United States Scientific Perspectives on Radiation Risk to Populations from Large-scale Radiation Events

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Health Physics Society

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Arakawa Campus of Tokyo Metropolitan University
Richland: Home to WNP2

Columbia Generating Station: 1,170 MWe
GE BWR-5 using a Mark II containment structure
Addressing difficult challenges

- As radiation professionals, we deal with radiation emergencies.
- Our broad science backgrounds and passion for technical accuracy:
  - help us put into proper perspective the relative magnitude of an incident,
  - provide us with methods for making correct radiation measurements and analyzing data, and
  - help us to determine engineered solutions for site recovery and waste management, containing and shielding radioactivity, packaging, transportation, and disposal.
We balance our perspectives on radiation risks and benefits

Our scientific perspective of radiation risks and health detriment, judged against the societal benefits of nuclear technologies, engenders credibility, and facilitates effective communication when dealing with government officials, the news media, and the public.
The principal issue today: risk

- What is the shape of the dose-response function at low doses and for low dose-rates?
- How does our understanding of dose-response functions translate to assessment of individual radiation risk?
Stated in other ways

- Is there a “safe” level of radiation exposure?
- Is there a safe level “below regulatory concern”?
- Are small amounts of radiation more beneficial than harmful? What does it mean for emergency response?
Related issues

- Dose and dose-rate effectiveness factor
- Adaptive response and other risk-modifying factors
- Risk coefficients (cancer induction)
- Deterministic effects: dose to lens of eye
- Correct use of effective dose
- Emergency response guidelines
We do not know the actual risks at very low levels.
Adaptive response to low-dose radiation

• As with some chemical agents, low doses of ionizing radiation (1 cGy) can induce mechanisms whereby cells become better able to cope with subsequent exposures to high doses (1.0 Gy) of radiation

Example: Chromosome aberrations in cultures of human lymphocytes were reduced by 50%

At very low doses, the transformation frequency is below that predicted by linear extrapolation.
Inhaled plutonium-239 oxide in beagle dog lungs


\[
\hat{Y} = 2.39 \times 10^{-3} (X) - 0.026, \quad r = 0.98
\]

Slope: 2390 lung tumors/\(10^6\) dog cGy
Leuraud, Richardson, Cardis et al., Lancet Haematol online June 22, 2015

ERR = 2.96 Gy$^{-1}$
- Negative for lymphoma and CLL

**Figure:** Relative risk of leukaemia excluding chronic lymphocytic leukaemia associated with 2-year lagged cumulative red bone marrow dose

The lines are the fitted linear dose–response model and the shading represents the 90% CIs.

**Interpretation** This study provides strong evidence of positive associations between protracted low-dose radiation exposure and leukaemia.
A major (but simple error) occurred in the publication by K. Leuraud et al. in *Lancet Haematol*. 2015):

The authors failed to consider the increase in medical radiation dose (e.g. from CT scans) that occurred during 1944-2005, the period of the study.

It was during this period of time that medical exposures increased and occupational exposures decreased.
Some problems with the linear no-threshold hypothesis

- The BEIR VII linear model assumes that the risk from instantaneous high dose rates from the Hiroshima/Nagasaki bomb data (gamma rays plus neutrons) is equivalent to the risk from protracted low-dose-rate, internally deposited (beta plus gamma-emitting) radionuclides.
- The linear no-threshold hypothesis may not hold for very low doses of radiation.
- An increasing dominant body of scientific evidence contradicts the linear non-threshold model.
Some problems with the linear no-threshold hypothesis (continued)

• The risk coefficients obtained therefrom have high uncertainty at very low doses

• Cancer is a low-probability event, so the linear model cannot predict individual risk

• Actual risks vary with cell type, biological end-point, radiation quality, age and sex, and competing risks

• The LNT hypothesis does not permit one to define what constitutes a “safe” radiation exposure

Conclusion: We do not know the actual risks of cancer at very low doses
WASHINGTON — A new report by the conservative Capital Research Center (CRC) indicates that the U.S. government’s policy on radiation exposure is premised upon “irrational fear” and “discredited science,” and denies Americans easy access to technology that could reduce the cost of electricity. The Green Watch report from CRS, entitled, Fear Itself, was distributed to conservative activists at a background briefing near Capitol Hill this morning. Called the “Linear No Threshold” theory by the Environmental Protection Agency (EPA), the Occupational, Safety and Health Administration (OSHA) and allies on the environmental Left, the theory posits that “any tiny amount” of radiation will kill “some number” of people per every million or billion exposed. “The idea, in essence, is this,” the June 2015 report indicates, “If 100 aspirins would kill the average person, then the same person would be killed by taking 100 aspirin at the rate of one a day for 100 days, or, if one day, 100 people each took one aspirin, then one of those hundred people would die.”

