequate to protect the people and environment while accounting for social and economic factors (ie, do they do more good than harm to society?). Does the LNT model withstand scientific scrutiny to link cancer with causation from LDDR exposures to ionizing radiation? Over 50 years ago, Sir Austin Bradford Hill established a set of objective criteria that help determine when causation can be legitimately concluded from an observed correlation.[20] These criteria are (1) temporal relationship (eg, exposure must occur before the disease), (2) strength (eg, size of the association between exposure and disease), (3) dose–response relationship, (4) consistency, (5) plausibility, (6) consideration of alternate explanation (eg, confounding effects), (7) experiment (eg, the condition can be altered by an appropriate experimental regimen), (8) specificity, and (9) coherence (eg, associated

compatible with existing theory and knowledge?). Hill's criteria have been specifically applied to LDDR,[21] and the case for LDDR increasing carcinogenic risk has been found lacking. In the current article, we point out when any of Hill's criteria can be applied to particular arguments or evidence.

In its comments on SARI's petition to the NRC, the USEPA stated,

> The U.S. Environmental Protection Agency strongly disagrees with the petition to the Nuclear Regulatory Commission (NRC) to cease using the linear no-threshold (LNT) model as a basis for regulating exposures to ionizing radiation. The USEPA's Carcinogen Assessment Guidelines specify that LNT should be used as a default assumption unless there is compelling evidence that the biological mechanism for carcinogenesis is inconsistent with LNT.[10]

This argument was also published by a senior official within the USEPA in a scientific article using a disclaimer that the article represented his own personal opinion. However, his article continues to be used by the agency to justify reliance on the LNT model. Puskin wrote:

> Radiation protection, like the regulation of other carcinogenic agents, is—in the absence of compelling evidence to the contrary—predicated on the linear, no-threshold (LNT) hypothesis . . . [5]

These explanations are not consistent with basic scientific study designs that accept a null hypothesis (eg, no effect at low doses[22]), unless there is strong evidence (eg, statistical significance $P < .05$) to suggest otherwise (eg, LNT is valid at low doses). *The burden of proof lies with those asserting the LNT model is correct, not on those asserting the null hypothesis of no effect at low doses*. These arguments inappropriately shift the burden of proof to proving that LNT is not valid, which is an impossible task.[23] It can always be argued that an LNT-predicted risk might exist but is too small to be detected, rendering the LNT hypothesis unfalsifiable. To be scientifically sound, compelling evidence must be provided that the valid null (no effect at low doses) should be rejected in favor of an alternative hypothesis (eg, there are detrimental health effects at low doses, as predicted by the LNT model; or there are no detrimental health effects at low doses but there are effects at higher doses, as predicted by the threshold model; or there are beneficial health effects at low doses, as predicted by the hormesis model). The current USEPA policy takes the position that the LNT model is accurate unless "compelling evidence to the contrary" is presented. This approach is included in the agency's guidelines that direct the use of the LNT even if the scientific evidence cannot substantiate that conclusion. This is a circular argument that excludes the option of other alternative models from being considered.

USEPA goes on to comment,

> Biophysical calculations and experiments demonstrate that a single track of ionizing radiation passing through a cell produces complex damage sites in DNA, unique to radiation, the repair of which is error-prone. Thus, no threshold for radiation-induced mutations is expected, and, indeed, none has been observed.[10]

This statement relies on a biological plausibility argument to support the use of the LNT dose–response model in LDDR environments. However, a biologically plausible argument based on more recent scientific evidence suggests that extensive protective biological processes are initiated upon initial DNA damage to prevent potential development of cancer (eg, cellular- and tissue-level defense mechanisms including not only DNA damage repair but also apoptosis, premature terminal differentiation, and immunosurveillance[9,24,25]). As explicitly acknowledged by the National Council on Radiation Protection and Measurements (NCRP) over 15 years ago,[26]

> Application of this [microdosimetric] argument to complex endpoints such as radiation-induced carcinogenesis is, however, more uncertain. *Based on these biophysical considerations about the shape of the dose-response relation for low-dose radiation-induced carcinogenesis, conclusions can be drawn if*: (1) radiogenic cancer induction is causally related to radiation induced damage in a single cell and (2) *the ways in which other cells or cell systems subsequently modify the probability that any given initially radiation-damaged cell becomes the clonal origin of a cancer do not vary with dose in a nonlinear fashion*. (emphasis added)

More and more scientific evidence has accumulated in recent years that neither of these underlying assumptions are valid.[24,27] In fact, even references cited by USEPA as supporting this position actually contradict it. For example, Trott and Rosemann stated,

> Since the cell is able to repair a very high level of endogenous DNA damage without frequent mutagenic consequences, a further small increment of such DNA damage from low dose rate irradiation should, equally efficiently, be repaired. Mutation rates will only increase if due to higher dose and dose rate, the capacity for high fidelity DNA repair is exceeded.[28]

And also,

> The mechanism which induces 'radiation-induced genomic instability' appears to involve a non-nuclear target and upregulation of oxidative stress, which also is the main mechanism of metabolic DNA damage. These experimental observations are not compatible with a single hit mechanism which is the basis for the microdosimetric justification of the linear-non threshold dose response hypothesis.[28]

Current evidence demonstrates that biological responses to LDDR radiation are distinct from those occurring at high doses.[21,24,29-33] Similarity of mechanisms is one of the fundamental assumptions underpinning the LNT extrapolation from

high-dose and high-dose rate (HDDR) to LDDR, and there is growing evidence that this assumption is inaccurate.

The USEPA's assertion that no threshold in radiation-induced mutations has been observed is inaccurate. Early data on mutations in fruit flies were very influential in adoption of the LNT model. These data actually indicated a threshold but was misrepresented as supporting the LNT model.[34-36] In similar experiments, more recent studies examining mutations in fruit flies confirm that the dose–response is characterized by a threshold or even hormesis.[37-41] These studies relate to another of Hill's criteria—Experiment which can greatly strengthen the case for causation.[20] However, these studies do not support the LNT model but rather a threshold or hormesis model.

A threshold for radiation-induced mutations has also been observed in mice,[42-46] human-hamster hybrid cells,[47] and human cells.[48] These findings also relate to another of Hill's criteria—Consistency, defined by Hill as generality or repeatability[20]—but here again, they do not support the LNT model; instead, they demonstrate thresholds.

The USEPA's own Scientific Advisory Board (SAB)[49] has cautioned the Agency on taking this position on LNT, stating,

> Radiation-induced genomic instability seems to be one of the early stages in the carcinogenesis process and has been seen both *in vitro* and *in vivo*. *These observations challenge the relative importance that initial mutations play in radiation-induced cancer*,[50]

and further,

> *Genomic instability and the ability to modify responses after the radiation exposure both challenge the linear relationship between initial DNA damage and cancer frequency*. (emphasis added)

The USEPA response suggests that unless cells repair DNA damage with 100% fidelity, the risk of cancer is increased.[5,10] This is not supported by current evidence.[24] DNA repair mechanisms act on both radiation-induced damage and on pre-existing spontaneous background DNA damage resulting from oxygen metabolism and other endogenous sources. If the resulting sum of radiation plus spontaneous DNA damage after radiation exposure is less than the level of damage that existed prior to radiation exposure, it is entirely reasonable and biologically plausible that radiation risks are not increased (consistent with a threshold) or may even be decreased (consistent with hormesis).

Nonetheless, USEPA continued,

> Of all the agents demonstrated to be carcinogenic, the evidence for LNT is particularly strong for ionizing radiation. Within limitations imposed by statistical power, the available (and extensive) epidemiological data are broadly consistent with a linear dose-response for radiation cancer risk at moderate and low doses.[10]

Strength of association is another of Hill's criteria.[20] The USEPA states the evidence is strong and consistent with the LNT response at moderate and low doses. However, radiation in general is a weak carcinogen,[51,52] and the evidence that LDDR radiation exposure in particular increases cancer risk is lacking.[21] In fact, many professional organizations have explicitly warned against estimating risks from low-dose radiation environments due to large uncertainties associated with the epidemiologic data.[53-55] The USEPA's position on this point appears to contradict their own guidance document,[6] which states,

> Generally speaking, epidemiology cannot be used to detect and quantify the carcinogenic effects of radiation at doses below about 100 mGy of low-LET [linear energy transfer] radiation because of limitations on statistical power.[56,57]

Is the USEPA Using the Concept of Collective Dose Appropriately?

International expert advisory bodies have repeatedly cautioned against application of the LNT model to calculate hypothetical risks from LDDR exposures.[53,55] For example, United Nations Scientific Committee on the Effects of ionizing Radiation (UNSCEAR) has stated,

> In general, increases in the incidence of health effects in populations cannot be attributed reliably to chronic exposure to radiation at levels that are typical of the global average background levels of radiation. . . . the Scientific Committee does not recommend multiplying very low doses by large numbers of individuals to estimate numbers of radiation-induced health effects within a population exposed to incremental doses at levels equivalent to or lower than natural background levels.[53]

Similarly, the ICRP has stated,

> Collective effective dose is an instrument for optimisation, for comparing radiological technologies and protection procedures. Collective effective dose is not intended as a tool for epidemiological studies, and it is inappropriate to use it in risk projections. This is because the assumptions implicit in the calculation of collective effective dose (*e.g.,* when applying the LNT model) conceal large biological and statistical uncertainties. Specifically, the computation of cancer deaths based on collective effective doses involving trivial exposures to large populations is not reasonable and should be avoided. Such computations based on collective effective dose were never intended, are biologically and statistically very uncertain, presuppose a number of caveats that tend not to be repeated when estimates are quoted out of context, and are an incorrect use of this protection quantity.[55]

Despite this guidance, the USEPA develops risk estimation tools based on the LNT model to determine cleanup policies and guidelines for its Comprehensive Environmental

Response, Compensation, and Liability Act (CERCLA) superfund sites. Because they multiply very small doses by large populations to predict excess cancer incidence or mortality, these tools conflict with the scientific guidance provided by other governmental or scientific organizations and professional societies. The impact to the United States is real, resulting in enormous cleanup costs that show no demonstrable benefit to society, creates a social stigma on affected communities, and foments fear among the public, causing unnecessary harm by promoting ill-advised decision-making. The USEPA's estimates of cancer incidence and mortality risks due to low doses of ionizing radiation for US population as well as their advice to the public and tools used to establish cleanup levels are at odds with UNSCEAR's and ICRP's guidance. For example, USEPA states,

> . . . overall, if each person in a group of 10,000 people exposed to 1 rem of ionizing radiation, in small doses over a life time, we would expect 5 or 6 more people to die of cancer than would otherwise. In this group of 10,000 people, we can expect about 2,000 to die of cancer from all non-radiation causes. The accumulated exposure to 1 rem of radiation, would increase that number to about 2005 or 2006.[58]

This advice to the public is inconsistent with the intended purpose of effective dose (prospective dose estimation for the purpose of optimization), which is inappropriate for predicting future cancer risk.[59]

Is There Scientific Consensus for Using the LNT Model to Estimate Risk in LDDR Environments?

USEPA's comments on the public petitions to the NRC[7,8] stated,

> *Given the continuing wide consensus on the use of LNT* for regulatory purposes as well as the increasing scientific confirmation of the LNT model, it would be unacceptable to the USEPA to ignore the recommendations of the NAS [US National Academy of Sciences] and other authoritative sources on this issue. The USEPA cannot endorse basing radiation protection on poorly supported and highly speculative proposals for dose thresholds or doubtful notions concerning protective effects from low-level ionizing radiation. Accordingly, we would urge the NRC to deny the petition.**[10]** (emphasis added)

And similarly,

> Over the last half century, numerous authoritative national and international bodies have convened committees of experts to examine the issue of LNT as a tool for radiation regulation and risk assessment. These include the U.S. National Academy of Sciences (NAS), the National Council on Radiation Protection and Measurements (NCRP), the International Commission on Radiological Protection (ICRP), and the United Nations Scientific Committee on the Effects of ionizing Radiation (UNSCEAR). Again and again, these bodies have endorsed LNT as a reasonable approach to regulating exposures to low dose radiation. One exception was a French National Academy Report, which found low-dose radio biological effects in vitro indicative of nonlinearity in the dose response.[10]

This argument was also repeated in[5]:

> To assist the Agency in its assessment of the health risks from ionizing radiation, EPA has often helped sponsor reports from these organizations, particularly from the NAS 'BEIR Committees'. The risk models and supporting evidence is then reviewed by EPA's Scientific Advisory Board of outside distinguished scientists before becoming final and being implemented. Thus, EPA's estimates of risk to low dose radiation reflect a broad scientific consensus.

In these arguments, the USEPA "appeals to authority,"[23] where the LNT model is asserted to be valid because some authority putatively endorses it. This is an academic point because there is in fact no consensus in favor of the LNT model among individual scientists, professional societies, expert advisory bodies, US regulators, nor even within USEPA itself. As acknowledged earlier, contradictory recommendations were issued by the French National Academies of Science and Medicine,[60] and evidence supporting the French conclusions has grown in the recent years. The French report contradicts the claim of consensus among expert advisory bodies in support of the LNT model.[5,10]

The USEPA's own SAB has expressed caution about applying the LNT at low doses as well. The USEPA has claimed that unfettered application of the LNT,

> . . . is the position adopted by the USEPA after review by the Agency's Scientific Advisory Board, an independent group of distinguished outside scientists.**[10]**

However, the SAB's Radiation Advisory Committee cautioned[49]:

> . . . a major issue with the choice of the LNT model is whether it is appropriately applied at low doses.
>
> . . . while the RAC endorses USEPA's use of the LNT model, the Agency is advised to continue to monitor the science of the biological mechanisms underlying cancer induction at low doses of ionizing radiation and of their influence on the biophysical models used to estimate the cancer risk in this dose range.
>
> At radiation exposures in the range of natural background, it is difficult to distinguish radiation-induced changes in risk from the baseline. Thus, as a cautionary note, the RAC recommends that the USEPA discuss potential problems associated with the use of LNT dose response model risk estimates in very low dose settings. Currently at these low doses, statistically significant differences between the cancer rates among 'exposed' (defined study populations) and 'non-exposed' (defined comparison populations) are not observed.

> As BEIR VII acknowledges, the epidemiological data below 100 mSv (0.1 Sv) are not sufficient by themselves for risk estimation, and considerable cellular and animal data suggest complexities beyond the application of a simplified DNA damage model which historically has been used as support for an LNT dose-response model.

It is important to note that since the SAB last took up this issue and advised USEPA to explicitly monitor developments on these topics, the NCRP has issued comprehensive reports on uncertainties in the measurement and dosimetry of external radiation,[61] internal radiation dose,[62] and in the estimation of radiation risks.[63]

There is also no consensus among US regulators. The US General Accounting Office (GAO) has on multiple occasions investigated whether or not there is a consensus among USEPA, the NRC, and the Department of Energy (DOE) on approaches to regulating LDDR radiation exposures to the public.[2,64-66] Over 20 years ago, the GAO found,

> the radiation standards that have been developed reflect a lack of overall interagency consensus on how much radiation risk to the public is acceptable

and also,

> Differences in radiation limits and risks, calculation methods, and protective strategies reflect the historical lack of a unified federal framework for protecting the public from radiation exposure.[65]

The situation had not been resolved by 2000, with GAO finding,[2]

> U.S. regulatory standards to protect the public from the potential health risks of nuclear radiation lack a conclusively verified scientific basis, according to a consensus of recognized scientists. In the absence of more conclusive data, scientists have assumed that even the smallest radiation exposure carries a risk. This assumption (called the 'linear, no-threshold hypothesis' or model) extrapolates better-verified high-level radiation effects to lower, less well-verified levels and is the preferred theoretical basis for the current U.S. radiation standards. However, this assumption is controversial among many scientists

and also,

> . . . USEPA and NRC have disagreed on exposure limits. Although we recommended as far back as 1994 that the two agencies take the lead in pursuing an interagency consensus on acceptable radiation risks to the public, they continue to disagree on two major regulatory applications: (1) the proposed disposal of high-level nuclear waste in a repository at Yucca Mountain and (2) the cleanup and decommissioning of nuclear facilities.

As recently as 2017, the GAO again recommended the DOE take the lead on reestablishing and coordinating federal research on the topic of low-dose radiation effects.[66]

There is also no consensus in support of the LNT model among relevant professional societies.[54,67-69] Extrapolation of LDDR risks via the LNT model is at odds with the advice of professional societies around the world. For example, the Australasian Radiation Protection Society has stated,

> There is insufficient epidemiological evidence to establish a dose-effect relationship for effective doses of less than a few tens of millisieverts in a year above the background level of exposure and further, . . . no inference may be drawn concerning the risk to health or risk of fatality of an individual from an effective dose below 10 mSv in a year. For individual doses less than some tens of millisieverts in a year, risk inferences are unreliable and carry a large uncertainty that includes the possibility of zero risk.[68]

In the United States, the HPS has concluded,

> The Health Physics Society advises against estimating health risks to people from exposures to ionizing radiation that are near or less than natural background levels because statistical uncertainties at these low levels are great . . . Substantial and convincing scientific data show evidence of health effects following high-dose exposures (many multiples of natural background). However, below levels of about 100 mSv above background from all sources combined, the observed radiation effects in people are not statistically different from zero. Scientists evaluate and estimate radiation risk using several assumptions that, taken together, may lead to a range of hypothetical health risk estimates for any given exposure scenario. For radiation protection purposes and for setting radiation exposure limits, current standards and practices are based on the questionable premise that any radiation dose, no matter how small, could result in detrimental health effects such as cancer or heritable genetic damage. Implicit in this linear no-threshold (LNT) hypothesis is the core assumption that detrimental effects occur proportionately with radiation dose received (NAS/NRC 2006). However, because of statistical uncertainties in biological response at or near background levels, the LNT hypothesis cannot provide reliable projections of future cancer incidence from low-level radiation exposures (NCRP 2001).[54]

Additional examples from medical physics and radiology professional societies are provided in "What Other Information Is Available in the Scientific Literature and Does It Support the Continued Use of the LNT Model for LDDR Environments?" section.

In addition to expert advisory bodies and professional societies, numerous individual scientists have argued against application of the LNT at low doses.[24,70-72] Studies have also been conducted of individual scientists' views regarding the accuracy of the LNT dose–response model for radiation effects[73,74] (Table 1). A survey of scientists employed at US national laboratories revealed that 70% believed that a threshold model

Table 1. Survey of Scientists Regarding the Most Accurate Radiation Dose–Response Model for Cancer.[73,74]

Surveys	Respondents	Percent Supporting LNT Model	Percent Supporting Threshold Model	Other
United States	National Labs	12	70	18[a]
	Union of Concerned Scientists	21	48	31[a]
Subscribers to *Science*	United States	19	75	6[b]
	Britain	21	71	8[b]
	France	18	70	13[b]
	Germany	22	64	13[b]
	Other European Union	23	69	8[b]

Abbreviation: LNT, linear no-threshold.
[a]The "other" category includes "supralinear" and "don't know" responses.
[b]The "other" category includes "supralinear" responses.

accurately reflected radiation effects, compared to only 12% who believed an LNT model is accurate.[74] Even among members of the Union of Concerned Scientists, a group that has expressed concerns about the US nuclear power industry, 48% believed a threshold model accurately describes LDDR effects while only 21% favored an LNT model. The results were similar when scientists from the United States and Europe who subscribe to the journal *Science* were surveyed[73]: (1) 75% of US scientists believed a sublinear threshold model accurately described radiation effects, compared to only 19% who favored an LNT model; (2) for British scientists, the breakdown was 71% for sublinear threshold and 21% for LNT models; (3) for French scientists, 70% and 18%, respectively; (4) for German scientists, 64% and 22%, respectively, and (5) for other European scientists, 69% and 23%, respectively. These studies indicate that a majority of individual scientists are skeptical of the accuracy of the LNT model—exactly the opposite of a pro-LNT consensus claimed by USEPA.[5,10]

Should the BEIR VII Report Continue to be Used to Justify the Use of the LNT Model for LDDR Radiation Environments?

In short, the answer is "no." The USEPA places great weight on a few scientific references to support its application of the LNT model, most notably, the BEIR VII report from the US NAS.[6] For example, USEPA states,

> The BEIR VII study, which was sponsored by several federal agencies including the USEPA and the NRC, determined that 'the balance of evidence from epidemiologic, animal and mechanistic studies tend to favor a simple proportionate relationship at low doses between radiation dose and cancer risk.'[10]

The NAS originally adopted the LNT model as the basis for its philosophy to protect against radiation-induced genetic mutations in the human population at the recommendation of its Biological Effects of Atomic Radiation Committee Genetics Panel in 1956.[75] This recommendation was made in spite of the fact that radiation-induced genetic effects in the offspring of irradiated parents have never been observed in humans. Recent historical research has revealed that this recommendation was made under questionable circumstances ([76-80] but see also[81-83]). Even so, the LNT model was later expanded and applied to radiation-induced cancer risks. Controversial from the beginning, this recommendation nevertheless initiated decades of institutional inertia, with multiple iterations of NAS Committees repeatedly reaffirming the suitability of the LNT model as the basis of radiation protection philosophy, most recently in the BIER VII report over a decade ago.[4] The BEIR VII Committee concluded,

> . . . current scientific evidence is consistent with the hypothesis that there is a linear, no-threshold dose-response relationship between exposure to ionizing radiation and the development of cancer in humans.

Although they acknowledged that a linear-quadratic model fit the data better than the LNT model at low doses, they reported the improvement was not statistically significant. In large part, because the NAS inappropriately treated the LNT model as if it were the null hypothesis rather than appropriately treating it as an alternative hypothesis to be tested against the null of no effect, the LNT model became the Committee's preferred recommendation. In turn, the USEPA incorporated BEIR VII risk models into their policy and guidance.[84]

However, two major pieces of evidence the BEIR VII Committee relied upon to support their endorsement of the use of the LNT model to estimate risks from low doses, the Lifespan Study (LSS) of the Japanese atomic bomb survivors and the 15-country study of nuclear workers, no longer support the LNT model.[85] We summarize the problems with continuing to cite these two pieces of evidence to justify risk estimates using the LNT model in LDDR environments below.

It is widely acknowledged (in the BEIR VII report and elsewhere) that the LSS was the most influential study in setting radiation protection guidelines around the world. It is also evident that even these data set do not provide definitive evidence of increased cancer risk after exposure to low radiation doses.[86] In fact, the most recent epidemiological study on cancer mortality in the Japanese survivors of the atomic bombings states,

> the estimated lowest dose range with a significant ERR [excess relative risk] for all solid cancer was 0 to 0.20 Gy.[87]

Another way of saying this is that no significant ERR was observed for doses below 0.20 Gy. The authors also concluded that,

> . . . statistically significant upward curvature was observed when the dose range was limited to 0–2 Gy . . . The curvature over the 0–2 Gy range has become stronger over time.

This means the argument for an LNT relationship has weakened over time. This is an example of epidemiological data possibly reflecting dissimilarity of biological responses to LDDR and HDDR; however, it is not discussed by the authors in spite of explicit calls to integrate biology and epidemiology.[88,89] Despite that evidence, these authors concluded,

> . . . a formal dose-threshold analysis indicated no threshold; *i.e.* zero dose was the best estimate of the threshold.[87,90]

Reviewing their threshold analysis, others found that they excluded the possibility of negative risk values despite eight of the 10 lowest data points having confidence intervals, including negative values. Alternative analyses that did not exclude negative values revealed the possibility of a nonzero threshold.[35,91-94]

Similarly, for cancer incidence in the LSS cohort,

> The lowest dose range that showed a statistically significant dose response using the sex averaged, linear ERR model was 0–100 mGy.[95]

In other words, there are no detectable health effects below 100 mGy. It is evident that statistical power limitations preclude the selection of one alternative hypothesis over another (eg, LNT vs linear with threshold); therefore, the assertion that the LSS data provide definitive evidence in support of the LNT is not accurate. A threshold model is also consistent with both the latest solid cancer incidence and the mortality data.

The second piece of evidence the BEIR VII Committee relied heavily upon was the so-called "15-country study."[96] This study initially concluded that,

> Significantly increased risks were found for mortality from all cancers excluding leukemia and from lung cancers.

However, further analysis revealed that this conclusion is also no longer valid. The Canadian Nuclear Safety Commission concluded that Atomic Energy of Canada, Ltd nuclear energy workers cohort included in the original 15-country study did,

> . . . not have an increased risk of solid cancer mortality. Incomplete dose records are likely the cause for the apparent increased risk of solid cancer mortality in AECL NEWs first employed before 1965 (1956-1964).[97]

Furthermore, Zablotska et al[98] concluded:

> Significantly increased risks for early AECL workers are most likely due to incomplete transfer of AECL dose records to the National Dose Registry. Analyses of the remainder of the Canadian nuclear workers (93.2%) provided no evidence of increased risk

and,

> Study findings suggest that the revised Canadian cohort, with the exclusion of early AECL workers, would likely have an important effect on the 15-country pooled risk estimate of radiation-related risks of all cancer excluding leukaemia by substantially reducing the size of the point estimate and its significance.

These findings should serve as a warning against relying on BEIR VII to justify the use of the LNT model for LDDR risk estimation purposes.

In summary, two influential pieces of evidence relied upon by the BEIR VII Committee (the LSS cohort and the 15-country study) no longer support the LNT model based on the latest scientific literature. However, the USEPA relies heavily upon the recommendations of the BEIR VII report on this issue and continues to use it to support is current policies and risk assessment strategies. This evidence alone is enough to warrant a new look at the science for risk assessment decision-making and determining radiation cleanup levels in LDDR environments.

What Other Information is Available in the Scientific Literature and Does it Support the Continued Use of the LNT Model for LDDR Environments?

The USEPA has cited studies published after BEIR VII, which they assert provides support for the LNT model in LDDR environments[99]:

> Since publication of BEIR VII, additional evidence has accumulated supporting the use of LNT to extrapolate risk estimates from high acute doses to lower doses and dose rates. In this connection, we would note, inter alia, results of epidemiological studies on: nuclear workers in the United States, France and the United Kingdom[100]; residents along the Techa River in Russia who were exposed to radionuclides from the Mayak Plutonium Production Plant[101,102]; and children who had received CT scans.[103] These studies have shown increased risks of leukemia and other cancers at doses and dose rates below those which LNT skeptics have maintained are harmless - or even beneficial.[10]

Follow-up studies of a selected part of the cohort included in the 15-country study has recently been published to examine leukemia[100] and solid cancer[104] risks. These studies, also known as the International Nuclear Workers Study (INWORKS)] studies, examined risk in worker cohorts from the United States, France, and the United Kingdom (a subset of the larger cohort included in the 15-country study). The leukemia study[100] concluded,

> This study provides strong evidence of positive associations between protracted low-dose radiation exposure and leukaemia.

Similarly, the solid cancer study[104] concluded,

> The study provides a direct estimate of the association between protracted low dose exposure to ionising radiation and solid cancer mortality.

Several methodological questions have been raised about these studies,[105,106] and the authors have replied.[107] In addition, numerous methodological objections have been raised in Sacks et al.[108] These include:

1. failure to account for natural background radiation exposure, the differences in which potentially dwarf the occupational exposures of the study cohort;
2. failure to account for medical exposures experienced by the public;
3. failure to account for dose–rate effects;
4. the a priori assumption of an LNT dose response;
5. mischaracterization of the *y*-intercept as 0 total dose when in fact it was 0 occupational dose;
6. arbitrary exclusion of all dose responses except LNT and linear-quadratic (which actually provided a better fit to their observed data, but the authors claimed the improvement was not statistically significant);
7. dismissing 6 of 7 disease outcomes as being highly imprecise rather than stating that they are not statistically significantly different from no-effect;
8. creating an artificial disease category by arbitrarily combining 3 forms of leukemia and excluding a fourth, then characterizing this artificial grouping as an additional statistically significant association;
9. providing misleading characterizations of the data above 200 mGy as statistically significant when in fact, only the 200 to 300 mGy dose category was significantly elevated, whereas the highest dose category was not (nor was any other dose category);
10. insufficient consideration of age as a possible confounder;
11. a priori and arbitrary consideration only of the possibility of increased risks and excluding the possibility of decreased risks; and
12. the arbitrary choice of a 90% confidence limit rather than the more conventional 95%, thus increasing the possibility of significance, then mischaracterizing the results as strong evidence of risk from LDDR radiation exposure.

