

## A Failed Ontology: The Linear No-threshold Model of Radiogenic Cancer

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### Overview

A debate within radiation protection science has been building over many years regarding whether low-dose, ionizing radiation (LDIR, in the range of 0 - 200 mGy) is known to be a carcinogen (1). That debate now approaches a fever pitch, and the time is ripe to assess what is known and how it is known to determine where truth must lie. A philosophy developed over centuries that applies to such an investigation, termed the philosophy of knowledge, or the philosophy of science (or scientific knowledge) as a subset (see, for example, the discussion by Wenning (2)), is useful in this debate. Within this philosophy, an ontology (and, specifically, a science ontology) may be simply defined as what we know in some particular niche or entity of science-space. But "knowing" in science requires strong foundations, and knowledge has been refined over the centuries to mean a 'justified, true belief.' Such a belief may be knowledge only under three conditions: it is certain; we believe it is true; and we have empirical justification to believe it. Stated differently, scientific knowledge arises when: we accept an objective reality; postulated rules govern that reality; and those rules may be demonstrated by empirical evidence. The empirical evidence supporting that belief or rule is called the epistemology - how we know the belief or rule is true - and epistemology for science must make use of reason constrained by verification through experimentation and observation. Putative facts deriving from misinformation and/or opinion can never be scientific knowledge.

Science history is a battlefield littered with rotted models, skewered evidence, and illogic once thought to be knowledge, but all discarded when better evidence and improved logic made for a new, more current knowledge. All scientific knowledge may be said to be presumptive or tentative, but the philosophy that guides its development makes scientific knowledge robust and remarkably stable. It is certainly true, therefore, that numerous scientific ontologies are road-side casualties of science's march towards truth, and these ontological failures trace assuredly to flawed and failed epistemologies. Accepted ontologies fail and decay in all scientific entities, but there is always a next one up, thanks to the philosophy of science. When the evidence indicates a belief is knowledge, it retains that *imprimatur* until it is overturned by empirical evidence supporting a new ontology. And so it is with the linear no-threshold model (LNT), so confidently claimed by advocates to predict accurately the subsequent generation of radiogenic cancer from LDIR exposure. The LNT extrapolates from *observed* high-dose harm to *assumed* low-dose harm, purportedly showing all ionizing radiation is harmful. It denies any biological response to damage, asserting *cumulative* lifetime harm, regardless of dose or dose rate.

Certainly, medical imaging science (e.g., nuclear medicine radionuclide imaging and x-ray/computerized tomography (CT) scans) and nuclear energy science should rely, not only upon a sound scientific foundation, but also upon the full rejection of false information and pure opinion in the development, licensing, deployment, and operation of their technologies. If the basis of all ionizing radiation exposure rules, regulations, guidance, and effects analyses (3) were shown to be a failed ontology supported by a false epistemology without merit after more than 70 years, one would expect a significant level of concern leading to an all-out effort to extirpate such a malignancy, long rooted in our knowledge system. Yet, it hasn't happened. This paper

## AUTHORS' VERSION

follows the findings and efforts endorsed earlier by Pennington and Siegel (4) and represents a full disclosure of precisely such a situation with respect to LDIR, rigidly supported within the philosophy of science. Herein, we will review the LNT ontology and examine some of the flawed and failed epistemology underpinning it, then examine the nature of the knowledge that LNT advocates are left with if its epistemology is shown to be lacking. Hopefully, such information helps both medical and nuclear industries find the determination to rid themselves of this affliction.

### **Principal Elements of the LNT Ontology**

The LNT's principal assertion of knowledge is that the type of harm from high-dose radiation is exactly mirrored with LDIR - that is, radiogenic cancer from LDIR follows the same simple cancer induction model as the cancer produced at high doses. Specifically, the LNT asserts that putative LDIR effects based on linear proportionality from high dose effects, where evidence shows it has legitimacy, manifest in two ways: that all acute exposures to LDIR produce radiogenic cancer and are proportional to that dose down to zero dose, regardless of dose rate; and that this effect is cumulative over a lifetime, regardless of dose rate. That is, the body responds in the exact same way to both high-dose and LDIR exposure. Additionally, these manifestations allow the extrapolation of proportionality and accumulated harm into another concept called "collective dose," which asserts a given radiation dose divided among any number of people will cause the same number of cancers and/or deaths from cancer - if a certain dose will produce an individual cancer death with high certainty, the same dose spread over 2 or more people will produce a cancer death with high certainty.

In summary, the LNT's ontology says: a radiogenic cancer-dose-response can be legitimately extrapolated linearly from high doses down to zero dose; a no-harm threshold dose does not exist; the body experiences only harm from radiation; such harm is cumulative; and any radiogenic, cancer-mortality-dose, spread among any number of people, produces a cancer mortality.

