

RESPONSE TO BEYEA

Dear Editors:

THIS LETTER rebuts Beyea's letter above, which offers a series of alternative interpretations to those in my article in *Health Physics* (Cuttler 2016). New evidence is presented that supports the need for urgent change in radiation protection policy. My letter identifies errors, omissions, and misleading information in the Beyea letter, which leads to incorrect conclusions and an invalid general perspective.

Contrary to the context given in Beyea's letter, my article is the summary of my invited presentation at the 2015 HPS Symposium on Health Risks from Low Doses and Low-Dose Rates of Ionizing Radiation, which was organized by Ludwig Feinendegen. The purpose of the symposium was to address "a most serious controversy of public concern ... from wide-ranging fear of exposures to low levels of ionizing radiation... This fear developed over decades, largely out of misinformation and controversies among scientists, and was also ideologically motivated. The current situation is paralyzing socioeconomic progress, medical applications, research and development, and the use and control of nuclear power" (Brooks et al. 2016).

Rebuttal of Beyea response

Contrary to Beyea's statement, my presentation was not about a paradigm; it contained a considerable amount of medical and biological facts and data, which should be the basis for the regulations that protect us against harm. However, protection policy contradicts the data and paralyzes all progress. The threshold for radiation-induced leukemia in humans was specified (Cuttler 2014), which Beyea did not recognize. A better discussion of the basis for this threshold appears in Cuttler and Welsh (2015).

Beyea attempts to discredit the very serious charges against the NAS of the 1950s by Calabrese (2015) without delivering specific challenges about any of the official transcripts of the meetings and the many signed items of correspondence that Calabrese listed, and does not challenge any of the analyses and conclusions. Instead, Beyea states that no one should believe what Calabrese has written until they read the rebuttals of Cicerone and Crowley (2014)

and Crowley et al. (2015). Beyea omits mentioning that this Cicerone and Crowley response did not address the factual basis of Calabrese's numerous assertions; it merely tries to defend the reputation of the NAS, led by Cicerone. Nor does he mention the Calabrese rebuttal (Calabrese 2014). Beyea does not clarify that the Crowley et al. rebuttal is totally unrelated to the Calabrese historical assessment; it only defends the BEIR VII report. Thus, this statement by Beyea is misleading in implying there was factual criticism of the Calabrese historical assessment when there was none.

There are no theories; the stated activities and decisions of the 1950s actually happened. Beyea's "most detailed" letter (Beyea 2016), which offered a series of alternative interpretations, has elucidated a 13,000-word rebuttal from Calabrese (2016). In this rebuttal, "Significant newly uncovered evidence is presented which supports and extends the findings of Calabrese (2015), reaffirming the conclusion that the Genetics Panel should be evaluated for scientific misconduct for deliberate misrepresentation of the research record in order to enhance an ideological agenda. This critique documents numerous factual errors along with extensive and deliberate filtering of information in the Beyea letter (2016) that leads to consistently incorrect conclusions and an invalid general perspective.

Beyea and many others ignored the very important data of Ogura et al. (2009), which reveal a threshold for excess fruit-fly mutation frequency at about 1 Gy. In my presentation and the references in my HPJ summary (Cuttler 2016), I focused on the extensive human and animal data that demonstrate beneficial health effects following low doses of radiation.

While radiation protection endeavors to estimate hypothetical cancer risks, the best statistic to study is radiation's effect on longevity. Continuous low level exposure and multiple low doses extend lifespan by upregulating adaptive protection systems (Cuttler 2013; Cuttler and Feinendegen 2015). Low doses of therapeutic radiation induce an anti-inflammatory phenotype, which is believed to mediate many of the clinical benefits associated with radiation treatments.

Yes, there are many epidemiological studies that try to link low radiation exposures to an elevated risk of cancer. Cancer incidence is likely affected by many confounding factors, which are neither controlled nor taken into account in most studies. Many data are not statistically significant. They are usually fitted by a linear function of radiation dose, and the analysis proceeds in a circular manner to demonstrate that cancer incidence fits the LNT model. Often, approaches and tricks are used, such as "dose lagging" or combining low-dose ranges, to conceal evidence of a

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threshold or of a reduced risk (Scott et al. 2008; Scott 2008). Such epidemiological studies sustain unwarranted fears and doubts.

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