To this list of methodological shortcomings, we add the omission of *occupationally required* medical imaging examinations (which are distinct from medical doses received by the public at large—raised as #2 above), resulting in potential significant underestimation of external radiation dose. With regard to potential confounding by diagnostic medical dose, the INWORKS authors state,

> . . . for confounding to occur, medical radiation exposures would need to be associated with occupational doses . . . which is unlikely to be the case.[107]

The basis for the authors' conclusion that such confounding is unlikely is not provided. The omission of dose from medical imaging received by workers as a condition of employment presents one of the most serious questions about the methodology of these studies, as it likely resulted in potentially significant underestimation of external radiation dose. At several of the US sites included in the study, workers were required to undergo a medical examination at least yearly, which included medical imaging examinations. Of particular concern is the use of photofluorography in the early years (eg, 1940s to 1950s). Photofluorography delivered high-dose rate radiation exposures to workers at the Savannah River Site (1951-1960, 0.46 mGy per examination to male red bone marrow),[109] Hanford (1943-1962, 1.41 mGy),[109] and the 3 Oak Ridge Sites: Y-12 (at least 1943-1947, 2.76 mGy),[110] X-10 (at least prior to 1947, 2.58 mGy),[111] and K-25 (1945-1956, 2.0 mGy).[112] So, for example, a worker at Hanford from 1943 to 1962 could have received a red bone marrow dose of ~27 mGy from photofluorography alone. Although these are not especially large doses, the authors reported recorded mean occupational external bone marrow doses of only 16 mGy and median doses of only 2.1 mGy, and they claim to have observed increased leukemia risks. If that is true, then even larger potential doses from occupationally required medical examinations cannot be casually dismissed. The impact of medical imaging examinations workers received as a condition of employment has been specifically studied at one of the sites included in the INWORKS study.[113,114] Work-related medical imaging examinations were the predominant source of radiation exposure among workers at the K-25 site. In fact, the work-related medical imaging dose was on average 50 times higher than the recorded occupational dose.[113] Occupationally required medical imaging could certainly influence the estimation of possible thresholds (which the authors of the INWORKS studies did not report), estimates of risk per unit dose, and the shape of the dose–response relationship.[113] Furthermore, at some sites, workers judged to be at high risk (eg, those performing jobs where they received higher occupational radiation dose) were examined more frequently, indicating nonrandom distribution of medical radiation exposure among the cohort and subsequent bias. Neglecting this important source of exposure seriously compromises the conclusions of the INWORKS study. At least for the US sites, workers' medical records are available, so including this dose should be feasible. The importance of this issue for the UK and French cohorts included in the INWORKS study should also be examined.

For the Techa River cohort, it is unclear why USEPA chose to cite an outdated reference[101] when there is a more recent update[115]; however, risk estimates in the most recent update are less than half of the estimates in the earlier reference USEPA cited. Furthermore, Krestinina et al[115] states,

> For the basic dose–response model, the ERR was assumed to be linear in dose but we also considered models where the dose response was taken as a linear-quadratic, a pure quadratic function of dose, or threshold models in which the ERR was assumed to be 0 up to some threshold dose and taken as linear for higher doses.

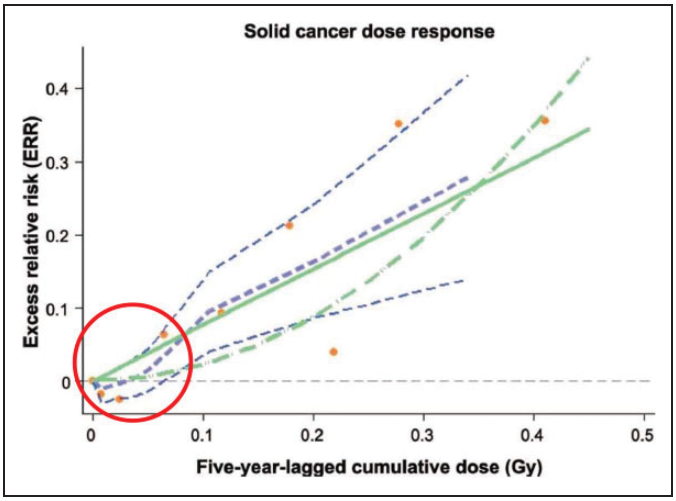

Figure 1. Solid cancer excess relative risk (ERR) estimates for the Techa River cohort plotted against stomach dose. Reproduced from figure 1 of Davis et al[102], used with permission, circle added for emphasis.

No further details are provided on their analysis of thresholds. It is not clear whether the authors allowed ERR to assume negative values, which would certainly be indicated given that the total leukemia rates reported for the 5 lowest dose groups were lower than the control group (those who received <0.01 Gy). Only the 2 highest dose groups (those receiving 0.5-1 Gy and 1+ Gy) exceeded controls. For leukemia excluding chronic lymphocytic leukemia, the rates for 2 of the 3 lowest dose groups were below that for the control group, suggesting a threshold or even potential hormetic effect which is often dismissed as a potential healthy worker effect. The authors reported that their data, "... are consistent with a linear dose response..."; however, they do not report whether or not their data are also consistent with a threshold or hormetic dose response, which would seem to be the case given these results. If multiple models adequately describe the observed dose response, then USEPA should not cite these results as supporting the LNT model and excluding the threshold model as petitioned by SARI.

For solid cancers in the Techa River Cohort, the situation is similar. The USEPA cited,[102] and again, the authors claimed,

> There is a statistically significant ($P = 0.02$) linear trend in the smoking-adjusted all-solid cancer incidence risks.

However, a closer look at the data in this study reveals that the two lowest dose categories have ERR estimates lower than the zero dose controls, consistent with a hormetic dose response or at least a threshold (Figure 1). This is another example of epidemiological data possibly reflecting the dissimilarity of biological responses to LDDR and HDDR, but again it is not discussed by the authors.

Within the past few years, new studies of pediatric patients receiving computed tomography (CT) medical imaging examinations claimed to observe increases in risks from relatively low doses (though delivered at a high-dose rate).[103,116] These studies received extensive press coverage, and almost immediately, claims were made that,

> ... the new data confirm that the cancer risk associated with the radiation from a CT scan is very small, but not zero.[117]

In presentations to the Interagency Steering Committee on Radiation Standards, USEPA has referenced these studies to suggest potential adverse health effects from LDDR radiation.[99] However, these early enthusiastic pronouncements have not held up to scientific scrutiny. A number of significant methodological issues have been identified in these studies,[118,119] including (1) individual doses were not directly assessed, but rather "typical" doses were assumed; (2) doses applied were for adults and assumed no decrease for pediatric patients, even though this is the standard of care; and (3) the reason for the CT was not considered, and it is possible that the underlying condition indicating the CT has associated cancer susceptibility (this point was acknowledged in one of the USEPA presentations[99,120]). On the latter point, as explained by Ulsh,[91]

> One of the strongest associations[103] observed was for gliomas, but they did not control for prior head injury. Head injuries are a common reason for head CT in children, and head injury may be associated with brain tumors.

This assessment agrees with UNSCEAR,[121] which concluded

> ... There are concerns about the risk estimates because of lack of information about indications for the CT scans and the consequent potential for 'reverse causation' (*i.e.,* cancers may have been caused by the medical conditions prompting the CT scans rather than by the CT dose).

The NCRP came to similar conclusions, stating:

> Children who receive frequent examinations may have some underlying disability related to the outcome of interest. That is, a child who receives multiple CT examinations of the head may have a central nervous system disorder that is prompting such examinations and it is these underlying disorders that are related to the cancer diagnosis and not the CT radiation dose.[63]

Furthermore, two recent studies from France[122] and Germany[123] have demonstrated that failing to account for the underlying reason requiring the examination can inflate risk estimates in studies of populations exposed to CT scans.

In spite of the UNSCEAR and NCRP conclusions, and multiple papers pointing out the limitations of these studies (eg,[91,119]), they continue to be cited by USEPA and others as providing strong or definitive evidence of risks of very low radiation doses and supportive of the LNT model.[99] However, the application of the LNT model and the As Low As Reasonably Achievable (ALARA) principle to medical imaging has come under heavy criticism.[72,124-126] Professional societies

with expertise in medical imaging continue to unanimously maintain that the carcinogenicity of low radiation doses has not been demonstrated, and estimates of risks from low doses like those associated with medical imaging examinations remain speculative and unproven. For example:

- American Association of Physicists in Medicine

  At the present time, there is no convincing epidemiological evidence of increased cancer incidence or mortality from low radiation doses (<100 mSv). Because medical imaging exposures are typically much lower than 100 mSv, when such exposures are medically appropriate, the anticipated benefits to the patient are highly likely to outweigh any small potential risks. Therefore, when discussions of risk occur, it is essential that the benefit of the clinical task also be discussed. Additionally, the AAPM discourages describing potential risks associated with medical imaging using predictions of hypothetical cancer incidence and deaths. These predictions are contrary to directives of radiation protection organizations, are highly speculative and can lead to sensationalistic coverage in the public media, leading some patients to fear or refuse appropriate medical imaging.[69]

- International Organization for Medical Physics

  Prospective estimates of cancers and cancer deaths induced by medical radiation should include a statement that the estimates are highly speculative because of various random and systematic uncertainties embedded in them. These uncertainties include dosimetric uncertainties; epidemiological and methodological uncertainties; uncertainties from low statistical power and precision in epidemiology studies of radiation risk; uncertainties in modeling radiation risk data; generalization of risk estimates across different populations; and reliance of epidemiological studies on observational rather than experimental data. Such uncertainties cause predictions of radiation-induced cancers and cancer deaths to be susceptible to biases and confounding influences that are unidentifiable.[127]

- The Society for Pediatric Radiology

  To prevent misconceptions and public alarm, it is important to realize that the radiation used in CT scans has not been proven to cause cancer during a child's lifetime. The very small risk of cancer from radiation exposure is an estimate and is based on information and statistics that are debatable.[67]

USEPA has also cited studies of natural background and other environmental LDDR radiation exposures. Studies to understand health effects on people exposed to LDDR radiation are especially important, since they more closely reflect the environment following a radiological cleanup effort. They also serve to help the agency determine whether the cleanup policies are adequate to protect human health and environment while accounting for social and economic factors (ie, do they do more good than harm to society?). USEPA cited a study of leukemia risk due to natural background radiation exposure[128] and noted that this study claimed to have observed significant excess risk associated with dose rates as low as 1 mGy/yr.[99] We reviewed[128] and have identified several methodological issues.

The authors conclude,

> The possibility of confounding by some unidentified factor can never be entirely disproved, and is of particular concern when dealing, as here, with small RRs. However, we were unable to identify any mechanism whereby such confounding might plausibly account for the observed magnitude and specificity of effect in this study.

Socioeconomic status was the only confounder considered. There is evidence that paternal smoking is also associated with increased risk of childhood leukemia,[129] yet the authors did not consider this. The USEPA presented[128] as evidence of an LNT relationship for LDDR exposures despite the fact that it ignored potential confounding due to exposure to tobacco smoke. It is also worth noting that USEPA explicitly criticized other ecological LDDR studies that contradicted the LNT model[130,131] for not accounting for smoking ([132,133] but see also[134,135]). In the same presentation citing,[128] USEPA acknowledged the potential role of confounding factors, stating "variations in cancer rates due to other causes tend to swamp out those due to [ionizing radiation] exposure," but apparently did not consider the potential for smoking to confound this study by noting this limitation.

This study[128] estimated background gamma and radon doses based on the residence location of the mother, using county measurements. This information was available for cases both at birth and at time of diagnosis. It was discovered that about half of the cases had moved between birth and diagnosis. For controls, only the residence location at time of birth was available, so the number of the controls who moved after birth is unknown. The UNSCEAR warned that,

> The study should be interpreted with caution because of the large uncertainties associated with using an ecological measure of dose.[121]

The study considers only radiation exposure from natural background gamma radiation and radon. It ignores other, potentially larger sources of radiation exposure, for example, medical exposure. This is in spite of the fact that one of the coauthors of this study (MPL) was a coauthor of a separate study which claimed that exposure of British children to CT scans has increased their leukemia risk.[103] If it is true that exposure to CT scans is an important risk factor for childhood leukemia in this population, then omitting it from Kendall et al[128] cannot be justified. This is not consistent with the author's stated inability to identify other possible sources of bias or confounding.

The number of cases with a γ-ray dose rate different from their control(s) was 14 308 (52% of all cases). This means that for 48% of the cases, the γ-ray dose rate was not different from their controls. This is not a result that strongly demonstrates a causal relationship between background γ-ray dose rate and

leukemia. This observation does not satisfy Hill's criteria of strength of association.[20]

The authors used a log-linear logistic model for data analysis. But the use of such a model to analyze dose–risk relationships contains the intrinsic assumption that dose is linearly related to leukemia risk without threshold. They did not report testing other possible dose–response relationships. The authors assumed the validity of the LNT model, and citing this study in support of the LNT model is therefore a circular argument.[23]

We also note that the USEPA presentations do not discuss the numerous studies of high natural radiation background areas that have observed no excess risks of cancer, even in populations exposed to dose rates well in excess of 100 mGy/yr (eg, [136-141]), except to categorically characterize them as "specious." An objective evaluation of these studies is warranted to better understand any health effects from LDDR exposure to ionizing radiation, especially following the large-scale accidents in Chernobyl and Fukushima.

A similar LDDR situation, but involving a man-made elevated radiation background, occurred in Taipei, Taiwan, where construction materials contaminated with ^{60}Cobalt were used to build hundreds of structures throughout the city.[142] These buildings included schools and nearly 1000 apartments. More than 4000 people were chronically exposed to elevated radiation levels in this incident, some estimated as high as 1.2 Gy of cumulative dose.[143] It has also been the basis of legal action against the Taiwanese government.[144] The USEPA cited a study of this population as supporting the LNT model.

Doses to the apartment dwellers were estimated by survey instrument measurements in the affected apartments and compared to doses measured by personal dosimeters.[145] This study found agreement to within 10% to 15% for adults but only to within 60% for children. Large uncertainties were also noted in other dose reconstruction efforts,[146] which found that children received the smallest radiation doses compared to other family members. Reconstructed doses were found to agree with measured doses to within a factor of 3.[147] Radiation doses have also been measured using thermoluminescent dosimeters (TLDs),[148] and studies have been conducted to determine how to convert TLD measurements to doses received by residents using phantoms.[149]

Epidemiological studies of this population reveal evidence that low doses of radiation not only failed to increase cancer risk but actually are consistent with a protective effect.[150] A study of cancer mortality in this population observed,

> The experience of these 10,000 persons suggests that long term exposure to radiation, at a dose rate of the order of 50 mSv (5 rem) per year, greatly reduces cancer mortality . . . .[151]

A separate study of cancer incidence was also conducted.[152] The abstract of this article highlighted the few specific cancer subtypes that yielded increased standardized incidence ratios (SIRs) based on very low numbers of cases (eg, leukemia, 7 cases vs 3.3 expected). No mention was made in the abstract of the lack of increase for the other 19 types of cancer which showed no statistically increased risks, nor more importantly, the observation of statistically significantly lower SIRs for all cancers (95 observed vs 114.9 expected), all cancers except leukemia (88 observed vs 111.6 expected) and all solid cancers (82 observed vs 109.5 expected). The USEPA's presentation highlighted only the result for leukemia and breast cancer from a follow-up study that arbitrarily excluded the possibility of lower risks in the exposed population and forced a linear fit to the data on selected cancers to estimate hazard ratios at 100 mGy.[153] The hazard ratio at 100 mGy for leukemia excluding chronic lymphocytic leukemia was just barely significant at the 90% α level (confidence interval [CI], 1.01-1.31) but not at the more conventional 95% level. The USEPA presentations did not discuss that no statistically significant increases were observed in all cancers, all cancers excluding leukemia, all solid cancers, or cancers of the cervix, lung, thyroid, liver, stomach, or rectum, even when the data were forced to follow an LNT model. Further, the USEPA presentation did not mention two other studies, including a larger study of cancer incidence by the same authors, which found statistically significantly *reduced* mortality[151] and incidence[152] of all cancers combined and all solid cancers, suggesting not only a lack of cancer risk from low radiation doses but possibly also a protective effect. This creates the misleading impression that the Taiwan studies support the LNT model when in fact they directly contradict it.

Another update on this cohort was recently published,[154] which claimed,

> Dose-dependent risks were statistically significantly increased for leukaemia excluding chronic lymphocytic leukaemia (HR [hazard ratio] 100 mSv 1.18; 90% CI 1.04-1.28), breast cancers (HR100 mSv 1.11; 90% CI 1.05-1.20), and all cancers (HR 100 mSv 1.05; 90% CI 1.0-1.08, $P = 0.04$).

However, as observed by Doss,[155]

> The Hsieh et al publication reports that 249 cancer cases were observed in the cohort up to the end of 2012. To calculate the SIR, we need to know the expected number of cancer cases for the same period. In the 2006 report, Hwang et al reported that the expected number of all cancers was 114.9, and the average age of the irradiated cohort was 33.3 at the end of 2002 (The average age of the population was 17.1 at the time of irradiation and the cohort was followed-up for an average of 16.2 years).[152] Hence, for the Hsieh et al publication, the average age at the end of the study period (end of 2012) would be 43.3. The cancer incidence rates for the ages of 33.3 and 43.3, obtained by interpolation of the average of male and female cancer incidence rates during 1998–2002 from Taiwan Cancer Registry (TCR, 2008), are 86.3 and 222.4, respectively, indicating there would be an increase in cancer incidence between these two ages by a factor of $\sim$2.58. Therefore, considering the 114.9 expected cases to the end of 2002 (Hwang et al, 2006), the expected cancer cases up to the end of 2012 would be 296.4, resulting in a SIR of 249/296.4.0.84 (95% CI: 0.74–0.95). ***Thus, the reduction of cancer rate in the irradiated cohort is***

> ***significant in the updated data also***. A similar analysis of the data published in 2008[153] shows that SIR for that study would be 0.75 (95% CI: 0.61–0.88), based on 117 observed and 156.8 expected cancers to the end of 2005, again indicating reduction of all cancers in the irradiated cohort. ***Hsieh et al have failed to discuss the significant reduction of overall cancers in the irradiated cohort***. (emphasis added)

Is it Appropriate to Regulate Ionizing Radiation in the Same Manner as Toxic Chemicals?

In 1992, the USEPA SAB provided guidance on ways to harmonize risk assessment and risk-reduction strategies for radiation and chemicals.[156] They noted that the regulations for radiation and chemical risks developed under different paradigms and stated:

> USEPA's priorities should be directed towards reducing the greatest risks first, especially when that can be accomplished economically. The corollary to that principle is that similar risks should be treated similarly, which calls for harmonization, in so far as is possible, of risk reduction strategies between chemical and radiation. *Harmonization does not necessarily imply identical treatment, but it does imply that any differences in treatment are clearly explained and justified*. (emphasis added)

The options noted in the SAB Commentary were:

1. bring risk-reduction strategies for excess radiation exposures consistently in line with the chemical paradigm, a direction that it noted that some parts of the agency were already headed;
2. bring chemical risk-reduction strategies more in line with the radiation paradigm; or
3. achieve harmony between the 2 systems by modifying both in appropriate ways, explaining residual differences, and placing more emphasis on what can reasonably be achieved. In this case, background risk could be incorporated, and the balancing of benefits and costs of risk-reduction measures could be strengthened while maintaining much of the Agency's current approach to chemicals.

The radiation paradigm approach to control radiation exposures is based on principles developed over many decades by the ICRP and the NCRP.[75] These principles are:

1. JUSTIFICATION: the need to justify any radiation exposure on the basis that the benefits to society exceed the overall societal cost;
2. ALARA (Optimization): maintain any exposures as low as reasonably achievable, economic and social factors being taken into account; and
3. LIMITATION: radiation exposures are kept to levels of *acceptable risk*.

As described by the ICRP,

> For any situation where intervention is considered, some protective actions might be justified while others are not justified. Of those protective actions which are justified, it is necessary to establish the level at which the best protection will be provided. In other words the radiation detriment averted by each protective action should be balanced against the cost and other detriments of the action in such a way that the net benefit achieved by the protective action is maximized (*i.e.* optimization of protection).[157]

The principles of ALARA (Optimization) and LIMITATION can be viewed as a "top-down" approach to limit radiation exposure and health risk (Figure 2). Therefore, radiation exposures are considered acceptable if they are less than a specific limit and they are as low as reasonably achievable. Compliance with a dose limit alone does not define acceptable exposures or risk.

The chemical paradigm approach can be viewed as a "bottom-up" approach. The historical use of this paradigm by the USEPA is based on the Delaney Clause of the Federal Food, Drug and Cosmetic Act Food Additives Amendment of 1958. This clause set a standard of *zero* risk to the public from carcinogenic food additives (eg, pesticides) that concentrate in processed foods. This was interpreted in terms of a "negligible" but nonzero lifetime cancer risk of 10^{-8}, which was later increased to 10^{-6} due to pesticide measurement difficulties at levels corresponding to the lower risk. This lifetime cancer risk criterion and the concept of risk goals were later incorporated into various USEPA regulations (eg, CERCLA, Safe Drinking Water Act, Clean Air Act, and Resource Conservation and Recovery Act). This paradigm has two basic elements:

1. a goal for acceptable risk and
2. allowance for an increase (relaxation) in risks above the goal, based primarily on considerations of technical feasibility and cost.

The USEPA made the decision to regulate radiation the same way it regulates toxic chemicals for consistency purposes,[158] despite advice from the SAB describing problems with such an approach[159]:

> To many radiation scientists, reducing excess exposures much below 100 mrem/yr seems unnecessary and in any case exceedingly difficult to monitor for compliance because it is within the natural variability of background.
>
> The application of standard chemical risk-reduction criteria to radionuclides in these situations leads to limitations on excess radiation dose that are small in comparison to natural background radiation.
>
> "In calculating excess risk from human sources of a chemical, background levels, if any, are therefore frequently seen as irrelevant . . . ." This is in marked contrast to radiation, which is universally distributed in the natural environment.

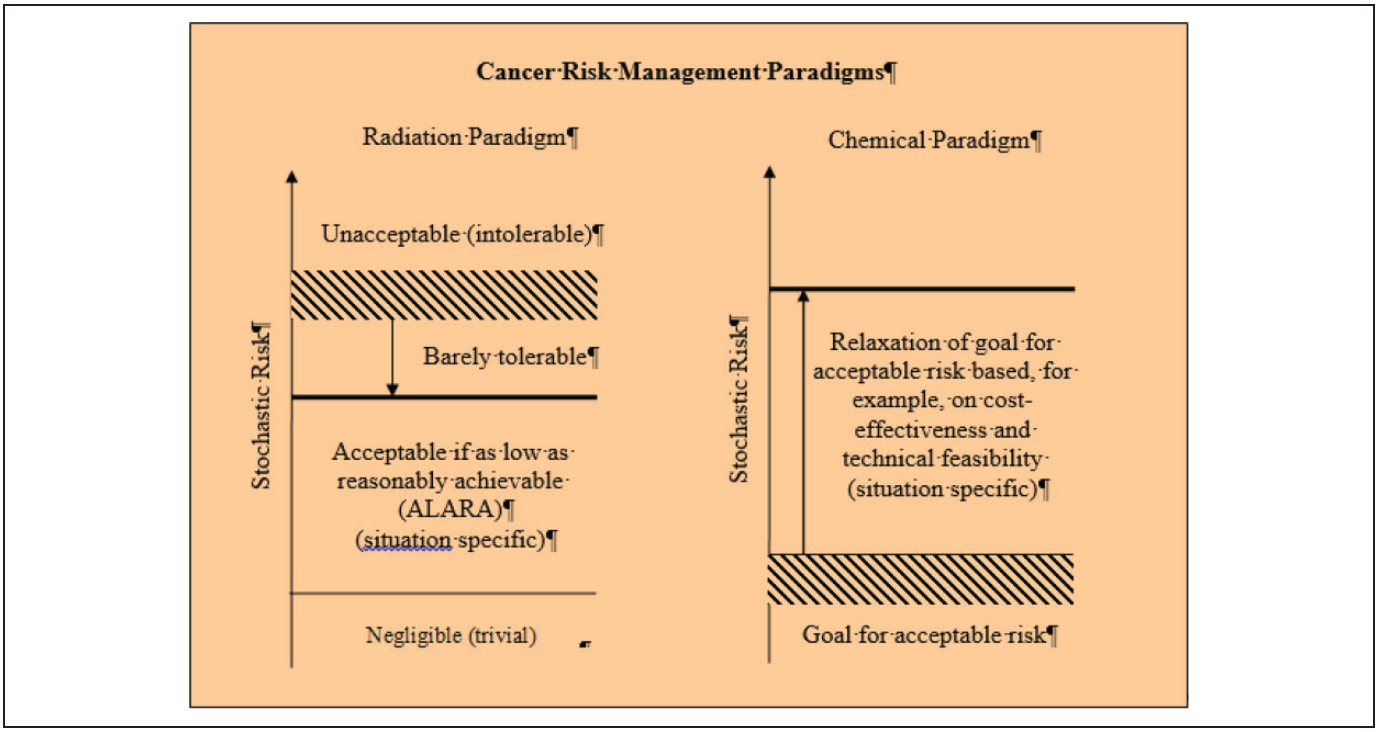

Figure 2. Cancer risk management paradigms. Reprinted with permission from the National Council on Radiation Protection and Measurements, http://NCRPonline.org.

The USEPA treats inorganic metals differently than other chemicals. In the assessment of human risks from exposures to inorganic metals,[160] USEPA takes into account metals that are naturally occurring and vary in concentrations across geographic regions. According to USEPA, the implications of these properties include:

> Humans, other animals, and plants have evolved in the presence of metals and are adapted to various levels of metals. Many animals and plants exhibit geographic distributions that reflect variable requirements for and/or tolerance to certain metals. These regional differences in requirements and tolerances should be kept in mind when conducting toxicity tests, evaluating risks, and extrapolating across regions that differ naturally in metals levels.

The USEPA also acknowledges that some metals are essential for maintaining proper health of humans, animals, plants, and microorganisms. As a result, USEPA considers the following implications for risk assessment[160]:

> Adverse nutritional effects can occur if essential metals are not available in sufficient amounts. Nutritional deficits can be inherently adverse and can increase the vulnerability of humans and other organisms to other stressors, including those associated with other metals.
>
> Excess amounts of essential metals can result in adverse effects if they overwhelm an organism's homeostatic mechanisms. Such homeostatic controls do not apply at the point of contact between the organism and the environmental exposure. Essentiality thus should be viewed as part of the overall dose-response relationship for those metals shown to be essential, and the shape of this relationship can vary among organisms. For a given population, 'reference doses' designed to protect from toxicity of excess should not be set below doses identified as essential. Essential doses are typically life-stage and gender specific.

These properties are analogous to those ascribed to radiation by the threshold and hormesis response models. An exception has been made to treat risk assessment for inorganic metals differently because of their essential characteristics or natural existence in background. Radiation has not been afforded the same consideration despite the similarities with inorganic metals. Instead, USEPA has stated,

> . . . as the purpose of a risk assessment is to identify risk (harm, adverse effect, etc.), effects that appear to be adaptive, non-adverse, or beneficial *may not be mentioned*.[161] (emphasis added)

and further,

> As a general principle, our practice is not to base risk assessments on adaptive, non-adverse, or beneficial events.[161]

Applying this guidance to radiation risk assessment excludes any scientific evidence on potential benefits from radiation exposures simply by policy mandate. That introduces bias by allowing only information claiming support for the LNT model

while prohibiting evidence that contradicts it. Excluding evidence of adaption or benefits, and only considering evidence of harm, is contrary to radiation protection philosophy as described by the ICRP.[162] National and international expert advisory bodies acknowledge adaptive and hormetic effects, and their consideration has even been formally included in new European standards for protection of the environment against radiation.[163]

Regulating radiation the same way as toxic chemicals also does not take into account that risks from radiation exposure have been established based largely on observations in humans exposed to well-known individual doses, whereas chemical risks are more often based on projections from experiments on animals or human epidemiology that suffer from poorly characterized individual exposures. Since background radiation is an underlying factor that isn't present for most toxic chemicals, the USEPA SAB acknowledged the existence of threshold models for radiation carcinogenesis (eg, the radium dial painters) or at least "practical thresholds" (eg, the idea that cancer latency was inversely related to dose such that manifestation of risks at low doses could be delayed so long that no cancers would occur during a normal lifetime).[156]

Radiation protection philosophy is distinct from toxic chemical protection philosophy:

> The precautionary principle is an alternative risk management strategy that gives disproportionate weighting to technological risks. It is often summarized by the phrase 'better safe than sorry' and requires forgoing, postponing or otherwise limiting a product or activity until uncertainty about potential risks has been resolved in favor of safety. ALARA, on the other hand, treats risks and benefits on a level playing field. Accordingly there is no prescribed dose goal. The end result of an ALARA practice is a residual dose and risk that is considered acceptable.[164]

The distinguishing hallmark of the ALARA philosophy is that interventions and radiation protection policies must be low, reasonable, and achievable. The USEPA application of the LNT model for determining risk and developing cleanup levels often result in very low numbers that are nearly three orders of magnitude below, where adverse effects are reliably observed and significantly lower than those recommended by national and international expert advisory bodies. For example, the USEPA suggests that radiation exposures above 3×10^{-4} risk (about 0.12 mSv/yr based on the LNT) is not protective of human health or the environment.[165]

Soil radiological cleanup criteria required by USEPA's preliminary remediation goals (PRGs), for example, as related to legacy uranium mining sites, are frequently within the statistical uncertainty of background and, in fact in some cases, less than natural background values. This often results in extensive remedial action costs with no demonstrable health benefits. In fact, cleanup standards as low as USEPA's PRGs often cannot be satisfied with current analytical capabilities. This is an example of where the toxic chemical approach is not appropriate for naturally occurring radionuclides, since the background contains naturally occurring radioactive material, in some cases at levels that exceed the PRG values. Additionally, there are large variations in natural background depending on altitude and geographic location.[166] This is in stark contrast to the background of most chemicals of concern.[156] As mentioned earlier, even BEIR VII acknowledges that epidemiological data below 100 mSv (0.1 Sv) are not sufficient by themselves for risk estimation, yet the USEPA maintains policies that require cleanup to levels where no net benefit to human health or the environment can be detected.