### **How the LNT Ontology Fails from Flawed Epistemology**

To show the failure of the epistemological underpinnings of the LNT, just a few epistemological features supporting "how we know" the LNT is true are briefly presented here. Following each, the epistemology that is, in fact, accepted science today is presented to demonstrate its failure to support the LNT ontology.

1. The LNT is based upon a model of LDIR interaction with cells, tissue, organs, and systems that is the same as at high-doses of ionizing radiation, thus making a linear extrapolation "logical" from high doses/dose rates to low doses/dose rates.

From its initial formulations more than 70 years ago, the LNT has maintained this linear extrapolation to project unobserved, low-dose effects from proven damage at high doses down to zero dose, without a no-harm threshold dose, producing predictions of cancer at all doses. But if the rules of radiation effects change at low doses/dose-rates regarding radiation interaction with cells/tissues/systems, so will the resulting dose-response relationship, such that some data (e.g., low-dose data on the order of <200 mGy) belong in one "box," while the higher dose data fall into a box where different rules apply.

## AUTHORS' VERSION

This putative linearity of dose-response, with one rule applying for high-dose and LDIR harm, is the broadest, most overarching fallacy of the LNT, since it presumes constancy of the cancer response of cells, tissues, and systems over the full dose range.

2. The LNT does not account for proven biological adaptive responses that change the shape of the LNT's dose-response line in the LDIR range.

Science has now proven that the body responds differently to radiation at high and at low doses, as demonstrated by a variety of studies: high-dose responses are associated with extensive cell/tissue/system damage, while at low doses the body repairs or eliminates the damage through a variety of protective mechanisms, termed biological adaptive responses. A cell's low-dose damage response may be linear, but the body's overall LDIR response is to mitigate this damage *as well as* some of the body's similar naturally-occurring exogenous damage due to oxygen metabolism, making the total response to radiation non-linear (5, 6). The body's biological adaptive responses effectively deal with the expected low-dose damage through a set of proven mechanisms (7, 8), which provide cancer protection through antioxidant production, apoptosis, immune system-mediated effects, and repair of DNA double-strand breaks (DSBs), the most significant damage producing radiogenic cancer. It has been demonstrated that DSB repair from LDIR occurs within hours of exposure, even after CT scans (9), with DSB levels decreasing over time and ending at lower values than the patients' baseline levels within 24 hours.

The same DSB repair responses have been reported for two nuclear medicine therapeutic studies, with DSB levels increasing at first, followed by decreasing levels over a short time, ending at values approaching, and even less than the baseline (10, 11). Other nuclear medicine imaging studies show that such doses do not produce radiogenic cancer increases. Thousands of children younger than 20 who received  $^{131}\text{I}$  for diagnoses ( $< 0.37$  MBq) that resulted in mean thyroid doses of about 1 Gy were tracked over many years, and some for 40 years. These studies, summarized and reported by Siegel and Silberstein (12), show no evidence of increased risk of thyroid cancer due to childhood intake of  $^{131}\text{I}$ .

More than 150 genes are involved in DNA repair. At least six mechanisms act to decrease cancer rates and enhance longevity from the stimulation of LDIR damage to an organism's constituent parts, and these biological responses are reported to produce benefit, i.e., hormesis (13).

Importantly, the science presented in the studies involving DSB damage and repair (9, 10, 11) shows why the LNT's predicted LDIR radiogenic cancer risk has never been observed or demonstrated by empirical evidence. LNT advocates excuse that fact by asserting that, at low doses, the ratio of the radiogenic cancer risk "signal" to the background spontaneous cancer risk "noise" is so small, the signal becomes statistically indistinguishable from the noise. Such a claim for radiogenic signal "invisibility" is inaccurate in the range of LDIR, however, since these and many other studies demonstrate reduced cancer risk and increased longevity, i.e. reduction in all-cause mortality (13, 14, 15) in the LDIR range. If harm is invisible, benefit should be, as well. But it's not.

## AUTHORS' VERSION

Further, science cannot distinguish the pure radiogenic signal since it cannot be differentiated from spontaneous cancer arising from the body's own metabolic damage by reactive oxygen species (ROS) that produces far more mutation events per cell per day than LDIR. Epidemiology's true "signal" is total cancer (spontaneous plus radiogenic), and the "gold standard" for epidemiological data of LDIR radiogenic cancer is the Life Span Study (LSS) cohort of A-bomb survivors (16) by the Radiation Effects Research Foundation, which, contrary to LNT advocates, has *always* purported to show radiogenic cancer is discernible. However, according to the latest update of LSS study results in 2017 (17), apparent threshold doses, below which there were no observable cancers, were 80 mGy for females (with upper 95% CI bound of 200 mGy) and 750 mGy for males (with upper 95% CI bound of 800 mGy), a decidedly non-LNT outcome. Of course, the LSS data do not account for the well-known cancer mortality/morbidity increases from high stress levels and significant deprivation experienced by those survivors, especially women. As reported earlier by Ozasa (18), radiation-related risk of disease at low-doses from atomic bomb radiation must be calculated on top of an uncertain background dose, and these two values can overlap, becoming indistinguishable.