The theory was put forth in the 1930s by a geneticist, Hermann Muller, who was an ardent socialist, and was guilty about working on the Manhattan Project to create the atomic bomb. Muller argued that there is “no” safe level of radiation exposure, an argument later exposed by researchers as a “lie,” according to the report.
Problems with radiation regulation using the linear no-threshold hypothesis

- Regulatory systems are “linear” and cannot accommodate any other model
- Primary and secondary standards for radiation protection are based on the linear model
- Regulatory systems assume that the linear model holds exactly at low doses, whereas substantial scientific evidence shows that this is not the case
- We cannot accurately measure, assign, and record radiation dose at low (background radiation) levels
Recent U.S. citizen petition for rulemaking

June 23, 2015. Federal Register

The Nuclear Regulatory Commission received three petitions for rulemaking requesting that the NRC amend its “Standards for Protection Against Radiation” and change the basis of those regulations from the Linear No-Threshold (LNT) model of radiation protection to the radiation hormesis model.

The radiation hormesis model provides that exposure of the human body to low levels of ionizing radiation is beneficial and protects the human body . . .
Dose and dose-rate effectiveness factor

A correction factor (DDREF) has been proposed to convert risk determined at relatively high doses and dose rates to risk at low doses (less than 100 mGy) and low dose-rates (less than 5 mGy per hour)

DDREF = 2.0 (ICRP Publication 60, 1991)

DDREF = 1.5 (BEIR VII Report, 2006)

DDREF = 1 to 2 (NCRP Report 171, 2012)

We do not know the DDREF with confidence!
Dose and dose-rate effectiveness factor


**FIG. 5.** Excess relative risk per Gy (ERR/Gy) for all solid cancer for selected dose ranges. The figure shows the ERR/Gy and 95% CI for a dose range from zero to a given dose based on the linear model for the full data that allowed for different ERRs below and above the given dose and taking radiation effect modifiers as common to the two dose ranges. The increased ERR/Gy in the low-dose levels less than 0.1 Gy corresponds to the estimates of ERR higher than the expected linear line in Fig. 4.
“The Dose and Dose-rate Effectiveness Factor (DDREF) is defined by dividing the slope of a non-linear function at low dose levels by the slope of the extrapolated linear non-threshold function . . . So this upward curvature may imply a DDREF greater than one.”
“The apparent upward curvature appears to be related to relatively lower than expected risks in the dose range 0.3 - 0.7 Gy, a finding without current explanation.”

Prospective risk assessments

- Cancer risk coefficients may be obtained directly from the dose-response function.
- It is a common practice to use the risk coefficients for predicting radiation risk in other populations exposed to:
  - environmental exposure to radiation sources
  - radiation at nuclear power plants
  - medical diagnostic radiological exams

If the dose-response function at low dose is highly uncertain, then what is the value of a prospective risk assessment derived therefrom?
**Nominal risk coefficients x 10^{-2} Sv^{-1}**

ICRP Publication 60, 1990 (cancer mortality)  
ICRP Publication 103, 2007 (cancer incidence)

<table>
<thead>
<tr>
<th>Population</th>
<th>Cancer</th>
<th>Heritable</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICRP 103</td>
<td>ICRP 60</td>
<td>ICRP 103</td>
</tr>
<tr>
<td>Whole</td>
<td>5.5</td>
<td>6.0</td>
<td>0.2</td>
</tr>
<tr>
<td>Adults only</td>
<td>4.1</td>
<td>4.8</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Calculation: person•Sv x risk (10^{-2} Sv^{-1}) = expected cases in a population
## Implied cancer risk*

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Effective Dose (mSv)</th>
<th>Risk (4x10^{-2} Sv^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head CT</td>
<td>2</td>
<td>1/12500</td>
</tr>
<tr>
<td>Abdominal CT</td>
<td>10</td>
<td>1/2500</td>
</tr>
<tr>
<td>Heart CT angiography</td>
<td>20</td>
<td>1/1250</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>0.1</td>
<td>1/250,000</td>
</tr>
<tr>
<td>Upper gastro-intestinal fluoroscopy</td>
<td>5</td>
<td>1/5000</td>
</tr>
<tr>
<td>Angioplasties</td>
<td>5</td>
<td>1/5000</td>
</tr>
<tr>
<td>Stents</td>
<td>40</td>
<td>1/625</td>
</tr>
<tr>
<td>Diagnostic arteriography</td>
<td>7</td>
<td>1/3571</td>
</tr>
<tr>
<td>Cardiac percutaneous intervention</td>
<td>23</td>
<td>1/1087</td>
</tr>
<tr>
<td>F-18-Fluorodeoxyglucose</td>
<td>14</td>
<td>1/1786</td>
</tr>
<tr>
<td>Tc-99m-MDP bone scan</td>
<td>6.3</td>
<td>1/3968</td>
</tr>
</tbody>
</table>