The USEPA SAB recognized in 1992[156] that the USEPA Superfund policy documents, like the risk assessment guidance for Superfund,[167] were being developed to be more consistent with the chemical risk paradigm. In contrast, it also noted that the USEPA radon policy was applying a rule of practicality based on the difficulty of reducing radon levels below 150 Bq/m (4 picocuries/L) within a reasonable budget. The associated risk for its radon policy translates to a lifetime risk of over 1 in 100 for an average person[168] based on the LNT model. More recently, USEPA's approach to radon regulation has been challenged.[169]

Should the Current USEPA Regulatory Radiation Policies Be Reconsidered and Harmonized With the Radiation Protection Philosophy Given the Lessons Learned From Fukushima?

The NCRP issued reports providing guidance on responding to a radiological or nuclear terrorism incident[170,171] and decision-making for late-phase recovery from nuclear and radiological incidents.[172] These recommendations from the NCRP endorse the strategy laid out by the ICRP[173] and apply them to the situation in the United States. This new strategy presents a:

> marked contrast to the current clean-up approach carried out under statutory regulatory provisions that focuses on radiological risk, precautionary decision making, and clean-up goals close to background.[170]

The ICRP suggests that the reference level should be selected in the lower part of the 1 to 20 mSv/yr range (100-2000 mrem/yr[173]). This is much more realistic and achievable than the LNT 10^{-6} risk-based PRGs developed by USEPA, which are approximately 2 to 3 orders of magnitude lower than other guidance provided by NCRP and ICRP.

Although the simplicity of the LNT model used for risk assessment has traditionally been thought to be reasonably conservative, its application has led many to believe that any amount of radiation brings unwarranted risk. This contributes to society's response to make personal decisions to avoid any radiation exposures at all costs, thus potentially resulting in

more societal harm than good. It also drives down cleanup levels, resulting in extraordinary cleanup costs. Furthermore, USEPA has provided guidance stating "approaches that do not follow the remedial program's policies and guidance should not be used at CERCLA remedial sites."[158] It specifically targets any guidance developed by other federal, state, or tribal agencies or by international or national organizations (eg, ICRP, NCRP, and other scientific or professional organizations) and leaves only USEPA guidance available for consultation.

A recent example of where LNT-based guidance may have caused more harm than good is the evacuation in Fukushima, Japan.[174] The Fukushima accident involved no deaths directly related to radiation exposure[175]; however, the evacuation itself caused increased mortality primarily among the elderly individuals.[176-178] Well over a thousand people died from causes related to the evacuation,[179] and the continued exclusion of residents from their homes for extended periods of time. This occurred in spite of the fact that "no significant contamination was found in the patients evacuated from the 20 km zone despite the fact that 48 h had passed between the first explosion and their evacuation."[180] During the Fukushima incident, the public exhibited distrust of radiation experts and confusion regarding what risks radiation from the accident actually presented.[181] The population that evacuated from the area around the Fukushima plant is now at increased risk for mental health problems and other social and psychological problems because of their continued exclusion from their homes, and they are subject to social stigma.[181,182]

The application of the LNT to estimate cancer risks associated with residual contamination, without appropriately considering the uncertainties involved (ie, LNT predictions represent an upper bound estimate of risks, and real risks might in fact be 0), has contributed to continued exclusion of the evacuated Fukushima population from their homes. The same situation occurred at Chernobyl.[183] In addition, recent research has indicated that even when hypothetical radiation risks from residual radioactive contamination are calculated via the LNT model, mass evacuations and relocations like those following Chernobyl and Fukushima have been unjustifiably extensive[184,185] and are almost never part of the optimal response strategy.[174,186,187] Therefore, it is reasonable to question the perceived protectiveness of the LNT model for setting protective standards in LDDR radiation environments.[72] The long-term response to the Fukushima accident will undoubtedly involve, and in fact emphasize, providing accurate information about radiation risks to returning residents and dealing with their fears.[188,189] These fears are exacerbated by strident statements that "there is no safe dose" and "doses outside the USEPA risk range are not protective" and by inaccurate and incomplete information about the uncertainties involved in estimating risks from very low residual radiation doses.[190]

While some of the remedial strategies in response to the Fukushima accident have been retrospectively analyzed and determined to be justified based on an LNT calculation of risk from residual contamination,[191] others response measures have been found to be unjustified.[192] Unrealistic cleanup standards, which fail to properly account for the real possibility that risks from such low doses, may very well be zero, exacerbate public fears, fail to optimize response strategies by ignoring the economic and public health consequences of these actions,[193] and can distort the allocation of resources in the recovery effort. The mission of the USEPA is to protect human health and the environment. The mission of the US Public Health Service is to protect, promote, and advance the health and safety of our nation. Both the USEPA and the USPHS develop policies to accomplish these missions. Although it is acknowledged that the determination of acceptable risk values is a matter of judgment and risk management policy,[194] the USEPA Scientific Integrity Policy explicitly states that science forms the backbone of its decision-making.[195] The science behind low-dose risk estimation and determining cleanup levels is showing that the LNT has the real potential to cause more economic, environmental, and public health harm than good to society.

A comprehensive review of the application of ICRP guidelines and the problems encountered at Fukushima has been documented[196] and offers many lessons. Among the highlights are the following:

> It has been noted that the uncertainties surrounding the crisis itself, in addition to the absence of demonstrated risk at the tiny exposures to the population and the uncertain validity of the linear extrapolation of risk down to such tiny doses, raise serious questions about whether these calculations could provide even an order-of-magnitude guess as to possible health consequences. Further, given the wide range of uncertainties in the risk models used, it is likely that zero effects should be included as a lower bound to the estimates, or even as a central estimate of the likely future effects.
>
> These hypothetical computations of effects are based on assumptions that cannot be validated because the estimated doses are substantially below the level where epidemiology has the ability to detect increases above the natural occurrence. The large number of deaths reported following these theoretical predictions, especially when not contrasted with the normal high occurrence of death, is alarmist and unfounded and has caused severe anxiety and emotional distress in the Japanese population.
>
> It should be recognized, however, that '*balancing' good and harm is not confined to issues associated with radiation exposure. Other non-radiation-related benefits and detriments arising from the protective action must also be considered,* thus going far beyond the scope of radiological protection. (emphasis added)

Fukushima and Chernobyl offer very rare opportunities to learn from the application of radiation protection guidance and strategies in challenging, real-world situations. A frank assessment of the successes and shortcomings of these strategies and how they may impact the agency's cleanup policies is necessary.

The USEPA has taken the position that any residual contamination concentration exceeding the upper risk range of 3×10^{-4} (a dose of about 0.12 mS/yr [12 mrem/yr]) is "not

protective."[165] Is this a valid interpretation, given the very different advice given by the ICRP? Gonzalez[196] state:

> Thus, the public has doubts about what type of exposure the inhabitants of the rehabilitated area will be subject to when the rehabilitation starts. If these people are regarded as members of the public and if the exposure situation is regarded as a planned one, the dose limit of 1 mSv $year^{-1}$ and the corresponding dose constraint could in principle be considered as applicable, therefore *requiring annual doses to the residents to be kept below a few tenths of a millisievert, a restriction that might be considered unrealistic and furthermore rather strange and unreasonable.*[196] (emphasis added)
>
> There was a particular misunderstanding about the appropriate use and application of the dose value of 1 mSv $year^{-1}$. The public tended to regard a dose above this value as dangerous, which created challenges in coping with the aftermath of the accident. The fact that there is little convincing evidence for human health effects below 100 mSv $year^{-1}$ (or 100 times the dose limit) appeared to hold little sway over the level of concern.

The USEPA's interpretation is clearly at odds with the views of the ICRP, which stated,

> The Commission's recommended limits are set at a level which is thought to be associated with a low degree of risk; thus, *unless a limit were to be exceeded by a considerable amount, the risk would still be sufficiently low as not to warrant such countermeasures as would themselves involve significant risks or undue cost*. It is therefore clear that it is not obligatory to take remedial action if a dose-equivalent limit has been or might be exceeded.[197] (emphasis added)

In answer to the question, "Is any Amount of Radiation Safe?," USEPA has explained,

> In setting limits, USEPA makes the conservative (cautious) assumption that any increase in radiation exposure is accompanied by an increased risk of stochastic effects.[58]

Similarly, USEPA has explained,

> LNT also has the great advantage of simplicity, risks from multiple exposures being proportional to the total dose. Given these features of protectiveness and convenience, there is very wide support for LNT in the context of radiation protection, even among scientists and regulators who harbor serious doubts about its scientific validity.[5]

Note that these explanations are based on the assumption that LNT is "conservative" and "cautious." In light of the Fukushima experience, these assumptions are no longer tenable. Others have argued that radiation protection guidelines are confusing and overly stringent, based on the application of LNT at doses far below where risks can actually be observed, and that this had directly observable negative public health consequences.[9,72]

Discussion

In the event of a large-scale domestic radiological dispersal device (RDD) attack, nuclear power plant (NPP) release, or an improvised nuclear detonation (IND), the long-term cleanup challenges will likely have a larger impact on the surrounding communities, cities, and regions, where factors other than potential radiation exposure may become the driving force behind the final cleanup levels. For example, psychosocial, economic, and speed-of-recovery issues all affect the long-term viability and survivability of the affected area. Risks associated with moving an entire population on a temporary or permanent basis may be higher than allowing some low-level exposures from residual contamination. Nondestructive cleanup technologies may prove to be too costly or applicable to only small portions of the recovery effort. Overall costs could become so expensive as to reduce the ability to protect human health and the environment if there are limited resources. Given the potential scope and urgency of the situation following an RDD/NPP/IND scenario, the preference to work toward an acceptable cleanup level (radiation risk paradigm) rather than having to raise a preliminary cleanup goal (chemical risk paradigm) has many political, economic, and societal benefits.

Both radiological and chemical risk paradigms warrant equal consideration when making cleanup decisions. The radiation risk paradigm was included in the Department of Homeland Security guidance with USEPA and other federal agencies' concurrence. The chemical risk paradigm is routinely used at USEPA superfund sites. Both employ risk-based methods and can lead to similar cleanup levels. However, risk is a metric that cannot be measured; only radiation exposure or radioactive surface contamination can be directly measured. Using the USEPA PRG calculators to meet the CERCLA, risk range suggests that the agency knows the risk with a much greater certainty than is scientifically possible. These are based on the LNT model and are inconsistent with the guidance from UNSCEAR, HPS, World Health Organization, and many others. They are tools that foment fear and uncertainty in the affected communities. Instead, a dose-based cleanup approach is more scientific and practical.

There is precedent for the USEPA to quickly change policy based on SAB recommendations. In 1992, the USEPA SAB changed its earlier 1988 recommendation from averaging the radon risk estimates from BEIR IV and ICRP 50 to just using those published in BEIR IV.[198] Recent findings from the ongoing Life Span Study and other peer-reviewed articles as late as 1990 were used to justify this change. This change to the USEPA's radon risk assessment policies is consistent with the goal and objectives of the existing USEPA Scientific Integrity Policy, which requires science to be the backbone of agency decision making.[195] Perhaps, findings or recommendations from a new USEPA SAB review will serve to justify changes

to the agency's existing policies on the use of the LNT model in LDDR radiation environments.

Conclusions

The USEPA is the lead federal agency responsible for protecting human health and the environment from hazardous agents. It carries out this mandate by applying scientific information to promulgate regulations and policies that other federal agencies (eg, NRC and DOE) and states incorporate into their regulations or policies where appropriate or applicable. Thus, the USEPA has a tremendous responsibility to ensure its radiation regulations, policies, and guidance are scientifically sound while providing adequate protection without placing an unnecessary burden on the affected population or organizations subject to them. An objective and unbiased reliance on scientific information to inform decision-making is an integral part of the agency's scientific integrity policy. It sets the foundation for objective discussions among all the affected stakeholders (eg, public, industry, professional organizations, international communities) for determining (1) what are acceptable radiation regulations and policies associated with determining cleanup levels following a large-scale radiological or nuclear incident and (2) what risk assessment model should be used to best represent the risks from LDDR radiation environments when a residual low-level contaminated environment becomes reality.

The scientific understanding of the effects of radiation exposures has evolved since its discovery in the late 19th century. The scientific information supporting the use of the LNT model for LDDR radiation environments developed over that past 70 years but is mainly extrapolated from HDDR environments. The application of the LNT model to determine health risks has created a culture where a few clicks on a radiation dose rate meter equate to cancer in the minds of the public. Society has become so fearful of radiation that unnecessary steps are taken, and other risks are accepted, to avoid even trivial radiation exposures at all costs. This includes potentially life-saving medical examinations, which is recognized as a problem by the many scientific and professional organizations specializing in radiation.

Since the Three Mile Island Nuclear Power Plant accident in 1979, the world has experienced several large-scale nuclear or radiological accidents (eg, Chernobyl, 1986; Goiania, 1987; Fukushima, 2011), affecting millions of people and contaminating millions of hectares of land. The 2011 Fukushima NPP accident is the most recent radiological accident. The accident itself caused no radiation-related deaths[175]; however, the evacuation in response to the accident, combined with the extended exclusion of area residents from their homes, has increased mortality from various stress-related causes. The elderly individuals are especially vulnerable to these effects,[176-178] and over 1600 people died as a result[179] of the response to the Fukushima accident. A retrospective evaluation has concluded that the risk from the evacuation outweighed any hypothetical risk of radiation exposure calculated using the LNT model,[184,185] particularly among the elderly individuals,[199] the evacuation did not protect human health, and was therefore unethical.[200]

Scientists and society continue to learn from these events by questioning how we can strengthen our resilience, reduce the time it takes to resume normal lifestyles, maintain economic viability, and minimize adverse psychological effects. The scientific literature is showing, and scientific organizations acknowledge, that adverse health effects from LDDR radiation exposures are not detectable and that there may be a threshold or even a beneficial effect. These findings contradict the use of LNT model-based predictions.

It is time for the USEPA to reconsider the use of the LNT model in LDDR radiation environments in the regulatory process, especially in the tools it has developed to determine cleanup levels. Change does not occur quickly or easily within government frameworks. It took decades of institutional inertia to arrive at the current regulatory framework. The USEPA SAB recommended "change in the agency culture, change in how the agency works, and increased support for scientists and managers in programs and regional offices responsible for science integration"[201] to occur and thereby improve its regulations and policies. Despite these recommendations by the EPA SAB, there's been no change in the agency's posture or policy associated with using the LNT model for risk assessment and determining cleanup levels in LDDR environments, nor a desire to have it reevaluated by the SAB for more than 20 years.

Objectively evaluating and incorporating the latest scientific evidence on LDDR dose–response relationships for application to the regulatory and policy-making process for risk assessment purposes will (1) ensure science remains the foundation for its decision making, (2) reduce the unnecessary burden of costly cleanups, (3) provide a much needed platform to educate the public on the risks or benefits from LDDR radiation exposures, and (4) harmonize the agency's policies with those recognized by the rest of the radiation scientific community. A continued resistance to conducting a comprehensive review of the latest science regarding LNT-based policies will only diminish the agency's credibility and influence to protect human health and the environment.

Authors' Note

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COMMENTARY Open Access

Low-dose radiation from A-bombs elongated lifespan and reduced cancer mortality relative to un-irradiated individuals

Shizuyo Sutou

Abstract

The US National Academy of Sciences (NAS) presented the linear no-threshold hypothesis (LNT) in 1956, which indicates that the lowest doses of ionizing radiation are hazardous in proportion to the dose. This spurious hypothesis was not based on solid data. NAS put forward the BEIR VII report in 2006 as evidence supporting LNT. The study described in the report used data of the Life Span Study (LSS) of A-bomb survivors. Estimation of exposure doses was based on initial radiation (5%) and neglected residual radiation (10%), leading to underestimation of the doses. Residual radiation mainly consisted of fallout that poured down onto the ground along with black rain. The black-rain-affected areas were wide. Not only A-bomb survivors but also not-in-the-city control subjects (NIC) must have been exposed to residual radiation to a greater or lesser degree. Use of NIC as negative controls constitutes a major failure in analyses of LSS. Another failure of LSS is its neglect of radiation adaptive responses which include low-dose stimulation of DNA damage repair, removal of aberrant cells via stimulated apoptosis, and elimination of cancer cells via stimulated anticancer immunity. LSS never incorporates consideration of this possibility. When LSS data of longevity are examined, a clear J-shaped dose-response, a hallmark of radiation hormesis, is apparent. Both A-bomb survivors and NIC showed longer than average lifespans. Average solid cancer death ratios of both A-bomb survivors and NIC were lower than the average for Japanese people, which is consistent with the occurrence of radiation adaptive responses (the bases for radiation hormesis), essentially invalidating the LNT model. Nevertheless, LNT has served as the basis of radiation regulation policy. If it were not for LNT, tremendous human, social, and economic losses would not have occurred in the aftermath of the Fukushima Daiichi nuclear plant accident. For many reasons, LNT must be revised or abolished, with changes based not on policy but on science.

Keywords: A-bomb survivors, Lifespan, Life Span Study, Linear no-threshold, LNT, Longevity, Residual radiation, Threshold

Background

Japan is the only country that has sustained a nuclear attack. The weapons dropped in 1945 killed approximately 200,000 people instantaneously. People around the world have been taught for decades since that ionizing radiation is limitlessly hazardous, This supposition is based on a linear no-threshold model (LNT): even the lowest doses of ionizing radiation are hazardous in proportion to their doses, Therefore, it is quite natural that most people think that ionizing radiation from the A-bombs killed people, shortened lifespan, and increased cancer mortality. The Fukushima Daiichi nuclear power plant accident presented an opportunity to study the effects of ionizing radiation on health, after which the author published associated books [1, 2] and papers [3, 4]. Through their composition, it became increasingly clear that LNT has a seriously flawed history [5]. The energy of A-bombs comprised 35% thermal radiation (heat and light), 50% blast energy (pressure shock waves), and 15%

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nuclear radiation [6]. In fact, instantaneous deaths were mostly ascribable to thermal and blast energy (85%), especially in the central area of the blast. People tend to forget that victims of heat and blast were affected in a moment or short period, whereas cancer induction has remained a menace even to the present day. For survivors of today, fear of A-bombs mostly overlaps with fear of cancer. It is less well known that ionizing radiation is not always hazardous. Low-dose radiation sometimes stimulates our defense mechanisms and beneficial (radiation hormesis) [7–10].

Taking these facts into consideration, the effects on lifespan and cancer incidence of A-bomb survivors were reexamined for the present analyses. Letting the data speak, one would hear that low-dose radiation from A-bombs has extended survivor lifespan and reduced cancer mortality on average for A-bomb survivors and not-in-the-city control subjects (NIC). The key to resolving the apparent discrepancy between the received notions and actual data is radiation hormesis and the radiation doses of a hormesis range to which a large fraction of A-bomb survivors and NIC were exposed. Of course, A-bomb survivors who received high doses exhibited shortened lifespan and increased cancer mortality, but they accounted for a minor fraction of all local residents. Therefore, results show that the "average lifespan" was longer and that "average cancer mortality" was reduced overall.

Radiation units such as rem, Sv, and Gy are used here as reference articles use, unless otherwise specified.

Longer lifespan of some people who were heavily irradiated by ionizing radiation

Reportedly the unhappiest man in the world, Mr. Tsutomu Yamaguchi, was A-bombed at Hiroshima. Later he relocated to Nagasaki, where he survived the second A-bomb attack [11]. He survived the two A-bomb attacks; he might be the happiest man in a sense that more than 70 people were evacuated from Hiroshima to Nagasaki: all except him were killed. More surprising is that the two A-bombs did not shorten his life: he died of stomach cancer at 93.

The Nikkei Shimbun reported on April 5, 2018 that Chairman Sunao Tsuboi of the Japan Confederation of A-Bomb and H-Bomb Sufferers Organizations was selected as an honorary citizen of Hiroshima City. When he was 20, the A-bomb attacks occurred when he was 1.2 km from the epicenter. He is 93 in 2018. He talked to then US President Obama to encourage efforts to abolish nuclear weapons. The occasion on May 27, 2017 was the first visit ever to Hiroshima by a serving president.

When he was 8, Shigeaki Mori was blown into a riverbed from a bridge and injured 2.5 km from the epicenter. He became a historian and discovered that American victims of the A-bomb were present in Hiroshima. His finding was reflected in President Obama's speech, "Why do we come to this place, to Hiroshima? We come to ponder a terrible force unleashed in the not so distant past. We come to mourn the dead, including over 100,000 Japanese men, women and children, thousands of Koreans and a dozen Americans held prisoner." After the speech, a tearful Mori was embraced by Obama. Born in 1937, he has lived longer than the average for Japanese men.

Dr. Don Wiles, Emeritus Professor of Chemistry at Carlton University, Canada, once engaged in extraction of radium from uranium ore for 16 months from 1947. Before the use of cobalt, radium ($20,000/g) encapsulated in a glass tube was used to treat cancer by embedding it into the malignant tissues. The crystallization process used by Marie Curie 50 years before included procedures that were apparently very lax and coarse compared to the present standard: encapsulation was performed with bare hands. Workers ignored the rule to wear rubber gloves because they were slippery. Radiation badges even under the lead shield became black at the daily check. Because radium is similar to calcium in terms of its chemical characteristics, radium was apparently accumulated in Dr. Wiles' bones. Born in 1925, he exhaled about 25 times the legal maximum of radon, a product of radium, at the age of 88. One might assume that he was seriously injured. He stated "About 65 years later, I am still healthy." [12]. These are some examples of increased longevity despite radiation exposure. Are they exceptional?

A-bomb survivors lifespans are unusually long

Figure 1 presents changes of the number of certificate holders who have been covered by the Law Concerning Relief to Atomic Bomb Survivors. The holders are regarded as A-bomb survivors. Until 1982, holders were more than expected because additional people had been admitted as holders; the holders' superiority in number does not necessarily mean that holders had a long lifespan. After 1982, the expected number became greater than the actual holders because few people were admitted as new and holders were getting steadily older year by year. The mortality ratio of the Japanese that was used to calculate the expected numbers was the average of infants, young people, adults, and elderly people, producing a result that is much less than that of the aged holders. Therefore, the holders' exposure and experience do not necessarily mean that their lifespan is short.

The average lifespan of certificate holders was 80.13 for 2014. The ratio of men to women is not available. The lifespan of Japanese men was 80.21 for 2013 and that for women was 86.61; the average was 83.49.

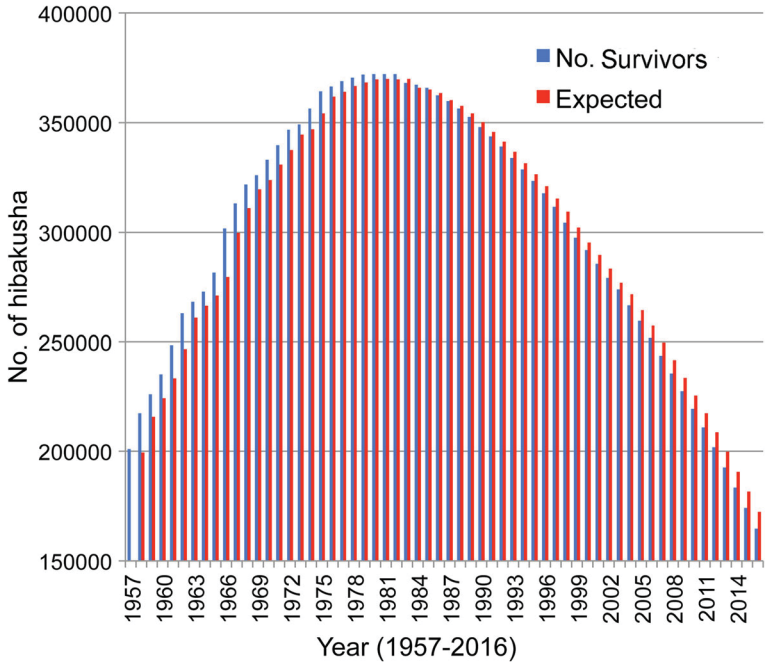

Fig. 1 Changes of people who have an A-Bomb Survivor's Certificates (Ministry of Health, Labour and Welfare [68] (blue). For example, a total of 183,519 certificate holders in 2014 comprised four classes: 1st class survivors, or direct victims (113,685); 2nd class survivors, or in-city victims who were within areas inside 2 km from the epicenter (42,529); 3rd class survivors, or rescue victims who engaged in rescue activities or physical treatments outside the 2 km areas and who were exposed to residual radiation (20,013); and 4th class survivors, or fetuses of people in one of the above three categories (7292). Their peak number was 372,264 in 1980. Expected numbers (red) were calculated as follows: holders in 1957 were 200,984; the death ratio of the Japanese in 1957 [69] was 0.008275 and 1663 (200,984 × 0.008275) were expected to die and 199,321 (200,984–1663) was the expected number in 1958 (the same hereinafter). Certificate holders are supported financially with six allowances and funeral fees. Some other benefits accrue: they can undergo free health examinations twice a year; and almost all sicknesses are treated at no charge. Patients with illness caused by a nuclear weapon were eligible to receive an allowance of 138,380 yen/m. The health control allowance is 34,030 yen/m. The funeral allowance is 206,000 yen. The total budget for fiscal year 2015 was 393,391,000,000 yen

The life expectancy (for remaining life) at age 80 is 8.61 for men and 8.19 for women. Therefore, the lifespan of A-bomb survivors is expected to be over 88, far exceeding the average. This elongated average lifespan of holders might be ascribable to good medical services offered by the Japanese government. This might have contributed to some degree, but apparently some other important factor has an influence: low-dose radiation stimulates human biological defense mechanisms.

A-bomb survivors lifespan was statistically shortened

Cologne and Preston investigated the longevity of 120,321 A-bomb survivors [13]. They concluded that "Median life expectancy decreased with increasing radiation dose at a rate of about 1.3 years per Gy, but declined more rapidly at high doses. Median loss of life among cohort members with estimated doses below 1 Gy was about 2 months, but among the small number of cohort members with estimated doses of 1 Gy or more it was 2.6 years. Median loss of life among all individuals with greater-than-zero dose estimates was about 4 months." Almost all readers of the summary sentences above must believe that ionizing radiation from A-bombs was hazardous and that it shortened A-bomb survivors' longevity to a greater or lesser degree. One must nevertheless be alert. The A-bomb survivors lifespan was not necessarily shortened, as described later. When a model cannot explain established facts, not the facts but the model must be wrong. What reasons are there in the discrepancy between actual life elongation and statistical shortening of lifespan? Apparently, three major factors engender wrong conclusions: 1) invalid LNT was promulgated – one never considers life elongation and cancer mortality reduction as effects of radiation; 2) a false assumption (zero exposure-zero risk) in NIC was used by neglecting residual radiation; and 3) radiation hormesis, the idea that low-dose radiation stimulates defense systems, was neglected. These three points are briefly examined before returning to discussion of Cologne and Preston's data [13] later.

LNT is not based on solid data

Muller's tenacity to maintain LNT

The origin of LNT dates back to 1927, when Muller found that X-rays induced sex-linked recessive lethality in *Drosophila melanogaster* [14]. This "data-poor/discussion-rich" paper was quite likely to have cleverly circumvented the normal peer review process [15]. Later, he presented related data. Apparent linearity at extremely high doses was extrapolated to lower doses without experimental data. He put forward the proportionality rule, an analog of LNT [16]. Then in 1939, World War II (WWII) broke out. The United States of America (USA) began production of the A-bomb under its Manhattan Project. Radiation effects on living organisms were investigated intensively. He learned of a threshold for positive excess risk in recessive lethality tests of *D. melanogaster* [17]. The US dropped A-bombs on Hiroshima and Nagasaki in 1945. Muller became a Nobel laureate in 1946 for his radiation research. Although he knew of thresholds to damage from radiation, he declared in his Nobel Prize lecture that there was "no escape from the conclusion that there is no threshold dose" [18].