Some LNT purveyors (large professional and regulatory organizations that arrogate radiation controls to themselves and still advance adherence to the LNT) sometimes try to adopt a middle-ground position. For instance, the National Academy of Sciences BEIR VII Report (19) acknowledges repair of initial DNA damage, but, without evidence, says that repair is incomplete and dismisses a radiogenic cancer threshold by ignoring additional mechanisms known to eliminate any unrepaired damage, such as by apoptosis and the immune system. BEIR VII cites the work of Rothkamm and Löbrich (20), but misrepresents its findings (13), arriving at conclusions contrary to the evidence that additional repair and/or cell-destroying apoptosis supports elimination of failed initial DNA repairs.

Lastly, the LNT tenet that radiation damage is cumulative, no matter the dose or dose rate, is directly contradicted by the proven practice of fractionation of high-dose radiation therapy, demonstrating that damage recovery occurs between treatments (21).

The LNT ontology that says damage producing radiogenic cancer results from LDIR all the way down to zero dose and that all such LDIR damage is cumulative is clearly not supported by the present epistemology addressing biological adaptive response.

3. The LNT relies upon the disproven somatic mutation model for radiogenic cancer, where cancer is the unalterable end-product of one or more driver mutations.

While mutations are necessary for cancer induction, they are now known to be insufficient to produce clinically-overt cancer. The body's adaptive response and its immune system are now recognized as being responsible for arresting the development of mutation-induced cancer.

Cancer is not simply the end-product of one or more enabling mutations. The immune system plays a much more significant role in the development of cancers, and it's functioning is now understood to make the "one mutation = one cancer" model obsolete. Cancers mainly develop from immune system suppression. Research shows mechanistic descriptions of

## AUTHORS' VERSION

radiation-induced cancer (e.g., DNA DSBs produce chromosome aberrations that inevitably produce cancer) to be inaccurate. Low-dose radiation stimulates the immune system, causing a reduction in cancer rates (22). Another study has reported that residents of high background radiation areas (3.3 mSv/year) had increased frequencies of chromosome aberrations compared to the control population in lower background areas (1.1 mSv/year), but had reduced all-cancer mortality (23).

The LNT ontology of radiogenic cancer induction, resulting from an outdated somatic mutation model that produces LDIR-radiogenic cancer down to zero dose and with all such LDIR damage being cumulative, is clearly not supported by the present epistemology of a more complex cancer induction process.

4. The LNT dose-response ontology cannot account for cancer induction that addresses the now-proven spontaneous rate of DNA alterations from a cell's normal oxidative metabolic processes, which dwarfs the DNA alteration rate due to LDIR.

The LNT's flaws become even more apparent through a broader, organism-level perspective. The spontaneous rate of DNA alterations resulting from a cell's metabolic processes, i.e., oxygen metabolism, dwarfs the DNA alteration rate due to LDIR (24, 25). Thus, oxygen produces more DNA damage than LDIR. From an average U.S. natural background dose of 3 mSv per year, 3 to 30 DNA alterations per cell per year result, almost 2.5 million times less than the body's metabolic spontaneous mutation rate. A CT scan's LDIR has been shown to produce DNA damage that is repairable to below baseline levels. The body processes both radiogenic and endogenous DNA damage through adaptive responses, as previously discussed, that are overwhelmed at high doses, but uniquely stimulated by LDIR (16). Therefore, the LNT is false, since high-dose effects (responses are associated with extensive damage) cannot accurately predict effects of LDIR (responses are protective) (5).

Further, a study comparing interventional cardiologists (median dose-rate of 4 mSv/year) to controls exposed only to background radiation showed chronic LDIR was associated with two responses: improved antioxidant defense and increased apoptotic response (26). An accompanying editorial noted these data may confirm the concept that LDIR induces a protective phenotype (27).

The LNT ontology regarding cancer induction is clearly unsupported by present epistemology, because the LNT epistemology flagrantly fails to account for the generation and disappearance of the DNA alterations from naturally-occurring metabolic processes.

