Commentary

Regulatory Application of the LNT Hypothesis and ALARA to Protect Radiosensitive People Is Misguided

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Advocates of the linear no-threshold (LNT) hypothesis assume that low-dose radiation exposure must be associated with some risk, justifying regulations that “protect” us from that risk. However, the LNT hypothesis is incorrect because it does not recognize the well-established evidence of radiation damage repair and elimination (Siegel and Welsh 2016). There is an abundance of biological evidence that a more plausible outcome or effect associated with low-dose exposure is a health benefit (Siegel et al. 2016). Nevertheless, even though many now admit that the LNT hypothesis is unfounded, it is still considered a valid basis for regulatory purposes, in part because its use supposedly protects radiosensitive people. Radiosensitive people may be thought of as those with genetic propensities to develop cancer at a greater rate than that of the general public, presumably due to family genetics or to mutation(s).

Cellular and in vivo animal data indicate there is a low-dose threshold that must be exceeded before an organism’s protective adaptive responses become functional (Mitchel 2010). These responses mitigate and/or eliminate low-dose, radiation-induced damage, as well as any spontaneously occurring damage due to natural and normal metabolic processes; the result is a beneficial outcome. One study, for example, has indicated that this low-dose threshold is likely higher if the TP53 gene is mutated than if the gene is normal, thereby increasing radiosensitivity in such individuals (Mitchel et al. 2008). As many as 50% of all human tumors contain TP53 mutants (Vogelstein et al. 2000). If an organism is not permitted to get the radiation exposure necessary to exceed this lower threshold because of the LNT-spawned, as-low-as-reasonably-achievable (ALARA) doctrine mandated in radiation regulations, the much more likely result will be harm and increased risk, not health benefit.

Radiosensitive individuals likely incur the same amount of radiation damage as those who are not particularly radiosensitive, but they have a higher cancer incidence and mortality because their adaptive protection systems are not fully functional at lower radiation exposures. In other words, their DNA repair mechanisms are sluggish. It is likely that these individuals have a low-dose threshold that may be significantly higher than in normal individuals (Mitchel 2010), and that threshold must be exceeded to more fully activate their protective systems and result in a beneficial health effect.

At least some of the genetic mutations found in radiosensitive people may affect protective adaptive responses, such as mutated TP53 genes that may reduce apoptosis (cellular self-destruction) of unrepaired abnormal cells. When damaged or abnormal cells, produced as a result of natural metabolic processes (oxygen metabolism) or induced to a much lesser extent by low-dose radiation exposure, are not repaired or removed by normally functioning adaptive protection mechanisms, they may go on to produce a cancer. Keeping radiation exposures ALARA will therefore more likely fail to maximally stimulate a radiosensitive person’s cells, compared to a normal individual’s cells with their innate protection mechanisms. So these individuals would exhibit “hyporepairability” rather than “hypeerradiosensitivity,” thereby rendering the ALARA concept useless and harmful.

It has been reported that African American women are more likely to die as a result of breast cancer than white women, and that these women had more TP53 gene mutations (Keenan et al. 2015). This may be only associative and not causative, but we suggest that a portion of the breast cancer incidence in African American women may be due to their increased radiosensitivity (hyporepairability). If true, the LNT hypothesis, as well as its inseparable companion, the ALARA principle, might have a greater detrimental effect on African American women than on white women.
To maximally protect radiosensitive (i.e., hyporepairing) people, we need to maximally stimulate their adaptive protection mechanisms. This would paradoxically involve a yet-to-be-defined, low-dose radiation exposure in the beneficial (hormetic) range that would mitigate the damage and lessen the threat of cancer incidence. Notably, the existence of a hormetric range is not only applicable to exposure to radiation but also to any and all physical and chemical stressors (i.e., a stressor-specific “Goldilocks range” of too little, just right, and too much). The mandated ALARA practice is also likely causing more cancer in normal people when carried to the extreme since everyone has hyporepairability at some level.

Ironically, the present radiation protection policies are probably resulting in more harm than protection. To end our reliance on the LNT hypothesis and the related ALARA doctrine, we must finally perform the necessary research to define the optimum beneficial (hormetic) dose range, or ranges, as different groups of people (dependent on, for example, genetic characteristics, age, and medical condition) are likely to exhibit maximal repair and/or damage elimination at somewhat different radiation doses. There are many uncertainties involved, but one thing is certain: no radiation exposure at all means not reaching the hormetic range in anyone and, therefore, jeopardizing everyone.

References