Oil industries felt uneasy about nuclear energy and took over the National Academy of Sciences

Standard Oil Co. Inc. was founded by John Rockefeller in 1870, who later established the Rockefeller Foundation (RF) in 1913. The oil industry might well have felt threatened by the discovery of atomic energy. The Republican Party had forged a close relationship with the oil industry, but the Democratic Party, led by F.D. Roosevelt (1933–1945) and H. Truman (1945–1953), governed the USA during and after WWII. When Republicans were reelected, Nelson Rockefeller was appointed as an important aide to President Eisenhower. Muller, in turn, had close ties to the RF. In 1954, the RF chose to finance a large project to evaluate ionizing radiation. RF asked the U.S. National Academy of Sciences (NAS) to organize the program, which was conducted under the auspices of NAS President Bronk of Rockefeller University, also an RF trustee. The Genetics Panel (GP) of the NAS Biological Effects of Atomic Radiation (BEAR) committee was established in 1954 and was chaired by Weaver, a mathematician and director of RF.

With no significant discussion, GP recommended LNT on June 12, 1956 [19]. The limit dose for nuclear workers of 500 mGy/y, which had been in place since 1934, was discarded. The next day, the front page of the New York Times, owned by an RF trustee, reported that radiation is dangerous. Other media followed suit. Soon, several leading biologists asked GP to provide documentation that supported LNT. GP refused to do so because they never possessed relevant data. This decision was cast, and reasonably so, as an ideologically motivated choice based on deliberate falsification and fabrication of research records [20]. Fossil fuel companies are opposed to nuclear energy even today.

Expansion of LNT from insect sperm to the human body

Lewis (a 1995 Nobel laureate) argued in 1957 that radiation-induced leukemia conformed to the LNT hypothesis [21]. This was a new deployment of LNT from germ cells (heritable effects) to somatic cells (cancer induction). Several prominent researchers criticized the Lewis' paper (Table 2 in ref. [22]). With no convincing data to support LNT reported for half a century, the Biological Effects of Ionizing Radiation committee of NAS published BEIR VII report in 2006 to support LNT [23]. This report includes several shortcomings, as discussed later. Moreover, LNT has been applied also to chemical carcinogens; the smallest amount of a carcinogen is hazardous without threshold for positive excess risk.

Radiation doses are underestimated by neglecting residual radiation or black rain

Residual radiation and the formation of black rain

The radiation doses for A-bomb survivors were estimated using radiation transport calculations based on radiation transport findings from tests conducted on the ground in the Nevada desert. The nuclear weapons dropped on Hiroshima and Nagasaki were detonated respectively at 600 m and 503 m heights. To obtain more accurate data, the ICHIBAN project was planned, for which a 510 m high tower was constructed in the Nevada desert [24]. A nuclear reactor or other radiation source was placed at the top of the tower and data were collected. The dosimetry of the ICHIBAN project was named tentative dose 1965 (T65D). Around the 1980s, results demonstrated that T65D did not correctly reflect A-bomb radiation intensity. Exposure doses were reexamined, after which the Dose System 1986 (DS86) was established. In the period around the 1990s, DS86 was revised again; Dose System 2002 (DS02) was established. DS02 was revised further as DS02R1, producing the current system used to estimate the exposure doses of A-bomb survivors [25]. Although dose systems have been revised several times, T65D is the basic one. Others are modified versions that do not deviate greatly from T65D. T65D was an outcome of a large-scale simulation model of A-bombs, but it included an important oversight, i.e., omission of residual radiation with a dose twice as large as the initial radiation on which the dose estimation was made.

The energy of a typical A-bomb comprises three components: 35% thermal radiation (heat and light), 50%

blast energy (pressure shock wave), and 15% nuclear radiation [6]. Of that latter 15%, 5% is initial radiation (released within 30 s). The remaining 10% is residual radiation, which consists of major fallout and minor induced radioactivity. Induced radioactivity is produced by the action of neutrons in making non-radioactive substances into radioactive ones, but its lifespan is very short and is mostly negligible. A large fraction of the fallout, 40–70%, is believed to settle onto the ground within a day, but this depends strongly on weather and geographical features. When T65D was established, Black rain never fell in the Nevada desert. At Hiroshima and Nagasaki, thermal radiation incinerated or scalded plants, animals (including humans), houses, and various organic substances, producing heat, carbon dioxide, and vapor and consuming oxygen. Heat killed people. A lack of oxygen contributed deaths by suffocation. Victims were therefore affected in various ways by the A-bombs. From many waterways in Hiroshima and Nagasaki, large volumes of water were evaporated. The water itself was sucked up as if by a tornado. The vapor and water went up into the sky and cooled, thereafter forming raindrops containing soot and other debris. The resultant black rain started to pour down 20–30 min after the detonation. The rainfall lasted for a few hours (Fig. 2). The heavy black rain is well known to be highly radioactive. The possibility exists that the black rain included the most fallout, two-thirds of the nuclear radiation energy, i.e., twice as much radiation as the initial radiation used to estimate the radiation doses.

Evidence that residual radiation fell to the ground with the black rain

An old Japanese article written in 1957 by G. Obo [26] was later translated into English [27]. For the article, approximately 4000 people who lived in a 7 km radius from the epicenter were interviewed personally if they entered the central area 1 km radius from the epicenter and if they had radiation acute effects such as skin burn, external injury, fever, diarrhea, sore throat, skin bleeding, or loss of hair. Students of Hiroshima University took part in this study. Fundamentally important data are presented in Fig. 3.

The left panel of Fig. 3 shows 1) positive relations between people with symptoms and distance from the epicenter, 2) outdoor people as more severely affected than indoor people as a matter of course, 3) people in the areas ≥3 km from ground zero (beyond the reach of γ-rays and neutrons from initial radiation) were affected, implying that this area was contaminated severely by residual radiation most probably carried by black rain, and 4) indoor and outdoor people who were at a distance ≥4 km and who entered the central areas were affected almost equally independent of their distance from the epicenter, strongly suggesting effects of residual radiation. The right panel of Fig. 3 shows that a large

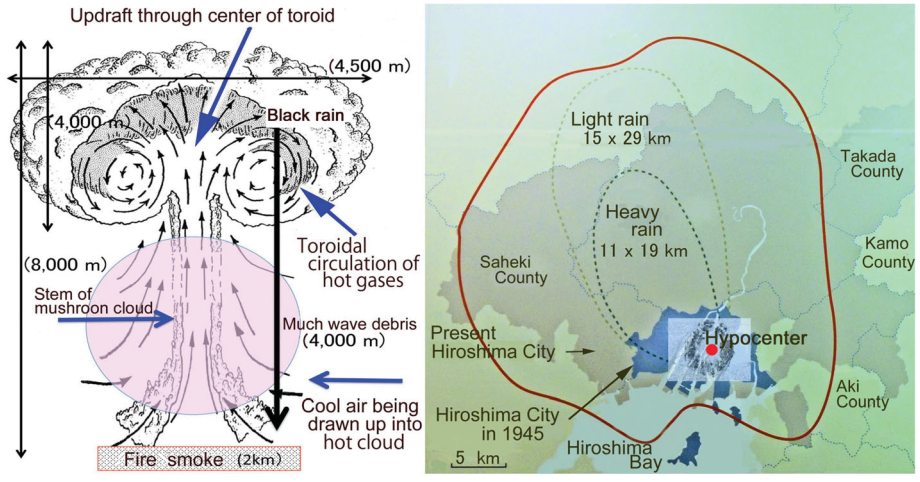

Fig. 2 Formation of black rain from the mushroom cloud (left), and black rain areas (right) [27]. Left: The A-bombs used to attack Hiroshima (16 kt TNT equivalent) and Nagasaki (21 kt TNT equivalent) were detonated respectively at 600 m and 503 m heights. A 500-m diameter fireball is formed by the detonation of a 20 kt bomb. The fireball rose like a skyrocket. During expansion of the ball, vaporized matter was condensed to a doughnut-shaped cloud with violent internal circulatory motion. Following the rising fireball, dirt and debris were sucked up from the Earth's surface. A Mach wave (the tip reaching 560 m 1.25 s after the blast) was reflected from the surface, whirling soil and debris up to form a Mach wave mass of 3800 t, providing black rain with raw materials together with the mushroom components. Trees, lumber, and other matter of 1.55×10^5 t were incinerated, forming a smoky fire 2 km in diameter, above the ground. Two references [70, 71] were used to draw this figure. Right: The probable heavy rain area reported in 1953 is shown as a thick broken line. That of light rain is shown as a thin broken line. The black rain area according to analyses of the "A-bomb Survivors' health awareness survey" in 2008 is shown as a solid red line (Hiroshima Peace Memorial Museum). A red circle off center denotes the epicenter. Black dots around the epicenter show locations of A-bomb survivors at the T65D survey [72]

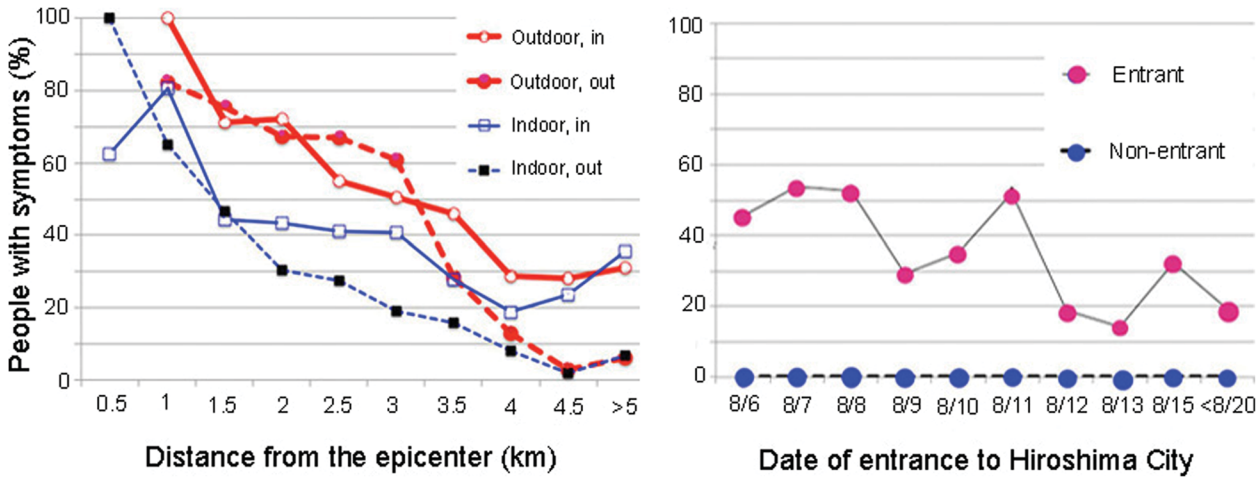

Fig. 3 Proportion of A-bomb survivors with symptoms (left) and that of non-A-bomb survivors with symptoms (right) [27]: Left, open circles (Outdoor, in), outdoor A-bomb survivors who entered the central area; filled circles (Outdoor, out), outdoor survivors who did not enter the central area; open squares (indoor, in), indoor survivors who entered the central area; and filled square (indoor, out): indoor survivors who did not enter the central area. Right, red circles (entrant), non-A-bomb survivors who entered the central area and blue circle (non-entrant), non-A-bomb survivors who did not enter the central area

fraction of non-A-bomb survivors entered the central area 2–3 weeks after detonation suffered from severe radiation sickness as if they were A-bomb survivors. This result indicates strongly that the area was heavily contaminated with residual radiation associated with black rain.

Report that black rain is negligible is refutable

The effects of black rain were studied using mortality data from 1950 to 2005 and cancer incidence data from 1958 to 2005 in Hiroshima and Nagasaki. The authors conclude that deleterious health effects from black rain exposure were not detected [28]. However, there is apparently a methodical fault. The authors asked people, "Was the person caught in Fallout Rain?" (Yes or No). According to the response, they were then divided into Yes or No groups. This grouping is almost meaningless because the important matter is not Yes or No, but if they had entered black rain affected areas within 2–3 weeks after detonation when residual materials remained active (Fig. 3). When solid cancer deaths and solid cancer incidence are extracted from the literature [28], excess relative risks (ERR) were smaller in the Yes group (caught in the rain) than in the No group (not caught in the rain) (Table 1). The data are suggestive of hormesis: slight radiation exposure is cancer-inhibitory.

The black rain affected areas were so wide that almost all A-bomb survivors and NIC must have been irradiated

Table 1 Excess relative risks for exposure to black rain for solid cancer death and solid cancer incidence (solid cancer incidence for 1950–2005 and solid cancer death for 1958–2005 were not available)

Data	Fallout rain status	No. of cases	Excess relative risk (ERR)
1962–2005			
Solid cancer death	No	3573	0.00
	Yes	1483	− 0.04
Solid cancer incidence No	5653	0.00	
	Yes	2283	−0.06
1950–2005			
Solid cancer death	No	3970	0.00
	Yes	1633	−0.02
1958–2005			
Solid cancer incidence	No	5982	0.00
	Yes	2430	−0.03

to a greater or lesser degree by residual radiation. The UNSCEAR 1958 report describes that almost all leukemia patients in zone C (1500–1999 m from ground zero) complained of severe radiation sickness in spite of an estimated dose of 50 rem (500 mSv in the International System of Units (SI)). Their doses must have been greater than 50 rem [29]. Exposure of around 2 Gy (close to 2 Sv in SI) is necessary to induce severe radiation sickness.

BEIR VII report fails to support LNT

BEIR VII report, the second problematic assertion by the National Academy of Sciences

Originally, LNT was based on Muller's experiments using repair-deficient *Drosophila* sperm [14]. He knew of the existence of thresholds for positive excess risk in *Drosophila* tests [17]. Indeed, later experiments by Japanese researchers indicate clearly that *Drosophila* irradiated with X-rays [30] or γ-rays [31] show not only thresholds but also hormesis. Hormesis has been observed in A-bomb survivors for solid cancers [32] and leukemia [33]. In spite of a large body of experimental data against LNT, NAS, the founder and advocator of LNT since 1956 [19], presented the BEIR VII report as basic LNT-supportive data (Fig. 4) [23]. The support, based on a Life Span Study (LSS) of A-bomb survivors, has been regarded as the gold standard to estimate radiation risk for human cancer. Nevertheless, this analysis presents serious flaws as explained below.

By the way, both Sv and Gy units are used according to original references in Fig. 4. Sv is a suitable unit for LNT and more generally acceptable Gy is used in this chapter.

Leukemia, a better indicator of radiation stochastic effects than solid cancer

Leukemia, a cancer of the blood cells, is a better indicator of radiation than problematic solid cancers because it is sensitive to radiation. It appears around 2 years after exposure and reaches a peak 6–8 years later, whereas solid cancers start to appear around 10 years after exposure and last for decades. Figure 4 (upper left insert, blue arrow) shows that ERR/Gy for leukemia is approximately 2, whereas that for solid cancer is approximately 0.55 (lower left, purple arrow). Therefore, leukemia is sensitive to radiation and a better indicator than solid cancers. The dose-response of leukemia is not linear but is instead linear-quadratic (upper left insert). That of solid cancer also fits better to linear-quadratic (red arrow) than linearity (orange arrow), but no statistical significance was found between the two; BEIR VII asserts linearity. This forcible logic is difficult to accept. Moreover, when taking into consideration neglected residual radiation, effects of blast/thermal wave injury on the immune system, and hormesis, dose responses might be deviated far from linearity.

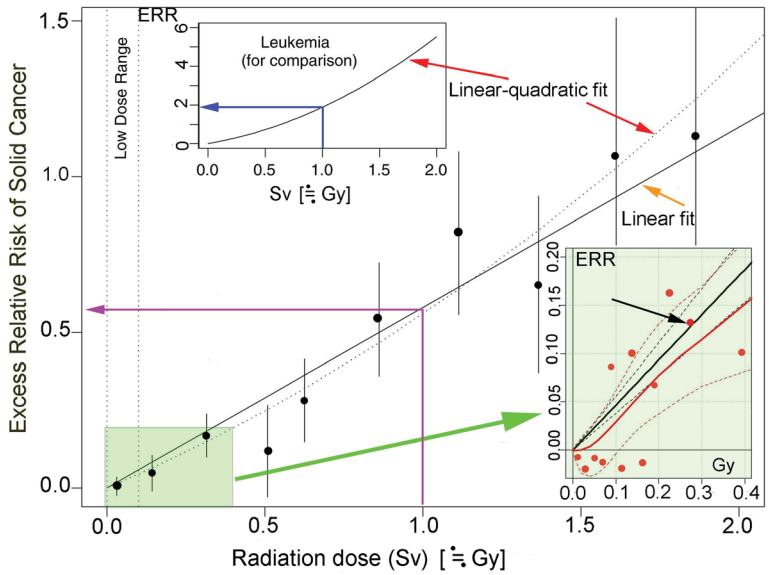

Fig. 4 Excess relative risk (ERR) of solid cancers for Japanese A-bomb survivors [23]. The plotted vertical lines represent approximate 95% confidence intervals. The thin blue, purple, red, and orange arrows respectively indicate ERR/Sv for leukemia, ERR/Sv for solid cancer, linear quadratic fit, and linear fit. The lower left area in pale green is enlarged as the lower right insert, which shows the results of two statistical analyses [35]. The black straight and dotted lines respectively show the linear fit of LNT and 95% confidence intervals. The red continuous and dotted lines respectively show the Bayesian fit and 95% credible interval. The black arrow indicates only one dot inside the 95% confidence intervals. Less than 100 mSv constitutes the low-dose range

Concealment of downturn

When radiation doses are much higher than 2 Gy, exposed people tend to die of adverse effects before reaching an age when cancer commonly occurs; ERR would show a downturn until finally reaching zero. The highest dose in Fig. 4 is 2 Gy, which conceals the downturn. Indeed, "The dose-response curve shows some downward bending in the high-dose range (2 + Gy in organ dose) for leukemia and even for all cancer except leukemia" [34]. When following the dots in Fig. 4 from low to high with downturn over 2 Gy in mind, one can easily imagine a sigmoid-like curve. This is shown by Bayesian analysis of LSS (Fig. 4, right below insert) [35]. A J-shaped curve is observed for solid cancers [36, 37] and leukemia mortality [33] in LSS. When hormesis and a downturn occur, the actual curve becomes instead an S-shaped curve [1].

Averaging of low-dose groups

Doses < 100 mGy are the most important for our risk analyses. No significant differences were found between the control subjects and A-bomb survivors at these doses. The BEIR VII report combined all data points < 100 mGy, to which more than 80% of all survivors belong, together into one point (Fig. 4). This has been explained as an old statistical trick. It was used by Lewis to insist on the validity of LNT [21]. This dishonest representation was successful in giving the impression that the dose response is linear and that no thresholds exist. The low-dose area < 400 mGy (Fig. 4, lower left in pale green) is presented in detail (lower right in pale green) [35]. The ERR dots are dispersed widely: only 1 dot (black arrow in lower right insert) out of 12 is inside the 95% confidence interval, indicating that dose responses are not linear in this area.

Inappropriate use of a false assumption (zero exposure-zero risk)

The line of LNT starts from zero according to the assumption that the exposure dose was zero and that ERR was zero in the control cohort (Fig. 4). This default model has been used to analyze LSS, but it is misleading because most A-bomb survivors and the control cohort people must have been exposed to residual radiation, as discussed later. The BEIR VII report based on that false assumption is therefore invalid. The dose-response line should not start from zero. Bayesian analysis does not assume this false assumption and allows more appropriate estimates. When the lower right insert of Fig. 4 is enlarged, crossing between the x-axis and the red line is roughly 25 mGy. An estimated zero dose might actually be 25 mGy. If these people were exposed to residual radiation, which was twice as great as the initial radiation, then A-bomb survivors and control subjects might have been exposed to additional 50 mGy: a total of 75 mGy.

LNT ignores hormesis and thresholds

Granted that A-bomb survivors and control NIC people were exposed to 25–75 mGy over the estimates, the false assumption (zero exposure-zero risk) must be abandoned. Bayesian analysis, which does not need this assumption, allows negative responses, i.e., cancer mortality is suppressed to below the background level. Figure 4 shows that six responses are indeed hormetic (red dots under the x-axis in lower right insert). Therefore, low-dose radiation can suppress cancer deaths. At the same time, hormesis indicates that thresholds for positive excess risk can be established between hormetic and carcinogenic doses.

Cherry picking of reference data

Siegel et al. [38] criticized The BEIR VII report in detail. One point is especially worthy of mention. The BEIR VII report cited that chromosomal aberrations induced by low-dose radiation in non-proliferating human cells were not repaired, thereby supporting LNT. However, that finding was a misrepresentation by failing to present that the aberrations in proliferating cells were repaired in several hours to the background level or less. Consequently, the result was opposite to what the BEIR VII argues.

Low-dose radiation elongates A-bomb survivors' lifespan

Earlier studies of lifespan elongation

Stewart and Kneale [39] showed that deaths in 1950–1982 from all non-malignant diseases in LSS population were significantly lower in survivors exposed to low doses than in unexposed persons. This U-shaped dose response relationship was refuted in comments by an LSS report [40], in which the mortality of A-bomb survivors was found to fit to the linear-threshold model (the estimated threshold is 1.4 Gy (DS86)) on the basis of LNT. Mine et al. [41] and Kondo [42] analyzed total deaths among about 100,000 A-bomb survivors in Nagasaki in1970–1988 and found that 290 males exposed to 0.5–1.49 Gy (T65D) showed significantly lower mortality. Although this beneficial effect was not found in female subjects, earlier studies [39, 41, 42] hint that A-bomb survivors exposed to low to intermediate doses live longer.

Contradiction 1: Excess relative mortality of early entrants is lower than that of late entrants

A-bomb survivors' lifespans were apparently shortened as discussed earlier. Cologne and Preston's analyses [13] were based on LNT using an assumption of zero exposure and zero risk, with no consideration of the

possibility that lifespans could be elongated and that cancer deaths might be reduced. Their results are reproduced in Fig. 5.

As depicted in Fig. 3, early entrants were exposed to higher doses of residual radiation than late entrants. Excess relative mortality of early entrants, however, is lower than that of late entrants (Fig. 5A and B). The key to resolve this contradiction can be explained by radiation hormesis-related mechanisms (e.g., enhanced DNA damage repair, apoptotic removal of aberrant cells, and anticancer immunity stimulation): the B group people were exposed to higher residual radiation than the A group people. Exposure doses of the B group must be in a hormetic dose range.

Contradiction 2: Excess relative mortality is inversely proportional to distance from the epicenter

Radiation doses are expected to be higher in proximal areas than in distal ones. If LNT is correct, then excess relative mortality must be higher in proximal areas. Data show inverse proportionality (Figs. 5C–F). Because the number of people is not small and mortality (death or life) data are accurate, the neat inverse proportionality must be close to the truth. Here again, this contradiction must be explained by radiation hormesis. People nearer the epicenter received more radiation than people farther away. Hormesis-related natural defense mechanisms also likely played a positive role in elongating the lifespan of survivors.

Excess relative mortality shows a typical J-shaped curve, indicating hormesis and a threshold

The radiation dose group of 0.005–250 mGy (Fig. 4, G group) comprises 40,403 people. Its excess relative mortality is almost equal to that of all in-city individuals ($n = 34{,}064$, a total of C to F groups) whose radiation doses are estimated to be zero or < 0.005 mGy (Fig. 4, control level Y). Considering the large population size, a lack of health hazard observed in group G would not be ascribable to a simple fluctuation: it must reflect actual effects of 0–250 mGy. If they were exposed to residual radiation, which was twice as strong as the initial radiation, then they might have been exposed to additional 0–500 mGy, a total of 0–750 mGy.

The excess relative mortality of H group (250–499 mGy is slightly higher than that of G group (0–250 mGy) and almost equal to D group (3–7 km from the epicenter). The mortality is below the control level X. These fluctuations are not random. At a glance from C to M in Fig. 5, one can see a beautiful J-shaped curve, an indicator of hormesis. When a J-shaped curve appears, we can establish a threshold at the crossing of the J and the x-axis. The threshold seems be between 250 and 499 mGy. Perhaps we could add 500–998 mGy of

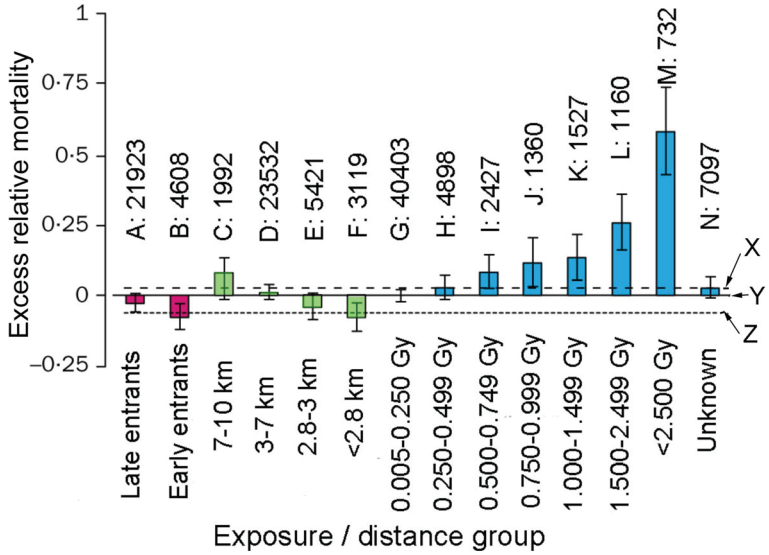

Fig. 5 Excess relative mortality by radiation dose or distance from the hypocenter. Figure 1 and Table 1 of an earlier report [13] are combined. The scale from left to right shows increasing proximity to the epicenter: A, late entrants (not in city, entered after 1 month); B, early entrants (not in city, entered within 1 month); C–F, in city at time of A-bomb, with different distance from the epicenter; G–M, seven dose groups with different doses; and N, distance from the hypocenter = 0.11–3 km with unknown doses. Numerals above A–N denote the number of people examined. The comparison group Y (baseline mortality, or excess relative mortality 0) is all in-city individuals ($n = 34{,}064$) with estimated doses of zero or < 0.005 Gy. Dashed line X is the in city zero dose distal groups C and D ($n = 25{,}524$). Dotted line Z is the in-city zero-dose proximal groups E and F ($n = 8540$). Y is the combined data of X and Z

residual radiation, twice as much radiation as estimated doses.

Cancer mortality of A-bomb survivors has been lower than the Japanese average

The US National Academy of Sciences proposed the spurious LNT in 1956 and put forward the problematic BEIR VII report in 2006 to support LNT (Fig. 4) [23]. The main reasons for the failure of the report are the use of LNT, use of the false assumption (zero exposure-zero risk), and neglect of hormesis effects. The Radiation Effects Research Foundation (RERF), a Japan–US scientific organization, has studied the health effects of A-bomb radiation. RERF has periodically reported research results and has insisted that the effects of radiation follow LNT in line with the BEIR VII report. The numbers of A-bomb survivors and solid cancer deaths are extracted from the latest three issues and are compared with Japanese averages (Table 2). The ratios of cancer deaths in both A-bomb survivors and NIC are smaller than those of Japanese averages. The numbers of people involved in Table 2 are not small. The differences are clear. Data must closely approximate reality. The finding that radiation of A-bombs reduces cancer mortality on average might be unexpected and incredible for LNT supporters. Nevertheless, such conclusions might be readily acceptable when one admits that low-dose radiation is hormetic under appropriate conditions and both A-bomb survivors and NIC who were exposed to low-dose radiation occupy a large fraction of the cohort. Consequently, low-dose radiation reduces cancer mortality on average and extends the lifespan (Fig. 5) as well.

Discussion

Earth has been exposed to ionizing radiation for billions of years

The current total heat flux from the Earth to space consists of half residual primordial heat and half radiogenic decay of uranium-238, thorium-232, and potassium-40, the respective half lives of which are 4.46, 14.0, and 1.28 billion years [43]. Therefore, radioactivity was much higher 4 billion years ago when life started to appear on the earth. Radioactivity at our university campus in the air is less than 100 cpm, as measured with a Geiger–Muller counter, but that of nearby granite is around 500 cpm or so. Radioactive substances from the birth of the earth are still abundant on the earth now. Radon-222, a daughter of uranium-238, and radon-rich hot springs are frequently found around uranium ore.

