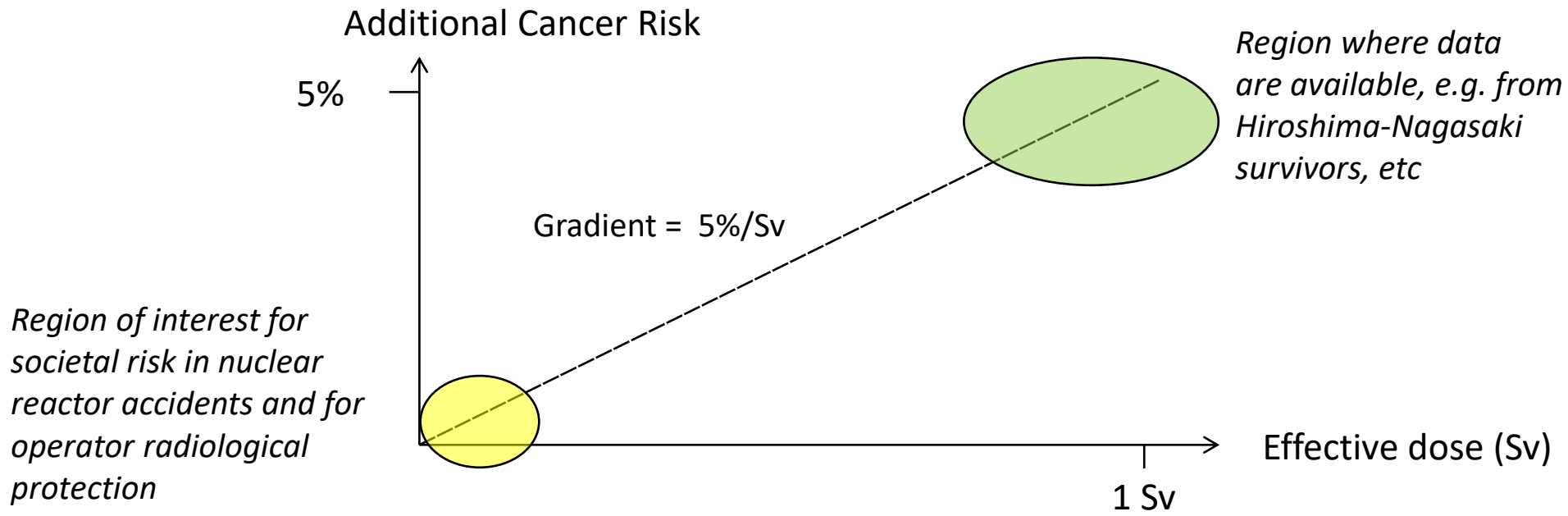


# Linear No Threshold (LNT) to be reconsidered?

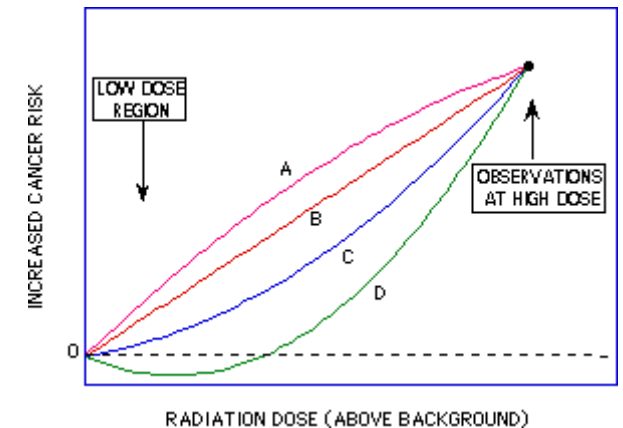
Jim Thomson, [www.safetyinengineering.com](http://www.safetyinengineering.com) , May 2023

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*More than 30% get cancer anyway, hence 'signal to noise' problem at low doses.  
This also makes disproving LNT by empirical methods very difficult, e.g. 'megamouse' experiments.*

**Delayed health effects (cancers) –**  
**Linear No Threshold (LNT) hypothesis**





### **Hermann Muller 1890-1967**

US geneticist, Nobel Prize for Physiology and Medicine 1946 for discovery of X-ray mutagenesis.

Primary advocate of LNT, following fruit fly/X-ray experiments published in 1926. (He was also a eugenicist.)

LNT became the accepted basis of radiological protection in the late 1940s, under Muller's leadership.

