

# LNT—The Guide for the Perplexed

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## 1. What is the Linear No-Threshold (LNT) Hypothesis?

First, what is a hypothesis? It is a proposition tentatively assumed in order to draw out its logical or empirical consequences and so test its accord with facts that are known or may be determined. It appears to be a condition of the most genuinely scientific *hypothesis* that it be ... of such a nature as to be either proved or disproved by comparison with observed facts.<sup>1</sup>

Second, why is the LNT hypothesis of interest in nuclear technology and medical applications of radiation? By a very unscientific process, the US National Academy of Science (NAS) adopted the LNT hypothesis in 1956 for assessing the excess risk of cancer from ionizing (nuclear or x-ray) radiation. The NAS decided to recommend a linearity dose-response policy for assessing risks to genetic material (DNA molecules) from radiation, replacing the threshold dose-response model. This formal recommendation initiated a series of advisory and regulatory dominoes in essentially all countries to adopt linearity and apply it to somatic late appearing effects, that is, cancer risk assessment, for radiation and later for chemical carcinogens (Calabrese 2009; 2013a; 2013b). The LNT hypothesis links any radiation exposure, no matter how small, to an increased risk of cancer.

Third, what is the LNT hypothesis? This idea assumes the risk of cancer death is proportional to the radiation dose over the full range from high to *zero dose*. The Life Span Study (LSS) of the LSS group of 86,611 Hiroshima-Nagasaki atomic bomb survivors (Ozasa et al. 2012, Table 9) identified several hundred excess cancer deaths<sup>2</sup> among several thousand who received high radiation doses (above 1 Gy or 100 rad). Plot the high-dose excess cancer deaths<sup>3</sup> (ordinate) on a graph against radiation dose (abscissa). Then fit a straight line, from zero dose, to the high-dose data. The LNT hypothesis predicts that the incidence of excess cancer death at any dose in the low-dose range is given by the ordinate on this straight line, corresponding to that dose. The concept of linear risk vs. dose leads to the idea of adding the small radiation dose received by each person in a large population, in a low radiation area, to evaluate the "collective dose." This is multiplied by a fatal cancer risk factor to predict the number of cancer deaths in this population due to the radiation exposure. Renowned radiation biologist Ron Mitchel (2007) has pointed out that a fundamental principle of radiation protection, the assumption of a linear dose response and dose additivity, is incorrect.

## 2. What is wrong with using the LNT hypothesis to predict cancer risk?

First, there is no statistically significant data that supports the use of this hypothesis to predict cancer risk at low dose, which is why it is still a hypothesis 58 years after it was adopted. The LNT hypothesis is employed to calculate hypothetical risks. It creates uncertainty and great fear about potential cancer risks from low radiation doses.

Second, there are enormous amounts of data on the biological effects of: a low radiation dose, repeated doses of radiation, and low radiation levels, which contradict the predictions of the LNT hypothesis (Cuttler 2013; 2014). These data were recorded over more than 115 years, from the

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<sup>1</sup> <http://unabridged.merriam-webster.com/>

<sup>2</sup> There are many non-radiation confounding factors that affected their cancer mortality.

<sup>3</sup> The statistical and other uncertainties of the low-dose data is large.

late 1890s until the present time. Compliance with the requirements of The Scientific Method should have led the NAS to reject the LNT hypothesis instead of adopting it in 1956.

Third, it continues to be defended as being a "conservative" means of radiation protection by requiring the minimizing of radiation exposures to as low as reasonably achievable (ALARA). This policy has led to precautionary measures, such as emergency forced evacuations, which cause many premature deaths and enormous psychological suffering due to fears of cancer.

### 3. How then does ionizing radiation produce health effects?

Feinendegen et al. (2013) point out that all living organisms possess very powerful adaptive protection systems that repair or remove cell, tissue and organ damage, and restore organism health. Radiation is one of the stressors that modulate the protection systems; high radiation impairs protection, while low radiation up-regulates many protection systems (>200 genes) that act to produce very important positive health effects over extended periods of time, including a *lower* incidence of cancer. This is the mechanism for the significant net beneficial effects of low doses even below ~200 mSv or 20 rem. At higher doses, additional protective mechanisms against cancer development operate.

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