The human body receive roughly 20,000 radiation hits each second

In addition, carbon-14 and tritium-3 are constantly produced by the action of cosmic rays in the atmosphere. They are incorporated into our bodies. Japanese foods contain polonium-210 and potassium-40 and commit an effective dose of 0.47 mSv [44]. Consequently, the total of our annual background exposure dose is 2.1 mSv: cosmic rays (0.3 mSv), ground radiation (0.33 mSv), foods (0.99 mSv from carbon-14, polonium-210, and potassium-40), and aerial radon (0.48 mSv) [45]. When these radiation levels are converted to Bq (disintegration/second) using an Sv-Bq conversion table, rough estimation is 20,000 Bq. Potassium, an indispensable nutrient, and its associated potassium-40 (0.0117% of all naturally occurring potassium) contribute 4000 Bq. Therefore, we are exposed to by and large 20,000 radiation hits a second from not only the environment but also from materials inside our body. We ourselves are radioactive entities. In actuality, sleeping next to someone exposes one to 0.00005 mSv, which is the equivalent of eating half of a banana (0.0001 mSv). Living within 80 km of a nuclear plant and a coal plant for a year are, respectively, 0.00009 mSv and 0.0003 mSv. The dose of a chest X-ray is 0.02 mSv (ca. 1,000,000,000,000 hits [46]). A jet-liner flight from New York to London is 0.04 mSv [47]. Of course, these estimates are quite rough with significant uncertainties.

Breathing is much more hazardous than low-dose radiation

The earth was anaerobic until 2.5 billion years ago when cyanobacteria started to add oxygen into the air. Oxygen

Table 2 Comparison of solid cancer mortality in the lifespan study of A-bomb survivors with Japanese cancer mortality. Japanese average cancer deaths were calculated by dividing cancer deaths by total deaths each year during 1958–2009 [69]. Averages corresponding to survey periods were determined

Reporters	Year	Survey period	No. *hibakusha* or [NIC[a]]	No. cancer deaths (%)	% Japanese average cancer deaths
Preston et al. [73]	2007	1958–1998	105,427	17,448 (16.6)	21.4 (1958–1998)
			[25,427]	[3,994 (15.7)]	
Ozasa et al. [74]	2012	1958–2003	86,611	10,929 (12.6)	22.3 (1958–2003)
			[26,529]	[NA[b]]	
Grant et al. [75]	2017	1958–2009	80,205	17,316 (21.5)	23.3 (1958–2009)
			[25,239]	[5,222 (20.6)]	

[a], not-in-the-city; [b], not available

is actually toxic, but it is useful to produce energy effectively through oxidative phosphorylation. Our ancestors started to use oxygen, but reactive oxygen species (ROS) are inescapable byproducts of the oxidative process. ROS themselves are toxic. Nine billion ROS are produced in a cell a day [48]. We developed systems to quench ROS instantaneously using radical scavengers such as glutathione and L-cysteine and using enzymes such as superoxide dismutase and catalase.

Hazards by both respiration and low-dose ionizing radiation are caused mainly by ROS, but ROS production by respiration overwhelms that by low-dose radiation by thousands to a million of times the magnitude. ROS-quenching systems developed under intensive ionizing radiation conditions for more than billion years before the appearance of oxygen in the air must be readily applied to quench ROS by respiration.

Low-dose radiation is not only beneficial but necessary

A benefit of oxygen beyond energy production is the shielding of ultraviolet (UV) light. We sometimes expose clothes and mats to the sunlight to dry them and simultaneously kill bacteria, fungi, and ticks. We are suntanned in the sun, by which dead epithelial cells are shed from the skin when UV is strong. When oxygen was not in the air, UV was so strong that organisms were unable to live on the ground. The ozone layer cuts most UV; organisms today can move across the ground. Although UV can kill some organisms, it is indispensable to produce vitamin D. We are using the toxic UV as a need. So are ROS. When leukocytes "eat" bacteria, they enzymatically produce large quantities of ROS to kill them. ROS are sufficient to kill bacteria, but cells are also killed later. We used to see pus, a pile of dead leukocytes, in or around the wound before antibiotics became popular. In fact, J.F. Miesher extracted DNA from pus for the first time in 1869.

Figure 5 and Table 2 respectively show radiation-hormesis-related benefits: 1) elongating of lifespan and 2) reduced cancer deaths. Other analyses of LSS show hormesis in solid cancers [32] and leukemia [33]. Hormesis has been reported for many organisms such as protozoa [49], Drosophila [30, 31], and mice [50]. Lung cancer incidence of humans exposed to radon-222 is also hormetic [51]. These are some examples, constituting only the tip of the iceberg. Radiation-hormesis-related health benefits are possibly universal among all living organisms. Low-dose radiation is apparently not only beneficial but also necessary. When human cells were cultured under unshielded (1.75 mGy/y) and 10 cm lead-shielded (0.3 mGy/y) conditions, heat shock proteins (products of adaptive responses) were produced more in shielded cells than in unshielded cells, indicating that reduced radiation was not relief, but was stressful to the cells [52]. When bacteria were cultured 650 m underground, where radiation levels were 1/80 those at ground level, bacterial growth was retarded [52, 53]. If LNT is correct, then growth should be enhanced by removal of hazardous ionizing radiation. The results were the opposite, indicating the failure of LNT. Low-dose radiation is sensed by bacteria and gene expression is changed greatly at the transcriptional level [54].

Systematically associated many-layered defense mechanisms that LNT ignores

The sanctuary zone of a 30 km radius in Chernobyl is a paradise for animals and birds. More than 315 species thrive there. Glutathione levels of rats are elevated, but no DNA lesions are found on the animals. Levels of this radical scavenger in birds of 16 species are also high [55]. The authors argue that hormesis is working there. Consequently, ROS are quenched before attacking DNA. If DNA is injured by a large amount of ROS, cells can repair most of them. If DNA injuries exceed the repair capacity, cells are killed by apoptosis and are removed. If cancerous cells are produced, then most of them are removed by vigilant survey of immune systems. These adaptive defense systems are only some examples acquired by living organisms through evolution as innate essential attributes. Humans have the ability to sense crisis and to prepare for defense. Even if ionizing radiation is neither seen nor sensed, its products, ROS, constitute signaling molecules for defense systems. Defense systems at various levels (cells, tissues, organs, etc.) by various mechanisms (ROS quenching, DNA repair, apoptosis, anticancer immunity, etc.) must be associated with hormetic dose-response relationship for radiation induced cancer. A fundamental failure of LNT is that it ignores these time-requiring biological systems. Indeed, LNT is aptly accused of "epidemiology without biology" [56].

Magic of epidemiology to change negative to positive

A large body of experimentally obtained results collectively indicates radiation hormesis, but LNT proponents ignore these data. Risk of death from leukemia and lymphoma in more than 300,000 radiation-monitored workers (INWORKS) was studied. Results indicate that the dose-response matched well with LNT [57]. This result was praised in an internationally prestigious journal: *Nature* [58]. Soon more than 20 researchers raised objections, some of which included 1) lack of negative control, 2) LNT-based analyses, 3) no consideration of natural background and smoking, 4) 90% confidence limits (usually 95%) to achieve easy statistical significance, 5) one-tailed tests ignoring possible hormetic response, and 6) primitive miscalculations a schoolboy would not make. Soon a correction appeared in *Nature*, "The original version of this article incorrectly calculated

an 'expected' death rate from leukaemia among the workers, and as a result, the risk posed by radiation increments was wrong. The story has been corrected to reflect this." At least two works have leveled detailed criticisms against INWORKS studies [59, 60]. Epidemiology is apparently the last foothold for LNT, but "flexibility in data collection and analysis allows presenting anything as significant" [61]. The present author required no sophisticated epidemiology to find the opposite of what the authors assert in elongation of lifespan in Fig. 5 and a decrease of cancer mortality in Table 2.

Tremendous human, social, and economic losses caused by obstinate application of the linear no-threshold model

The individual external doses of 421,394 Fukushima residents for the first 4 months after the 2011 earthquake and tsunami were the following: 62.0%, < 1 mSv; 94.0%, < 2 mSv; 99.4%, < 3 mSv. The arithmetic mean and maximum for individual external doses were 0.8 and 25 mSv, respectively [62]. When actual external exposure doses estimated by individual glass-badge measurements in Date City, Fukushima, were compared with official ambient doses presented by the Japanese government, the ratio was 0.15 [63]. If this figure is applied to the data above [62], then the effective doses can be calculated as follows: 62.0%, < 0.15 mSv; 94.0%, < 0.3 mSv; 99.4%, < 0.45 mSv. The respective mean and maximum doses were 0.12 and 3.75 mSv. Even the maximum external dose is below the Japanese average medical exposure dose: 4 mSv. At the time of the Fukxushima nuclear accident, the International Commission on Radiological Protection (ICRP) recommended reference levels of 20–100 mSv [64]. Less than 100 mSv, the so-called low-dose range (Fig. 4), is accepted as representing no difference between exposed and non-exposed people. These are acute doses. Hazardous effects can be reduced to 1/16.5 by prolonged radiation such as in Fukushima [65], meaning that 1.65 Sv (100 × 16.4 mSv) might be non-hazardous. If it were not for LNT, evacuation would not have been necessary in Chernobyl or Fukushima [37]. In Ramsar, Iran, people have lived continuously in environments of 260 mSv with no health problems [66]. Tremendous human, social, and economic losses caused by obstinate application of the failed LNT could have been avoided [3]. In truth, LNT is a deeply immoral. Prof. G. Walinder's words, "The LNT hypothesis is a primitive, unscientific idea that cannot be justified by current scientific understanding. As practiced by the modern radiation protection community, the LNT hypothesis is one of the greatest scientific scandals of our time." Madame M. Curie's words, "Nothing in life is to be feared, it is only to be understood. Now is the time to understand more, so that we might fear less." It is the time to reconsider the use of the LNT [67]. The author's sincere hope is that some unmasking of LNT can help Fukushima people and others to live their lives free of irrational fear.

Conclusion

The linear no-threshold hypothesis (LNT) was recommended without solid data by the National Academy of Sciences in 1956. The academy put forward the BEIR VII report in 2006 as supporting evidence of LNT. This report was based on the Life Span Study (LSS) of A-bomb survivors. LSS has three major defects: 1) Residual radiation to which both A-bomb survivors and control subjects were exposed was neglected. Specifically, the control subjects were not valid as representing the negative control. 2) LNT is the basis of risk analyses. The failed model cannot be used. 3) Radiation hormesis is beyond the scope of LSS, but it actually occurs. The average lifespan of A-bomb survivors is longer than the Japanese average. Solid cancer deaths of A-bomb survivors and control subjects were fewer than the Japanese average. Consequently, one can reasonably infer that radiation of A-bombs elongated their lifespan and reduced cancer deaths on average, indicating a failure of LNT. Unfortunately, LNT has served as the basis of radiation regulation. If it were not for LNT, then evacuation of Fukushima people would not have been mandated and tremendous human, social, and economic losses would have been avoided. To avoid unnecessary losses and fear, humanity must learn as soon as possible that low-dose radiation is not only harmless but beneficial.

Abbreviations

BEAR: Biological Effects of Atomic Radiation; BEIR: Biological Effects of Ionizing Radiation; ERR: Excess relative risk; GP: Genetics Panel; ICRP: International Commission on Radiological Protection; LET: Linear energy transfer; LNT: linear no-threshold model; LSS: Life Span Study; NAS: National Academy of Sciences of the United States of America; NIC: not-in-the-city; RERF: Radiation Effects Research Foundation; RF: Rockefeller Foundation; SI: International System of Units; USA: United States of America

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Author's contribution
The author read and approved the final manuscript.

Author's information
The author was taught that ionizing radiation is limitlessly hazardous until March 2011. On the occasion of volunteer activity in Fukushima to measure contamination of evacuees, I examined the effects of ionizing radiation and found that LNT is invalid. As a scientist, I would like to relieve as many people as possible from unreasonable belief in LNT.

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Not applicable.

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The author declares that he has no competing interest related to this report.

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OBSERVATIONS ON THE CHERNOBYL DISASTER AND LNT

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□ The Chernobyl accident was probably the worst possible catastrophe of a nuclear power station. It was the only such catastrophe since the advent of nuclear power 55 years ago. It resulted in a total meltdown of the reactor core, a vast emission of radionuclides, and early deaths of only 31 persons. Its enormous political, economic, social and psychological impact was mainly due to deeply rooted fear of radiation induced by the linear nonthreshold hypothesis (LNT) assumption. It was a historic event that provided invaluable lessons for nuclear industry and risk philosophy. One of them is demonstration that counted per electricity units produced, early Chernobyl fatalities amounted to 0.86 death/GWe-year), and they were 47 times lower than from hydroelectric stations (~40 deaths/GWe-year). The accident demonstrated that using the LNT assumption as a basis for protection measures and radiation dose limitations was counterproductive, and lead to sufferings and pauperization of millions of inhabitants of contaminated areas. The projections of thousands of late cancer deaths based on LNT, are in conflict with observations that in comparison with general population of Russia, a 15% to 30% deficit of solid cancer mortality was found among the Russian emergency workers, and a 5% deficit solid cancer incidence among the population of most contaminated areas.

Keywords: Chernobyl, irradiation, LNT, health effects, remedial measures, social consequences

INTRODUCTION

Ten days after two steam and hydrogen explosions blew up the Chernobyl nuclear reactor the fire that melted its core died out spontaneously. But the drama of this catastrophe still flourishes, nourished by politics, authorities, media and interest groups of ecologists, charitable organizations and scientists. It lives in the collective memory of the world and propagates real health, social and economic harm to millions of people in Belarus, Russia and the Ukraine. It is exploited in attempts to strangle development of atomic energy, the cleanest, safest and practically inexhaustible means to meet the worlds energy needs. The world's uranium resources alone will suffice for the next 470,000 years (IAEA 2008). Chernobyl was indeed a historic event, but it is the only nuclear power station disaster that ever resulted in an occupational death toll, albeit a comparatively small one. A vast environmental dispersion of radioactivity occurred that did not cause any scientifically confirmed fatalities in the

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general population. *The worst harm to the population was caused not by radiation, and not to flesh, but to minds.*

This catastrophe provided many invaluable lessons. One of them is a recognition of the absurdity of the linear non-threshold hypothesis (LNT) which assumes that even near zero radiation dosage can lead to cancer death and hereditary disorders. Chernobyl was the worst possible catastrophe. It happened in a dangerously constructed nuclear power reactor with a total meltdown of the core and ten days of free emission of radionuclides into the atmosphere. Probably nothing worse could happen. Yet the resulting human losses were minute in comparison with catastrophes from other energy sources.

Highly sensitive monitoring systems that had been developed in many countries for the detection of fallout from nuclear weapons enabled easy detection of minute amounts of Chernobyl dust even in remote corners of the world. This added to global epidemics of fear induced by the accident. Radioactive debris was dispersed into the troposphere and stratosphere of the Northern Hemisphere up to at least 15 km altitude (Jaworowski and Kownacka 1994). On the first few days after the accident the concentrations of radiocesium measured at this altitude over Poland (maximum 36.1 mBq/m^3 STP) was 2 to 6% of that at the ground level. Such a high vertical distribution and mixing enabled a small portion of Chernobyl debris to pass over the equatorial convergence and into the Southern Hemisphere and on to the South Pole (Dibb et al. 1990; Philippot 1990). This was not in agreement with computer models of nuclear accidents that projected a maximum uplift of fission products to below 3000 m altitude (ApSimon et al. 1985; ApSimon and Wilson 1987).

Enormous amounts of radionuclides entered the air from the burning reactor. Yet the total emission was 200 times less than from all of the 543 nuclear warheads exploded in the atmosphere since 1945. The highest estimated radiation dose exposure to the world population from these explosions was 0.113 mSv recorded in 1963 (UNSCEAR 1988). The radiation doses from Chernobyl dust were estimated and compared with natural doses by UNSCEAR (2000a). During the first year after the accident the average individual dose received by inhabitants of the Northern Hemisphere was estimated by UNSCEAR as 0.045 mSv, *i.e.*, less than 2% of the average global annual natural dose (2.4 mSv per year). During next 70 years the global population will be exposed to a total Chernobyl dose of approximately 0.14 mSv, or 0.08% of the natural lifetime dose of 170 mSv. People living in the most contaminated areas of the former Soviet Union received an average annual whole body radiation doses in 1986 – 1995 of 0.9 mSv in Belarus, 0.76 mSv in Russia, and 1.4 mSv in Ukraine (UNSCEAR 2000b). Average doses estimated for the period 1986 – 2005 are 2.4 mSv in Belarus, 1.1 mSv in Russia, and 1.2 mSv in Ukraine (UNSCEAR 2008). All these doses dwarf in comparison with natural radi-

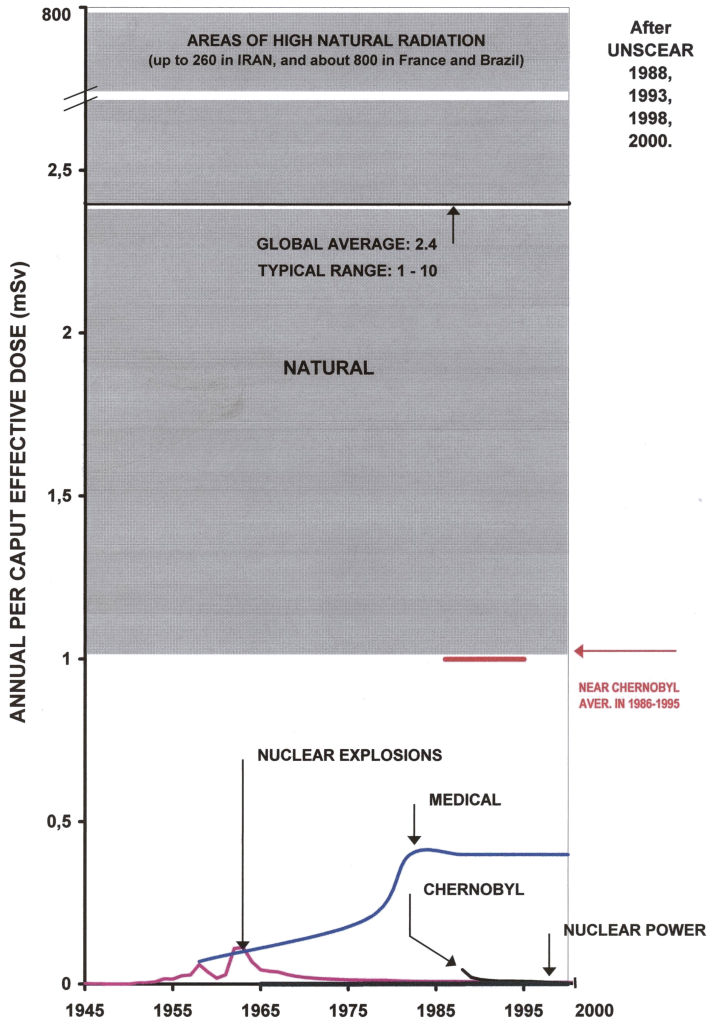

FIGURE 1. Worldwide and local (near Chernobyl and in areas of high natural radiation) average annual radiation doses from natural and man-made sources. Based on UNSCEAR (1988, 1993, 1998, 2000b).

ation doses in some parts of the world which, for example, in Ramsar, Iran reach >400 mSv/year (Mortazawi et al 2006) and in Brazil and south-western France reach up to more than 700 mSv per year (UNSCEAR 2000b) (Figure 1).

Comparison of these doses and epidemiological observations should be a basis of realistic estimates of the latent medical consequences of the Chernobyl accident, rather than risk factors based on LNT. This, and comparatively minute health consequences were apparent soon after the catastrophe (Jaworowski 1988), but this information was not shared with the

public. Recently the well-known environmentalist James Lovelock spent a lot of time dispelling all usual myths that surround the Chernobyl accident and stated that for many years the scientists who could have challenged the nonsense about the catastrophe chose to keep quiet (Murphy 2009).

No harmful health effects have ever been detected in high natural radiation background areas. This is consistent with other studies of the incidence of cancers in exposed populations. In the United States and in China, for example, the incidence of cancers was found to be lower in regions with high natural radiation than in regions with low natural radiation (Frigerio et al. 1973; Frigerio and Stowe 1976; Wei et al. 1990). Among British radiologists exposed mainly to x-rays the all causes and cancer mortality is lower by about 50% than that in the average male population of England and Wales (Berrington et al 2001). Also, in other population groups exposed to low doses of ionizing radiation (*i.e.*, patients diagnosed with ^{131}I and X-rays, dial painters, chemists and others exposed to ingested or inhaled radium or plutonium, persons exposed to higher levels of indoor radon and A-bomb survivors) a lower percentage of neoplastic malignances was observed (Cohen 2000; Luckey 2003; UNSCEAR 1994). A Taiwan study of several thousand residents of apartments contaminated with cobal-60 who had been chronically exposed to gamma rays for up to 20 years with total doses estimated to range from 120 to 4000 mSv revealed that the cancer mortality and congenital malformations of these residents substantially decreased rather than increased (Chen et al 2004), suggesting a stimulating or hormetic effect of low doses of low linear-energy-transfer (LET of ionizing radiation. This finding was partially confirmed by a later study on cancer incidence in a similar Taiwan cohort, in which in groups of all cancers, all cancers except leukemia, and solid cancers, with number of cancer cases ranging from 119 to 190, a deficit of incidence was found in comparison with unexposed population. Such deficit, however, was not found in groups of all types of leukemia and of some solid cancers of particular organs, in which the number of cases was 1 to 2 orders of magnitude smaller than in the first three groups (Hwang et al 2006). About 3000 reports on radiation hormesis were recently reviewed (Luckey 2003).

Among approximately 200,000 American, British and Canadian nuclear workers exposed to radiation total cancer deaths ranged from 27% to 72% of total cancer deaths in control workers (Luckey 2003). Such hormetic deficit invalidate LNT, because the concept of hormesis transcends difficulties of a dose threshold for excess cancers. In the absence of hormesis, the existence of a true threshold for excess cancers might be impossible to demonstrate rigorously because of the statistical problems of proving an absolute equality of effect in an epidemiological study at a very low dose level. If however a deficit is observed in the population irradiated at relatively high dose level, as in hormesis, there is

often a statistically significant difference at an acceptable confidence level (Webster 1993). This remark of Webster, an UNSCEAR member, reflects discussions in the Committee during preparation of its "hormetic report" (UNSCEAR 1994).

A more recent study based on collective doses for about 400,000 nuclear workers concluded that the cancer death data are consistent with the LNT relationship, although the authors found a 31% decrease in relative cancer mortality (Cardis et al 2007). This conclusion was based on an *ad hoc* accepted assumption of a confounding healthy worker effect for the studied cohort. However, the existence of this effect was not supported by their data or by any other factual evidence. This effect could be correctly assumed only if the cancer marker diagnostics (ACS 2009) and genetic tests were used in pre-employment screening and selection of these workers. But these procedures were not applied in the (Cardis et al 2007) cohort, and even now they are not recommended by ICRP, directives of European Union or IAEA International Basic Safety Standards. Thus this assumption is invalid and explains nothing. On the other hand, the statistical reanalysis of Cardis et al (2007) data clearly documents that their assumption of a healthy worker effect was incorrect, and their data indicated that low doses of ionizing radiation induced a hormetic effect in the exposed nuclear workers (Fornalski and Dobrzynski 2009).

In terms of human losses (there were 31 early deaths) the accident in the Chernobyl nuclear power plant was a minor event compared with many other major industrial catastrophes. In the 20th century more than ten such catastrophes have occurred, with several hundreds to many thousands fatalities in each. For example, coal smog killed approximately 12,000 people in London UK between December 1952 and February 1953 (Bell and Davis 2001). The annual death toll from accidents in Chinese coal mines reached 70,000 deaths in the 1950s and 10,000 in the 1990s (WNA 2009). In 1984 about 20,000 people perished due to an eruption in a pesticide factory in Bhopal (India) (Dhara and Dhara 2002), and the collapse of a hydroelectric dam on the Banqiao river in China in 1975 caused 230,000 fatalities (Altius 2008; McCully 1998; Yi 1998).

The world does not celebrate the anniversaries of these enormous man-made disasters, but year after year we do so for the hundreds and thousands of times less deadly Chernobyl accident. Ten years ago I discussed the possible causes of this paranoiac phenomenon (Jaworowski 1999). Measured as early deaths per electricity units produced by the Chernobyl facility (9 years of operation, total electricity production of 36 GWe-years, 31 early deaths) yields 0.86 death/GWe-year). This rate is lower than the average fatalities from a majority of other energy sources. For example the Chernobyl rate is 9 times lower than the death rate from liquefied gas, (Hirschberg et al 1998) and 47 times lower than from hydroelectric stations (40.19 deaths/GWe-year including Banqiao disas-

ter). But the political, economic, social and psychological impact of Chernobyl was enormous. Let's examine what happened starting with my personal experience.

PSYCHOLOGY TUNED BY LNT

At about 9 A.M. on Monday, April 28, 1986 at the entrance to my institute in Warsaw I was greeted by a colleague with a statement, "*Look, at 7:00 we received a telex from a monitoring station in northern Poland saying that the beta radioactivity of the air there is 550,000 times higher than the day before. I found a similar increase in the air filter from the station in our backyard, and the pavement here is highly radioactive.*"

This was a terrible shock. My first thought was: A NUCLEAR WAR! It is curious that all my attention was concentrated on this enormous rise of "total beta activity" in air used to monitor radiation emergencies from nuclear test fallout. Many years spent during the Cold War on preparations to defend the Polish population against the effects of a nuclear attack had conditioned my colleagues and me to such an exaggerated reaction. We reacted that way although we knew that on this first day of "Chernobyl in Poland" the dose rate of external gamma radiation penetrating our bodies was higher only by a factor of 3 from the day before, and it was similar to the average natural radiation doses which since time immemorial we have received from ground and cosmic radiation. At 11 A.M., after we had collected enough dust from the air for gamma spectrometry measurements, we discovered that it contained cesium-134, and thus that its source was not an atomic bomb but a nuclear reactor. This was tranquilizing news, which did not, however, calm our frantic behavior.

In 1986 the impact of a dramatic increase in atmospheric radioactivity dominated my thinking and everybody else's. This state of mind led to immediate consequences. First there were various hectic actions, such as *ad hoc* coining of different limits for radionuclides in food, water and other things. In particular countries these limits varied by a factor of many thousands, reflecting various political and mercenary factors and the emotional states of the decision makers. For example, Sweden allowed for 30 times more activity in imported vegetables than in domestic ones, and Israel allowed less radioactivity in food from Eastern than from Western Europe. The cesium-137 concentration limit in vegetables imposed in the Philippines was 22 Bq per kg, 8600 times lower than in the more pragmatic United Kingdom (Salo and Daglish 1988). In Poland a group of nuclear physicists and engineers proposed a cesium-137 limit of 27 Bq in 1 kilogram for any kind of food, but, fortunately, the authorities decided more soberly and imposed a 1000 Bq limit.

Behind these restrictions, meaningless from the point of view of human health, stood three factors: (1) emotion; (2) the LNT mindset

and international recommendations based on it; and (3) a social need to follow an old medical rule, "*Ut aliquit fecisse videatur*" (to make it appear that something is being done). The third factor was a placebo used by the authorities to dodge the worst kind of criticism, *i.e.*, accusations of inactivity in the face of a "monstrous disaster". This led to an overreaction in Europe and in some other countries, but at the greatest scale and with the most severe consequences in the Soviet Union. The costs of these regulations were enormous. For example, Norwegian authorities introduced a cesium-137 concentration limit of 6000 Bq/kg in reindeer meat and game, and a 600 Bq/kg limit for sheep (Henriksen and Saxebol 1988). A Norwegian eats an average of 0.6 kg of reindeer meat per year. The radiation dose from this meat would be 0.047 mSv per year. Thus this measure was aimed to protect Norwegians against a radiation dose about 200 times lower than the natural dose in some regions of Norway (11 mSv per year) (UNSCEAR 1982). The costs of this "protection" climbed to over $70 million in 1986, and in the 1990s it was still about $4 million per year (Christensen 1989; Idas and Myhre 1994). This means that unnecessary and wasteful restrictions, once implemented under the influence of the above three factors, have a long lifetime.