**Muller's 1926 findings of X-ray mutagenesis had been published in the journal Science without accompanying data and thus avoided the peer review process. Muller nonetheless received great acclaim and created a false narrative that he had produced gene mutation in fruit flies. It took some 30 years before Muller was forced to publicly admit that he was wrong but only after he had received the Nobel Prize some 10 years earlier.**

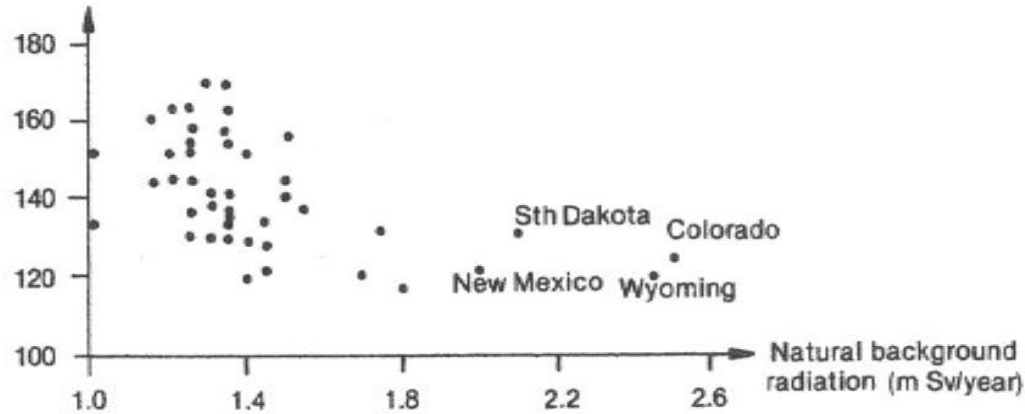
*The History of the Linear No-Threshold Model and Recommendations for a Path Forward*,  
John Cardarelli II, Barbara Hamrick, Dan Sowers, and Brett Burk  
Journal of Health Physics, 2023, pp131-135 DOI: 10.1097/HP.0000000000001645

- The International Commission on Radiological Protection (ICRP) has embarked on a mission to review the entire system of radiation protection.
- “The scientific community made a fundamental mistake on the nature of the dose response curve—a mistake built on scientific errors, profound bias, professional self-interest, and scientific misconduct.”
- “Some members of the radiation genetics community had a vested interest in maintaining this ideology so that they would continue to receive research funding.”
- “Muller misled the Nobel Prize audience in his December 1946 acceptance lecture by stating there is ‘...no escape from the conclusion that there is no threshold dose....’. He based this on the flawed and, once again, non-peer reviewed research of one of his Ph.D. students while ignoring a key threshold finding of a then-recently completed study by Ernst Caspari.”

## Some evidence against LNT

Annual malignant mortality rate per 100,000

Empirical (*one example of many*)



(Frigerio et al., 1973)

Fig. 5.25 Malignant mortality rate for USA white population 1950–67, by state and natural background radiation

(excludes radon effects)

*No examples of mutagenesis amongst Hiroshima -Nagasaki survivors have been discovered.*

Theoretical

Over the last 20 years, DNA repair mechanisms have been discovered. These undermine the theoretical basis for LNT, which was a 'one-hit' model of DNA damage.

With DNA repair mechanisms now well established, **a theoretical basis for a radiation threshold for carcinogenesis can be proposed.**

*See 'Additional Material' at end of presentation for details of DNA repair mechanisms.*

### The way forward? (as proposed by Cardarelli et al.)

- Re-evaluate the regulatory paradigm of “as low as reasonably achievable” (ALARA) to determine its merit on a scientific basis; and
- Harmonize radiation protection by considering all dose response models and applying the LNT model only to the point where adverse health effects are observed (e.g., above 100 mSv or 50 mSv y<sup>-1</sup>).

### Potential Implications for Nuclear Engineering????

- Public acceptability of changing from current standards?
- The death of ALARP?
- Design (shielding, decommissioning, radwaste, etc)
- Accident studies (e.g. Chernobyl “thousands” of deaths becomes <100)
- Operational dose management
- Protection system requirements?

## 1. Current UK annual operational exposure limits for employees

	<u>BSL</u>	<u>BSO</u>	
Radiation classified workers	20	1	mSv/yr
Other employees	2	0.1	mSv/yr