*not for individual risk analysis
Comparable risks: Lifetime risk of death from everyday activities

<table>
<thead>
<tr>
<th>Activity</th>
<th>1/risk</th>
<th>Examination</th>
<th>1/risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Riding in a car</td>
<td>304</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crossing the street</td>
<td>652</td>
<td>CT abdomen (10 yr)</td>
<td>1600</td>
</tr>
<tr>
<td>Choking on food</td>
<td>894</td>
<td>CT abdomen (40 yr)</td>
<td>2900</td>
</tr>
<tr>
<td>Drowning</td>
<td>1127</td>
<td>¹⁸FDG-PET (10 yr)</td>
<td>1515</td>
</tr>
<tr>
<td>Falling down stairs</td>
<td>2024</td>
<td>¹⁸FDG-PET (40 yr)</td>
<td>2700</td>
</tr>
<tr>
<td>Riding bicycle</td>
<td>4734</td>
<td>⁹⁹mTC-MDP (10 yr)</td>
<td>2560</td>
</tr>
<tr>
<td>Airplane crash</td>
<td>7058</td>
<td>⁹⁹mTC-MDP (40 yr)</td>
<td>4760</td>
</tr>
<tr>
<td>Hit by lightning</td>
<td>84,388</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusion: It is not appropriate to extrapolate cancer risks obtained from one organ or tissue, biological end-point, dose rate, and cancer type to another organ or tissue, end-point, or cancer type

“Misuse of the linear no-threshold model for predicting radiation effects in exposed individuals and populations should be discontinued.”

-- Siegel JA and Stabin MG, 
Health Physics 102(1):90-99; 2012
Equivalent dose, $H_T$ (ICRP 60)

$$H_T = \sum w_R \, D_{T,R}$$  
(to an organ or tissue)

where $D_{T,R}$ is the absorbed dose (averaged over a tissue or organ $T$) due to radiations of type $R$, and $w_R$ is the radiation weighting factor

- $D_{T,R}$ can not be measured experimentally
- The weighting factor adjusts absorbed dose for biological effectiveness

Unit: J kg$^{-1}$ (not a physical quantity)
Special name is sievert (Sv)
Effective dose, $E$ (ICRP 60)

\[ E = \sum w_T H_T = \sum w_T \sum w_R D_{T,R} \]

where $w_T$ is a tissue weighting factor to adjust for total detriment to health

- Effective dose represents a doubly-weighted surrogate of risk based on energy imparted multiplied by two correction factors

Unit: J kg\(^{-1}\)  (not a physical quantity)
Special name is sievert (Sv)
Problems with the concept of effective dose

• Represents a “surrogate” of risk; it is not a physical quantity
• Not a good measure for retrospective risk analysis
• Designed only for establishing radiation protection standards
• Presupposes validity of the linear model
• Applies only to an age-averaged, sex-averaged (male plus female), region-averaged population group

✓ Does not apply to individual medical patients
✓ Cannot be used to predict individual risk
✓ Cannot predict population risks at low doses
Deterministic effects

- Tissue reactions (ICRP 2012) due mainly to cell killing
- Examples: radiation skin burns, ulceration, necrosis
- Characterized by a clinical threshold dose
- Severity increases with dose
- Effects may appear from minutes to weeks
- Chronic changes (over months) may be due to fibrosis and small blood vessel narrowing or occlusion leading to necrosis
- Sensitivity and expression vary with age at exposure (UNSCEAR 2013)
Dose to lens of eye

Vision-impairing cataracts may occur after high radiation doses (more than 5 Gy) to the lens of the eye.

Radiation cataracts may sometimes be differentiated from senile cataracts by first appearing in the posterior aspect of the lens.
Dose to lens of eye

- The ICRP recommended a large reduction in dose limits to the lens
  - Previous limit: 150 mSv
  - New limit: 20 mSv per year averaged over five consecutive years, and 50 mSv in any single year

Is this change in dose limits justified?
Position of the Health Physics Society and NCRP Scientific Committee 1-23

- It is not clear that the proposed ICRP dose limit is justified by scientific data
- Recent data on threshold appearance (0.5 Gy) apply to lens opacities, not cataracts
- Supporting studies lack robust dosimetry
- Dose fractionation alters the biological response
- Cataracts may be corrected surgically (and does not represent a life-threatening risk to health)
- New dose limits will impact ability of radiologists to perform essential medical procedures

Conclusion: the change is premature and not based on sufficient scientific evidence
Emergency response

- *Effective dose* is not an accurate predictor of future risk of cancer for individuals or populations.

- Emergency response actions should **never** be based on statements involving *estimated future risk of cancer* to workers or to the public.

- Regulators need to begin thinking in terms of “safe” or “not harmful” dose levels.
Thank you!

I have enjoyed meeting with the Japan Health Physics Society

Kanazawa Park 2014