The hysterical reaction of authorities, further excited by extremely exaggerated media reports, is well exemplified by the Japanese government's cancellation of a several hundred million (in US$) contract for shipping Polish barley for the production of Japanese beer. This happened in May, 1986 a few days after completely false information of extreme contamination of Poland by Chernobyl fallout appeared on the front page of the biggest Japanese daily, Asahi Shimbun. It screamed with block letters, "DUST OF DEATH IN POLAND", and it cited my name as the source of the information. I was asked by the Polish government to write a text in English which might be used to avert this loss of money. I did this during a weekend spent with my wife in our cottage on the banks of the Vistula together with John Davis, the American ambassador to Poland, and his charming wife Helene. When I finished my writing assignment I asked John to correct the language. He said that the English was almost OK, but not exactly in proper diplomatic style. He then proceeded to change the text completely. On Monday a spokesman for the communist government asked me to read the text at his press conference. I presented the talk, but after I finished he distributed copies of the talk to the waiting flock of journalists. He was totally unaware that it had been prepared by the US ambassador. A visit by the Japanese ambassador to our institute managed to salvage the contract. A few days later ambassador Davis arranged an international deal for shipment by air of large quantities of powdered milk for Polish children to replenish strategic reserves that were rapidly being depleted. This was not an easy task because other European countries, in a similar position to ours, refused

to sell their milk. As we now know, during the next four years the Davises played a delicate but pivotal role in realizing a major goal for the people of Poland, Solidarity's victory over communism (Davis 2009; Davis et al 2006). As explained below Solidarity's triumph was related to the Chernobyl accident.

A classic example of wastefully applying the LNT principle to the Chernobyl emergency was provided by Swedish radiation protection authorities. When the farmers near Stockholm discovered that the Chernobyl accident had contaminated their cow's milk with cesium-137 above the limit of 300 Bq per liter imposed by authorities, they wrote asking if their milk could be diluted with uncontaminated milk from other regions to bring it below the limit. This would be done by mixing 1 liter of contaminated milk with 10 liters of clean milk. To the farmers' surprise and disappointment the answer was "no", and the milk was then to be discarded. This was a strange ruling since it has always been possible to reduce pollutants to safer levels by dilution. We do this for other pollutants in foodstuffs, and we dilute fumes from fireplaces or ovens with atmospheric air in the same way that nature dilutes volcanic emissions or forest fire fumes. The Swedish authorities explained that even though the individual risk could be reduced by diluting the milk, at the same time the number of consumers would be increased. Thus the risk would remain the same, but now spread over a larger population (Walinder 1995).

This was a faithful application of the ICRP recommendations based the LNT assumption and its offspring, the concept of "collective dose", *ie.*, reaching terrifyingly great numbers of "man-sieverts" by multiplying tiny innocuous individual radiation doses by large number of exposed people. In an earlier paper I exposed the lack of sense in and negative consequences of the LNT assumption and of the collective dose and dose commitment concepts (Jaworowski 1999). The application of these principles has caused the costs of the Chernobyl accident to exceed $100 billion in Western Europe (Becker 1996) and much more in post-soviet countries where it has led to unspoken sufferings and the pauperization of millions of people. The international institutions standing behind this assumption and these concepts certainly will not admit responsibility for their disastrous consequences. They should.

The linear no-threshold hypothesis was accepted in 1959 by the International Commission on Radiological Protection (ICRP 1959) as a philosophical basis for radiological protection. This decision was based on the first report of the newly established United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1958). A large part of this report was dedicated to a discussion of linearity and of the threshold dose for adverse radiation effects. Fifty years ago UNSCEAR's stand on this subject was formed after an in-depth debate that was not without influence from the political atmosphere and issues of the time.

The Soviet, Czechoslovakian and Egyptian delegations to UNSCEAR strongly supported the LNT assumption and used it as a basis for recommendation of an immediate cessation of nuclear test explosions. LNT was also supported by the Soviet Union during the later years of the Cold War (Jaworowski 2009), and this was consistent with the thinking of American authorities. The target theory prevailing in the 1950s and the then new results of genetic experiments with fruit flies irradiated with high doses and dose rates strongly influenced this debate. In 1958 UNSCEAR stated that contamination of the environment by nuclear explosions increased radiation levels all over the world and thus posed new and unknown hazards for present and future generations. These hazards cannot be controlled and "even the smallest amounts of radiation are liable to cause deleterious genetic, and perhaps also somatic, effects". This sentence had an enormous impact in subsequent decades and has been repeated in a plethora of publications. Even today it is taken as an article of faith by the public. However, throughout the entire 1958 report the original UNSCEAR view on LNT remained ambivalent. As an example, UNSCEAR accepted as a threshold for leukemia a dose of 4000 mSv (page 42), but at the same time the committee accepted a risk factor for leukemia of 0.52% per 1000 mSv, assuming LNT (page 115). The committee quite openly presented this difficulty and showed its consequences in a table (page 42). Continuation of nuclear weapons tests in the atmosphere was estimated to cause 60,000 leukemia cases worldwide if no threshold is assumed, and zero leukemia cases if a threshold of 4000 mSv were in place. In the final conclusions UNSCEAR pinpointed this dilemma. "*Linearity has been assumed primarily for purposes of simplicity*", and "*There may or may not be a threshold dose. The two possibilities of threshold and no-threshold have been retained because of the very great differences they engender*". After a half century we still discuss the same problem. In 1958 UNSCEAR had no doubts about major genetic defects in the world population that could be caused by nuclear test fallout, and estimated them as high as 40,000. But now the Committee has learned that even among the children of highly irradiated survivors of atomic bombings no statistically significant genetic damage could be demonstrated (UNSCEAR 2001).

However, in the ICRP document of 1959 no such controversy and no hesitations appeared. LNT was arbitrarily assumed, and serious epistemological problems related to the impossibility of finding harmful effects at very low levels of radiation were ignored. Over the years the working assumption of ICRP of 1959 came to be regarded as a scientifically documented fact by the mass media, public opinion and even many scientists. The LNT assumption, however, belongs in the realm of administration and is not a proved scientific principle (Jaworowski 2000).

The absurdity of the LNT was brought to light in 1987 when minute doses of Chernobyl radiation were used to calculate that 53,000 people

would die of Chernobyl-induced cancer over the next 50 years (Goldman et al 1987). This frightening death toll calculation was derived simply by multiplying the trifling Chernobyl doses in the US (0.0046 mSv per person) by the vast number of people living in the Northern Hemisphere and by a cancer risk factor based on epidemiological studies of 75,000 atomic bomb survivors in Japan. But the A-bomb survivor data are irrelevant to such estimates because of the difference in the individual doses and dose rates. A-bomb survivors were flashed within less than a second by radiation doses at least 50,000 times higher than any dose that US inhabitants will ever receive over a period of 50 years from the Chernobyl fallout. We have reliable epidemiological data for a dose rate of perhaps 1000 or 6000 mSv per second in Japanese A-bomb survivors. But there are no such data for human exposure at a dose rate of 0.0045 mSv over 50 years, nor will there ever be any. The dose rate in Japan was larger by a factor of about 10^{12} than the Chernobyl dose rate in the US. Extrapolating over such a vast span is neither scientifically justified nor epistemologically acceptable. It is also morally suspect (Walinder 1995). Indeed, Lauriston Taylor, the late president of the US National Council on Radiological Protection and Measurements, deemed such extrapolations to be a "deeply immoral use of our scientific heritage" (Taylor 1980).

In its document on protection of the public in a major radiation emergency ICRP recommended administration of stable iodine in form of tablets to be taken before or as soon as possible after the start of exposure to radioactive iodine-131 (ICRP 1984). The commission advised applying this prophylactic measure to everybody, pregnant women, neonates, young infants and adults, starting at the projected thyroid dose of 50 mSv. This recommendation was based on the LNT dogma. We followed it in Poland.

In the late afternoon of April 28, 1986 we learned from the BBC that there was a reactor accident in Chernobyl. We had seen the radioactive cloud flowing over Poland from east to west, and we had the first data on concentration levels of radioiodine in grass and soil in eastern Poland and in Warsaw. Using these data I calculated that contamination of thyroid glands of Polish children might reach a limit of 50 mSv, and much more if the situation in Chernobyl and weather conditions further aggravated the situation. In our institute we had no information from the Soviet Union on the current state of affairs or of any projections regarding the behavior of the destroyed reactor. Therefore we assumed that in the next few days the radioactivity in the air would increase and cover the whole country. We prepared a portfolio of countermeasures to be implemented by the government. I presented this project at a meeting of the deputy prime minister, several ministers and high ranking secretaries of the Central Committee of the PZPR (Polish United Workers Party) at

about 4 A.M. on April 29th. The most important measure recommended, and also accepted after a short discussion by this mixture of government and party, was stable iodine prophylaxis to protect the thyroid glands of children against iodine-131 irradiation. Administration of stable iodine in liquid form (as a "solution of Lugol") was initiated in the northeastern part of Poland approximately 38 hours after we discovered the Chernobyl fallout (at approximately midnight on April 28th). Treatment was given for the next three days, and about 18.5 million people, including adults, received the stable iodine drug.

We were able to perform this action successfully because we had already made plans for implementing nuclear war emergency measures. In the 1960s our institute had recommended that the government prepare for such an event by distributing strategic stores of stable iodine at sites all over the country as the only reasonable measure against body contamination from fission products. The program was implemented in the early 1970s, and each Polish pharmacy, hospital and various other institutions had large supplies of iodine. At the time of the Chernobyl accident Poland had more than enough iodine ready for use for approximately 100 doses for each Polish citizen. A few years after the catastrophe it was estimated that in the more contaminated parts of the country the average thyroid radiation dose in the 1 to 10 year old age group was about 70 mSv, and in about 5% of children the maximum dose was about 200 mSv (Krajewski 1991). A decade later we learned that among those of more than 34,000 Swedish patients who were not suspect for thyroid cancers, and whose thyroids were irradiated with iodine-131 up to dose of 40,000 mSv (average dose 1,100 mSv), there was no statistically significant increase in thyroid cancers, but rather a 38% decrease in their incidence (Dickman et al. 2003; Hall et al. 1996; Holm et al. 1988). If I knew then what I know today I would not have recommended to the Polish government such a vast prophylactic action, not because of its allegedly adverse medical effects - there were none (Nauman 1989) - but because its practical positive health effect was meaningless.

The most nonsensical, expensive and harmful action, however, was the evacuation of 336,000 people from contaminated regions of the former Soviet Union, where the radiation dose from Chernobyl fallout was about twice the natural dose. Later this limit was decreased to even below the natural level and was some five times lower than a radiation dose rate of 5.25 mSv/year at Grand Central Station in New York City (Benenson et al 2006). "Contaminated areas" were defined as being those where the average cesium-137 ground deposition density exceeded 37 kBq per m^2. In the Soviet Union these areas covered 146,100 km^2. The Chernobyl fallout of about 185 kBq/m^2 or more also covered large areas of Austria, Bulgaria, Finland, Norway and Sweden (UNSCEAR 2000b). Small areas with Chernobyl fallout reaching up to about 185 kBq/m^2 were also found

in other countries (Great Britain, Greece, Romania, Switzerland and Turkey (EUR 1996). Radiation doses received in areas with a cesium-137 deposition density of about 37 kBq/m^2 were about 1.6 mSv during the first year after the Chernobyl accident, and the lifetime dose (after 70 years) was predicted to reach 6 mSv (UNSCEAR 1988). This activity level is ten times lower than the average amount (400 kBq per m^2) of about 50 natural radionuclides present in a 10 cm thick layer of soil (Jaworowski 2002). The corresponding Chernobyl lifetime radiation dose is 28 times lower than the average natural lifetime dose of about 170 mSv. But the annual dose from 37 kBq of cesium-137 per m^2 was similar to the 1 mSv/year dose limit recommended by ICRP for the general population, and this is why it was accepted by the Soviet authorities as a yardstick for remedial measures.

The evacuation caused great harm to the populations of Belarus, Russia and the Ukraine. It led to mass psychosomatic disturbances, great economic loss and traumatic social consequences. According to Academician Leonid A. Ilyin, the leading Russian authority on radiation protection, the mass relocation was implemented by the Soviet government under the pressure of populists, ecologists and self-appointed "specialists", and it was done against the advice of the best Soviet scientists (Ilyin 1995; Ilyin 1996). The really dangerous air radiation dose rate of 1 Gy/h on 26 April 1986 (0.01 Gy/h 2 days later) covered an uninhabited area of only about 0.5 km^2 in two patches reaching up to a distance of 1.8 km southwest of the Chernobyl reactor (UNSCEAR 2000b).

Based on these data there was no valid reason for the masterly evacuation of 49,614 residents from the city of Prypyat and the village of Yanov situated about 3 km from the burning reactor. In these settlements the radiation dose rate in the air on 26 April 1986 was 1 mSv/h (UNSCEAR 2000b), and two days later it was only 0.01 mSv/h. Thus with a steadily decreasing radioactivity fallout the dose rate was not dangerous at all. However, according to L.A. Ilyin, one of the leaders of the Chernobyl rescue team, there was a danger that the "corium" (the melted core of the reactor, with a total volume of ~200 m^3, a mass of ~540 tons and a temperature of about 2000°C,) might penetrate down through the concrete floor and spread to rooms below. The team suspected that in these rooms there could have been a great volume of water with which the corium could come into contact. This would have led to a much more powerful explosion than the initial one and caused a vastly greater emission of radioactivity that could have covered Prypyat and Yanow with lethal fallout. Therefore, the evacuation of the whole population of these localities was a correct precautionary measure that was carried out in an orderly manner in only two hours. But the evacuation and relocation of the remaining approximately 286,000 people, of which there were about 220,000 after 1986 (UNSCEAR 2000b), was an irrational overreaction

FIGURE 2. Measuring radiation on April 10, 2008 at a sport stadium downtown of Pripyat, about 4 km NW from Chernobyl reactor. The dose rate was 0.28 µSv/h (2.5 mSv/year). Based on Fornalski (2009).

induced in part by the influence of the ICRP and IAEA recommendations based on the LNT (Ilyin 1995). The current reluctance of the Ukrainian authorities to resettle the residents back to Prypyat (now a slowly decaying ghost town and tourist attraction) does not seem rational. The radiation dose rate measured on April 10, 2008 in the streets of this city ranged from 2.5 to 8.4 mSv/year, *i.e.*, more than 10 times lower than natural radiation in many regions of the world (Fornalski 2009) (Figure 2).

Besides the 28 fatalities among rescue workers and employees of the power station due to very high doses of radiation (2.9 – 16 Gy), and 3 deaths due to other reasons (UNSCEAR 2000b), the only real adverse health consequences of the Chernobyl catastrophe among approximately five million people living in the contaminated regions were the epidemics of psychosomatic afflictions that appear as diseases of the digestive and circulatory systems and other post-traumatic stress disorders such as sleep disturbance, headache, depression, anxiety, escapism, "learned helplessness", unwillingness to cooperate, overdependence, alcohol and drug abuse and suicides. These diseases and disturbances could not have been due to the minute irradiation doses from the Chernobyl fallout (average dose rate of about 1 – 2 mSv/year), but they were caused by radiophobia (an deliberately induced fear of radiation) aggravated by wrongheaded administrative decisions and even, paradoxically, by increased medical attention which leads to diagnosis of subclinical changes that persistently hold the attention of the patient. Bad administrative decisions made several million people believe that they were "*victims of Chernobyl*" although the average annual dose they received from "Chernobyl" radiation was only about one third of the average natural dose. This was the main factor responsible for the economic losses caused

by the Chernobyl catastrophe, estimated to have reached $148 billion by 2000 for the Ukraine and to reach $235 billion by 2016 for Belarus.

Psychological factors and a failure to teach radiological protection in medical school curricula might have led to abortions of wanted pregnancies in Western Europe during the period soon after the accident where physicians wrongly advised patients that Chernobyl radiation posed a health risk to unborn children. However, numerical estimates of this effect (Ketchum 1987; Spinelli and Osborne 1991) cast doubt on this assumption. Similarly uncertain are estimates of the number of decisions against fecundation probably taken in Europe during the first few months after the accident (Trichopoulos et al 1987). This problem was discussed in 1987 by an IAEA Advisory Group that concluded that medical practitioners having direct contact with the population at large are among the most important persons who might develop the right perception of risks in nuclear emergencies, prevent social panic and overreactions, and help to ensure the rational behavior in the society. After the Chernobyl accident the public very often turned for help to medical practitioners, but physicians were unable to provide realistic advice even on minor problems. This was because medical curricula did not at that time prepare doctors for nuclear emergencies. In none of the nine countries represented at the meeting were the principles of radiobiology and radiation protection included in medical school curricula (IAEA 1987). Lack of knowledge in this important group was among the factors that increased public anxiety and stress. It seems that now, two decades later, the situation in this respect is very much the same.

EFFECTS OF CHERNOBYL FALLOUT ON THE POPULATION

In 2000 the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 2000b) and in 2006 the United Nations (UN) Chernobyl Forum (a group composed of representatives from 8 UN organizations, the World Bank and the governments of Belarus, Russia and the Ukraine) stated in their documents that, except for thyroid cancers in the population of highly contaminated areas, no increase in the incidence of solid tumors and leukemia, and no increase in genetic diseases was observed. An increase in registration of thyroid cancers in children under 15 years old was first found in 1987, one year after the accident, in the Bryansk region of Russia, and the greatest incidence, of 0.027% was found in 1994. Both of these studies were made too early to be in agreement with what we know about radiation induced cancers. The mean latency period for malignant thyroid tumors in adults and children exposed to external and internal medical irradiation with <20 to >40 Gy is about 28 years (Kikuchi et al; 2004; UNSCEAR 2000b). Kikuchi et al (2004) tried to explain the discrepancy between the clinical experience

and the Chernobyl findings by some exotic ideas, such as, for example, "radiation leakage or other environmental conditions, exposure to carcinogens that occurred near Chernobyl prior to the nuclear accident, or that the population is genetically predisposed to thyroid cancer". However, mass screening and diagnostic suspicion, already flourishing in 1987, is a more serendipitous explanation.

The number of 4000 new thyroid cancers registered among the children from Belarus, Russia and the Ukraine should be viewed with respect to the extremely high occurrence of these dormant subclinical malignant tumors that contain transformed tumor cells and are quite common in the population (Akslen and Naumov 2008; Weinberg 2008). This is exemplified by occult thyroid cancers, the incidence of which varies from 5.6% in Colombia, 9.0% in Poland, 9.3% in Minsk, Belarus, 13% in the United States, 28% in Japan, to 35.6% in Finland (Harach et al 1985; Moosa and Mazzaferri 1997). In Finland occult thyroid cancers are observed in 2.4% of children (Harach et al 1985), *i.e.*, some 90 times more than the maximum observed in the Bryansk region. In Minsk, Belarus the normal incidence of occult thyroid cancers is 9.3% (Furmanchuk et al 1993). The "Chernobyl" thyroid cancers are of the same histological type and are similar in invasiveness to the "occult cancers" (Moosa and Mazzaferri 1997; Tan and Gharib 1997). Since 1995 the number of registered cancers has tended to decline. This is not in agreement with what we know about radiation induced thyroid cancers whose latency period is about 5 - 10 years after irradiation exposure (Inskip 2001) and whose risk increases until 15 - 29 years after exposure (UNSCEAR 2000a). In the United States the incidence rate of thyroid tumors detected between 1974 and 1979 during a screening program was 21 times higher than before the screening (Ron et al 1992), an increase similar to that observed in three former Soviet countries. It appears that the increased registration of thyroid cancers in contaminated parts of these countries is a classical screening effect.

According to the regulations of the Belarusian Ministry of Health the thyroids of all people who were younger than 18 in 1986 and those of each inhabitant of "contaminated areas" must be diagnosed every year (Parshkov et al 2004). More than 90% of children in contaminated areas are now diagnosed for thyroid cancers every year with ultrasonography (USG) and other methods. It is obvious that such a vast scale screening, probably the greatest in the history of medicine, resulted in finding thousands of the "occult" cancers, or "incidentalomas", expanded to forms detectable by modern diagnostic methods that were not in routine use in the Soviet Union before 1986.

Data for the past 20 years published by (Ivanov et al 2004) and cited in the UNSCEAR and Chernobyl Forum documents (Forum 2005; Forum 2006; Ivanov et al 2004; UNSCEAR 2008) show, in comparison to the Russian general population, a 15% to 30% lower mortality from solid

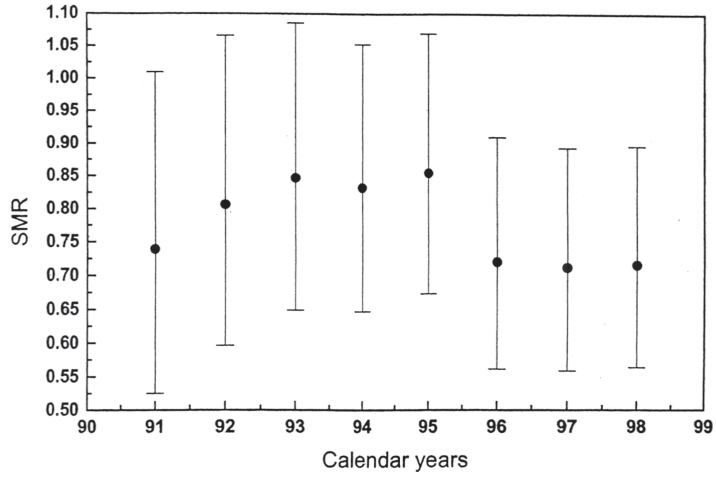

FIGURE 3. Standard mortality ratios (SMR) for solid cancers among the Russian emergency workers. The values of SMR indicate how cancer mortality of emergency workers differs from that in general population of Russia used as a control group (1.0). The deficit of cancers among these workers between 1990 and 1999 ranged between 15% and 30%. Based on Ivanov et al. (2004, page 225).

tumors among the Russian Chernobyl emergency workers and a 5% lower average solid tumor incidence among the population of the Bryansk district, the most contaminated in Russia (Figures 3 and 4). In the most exposed group of these people (with an estimated average radiation dose of 40 mSv) a 17 % decrease in the incidence of solid tumors of all kinds was found. In the Bryansk district the leukemia incidence is not higher than in the Russian general population. According to (UNSCEAR 2000b) no increase in birth defects, congenital malformations, stillbirth or premature births could be linked to radiation exposures caused by the Chernobyl fallout. The final conclusion of the UNSCEAR 2000b report is that the population of the three main contaminated areas with a cesium-137 deposition density greater than 37 kBq/m^2 "*need not live in fear of serious health consequences*", and forecasts that "*generally positive prospects for the future health of most individuals should prevail*".

The publications of the UN Chernobyl Forum (2005, 2006) present a rather balanced overview of the Chernobyl health problems, but with three important exceptions. The first is (mainly after (Cardis et al. 2005) ignoring or downplaying the effect of screening for thyroid cancers of about 90% population (see discussion above), and interpreting the results with a linear no-threshold dose-response model. This Cardis et al (2005) paper, however, was criticized by (Scott 2006) for this interpretation, not confirmed by the data presented. Both the Chernobyl Forum (2005, 2006), and Cardis et al (2005, 2006) papers, ignore the aforementioned fundamental problem of occult thyroid cancers in the former

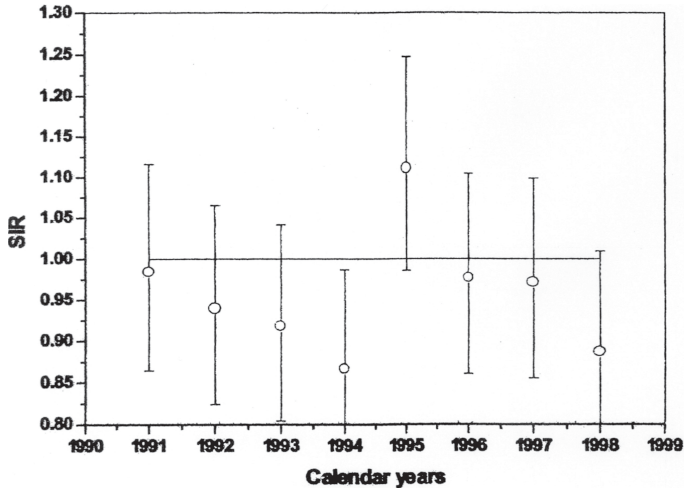

FIGURE 4. Standard incidence ratios (SIR) for solid cancers among inhabitants of Bryansk region, Russia. The average deficit of cancers in Bryansk region was 5%, and in the most exposed group (mean radiation dose of 40 mGy) 17%. Based on Ivanov et al. (2004, pages 373 and 374).

Soviet Union and elsewhere in Europe. The incidence of thyroid occult cancers increased rapidly after advent of new USG diagnostics (Topliss 2004). Reaching up to 35.6% (see above) this incidence is more than 1300 times higher than the maximum thyroid cancer incidence found in Bryansk Region, Russia in 1994 (UNSCEAR 2000b), what implies a vast potential for bias. It seems that up until now an epidemiological study on temporal changes of intensity of thyroid screening in the former Soviet Union was not performed. The conclusions from epidemiological studies not taking into account these changes in screening may be invalid. In Bryansk region, Russia the thyroid cancer incidence was found 45% higher in males and 90% higher in females, than for the whole Russian population. However, when dose-response analyses were performed using external and internal comparisons, no positive association of thyroid cancers with radiation dose was observed, but a negative one, i.e. a hormetic effect (Ivanov et al 2004). These results strongly suggest that the increased cancer rates in Bryansk (and by implication in other contaminated regions) compared with general population rates are due to thyroid cancer screening and better reporting rather than radiation exposure (Ron 2007). In her interpretation of thyroid cancer data Ron also did not take into account the occult thyroid cancer issue. Even more important, however, was perhaps ignoring both in her and Cardis et al (2006) papers a decrease of thyroid cancer incidence of up to 38%, after diagnostic irradiation with iodine-131 of many thousands of non-cancer Swedish patients with doses similar to or higher than those received from

the Chernobyl fallout by inhabitants of post-soviet countries (Dickman et al 2003; Hall et al 1996;Holm et al 1991; Holm et al. 1988).

The second problem with the Chernobyl Forum (2005, 2006) reports is estimation of deaths among the patients with acute radiation disease. From among 134 persons with this disease who had been exposed to extremely high radiation doses, 31 died soon after the accident. Among the 103 survivors, 19 died before 2004. Most of these deaths were due to such disorders as lung gangrene, coronary heart disease, tuberculosis, liver cirrhosis, fat embolism and other conditions that can hardly be defined as caused by ionizing radiation. But the Chernobyl Forum (2005, 2006) presents them as a resulting from high irradiation and sums them up to a total of approximately 50 victims of acute irradiation. After many summers all the 103 survivors will eventually die. The Chernobyl Forum (2005, 2006) philosophy would then count them all, yielding a round total of 134 victims of high irradiation. In fact, the mortality rate among these 103 survivors was 1.08% per year, *i.e.*, less than average mortality rate of 1.5% in the three affected countries in 2000 (GUS 1991).

And finally, the third "Forum problem" is the projections of future fatalities caused by low level Chernobyl radiation from 4000 up to exactly 9935 deaths. These numbers are not based on epidemiological data of cancer mortality observed during the past 20 years by (Ivanov et al 2004) that demonstrated no such increase, but rather a decrease of solid tumor and leukemia deaths among exposed people. These epidemiological data, rather than the LNT assumption, should be used as the basis for a realistic projection of the future health of the millions of people officially labeled "victims of Chernobyl". However, the Chernobyl Forum (2005, 2006) instead chose to use the LNT radiation risk model (ICRP 1991) and performed a simplistic arithmetical exercise by multiplying small doses by a great number of people and including a radiation risk factor deduced from the Hiroshima and Nagasaki studies. People living in areas highly contaminated by the Chernobyl fallout were irradiated during a protracted time. The dose rates in Hiroshima and Nagasaki were higher by a factor of about 10^{11} than the average dose rate of the "Chernobyl victims" that was used in Forum's projections. The result of this exercise is nothing more than a fibbing fantasy. Several scientific and radiation protection bodies, including UNSCEAR, the Health Physics Society (Mossman et al 1996), the French Academy of Science (Tubiana 1998), and even the chairman of the International Commission on Radiological Protection (Clarke 1999), advised against making such calculations. Merely publishing these numbers is harmful and petrifies the Chernobyl fears. Any efforts to explain the intricacies of radiation risk assessments to the public or to compare these numbers with the much higher level of spontaneous cancer deaths will be futile excercises. The past twenty years proved that such efforts are worthless. Making such cal-

culations keeps a lot of people busy and well but has no relation to reality and honesty. The Chernobyl Forum (2005, 2006) elucubrations pale in the face of recent estimates by other bodies (Greenpeace 2006; Vidal 2006) predicting the incidence of millions Chernobyl cancers and hundreds of thousands deaths.