BSL = Basic Safety Limit

BSO = Basic Safety Objective

ALARP = As Low As Reasonably Practicable

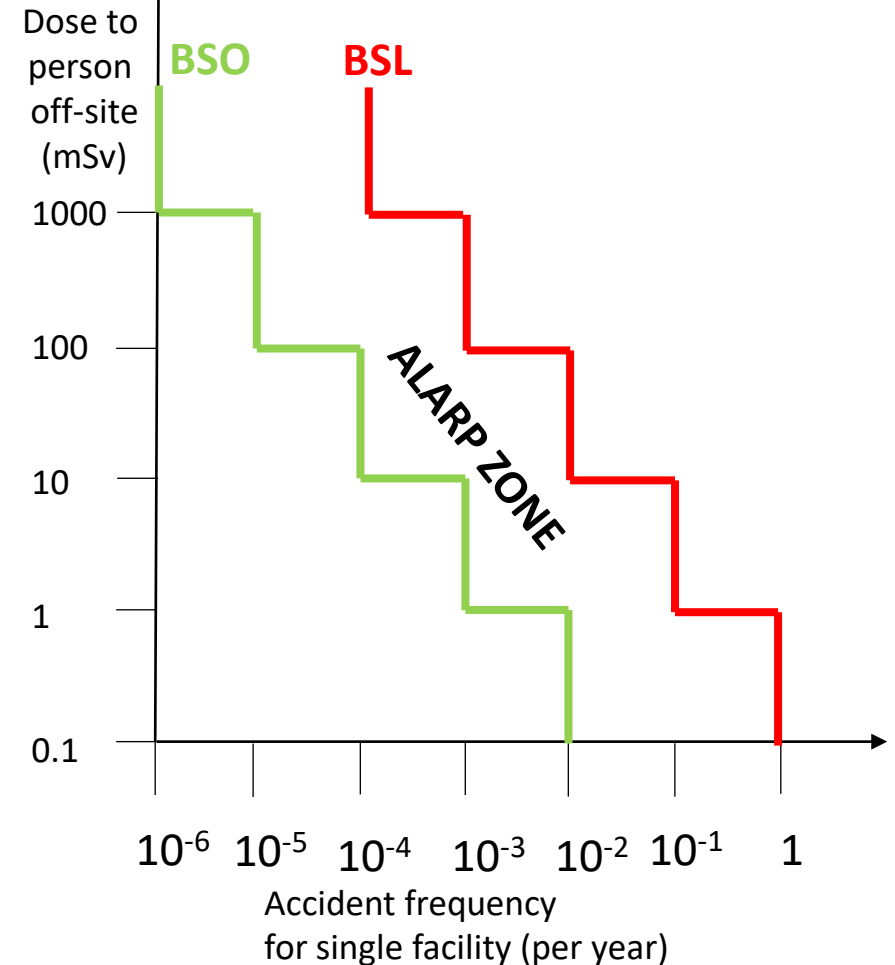
## Potential effects of Cardarelli et al proposals:

1. Increased annual operational exposure limits (to 50 mSv/yr).
2. Increased frequency targets for all accidents <100 mSv.
3. Removal of ALARP requirements below these dose limits.

# Current UK radiological exposure limits for nuclear facilities, and some potential effects of possible revised regulations

Source: ONR SAPs (2020 ed)

## 2. Frequency-dose targets for accidents (single facility, persons off-site)



In addition:

10<sup>-6</sup>/yr = BSO for accidents with >100 fatalities

10<sup>-5</sup>/yr = BSL for accidents with >100 fatalities

## ADDITIONAL MATERIAL

Chat GPT response to “Please describe the principal mechanisms of DNA repair following damage, for example due to ionizing radiation”, 3<sup>rd</sup> March 2023

DNA damage can occur due to various reasons such as exposure to ionizing radiation, chemical mutagens, or errors during DNA replication. To maintain the integrity of the genome, cells have evolved several mechanisms of DNA repair, including:

1. Base Excision Repair (BER): This mechanism repairs single base damage that occurs due to chemical mutagens, reactive oxygen species or spontaneous damage. In BER, a specific DNA glycosylase enzyme recognizes and cleaves the damaged base, creating an apurinic/apyrimidinic (AP) site. An AP endonuclease cleaves the DNA strand on either side of the AP site, and the resulting gap is filled with the correct base by DNA polymerase and ligase enzymes.

2. Nucleotide Excision Repair (NER): NER repairs damage caused by bulky lesions, such as those induced by UV radiation or chemical mutagens. In NER, a complex of proteins scans the DNA for distortions in the double helix, cleaves the DNA strand on either side of the damaged site, removes the damaged segment, and then fills in the gap with the correct nucleotides.

3. Mismatch Repair (MMR): MMR repairs errors that arise during DNA replication, such as the misincorporation of an incorrect base into the newly synthesized strand. MMR relies on the recognition of the mismatched base by a complex of proteins, followed by the excision of the incorrect base and its replacement with the correct base.

4. Double-strand Break Repair (DSBR): This mechanism repairs the most severe form of DNA damage, which occurs when both strands of the DNA molecule are severed. DSBR involves two main pathways: Homologous Recombination (HR) and Non-Homologous End Joining (NHEJ). In HR, the undamaged homologous chromosome is used as a template to repair the break, resulting in error-free repair. In contrast, NHEJ directly ligates the broken DNA ends, resulting in the loss or gain of nucleotides at the repair site, which can lead to mutations.

In summary, the mechanisms of DNA repair are complex and involve a variety of proteins and enzymes. The choice of repair mechanism depends on the type of DNA damage, and the accuracy of repair is critical for maintaining the integrity of the genome and preventing the onset of disease.