It is reassuring, however, that sixteen years after the Chernobyl catastrophe another group composed of four UN organizations (United Nations Development Programme – UNDP; United Nations International Children's Emergency Fund – UNICEF; World Health Organization – WHO; United Nations Office for the Coordination of Humanitarian Affaires – UN-OCHA) dared to state in its 2002 report based on UNSCEAR studies that a great part of the billions of dollars used to mitigate the consequences of the Chernobyl accident was spent incorrectly. The dollars spent in these efforts did not improve but actually worsened a deteriorating situation for 7 million so-called "victims of Chernobyl" and petrified the psychological effects of the catastrophe and the wrong decisions of the authorities. The report (UNDP 2002) recommended that the three post-soviet countries and the international organizations abandon the current policy. The misguided basis of this policy, *i.e.* expectation of mass radiation health effects, was responsible for the enormous and uselessly expended resources sacrificed for remediation efforts. The report presented 35 practical recommendations needed to stop the vicious cycle of Chernobyl frustrations, social degradation, pauperization and the epidemic of psychosomatic disorders. The recommendations suggest a reversal from the position of concentrating attention on nonexistent radiation hazards and that the relocated individuals should be allowed to return to their old settlements, *i.e.*, that essentially all of the restrictions should be removed.

But here we enter a political mine-field. How well will people accept losing the mass benefits (equivalent to about $40 a month) that they poetically call a "coffin bonus"? How can it be explained to them that they were made to believe that they were the "victims" of a non-existing hazard, that the mass evacuations were an irresponsible error, that for twenty years people were unnecessarily exposed to suffering and need, that vast areas were unnecessarily barred from use, and that their countries' resources were incredibly squandered? One can read in many publications that the Chernobyl catastrophe had serious political implications by becoming an important factor in the dismantling of the Soviet Union and in attempts to control nuclear arms. As Mikhail Gorbachev stated : "*The nuclear meltdown at Chernobyl 20 years ago … even more than my launch of perestroika, was perhaps the real cause of the collapse of the Soviet Union five years later. … Chernobyl opened my eyes like nothing else: it showed the horrible consequences of nuclear power …One could now imagine much more clearly what might happen if a nuclear bomb exploded …one SS-18 rocket could contain a hundred*

Chernobyls. Unfortunately, the problem of nuclear arms is still very serious today." (Gorbachev 2006).

Would fulfilling the recommendations of the UNDP 2002 report again result in a political catharsis and perhaps induce violent reactions? Probably not in Russia, where a more rational approach to Chernobyl prevails. But the political classes of Belarus and Ukraine have for years demonstrated a much more emotional approach. When the (UNSCEAR 2000a) report documenting the low incidence of serious health hazards resulting from the Chernobyl accident was presented to the UN General Assembly, the Belarus and Ukraine delegations lodged a fulminating protest. This in 2002 set the stage the Chernobyl Forum and helped to focus its agenda.

The Chernobyl rumble and emotions are beginning to settle down. In the centuries to come the catastrophe will be remembered as a proof that nuclear power is a safe means of energy production. It even might change the thinking of ICRP.

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Symposium Paper

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Genetic studies at the Atomic Bomb Casualty Commission–Radiation Effects Research Foundation: 1946–1997

(genetic effects of atomic bombs/radiation genetics/genetic epidemiology/critique of Dubrova report/Hiroshima and Nagasaki)

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Beginnings

It is difficult, some 52 years later, to recreate the intensity of the concern about the delayed effects of exposure to the atomic bombs, as well as other radiation exposures, that surfaced in the first few months after the bombings. It is not generally appreciated that the survival in Japan of so many persons receiving exposures to ionizing radiation up to the amount compatible with survival was unexpected. The physicists on the Manhattan Project had assumed that anyone close enough to the hypocenter of the explosion to have received significant amounts of radiation would have been killed by the blast or thermal effects of the bombs (1). The survivors within 2 km of the hypocenter of the explosion, this being the radius of significant radiation, were, therefore, a group without parallel in human history, regardless of individual feelings about the use of the two bombs, and the significance of an intensive follow-up of this group was at that time immediately apparent to laypersons and scientists alike of all nationalities.

Dr. Putnam (2) has outlined the developments that led to the involvement of the Academy in the organization of the long-term studies of the atomic bomb survivors carried out by the Atomic Bomb Casualty Commission (ABCC). Elsewhere, I have described the somewhat unusual circumstances that resulted in then First Lieutenant Neel, Medical Corps, U.S. Army, being assigned to the small survey team that first touched down in Japan on November 25, 1946, charged with advising the Academy's new Committee on Atomic Casualties concerning both the potentialities and the problems inherent in any study the Academy might undertake (3).

The Genetic Challenge of Hiroshima and Nagasaki

Because my background at that time included a Ph.D. in genetics as well as my medical training, I covered the genetic beat for the group. It was obvious from the outset that the obstacles to a proper study were formidable. The task was clear: to ascertain all births occurring in the two cities and then examine every single one of those children. But the devastation in the two cities was daunting, all services badly disrupted and facilities in ruin; the vast majority of deliveries were at home—and in the Japanese culture, the birth of an abnormal child was considered a disgrace and concealed whenever possible. Human genetics as a discipline at that time was still a very fledgling science in the U.S., and almost nonexistent in Japan: there was no pool of expertise from which to recruit for the study, and the Academy, for all its well-deserved prestige as an advisory body, was not accustomed to operating field studies, let alone studies of that magnitude and difficulty. Finally, working out the appropriate relationship with the funding agencies, from the Atomic Energy Commission to the Department of Energy, presented issues that persist right down to the present. To say there was a certain amount of stumbling around in the beginning would be a kind appraisal of the situation. In particular, the resources necessary for a proper follow-up were grossly underestimated at first.

The Study

The key to the decision that a proper genetic study might be feasible materialized when, early on, Dr. I. Matsubayashi, chief of public health for Hiroshima City, informed me that during those difficult days, the Japanese still maintained their wartime rice-rationing system, with a special provision for pregnant women. A survey determined that the registration of pregnant women at the completion of the fifth lunar month of pregnancy was almost 100% complete, and, by coordinating the Atomic Bomb Casualty Commission effort with that registration, the basis for a prospective ascertainment of the total population of newborns-to-be in Hiroshima and Nagasaki was established. That procedure minimized the opportunities for the concealment of birth defects and other unfortunate pregnancy outcomes.

The initial battery of observations on each newborn included occurrence of major congenital defect/sentinel phenotype, stillbirth, survival of liveborn children through the neonatal period, sex of child, and birth weight. There was a further clinical examination of a subsample of these children at age 9 months (cf. ref. 4). In 1953, that major clinical program was discontinued, but births in the two cities were, as they were registered for civil purposes, screened for parental radiation history, and, where indicated, added to a growing cohort for future study. By 1984, there were very few births in the two cities to exposed parents, and the study cohort was closed out, with 31,150 children in the cohort of children one or both of whose parents had been within 2 km of the hypocenter of the bombings, the so-called proximally exposed. A suitably matched control cohort, which had been accumulating over the years, of 41,066 children, also was closed. In 1967, Dr. A. A. Awa and associates launched major cytogenetic studies of a subset of this cohort. In the 1970s, Dr. T. Furusho and Dr. M. Otake analyzed from school records the physical development of a subset of these children who were in middle and senior high school. In 1972, a search for mutational damage in a battery of serum proteins and erythrocyte enzymes was launched, using this cohort, which study came under the direction of Dr. C. Satoh. Finally, the children in these cohorts

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were followed for survival and malignancy, the studies on malignancy using the newly established Cancer Registries in Hiroshima and Nagasaki, with Dr. H. Kato playing a major role in those studies. It must be obvious that a study of this magnitude is the work of many more hands than those just mentioned. On the U.S. side, Dr. W. J. Schull, with whom I have been associated in these studies since 1949, played an especially prominent role. Altogether there were perhaps 100 professionals involved in these studies over the years, participants in the many scientific papers that have appeared.

In 1986, new estimates of the radiation exposures sustained by the survivors of the bombings became available, and the entire data set, which had been subject to numerous previous reports, was reanalyzed on the basis of these new dose schedules. The most relevant of the resulting papers were collected in a volume published by the National Academy Press in 1991 (5). There was no statistically significant effect of parental exposure on any of the indicators of possible genetic damage mentioned above, but, pooling the results of the analysis of all the indicators, where pooling was feasible, the net regression of the pooled indicators on parental exposure was slightly positive. Inasmuch as there seems no doubt some genetic damage resulted from the A-bomb exposures, we essayed to explore the implications of this small positive regression for the estimation of the genetic doubling dose of acute ionizing radiation for humans (6). The doubling dose is the exposure of a population to ionizing radiation that will produce the same amount of genetic damage as occurs spontaneously each generation. It can be expressed either as per haploid gamete or per diploid zygote (i.e., person); the studies in Japan yielded a zygotic estimate (7) whereas most of the experimental studies resulted in gametic estimates. The doubling dose is a convenient concept, but the many assumptions and practical difficulties in actually deriving a doubling dose were well enumerated by Muller (8). The situation has not changed materially in the ensuing 39 years (cf. ref. 9). Ideally, the concept embraces the whole spectrum of mutational morbidity, from mutations involving entire chromosomes to single nucleotide substitutions, thus requiring the study and integration of a wide range of genetic damage. In addition, for the Japanese data this calculation required specifying the contribution of spontaneous mutation in the preceding generation to such indicators as congenital defect and early death. Nevertheless, in an imperfect world, the doubling-dose concept supplies a perspective, if blurred, difficult to obtain by any other approach. Because of the mixed spectrum of radiation delivered by the atomic bombs, dose must be measured in sieverts (Sv).

The doubling-dose estimate suggested by these studies was an acute gonadal exposure of approximately 2.0 Sv equivalents, with a wide but, for several reasons, essentially indeterminate error (6). We believe that, as befits the situation, the assumptions in reaching this estimate have been very conservative. This estimate may be biased downward by the somewhat lower socioeconomic status of the proximally exposed parents than that of the control population in the decade after the bombing (10). For instance, if only 50% of the small increase in mortality among the children born to survivors of the bombing were socioeconomic in origin, the estimate of the doubling dose would become 4.0 Sv equivalents. This is a zygotic rather than gametic doubling dose. The calculations revealed that the doubling dose was unlikely to be less than 1.0 Sv equivalents, but in the absence of statistical significance an upper bound could not be assigned to the estimate. To be specific, the data do not exclude estimates of the zygotic doubling dose of acute radiation as high as 3 or 4 or even 5 Sv equivalents.

Most of the radiation human populations receive is in small dribbles, or even more or less continuously as from cosmic radiation or radon. In the mouse, at the experimental doses used, such chronic radiation is genetically only about ⅓ as effective in producing mutations as acutely delivered radiation, such as was involved in the Japanese exposures (11). For technical reasons discussed elsewhere (6), we have argued that with the radiation exposures in Japan, the appropriate conversion factor is ½. The zygotic doubling dose for chronic radiation thus becomes in the neighborhood of 4 Sv equivalents. For those for whom these radiation units are unfamiliar, some perspective to the numbers being used in this presentation is provided by the following: The average U.S. citizen is receiving about 0.004 Sv equivalents a year from all sources of radiation in the environment but especially from radon (12). This annual exposure is about 1/1,000 of a doubling dose. Otherwise stated, it would require some 1,000 years to accumulate a doubling dose of radiation in our industrialized society—and there is a long-running debate as to whether at these very low doses of radiation, the body's DNA repair mechanisms may be able to heal all the potential genetic damage caused by the radiation. In an additional effort to provide perspective, let me point out that in the decade after the atomic bombings, no less a scientific figure than the geneticist J. B. S. Haldane could speculate that the doubling dose of radiation for humans could be as low as 0.05 Sv equivalents (13); from this you can readily grasp the perspective brought to this issue by the studies in Japan.

The Scientific Spin-Off of the Study

Although the dominating objective in the conduct of the genetic studies in Japan has been a comparison of the children born to A-bomb survivors exposed within 2 km of the hypocenter and the children of suitable controls, it was realized from the outset that the children of unexposed parents would provide data of interest in their own right. For instance, these studies have resulted in the first extensive normative data on the pattern of major congenital malformations in a mongoloid (Japanese) population and in similar data with respect to cytogenetic abnormalities in the general population and on inherited variation in a series of some 30 human proteins (14–16). However, the genetically most interesting data were on the effects of inbreeding. In the work preliminary to setting the design of the major program, it became clear that cousin marriage was by Western standards quite frequent in Japan, 6% of the newborns in Hiroshima and 8% in Nagasaki resulting from consanguineous marriages. Because if this difference in frequency between the two study groups of children was unequally distributed in the two cities it would be a confounding factor in the results, the consanguinity status of the parents of each child in the study was determined. In 1958–1960, a special study was undertaken of this extensive and unbiased sample of inbred children and suitable controls (17). This study, probably the most complete study of consanguinity effects ever performed, revealed smaller consanguinity effects than the prevailing opinion; the data have been extensively used not only in genetic counseling but also for insights into the biological significance of the surprising amount of variation encountered at the DNA level.

A Comparison with the Relevant Studies on Mice

When the atomic bomb project, the Manhattan Engineering District, was initiated during World War II, it was recognized that some increase in "occupational" exposures to radiation was inevitable, and studies to anticipate worker's health hazards were undertaken. At that time, most of the data available on the genetic effects of ionizing radiation were derived from experiments with *Drosophila*. The mouse met the obvious need for an experimental organism whose physiology was closer to the human, and although further experiments on *Drosophila* were sponsored by the District (and its successor agencies), major experiments on mice were initiated, experiments that after the war were amplified by additional efforts in many

countries. While the human data have been accumulating, the experimental data from mice have been the chief guide to human risks.

When the data from humans just summarized indicated less of a genetic radiation risk than the then-prevailing extrapolations to human from the mouse experiments, Susan Lewis and I (18) undertook a point-by-point comparison of the two data sets. This comparison emphasized those data from the mouse most nearly comparable to the human data. Unfortunately, for reasons discussed in some detail elsewhere (18), most notably the immaturity of the mouse fetus at birth and the intra-litter competition effect both before and after birth, although effects of paternal radiation on the frequency of congenital defects, stillbirths, and early survival were demonstrated in the offspring of radiated males, the data cannot be directly compared with the human data. A further reason for great care in extrapolating from mice to humans derives from all the differences between the exposure of a total population to instantaneous radiation, as in Japan, and the pattern of exposure usually used in the mouse experiments, namely, the exposure of members of a single inbred line at a predetermined age, followed by a controlled mating system in which a relatively few treated males father many offspring.

The most appropriate data for comparison with the human data would seem to be the results of the various specific locus-specific phenotype test systems. The results from eight different attempts to develop data from which such a radiation doubling dose for mice could be calculated, based on more or less specific locus (or specific phenotype) approaches, are shown in Table 1. (For present purposes, 1 Gy of radiation is for genetic purposes the same as 1 Sv equivalent.) Note the wide range in the various estimates, to which we found it impossible to attach errors in the usual statistical sense. Not shown there (because the data do not lend themselves to the calculation of a doubling dose) are the important results of Roderick (19), who estimated for mice a per locus recessive lethal mutation rate in postspermatogonial cells per locus from ionizing radiation of only 0.35×10^{-8}/0.01 Gy, whereas for the Russell 7-locus system, the corresponding rate for all postspermatogonial mutations was 45.32×10^{-8}/0.01 Gy, approximately 80% of these mutations being homozygous lethal. As Roderick pointed out, these results indicate about a 100-fold lesser sensitivity than the Russells' studies (20), although the error term to be attached to Roderick's estimate was large but difficult to calculate. The simple average of all the estimates in Table 1, unweighted because of the differing natures of the individual studies, was a male gametic doubling dose of 1.35 Gy, with an indeterminate error.

Table 1. A summary of the gametic doubling doses for acute, "high-dose" radiation of spermatogonia yielded by the various specific-locus/specific-phenotype systems developed in the laboratory mouse, after Neel and Lewis (17)

System	Doubling dose, Gy		Origin of treated males
Russell 7-locus	.44		101 × C3H
Dominant visibles	.16		Various
Dominant cataract	1.57		101/E1 × C3H/E1
Skeletal malformations	.26		101
Histocompatibility loci	>2.60		C57BL/6JN
Recessive lethals	.51		DBA
(3 studies)	.80	1.77	C3H/HeH × 101/H
	4.00		CBA, C3H
Loci encoding for proteins	.11		Various
Recessive visibles	3.89		C3H/HeH × 101/H
	Av. 1.35		

References to the sources of the data and the doubling-dose calculations will be found in Neel and Lewis (17).

There are several reasons to approach this estimate with caution. First, the data from many of the systems used in Table 1 are absolutely minimal for the generation of a doubling dose. Because of their magnitude, the data obtained by W. L. Russell at Oak Ridge (21, 22), yielding one of the lower estimates of the doubling dose, should have and did dominate the estimates, forcing us to look at them with great care. Second, Russell in his very first papers (23) recognized that the assumption that the loci he studied were representative of the genome was key. There are now data for the mouse indicating a 7-fold range in the rate per locus with which spontaneous mutation results in phenotypic effects (24, 25). In Russell's data (21), radiation produced 18 times more mutations at the s locus than at the a locus, surely a signal to extrapolate with caution (reviewed in ref. 21). Furthermore, in the test system developed by Lyon and Morris (26, 27) involving six different loci than those used in the Russell system, the radiation-induced rate was only about one-third of the rate in the Russell experiments. It is really not clear how best to treat these locus differences in spontaneous and induced mutation rates. The situation is further complicated in that the detailed analyses of L. B. Russell and colleagues (cf. refs. 28–30) reveals that the "specific locus system" is detecting events ranging from deletions of up to 11 cM, corresponding to physical lengths ranging to perhaps 20 nucleotide megabases, down to single nucleotide substitutions.

Third, the mouse doubling-dose estimates of Table 1 are male-based. The demonstration (31) that although in the first few litters posttreatment the offspring of radiated female mice exhibited about the same amount of genetic damage as the offspring of radiated male, there was no apparent damage in the later litters of these females, created a dilemma for risk setting. Was the human female similar to the mouse female in this respect? To be conservative, in extrapolating to the human situation, the mouse male-derived risks usually have been applied to both sexes. Thus, from Table 1 the zygotic doubling dose would become 2.7 Gy, but because of the lack of induced mutations in the late litters of females, this is almost certainly an underestimate of the mouse zygotic doubling dose. In the Japanese data, by contrast, radiated females contribute about half the dose on which the doubling dose estimate is based.

The fourth reason the murine-based estimate of 1.35 Gy may be conservative is the apparent omission of the observed "cluster" and "mosaic" mutations in the doubling-dose estimates derived from the Russell system. More than 30 years ago L. B. Russell (32) described some 40 specific locus mutations that in the course of the experiment at Oak Ridge occurred in the offspring of both irradiated and control mice as clusters of two or more. Of these, 21 had one irradiated parent and 19 came from a contemporary control population of slightly smaller size. It is not clear how many of these occurred in the basic 7-locus series that provided the mutation rates quoted above. More recently, Russell and Russell (20) also have described a series of some 37 mosaic mutants that appeared in the F_1 of both radiated and control mice, none of which apparently have been incorporated into the doubling-dose calculations of the past that used the Russell data. Selby (33) in a brief abstract has suggested that because of the failure to incorporate clusters into the calculations, "the size of the doubling dose has been underestimated by at least a factor of three." No similar estimate is yet available for the effect of noninclusion of the mosaic mutants, but it could be a factor of two. These clusters, apparently reflecting a relatively high mutation rate in the "perigametic—very early zygote" interval (see ref. 8), are well documented in humans and *Drosophila* and have been, by purpose or default, included in past doubling-dose estimates for these species (reviewed in ref. 34). The *Drosophila* data, however, suggest that only some 40% of all spontaneous mutations occur as clusters, so that although their omission from a calculation of the doubling dose for *Drosoph-*

ila would have biased the estimate downward, it would not be by a factor of three. From the standpoint of the population geneticist, there are both theoretical and practical reasons cluster mutations must be properly incorporated into the doubling-dose issue. First, when Mother Nature views a newly fertilized egg carrying a mutant gene not present in either parent, she (or, more technically, the process of natural selection) does not ask exactly when and how that mutation originated. Selection must reckon with the totality of all the newly arisen mutations represented in the zygote, which is what we have in effect attempted to emulate in the study in Japan. Selection does not stop to ask whether the mutation occurred as a member of a cluster. Second, although the frequency of clusters may not be altered by radiation under the special circumstances of the design of the Russell study (23), with the radiation usually delivered at the 12th week of age, in the human experience, such as the exposures from the atomic bombs or the Chernobyl disaster, exposure is to both sexes at all ages and all stages of gametogenesis or fetal development, including the period particularly susceptible to the occurrence of what will become "clustered mutations." Unfortunately, because of aspects of the design of the mouse studies, namely, the repeated use of a relatively few radiated males to impregnate many females, and the resulting favorable circumstances for the detection of clusters, the proper comparison of the mouse with the human data must be approached with care. Nevertheless, it is of some importance that the mouse data be presented in such a way that this comparison can be undertaken.

At this point in time, then, given all the difficulties in the calculations and the wide errors to be attached to these calculations, the estimates of the doubling dose of radiation for humans and mice appear to be converging. There is no theoretical reason for this agreement between two animals as disparate as humans and mice, but some nevertheless may find this agreement somewhat reassuring with respect to the validity of the conclusions from the epidemiological studies in Japan. Furthermore, inasmuch as the suggested permissible population and occupational exposures for genetic reasons, set by the Academy's Committee on the Biological Effects of Atomic Radiation in 1956, were—quite properly at the time—highly influenced by W. L. Russell's early studies (23) on mice, the adjustments suggested, as well as the studies in Japan, imply that these guidelines are even more conservative than we committee members thought at the time.

Two Recent Challenges to the Validity of the Mouse/Human Data Just Reviewed

Within the past 7 years, there have been two very well-publicized challenges to the view of the genetic risks of radiation just developed. The first was the suggestion by Gardner *et al.* (35, 36), after an extensive epidemiological study, that the previously reported cluster of childhood leukemia in Seascale, West Cumbria, England, was associated with paternal employment in the nearby Sellafield Nuclear Reprocessing Plant, a finding given a genetic interpretation. Shortly thereafter, a suit claiming damages for personal injuries was initiated on behalf of two of the individuals who had developed leukemia. The suit was filed by a well-known British law firm, Leigh, Day, and Company, and directed against British Nuclear Fuels plc, the firm that operated the plant. The suit, heard before the Royal High Courts of Justice of England, was record-breaking in its estimated costs. A verdict for the plaintiffs would have challenged all of the present guidelines concerning occupational exposures. There is no time to lead you through the intricacies of the case (for reviews cf. refs. 37–40). After an extended trial, the judge found resoundingly for the defendant. The crucial evidence in reaching this verdict was supplied by the studies in Japan, which yielded results in flat contradiction with the possibility that the increase in leukemia in Seascale could be a genetic radiation effect.

The second of these challenges is still ongoing. In 1996, Dubrova *et al.* (41) reported that the rate of mutation involving a battery of DNA minisatellites was twice as high in children whose parents had been exposed in the Mogilev district of Belarus to fallout from the Chernobyl disaster than in controls. Minisatellites are regions of DNA characterized by identical tandem DNA repeats, the repeat unit usually varying between 5 and 45 bp in length. The function of this type of DNA is unknown; it has an extraordinarily high spontaneous mutation rate. The maximum cumulative exposures to these parents from fallout can be estimated at .08 Sv equivalents of chronic radiation, and the average may be half of that. Thus, the results suggest radiation sensitivities far, far greater than observed in the Japanese studies, and has been enthusiastically hailed by the press for the new insights they provide. Fortunately or unfortunately, depending on your viewpoint, the study is badly flawed (cf. refs. 9 and 42). First, the controls are drawn from England, a violation of all the canons of design for a study of this nature. Second, the alleged effect is several hundred times greater than would be anticipated from experimental studies on minisatellites in mice (43, 44). But third, and most convincingly, these results are flatly contradicted by a study by Kodaira *et al.* (45), at the Radiation Effects Research Foundation (RERF), successor agency to the Atomic Bomb Casualty Commission, a study even now being extended. H. Mohrenweiser (unpublished work), in a preliminary study, also finds no effect of parental radiation on minisatellite mutation rates in the children of the so-called Chernobyl liquidators, in whom the radiation dose was substantially higher than for the parents reported by Dubrova *et al.* (41). Again the role of RERF in establishing a sane view of radiation risks has been underlined.

These recent episodes underscore the wisdom of continuing, or even initiating, several types of genetic studies in Japan. Chief among these is the completion of a resource for genetic studies at the DNA level. This latter undertaking, initiated some 10 years ago, involves establishing Epstein–Barr virus-immortalized cell lines organized into mother/father/child trios, some 600 with respect to which one or both parents were proximally exposed to the bombs, another 600 in which neither parent received significant radiation at the time of the bombings. It was these cell lines that already have served as the basis for the above-quoted studies of Kodaira *et al.* (45). Establishing these cell lines has been a very major undertaking.

At the moment, a variety of approaches to the efficient use of these cell lines for mutation studies is being explored. Chief among them is the application of electrophoresis to produce two-dimensional agarose gels of enzyme-digested, isotope-labeled genomic DNA. In such gels, some 2,000 DNA fragments can be recognized (46, 47). Computer algorithms have been developed to assist in the analysis of these complex patterns (48–50). A mutation would be detected as a feature of the child's gel not present in either parent. The approach should be most efficient in the detection of mutations resulting in DNA insertions/deletions/inversions.

Should the pilot studies now underway at the Radiation Effects Research Foundation and the University of Michigan concerning the potential of this system be expanded into full-scale studies, and a definitive body of data begin to emerge, then, I suggest, the question arises of whether extensive parallel experimental studies involving the mouse should be undertaken. On the one hand, it can be argued that in this situation, the proper study of humans is humans, and at this level of genetic resolution, there is no need for animal experimentation. On the other hand, almost surely some will argue the need for parallel studies on mice and *Drosophila*. Then, for the first time, society would have homologous indicators across species, the resulting data of great theoretical and practical

value. Were such studies initiated, however, it would seem desirable that the circumstances of the radiation exposure in the animal work be made much more comparable to the human exposures in Hiroshima and Nagasaki than has been the case in the past. Specifically, it would seem highly desirable that the experimental radiation doses be lower than in the past, and that the experimental breeding pattern be better approximated to that of a human population. Finally, for cross-species comparisons, it would be highly desirable that in any further experiments the radiation exposures more equally involve both sexes and a variety of life stages, rather than being concentrated, as in the past, on one sex, the male, and exposure at one brief window during the life cycle, usually the 12th week.

This brief presentation attempts to summarize the most extensive and longest running study in genetic epidemiology ever undertaken. In retrospect, it seems clear that the data the study has yielded, together with the current revisions of the murine data, have resulted in a much more rational view of the genetic risks of exposure to ionizing radiation than existed in the first several decades after the bombings. Yes, there are genetic risks in exposure to ionizing radiation, but current national and international recommendations regarding permissible exposures now can be seen as incorporating an even wider margin of safety than appeared to be the case when they were promulgated. In closing, I reiterate that whatever success the study has enjoyed has been the result of an unparalleled collaboration between scientists of two nations and, on the U.S. side, a remarkable coordination between administrative support at the Academy and the field work in Japan. And isn't it a revealing commentary on the speed of scientific advance, that when these genetic studies began, the "gold standard" for an epidemiological study such as this was frequency of congenital defect and "sentinel" phenotypes resulting from single gene mutations, now it has become, damage to DNA.

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HIGHLIGHTED ARTICLE
GENETICS | PERSPECTIVES

The Hiroshima/Nagasaki Survivor Studies: Discrepancies Between Results and General Perception

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ABSTRACT The explosion of atom bombs over the cities of Hiroshima and Nagasaki in August 1945 resulted in very high casualties, both immediate and delayed but also left a large number of survivors who had been exposed to radiation, at levels that could be fairly precisely ascertained. Extensive follow-up of a large cohort of survivors (120,000) and of their offspring (77,000) was initiated in 1947 and continues to this day. In essence, survivors having received 1 Gy irradiation (~1000 mSV) have a significantly elevated rate of cancer (42% increase) but a limited decrease of longevity (~1 year), while their offspring show no increased frequency of abnormalities and, so far, no detectable elevation of the mutation rate. Current acceptable exposure levels for the general population and for workers in the nuclear industry have largely been derived from these studies, which have been reported in more than 100 publications. Yet the general public, and indeed most scientists, are unaware of these data: it is widely believed that irradiated survivors suffered a very high cancer burden and dramatically shortened life span, and that their progeny were affected by elevated mutation rates and frequent abnormalities. In this article, I summarize the results and discuss possible reasons for this very striking discrepancy between the facts and general beliefs about this situation.

THE first (and only) two A-bombs used in war were detonated over Hiroshima and Nagasaki on August 6 and 9, 1945. Casualties were horrendous, approximately 100,000 in each city including deaths in the following days from severe burns and radiation. Although massive bombing of cities had already taken place with similar death tolls (*e.g.*, Dresden, Hamburg, and Tokyo, the latter with 100,000 casualties on March 9, 1945), the devastation caused by a single bomb was unheard of and remains one of the most horrifying events in the past century. The people who had survived the explosions were soon designated as *Hibakusha* and were severely discriminated against in Japanese society, as (supposedly) carriers of (contagious?) radiation diseases and potential begetters of malformed offspring. While not reaching such extremes, the dominant present-day image of the aftermath of the Hiroshima/Nagasaki bombings, in line with the general perception of radiation risk (Ropeik 2013; Perko 2014), is that it left the sites heavily contaminated, that the survivors suffered very serious health consequences, notably a very high rate of cancer and other debilitating diseases, and that offspring from these survivors had a highly increased rate of genetic defects. In fact, the survivors have been the object of massive and careful long-term studies whose results to date do not support these conceptions and indicate, instead, measurable but limited detrimental health effects in survivors, and no detectable genetic effects in their offspring. This Perspectives article does not provide any new data; rather, its aim is to summarize the results of the studies undertaken to date, which have been published in more than 100 papers (most of them in international journals), and to discuss why they seem to have had so little impact beyond specialized circles.

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Bombings and Implementation of Cohort Studies

Characteristics of the bombs and the explosions

The device used at Hiroshima was based on enriched uranium and exploded at an altitude of 600 m with an estimated yield

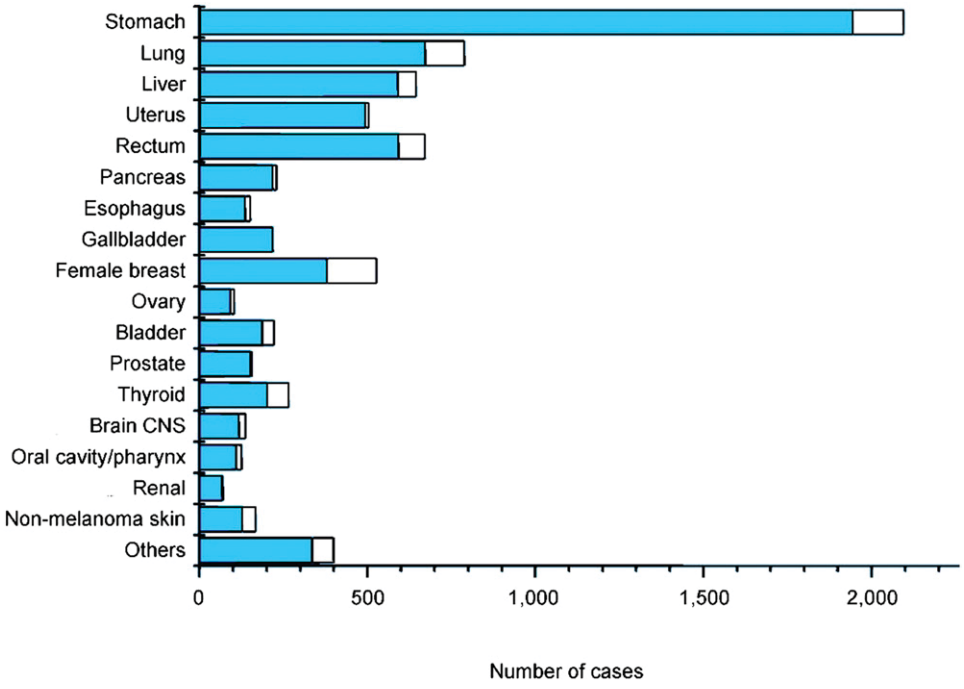

Figure 1 Number of solid cancers observed up to 1998 in the exposed group; the white portion indicates the excess cases associated with radiation (comparison with the unexposed group). Data are from Preston *et al.* (2007).

equivalent to 16 kilotons of high explosive. The bomb at Nagasaki was based on plutonium and exploded at 500 m with a yield of 21 kilotons. The major effect of both bombs was an extreme heat and pressure blast accompanied by a strong burst of gamma radiation and a more limited burst of neutrons. The heat blast set the (mostly wooden) buildings on fire in a radius of several kilometers and resulted in an extensive firestorm centered on the explosion site (also called the hypocenter). People were exposed to the combined heat and radiation blasts, with little shielding from the buildings; most of those located within 1.5 km of the hypocenter were killed. The contribution of fallout from these explosions, which occurred mostly as "black rain" in the following days, is not precisely known: few measurements were taken due to scarcity of equipment, and investigations in the first months were performed by the US army and subsequently classified. It was probably limited: the bombs exploded at a significant altitude, the resulting firestorm carried the fission products into the high atmosphere, and the eventual fallout was spread over a large area. In addition, a strong typhoon occurred 2 weeks after the bombings and may have washed out much of the materiel. The major health effects (other than the heat blast and accompanying destruction) were almost certainly due to the gamma and neutron radiation from the blasts themselves, and these doses can be quite reliably estimated from the distance to the hypocenter. Thus studies on the survivors can ascertain the health effects of a single, fairly well-defined dose of gamma radiation with a small component from neutrons.

The Atomic Bomb Casualty Commission and the Radiation Effects Research Foundation

Initial studies (1945–1946) on survivors from the bombings were performed under the authority of the occupying US army and their results remained classified; the number of delayed deaths from radiation or, possibly, contamination is therefore not known precisely, although it is probably of the order of 10,000 for each site. Open studies were started in 1947, with the establishment of the Atomic Bomb Casualty Commission (ABCC) by the US National Academy of Sciences, joined a year later by the Japanese National Institutes of Health, and including well-known geneticists such as James Neel and William Schull. It initiated extensive health studies on the survivors and was reorganized in 1975 to form the Radiation Effects Research Foundation (RERF), a Japanese foundation funded by both Japan (Ministry of Health) and the United States (Department of Energy). Both institutions have been criticized by the Japanese public for observing the victims but not providing medical assistance to them. They have, however, fulfilled an extremely useful role in establishing reliable data on radiation effects. A general description of the RERF and its activities (including references to published studies) is accessible through the RERF Web site (RERF 2014). The RERF currently employs ~170 persons at its main location in Hiroshima, as well as 50 in Nagasaki, with staff from both Japan and the United States. The ABCC and, later, the RERF, assembled a "Life Span Study" (LSS) cohort of 120,000 individuals [~100,000 exposed at various (known) levels and ~20,000 controls, "not in city" at the time of the bombings], and a cohort of 77,000 children born between 1946 and 1984 and for which at least one parent had been exposed. These have been followed now for over 60 years in most cases, and their general health, life expectancy, cancer incidence, and mortality ascertained. In addition, cytogenetic, biochemical, and molecular genetic studies have also been performed on significant subsets. The population followed represents approximately half of the people

Table 1 Observed and excess solid cancers observed up to 1998 in the exposed group, according to radiation dose

Weighted colon dose (Gy)	LSS subjects	Cancers		Attributable risk (%)
		Observed	Estimated excess	
0.005–0.1	27,789	4406	81	1.8
0.1–0.2	5,527	968	75	7.6
0.2–0.5	5,935	1144	179	15.7
0.5–1.0	3,173	688	206	29.5
1.0–2.0	1,647	460	196	44.2
>2.0	564	185	111	61.0
Total	44,635	7851	848	10.7

This is a simplified version of Table 9 in Preston *et al.* (2007), which tabulates all cancers observed from 1958 through 1998 among 105,427 LSS cohort members. LSS, Life Span Study.

who were exposed in the bombings, and the fact that they received a single dose of radiation that can be consistently estimated makes the conclusions much more reliable than in more complex situations such as the Chernobyl disaster (see later). A detailed general overview of the results as of 2011 has been published (Douple *et al.* 2011). Current results from these studies (that are still ongoing) are summarized below, first for survivors and then for their offspring.

Studies on Survivors

In both Hiroshima and Nagasaki, there was extensive mortality in the days and weeks following the bombings, representing perhaps 10% of the casualties. It is difficult to separate the effect of radiation (acute radiation syndrome, ARS) and, possibly, of contamination from the consequences of burns since most victims suffered both. Early studies, however, indicated that the median lethal dose (LD_{50}) from whole-body gamma radiation is ~2.5 Gy[1] when little or no medical assistance is available (5 Gy with extensive medical care). This estimate is based on early studies at the bomb sites, but with dose estimates refined according to later studies.

Cancer

In 1950, a survivor cohort was defined and detailed medical follow-up established. From then on, the causes of death and the excess due to radiation exposure could be ascertained. This excludes mortality caused by ARS and other bomb-related trauma, but not most delayed effects, except for a small number of leukemia deaths since this is the earliest neoplasm to appear. The results of these studies have been published in a large number of papers, mostly in specialized journals (such as *Radiation Research* or the *Journal of Radiation Research*) but occasionally in more widely read journals (*The Lancet*, the *American Journal of Human Genetics*, *Nature*, *etc.*). Figure 1 shows the solid cancer cases in the whole exposed group from the LSS survivor cohort, with the excess cases (in white) attributable to radiation (by comparison with the control group "not in town" from the same cohort). It is quite obvious from Figure 1 that there is a measurable excess of cancer cases in the exposed group, but also that this excess is relatively limited, amounting at most to an increase of ~30%, often much less.

Figure 1, however, tabulates results for the whole exposed group, most of whose members have experienced a relatively low dose of radiation: half of them received less than 0.1 Gy. It is therefore more meaningful to look at the percentage of excess cancers according to dose received, as shown in Table 1 (Preston *et al.* 2007). This time, all solid cancers are lumped together, but the cases are broken down according to radiation exposure. As expected, the fraction of excess cancers increases with radiation dose, from a nearly negligible 1.8% below 0.1 Gy to 61% at 2 Gy or above. For a quite sizeable exposure of 0.5–1 Gy,[2] the figure is 29.5%, corresponding to 206 excess solid cancer cases (all types) in a group of 3173 persons. In other words, there is a clear excess of cancer cases in strongly irradiated survivors, but this involves less than 10% of the total. It is also important to note that the excess risk is higher for people exposed at a young age, that this risk persists through the subject's lifetime, and that it is ~50% higher in women than in men (Douple *et al.* 2011).

For leukemias (Table 2), the outlook is both worse and better (Preston *et al.* 2004): worse, as the fraction of excess cases is larger (63% in the 0.5–1 Gy group), and better since, given the rarity of the disease, this translates into a much smaller number of excess cases, 19 for 3963 individuals. Leukemias also appear earlier than solid cancers, as early as 4 or 5 years after exposure; thus, some of them may have been missed in this accounting that started 5 years after the bombing.

Altogether, the picture that emerges is that, for quite heavily irradiated survivors (*e.g.*, the 0.5–1 Gy group), there is a sizeable increase of neoplasms, especially leukemia but also most solid cancers. It would be wrong, however, to assume that all survivors are hit by this disease, since even in this group the fraction affected is slightly above 20%, less than one-third of this being attributable to radiation exposure. The most recent report on the LSS cohort of survivors (Ozasa *et al.* 2012), covering the period up to 2003 (by which

[1] Throughout this paper, radiation exposure is expressed using the gray unit (irradiation resulting in the absorption of 1 joule per kilogram), which is the unit appropriate for whole-body irradiation; for low levels of radiation and taking into account the nature of radiation and the exposed tissue, the unit generally used is the sievert (mSv, millisievert). For whole-body, mostly gamma-ray exposure, the two units are roughly equivalent, *i.e.*, 1 Gy ~1 Sv = 1000 mSv.

[2] Let us remember that 2.5 Gy is the LD_{50}, and that the limit for annual exposure for the general public is 1 mSv, *i.e.*, ~1 mGy.

Table 2 Observed and excess leukemia deaths observed up to 2000 in the exposed group according to radiation dose

Weighted marrow dose (Gy)	Subjects	Deaths		Attributable risk (%)
		Observed	Estimated excess	
0.005–0.1	30,387	69	4	6
0.1–0.2	5,841	14	5	36
0.2–0.5	6,304	27	10	37
0.5–1.0	3,963	30	19	63
1.0–2.0	1,972	39	28	72
>2.0	737	25	28	100
Total	49,204	204	94	46

Data are from Preston *et al.* (2004), as reported in RERF (2014).

time 58% of the survivors had died) confirms these results while increasing somewhat the excess relative risk (ERR) associated with radiation; these results also indicate a dependence on age at irradiation, with elevated risk for those irradiated when young. Overall, the ERR for all solid cancers corresponding to a (very sizeable) irradiation of 1 Gy works out as 0.42, and the ERR/radiation dose relationship appears to be linear, with no indication of a threshold.

Other diseases and life span

Of course, cancer is not the only possible detrimental effect of radiation exposure, which could have an influence on cardiovascular diseases, autoimmune syndromes, and other ailments. Because of the size of the LSS cohort and the exceptional quality[3] and duration of its follow-up, it is possible to look at the end result, *i.e.*, the longevity of individuals according to the radiation dose received. The result is shown in Figure 2, reproduced from the 2000 Lancet paper by Cologne and Preston (2000) and showing data from the LSS cohort up to 1995. At that time, approximately half of the original cohort had died, allowing a reliable evaluation of life span. From Figure 2, it is clear that the separation between the curves is limited, even for the one that corresponds to a dose of >2 Gy. The median loss of life span at 1 Gy irradiation is 1.3 years, and decreases to 0.12 years at 0.1 Gy. Again, the effect is measurable, and follows the expected dose/effect relationship, but its magnitude is quite limited. As a comparison, note that in Russia, life expectancy decreased by 5 years between 1990 and 1994, essentially because of social disruption impacting on living conditions and healthcare (Notzon *et al.* 1998). As noted above, the survivors may have been exposed to an additional (but unknown) irradiation due to fallout from the bombs; this would lead to an overestimate of the gamma and neutron radiation effects. In other words, it would not affect the major conclusion from this section, that these effects are detectable but relatively limited even for radiation doses of the order of 1 Gy.

Studies on the Offspring of Survivors

The large cohort of children of survivors (77,000 individuals) is of particular interest: it should allow reliable estimation of detectable genetic effects resulting from parental irradiation thanks to its large size and to detailed follow-up over several decades. It must be emphasized, however, that some members of this group are still quite young: the cohort includes children born from 1946 to 1984, and the latest published results (Grant *et al.* 2015) are based on data as of December 31, 2009. Thus late events (*e.g.*, excess cancer cases) are likely to be underrepresented. In addition, the assessment of mutation rate has not yet been performed directly by whole-genome or whole-exome DNA sequencing. The indirect evaluation through examination of the phenotype (incidence of malformations, age-specific mortality) and the limited molecular data (gross chromosome aberrations, mutations at microsatellite loci) lack sensitivity to detect small increases in mutation rate or large increases of point mutations of subtle phenotypic effect.

Malformations and mutations

Within the limitations indicated above, and setting apart the case of children exposed *in utero*, who display growth deficiencies, intellectual impairment and neurological effects

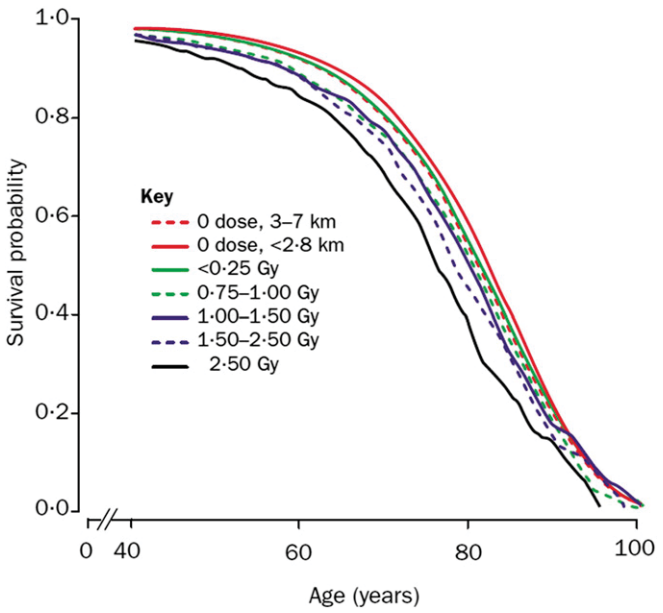

Figure 2 Survival curve (up to 1995) according to level of exposure to radiation (Cologne and Preston 2000). Note the limited separation between the curves for zero (red) and 2.5 Gy (black) exposure.

[3] Thanks to the Japanese *koseki* family registration system, only 121 individuals were lost to follow-up among the 120,321 cohort members.

Table 3 Malformation frequency at birth (including stillbirths and perinatal deaths, but not early miscarriages) in relation to parental exposure

	Father's exposure condition		
Mother's exposure condition	Not in cities (%)	Low-to-moderate doses (%)	High doses (%)
Not in cities	294/31,904 (0.92)	40/4,509 (0.89)	6/534 (1.1)
Low-to-moderate doses	144/17,616 (0.82)	79/7,970 (0.99)	5/614 (0.81)
High doses	19/1,676 (1.1)	6/463 (1.3)	1/145 (0.7)

Data are from Neel and Schull (1991). The early miscarriages have apparently not been recorded.

(Douple *et al.*, 2011), the children of survivors show no detectable radiation-related pathology. The incidence of malformations at birth does not increase if both parents have been exposed (Neel and Schull 1991; Table 3). Of course, such studies may not reveal recessive mutations that would only become apparent in subsequent generations.

In addition, all attempts to detect increases in mutation rate (looking at chromosome aberrations, blood protein changes, and minisatellite mutations at various loci) have so far given negative results (Table 4).

Naturally, this does not mean that the radiation received by parents has no genetic effect, only that this is not detectable with the techniques used: in particular, microsatellite variation may not be a reliable indicator of mutation rates. Current technology should allow much more extensive investigations using DNA sequencing, which might allow the detection of a small increase in mutation rate. The fact that samples are available from individuals whose parents have received quite diverse (but fairly well-known) doses would be a great asset to make sure that whatever is observed is actually radiation dependent. It is somewhat surprising that detailed sequencing studies have not yet been performed or, at least, reported—this may reflect both funding issues at RERF and possibly reluctance to provide samples to US collaborators. These investigations could take the form of full genome sequencing on parent/offspring trios, which would enable a more direct estimate of the mutation rates. It is true that such studies are technically demanding, as they require extremely high accuracy to eliminate false positives and to provide a true evaluation of mutation rates, but they are doable (see *e.g.*, Roach *et al.* 2010). In any case, it is clear that—contrary to popular belief—the genetic effects in humans of quite significant radiation doses (of the order of 1 Gy) seem to be small, indeed so far undetectable. This is in contrast with some animal studies: for example, gamma irradiation of BALB/c and CBA/Ca mice at, respectively, 1 and 2 Gy has been found to double the mutation frequency in their progeny (Barber *et al.* 2006), at least within expanded simple tandem repeat sequences. It thus appears that humans are less radiosensitive than mice, which makes evolutionary sense in view of their much longer reproductive life span. The mechanisms responsible for this difference, however, are not clear. It is interesting to note that a recent study (Abegglen *et al.*, 2015) found multiple copies of the TP53 gene in the elephant genome and interpreted this as a potential mechanism for cancer resistance in this large-bodied and long-lived species.

Risk of death due to cancer or noncancer diseases in offspring of irradiated survivors

A recent assessment of the risk of death among these offspring (after 62 years of follow-up for the oldest members of this cohort) (Grant *et al.* 2015) confirms these results and shows no discernible effect of the radiation dose received by parents on the risk of death either by cancer or other causes—*i.e.*, no indication of strongly deleterious health effects. More precisely, the risk of either cancer or noncancer mortality is not correlated with maternal or paternal exposures, and all hazard ratios are in the 0.9 to 1.1 range, even when the mother and/or father have received an exposure of 1 Gy. In other words, as the paper states, there is "no indication of deleterious health effects after 62 years"[4] of follow-up. It is too early to have lifespan results similar to those reported above for survivors, since >90% of the offspring were still alive at the cut-off date for the study (end of 2009), and, as already mentioned, an excess of late-appearing pathologies such as cancer may be still undetected. Nevertheless, as of today, there is no discernible effect of the parental irradiation on the health of their offspring, even for quite significant exposures of 1 Gy (~1000 mSV), to be compared with current safety standards of 1 mSv per year for the general population.

To conclude this section, the studies on the offspring of irradiated survivors have so far not demonstrated excess mutations or decrease of fitness in this group. These studies are ongoing and may eventually reveal effects that have been missed because of the relatively young age of most members of the cohort as well as the limitations of the assay methods used. In light of the data already obtained, however, these effects are likely to be very limited.

Coming back to the issue of the possible contribution of fallout to health effects in both exposed individuals and their offspring, this would—if found to be more significant than previously indicated—worsen the outcome in all cases and thus lead to an overestimate of radiation effects. This would not, however, affect the major conclusion of these studies, *i.e.*, the limited impact of significant irradiation on the longevity of survivors and the absence of detectable genetic effects in their offspring (apart from children irradiated *in utero*).

[4] For the youngest of these offspring (born 1984) the follow-up is only 25 years.

Table 4 Mutations at minisatellite loci in relation to parental exposure

	Controls (<0.01 Gy)	Exposed[a] (≥0.01 Gy)
Number of children examined	58	61
Minisatellite loci tested	1403	496
Mutations detected	39	13
Mutation rate/loci/generation	2.8%	2.6%

Data are from Kodaira *et al.* (2010). Note that the irradiation level for the exposed parents is quite high.
[a] Mean parental gonadal dose = 1.47 Gy.

What These Results Tell Us

A very clear-cut set of studies

Compared to subsequent nuclear disasters involving nuclear power stations (Chernobyl and Fukushima), the Hiroshima/Nagasaki bombings provide data that are much more clear cut and reliable. The Chernobyl disaster involved quite differentiated populations: the "liquidators" who attempted to quench the fires and dump shielding material onto the reactor, the local inhabitants, and the much larger population potentially affected by the plume of radioactive fallout. There were contributions from direct irradiation and from contamination. In addition, extensive but disordered redistribution of people took place, all in the framework of a largely dysfunctional administrative and political system. As a result there has been no exhaustive and systematic follow-up, the exact radiation exposure of most people is unknown, and the estimates of the associated health effects vary wildly (Williams 2008). The Fukushima accident also resulted in the release of large amounts of radioactivity, and in exposure of the surrounding population to a combination of irradiation and contamination (Hasegawa *et al.* 2015). Thorough follow-up studies have been initiated but uncertainties in the estimation of radiation exposure and the fact that this has been quite low (<10 mSv) for most of the exposed persons (excluding the personnel working in the nuclear facility) (Tsubokura *et al.*, 2012) will limit the possible conclusions. In contrast, the RERF studies include a large and representative population sample, rely on a fairly accurate estimation of a single irradiation dose, with a wide range of exposure within the cohort, and have been able to follow in detail this population (as well as its offspring) for more than half a century. They have, in fact, been essential to defining the legal limits for radiation exposure from nuclear activities, which are currently 1 mSv/year for the general public and 20 mSv/year for workers in the nuclear industry.[5]

The picture obtained from these extensive and careful studies is very different from the impressions that prevail in the general public and even among many scientists (Perko 2014). The general perception is that survivors from these cities were heavily affected by various types of cancer, and suffered much shorter lives as a result. While it is true that the rate of cancer was increased by almost 50% for those who had received 1 Gy of radiation, most of the survivors did not develop cancer and their average life span was reduced by months, at most 1 year. Likewise, it is generally thought that abnormal births, malformations, and extensive mutations are common among the children of irradiated survivors, when in fact the follow-up of 77,000 such children (excluding children irradiated *in utero*) fails so far to show evidence of deleterious effects (Douple *et al.* 2011; Grant *et al.* 2015). These studies should, of course, not lead to complacency about the effects of accidents at nuclear power plants, and even less with respect to the (still possible) prospect of a nuclear war, that would involve huge amounts of fallout and very large exposed populations. Nevertheless, concerning the Hiroshima/Nagasaki bombings, there is indeed a large gap between the results of careful studies backed by more than 100 scholarly publications, and the perception of the situation as seen by the general (and even scientific) public (Ropeik 2013).

Why this disparity?

This contradiction between the perceived (imagined) long-term health effects of the Hiroshima/Nagasaki bombs and the actual data are extremely striking. Part of this distortion must stem from the fact that radiation is a new and unfamiliar danger in the history of mankind, an agent that is unseen and unfelt, whose nature and mode of action are mysterious. Familiar dangers are more easily tolerated, as shown by the absence of concern about deaths due to the use of coal, whether they are direct, due to extraction activities (dozens of casualties every year in the United States, thousands in China) or indirect, through atmospheric pollution (several 100,000 premature deaths per year according to the World Health Organization). In addition, radiation is associated with the instant obliteration of two cities and 200,000 people, and with several decades during which the risk of an all-out nuclear war, either by design or by accident, was quite high and present in all minds.

On a more scientific level, the extreme sensitivity of radioactivity detection systems also plays a role. Depending on the type of radiation, a simple Geiger counter can detect radioactivity levels as low as a few becquerels (1 Bq = 1 disintegration per second) that would correspond in most circumstances to very low irradiation levels, orders of magnitude below 1 μSv/day.[6] In other words, even simple handheld counters can detect minuscule levels of radioactivity and cause alarm, even though they pose no actual danger. If detection systems for pollutants and poisons were similarly

[5] Note that annual exposure from medical devices is currently estimated at 3 mSv in the United States (Leuraud *et al* 2010).

[6] The correspondence is not trivial; it depends on the type of radioactivity considered and the geometry of the layout; the figure quoted is just an order of magnitude. A calculation tool can be found at http://www.radprocalculator.com/.

sensitive, we would realize that these molecules are ubiquitous, albeit at very low concentrations. Another contribution to anxiety has been the uncertainty about extrapolation of radiation effects toward very low doses: since these effects are only measurable for fairly high irradiation levels, they have to be estimated by extrapolation for low doses. There have been debates on this point, some arguing that there is a threshold below which no biological effect occurs (assuming that DNA repair mechanisms kick in and have ample time to repair any damage), and others asserting that very low levels over long periods are somehow more damaging than expected from linear extrapolation. Both the latest RERF studies (Ozasa *et al.* 2012) and recent very large-scale cohort studies covering 300,000 individuals working in the nuclear industry (Leuraud *et al.* 2015; Richardson *et al.* 2015) indicate that the relationship between irradiation levels and biological effects is probably linear down to zero exposure—so there *is* an effect from very low doses, even though it is very small: 10 mSv of accumulated exposure are estimated to raise the risk of leukemia by 0.002% (Leuraud *et al.* 2015). Thus even low doses of radiation entail some health risks, but the magnitude of these risks is extremely small.

Finally, the handling of recent nuclear incidents by the authorities has been particularly inept and has provided strong grounds for public distrust. The Chernobyl disaster was denied for several days by Soviet authorities while a strongly radioactive plume was being swept by winds over Eastern and then Western Europe; the French government repeatedly asserted that this plume did not spread over France, while it actually was depositing significant (but relatively harmless) amounts of radioactivity on the vegetation. More recently, the seriousness of problems at the Fukushima power station was repeatedly denied by Tepco, the company in charge of this plant, until the scope of the disaster became evident to all. The credibility of authorities over nuclear matters has become very low, and sensational news stories abound, in which irradiation levels are often expressed in microsieverts, which makes for impressive figures. Conspiracy theories argue for a massive cover-up of catastrophic health information and sometimes make their way into allegedly scientific papers (Sawada 2007; Yablokov 2009). Furthermore, there is indeed a gray area in the history of the Hiroshima/Nagasaki bombings: during the first 2 years (1945–1947), before the establishment of the ABCC (later RERF), medical studies were performed by the US army and their results were not disclosed. There may have been significant casualties in this period from the fallout and radioactive contamination that occurred in these two cities. At that time, at the beginning of the Cold War, the US military-industrial complex advocated the potential use of A-bombs as tactical weapons, and would definitely have wanted to suppress evidence of risks from fallout, in order to present them as "clean" weapons differing from conventional explosives only in their potency. Thus it is indeed possible that our knowledge on the aftermath of Hiroshima and Nagasaki is incomplete. This does not, however, affect the conclusions discussed in this Perspective article, which cover the more than 60 years following the explosions, rely on comparison of well-defined exposure groups, and show effects that are clearly related to radiation dose.

A duty to correct distortions

The tremendous gap between public perception and actual data is unfortunately not unique to radiation studies. It is easy to list a number of cases where dangers are grossly exaggerated (*e.g.*, foods from genetically modified organisms being supposedly detrimental to health, on the basis of essentially zero scientific evidence), or, on the opposite side, not recognized in spite of strong and convergent scientific evidence (anthropogenic climate change, until recently at least). Sometimes, as in the topic of this article, these misrepresentations are also present within the scientific community. These distortions can be very damaging as they skew important public debates, such as the choice of the best mix of energy generating options for the future;[7] I believe it is important to try to clear up these questions, and to disseminate widely the scientific data when they exist, in order to allow for a balanced debate and more rational decisions.

Acknowledgments

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