5. Today's LNT advocates and purveyors often rely upon sophisticated epidemiological methods that are incorrectly applied, attributing significance to purported LNT compliance where none actually exists.

The preceding sections demonstrate there is no validated and accepted empirical research to support a reasonable epistemology for the LNT ontology. Yet, LNT advocates and purveyors still cling to epidemiological studies that purportedly support the LNT ontology.

As is well-known, investigational methods used to test models must be scientifically appropriate and rigorously applied. Sacks et al. (13) provides a thorough analysis of a

## AUTHORS' VERSION

substantial portion of the most recent published epidemiological studies supportive of LNT, showing huge flaws in the science, thereby invalidating their conclusions. These studies generally confine their investigations to mathematical and statistical methods without reference to the supporting sciences and their well-established empirical findings. This separation of the epidemiological from the empirical science, examining damage and ignoring the biological response to that damage, are central components of the major errors highlighted in the preceding sections. Further, such studies implicitly invoke the LNT as an *a priori* assumption and, based on circular reasoning, arrive at a self-fulfilling conclusion that the LNT is valid, while presenting the “measured” slope of a presumed linear dose-response relationship as an uncritically reviewed certainty. Assuming the LNT *a priori* permits study authors to avoid any biological considerations when calculating cancer risk from LDIR. It further forces the LNT as the null hypothesis, a failure of the burden-of-proof test, which invalidates any claim of cancer *causation* by radiation. Typical additional errors in such studies include:

- Defining the cumulative dose origin as zero dose, neglecting all other sources of LDIR received by the population;
- Failure to invalidate reverse causation or confounding by indication, key considerations in medical imaging, particularly for repeated imaging;
- Failure to obtain accurate dose measurements for investigation, assuming doses from geographic models or average doses for a given imaging technology and patient body size;
- Failure to consider confounders such as smoking, socioeconomic effects, environment, etc.

The LNT's ontology now relies only on studies having illegitimate bases for supporting the LNT's conclusions, and such studies are the LNT's last bastion of defense. However, their use is a known application of false empiricism.

### **What Is Now the LNT's Position Within Science?**

The LNT's ontology is without empirical basis, is unsupported by currently accepted epistemology, and is, therefore, no longer science, if ever it was. Having the LNT as the basis for regulation and practice within medicine and nuclear energy is, therefore, an on-going and outrageous *non sequitur*. The evidence demonstrating a failed LNT ontology is a robust counter-epistemology. Regarding robust evidence for such ontologies, research on scientific ontologies from Burian and Trout (28) observes, "What makes the evidence robust is that it is so multifarious and that it has withstood integration into a body of practice quite thoroughly removed from the theoretical frontiers." This is amply shown in the preceding failed epistemological features supporting the LNT's ontology.

This leaves LNT advocates and purveyors simply outside of science. Nevertheless, they still cling to it as if it were holy writ, asserting that even if the LNT is wrong, it produces conservative (that is, "safe") projections of outcomes. Yet, this is demonstrably false. As the basis for all medical and nuclear energy radiation-regulations and policies, the LNT is well-documented for generating intense radiophobia, responsible for millions who have suffered

## AUTHORS' VERSION

from radiophobia-induced mental disability over the last four decades (eg, from Chernobyl, Fukushima, and medical imaging), and many thousands have died from LNT-based policies tied to evacuations rather than using safer shelter-in-place policies (29-36). Still the LNT debate rages on, especially within the medical imaging community. A recent point-counterpoint debate (37) on medical imaging produced but Parthian volleys of LNT-based misinformation and opinion from ALARA-dosing advocates within the Image Gently Alliance vs. rigorous, scientific epistemology from its critics.

Another possible explanation, especially applicable for LNT-based misinformation from LNT purveyors, is ignorance. Van Nostrand (38), writing on a different topic, says that physicians must more critically read the literature and not just accept the "findings" of studies as fact. Medical imaging and nuclear energy experts must personally review and understand the primary literature (not relying on their beliefs or less-informed, second-hand versions) that controls the advancement of their technologies and the welfare of humanity. If putative experts do not self-educate, LNT advocates, purveyors and the media will continue to misinform the public about the effects of LDIR exposure, causing much harm; the problem is radiophobia, not LDIR exposure.

The LNT has degenerated within the philosophy of science to a lower level of "knowing" than empirical knowledge. Other disciplines, where knowledge must still be accepted and defended, cannot rely on empiricism. They predominantly use a path to knowledge called coherentism. Empiricism combines rationalism with observation and experimentation (logic constrained by verification) to arrive at knowledge. Coherentism follows a path to knowledge where individual concepts, without the use of empirical support, may form some logically connected structure whose ascent to knowledge occurs if many agree it's true. This is not empirical science, since it fails to rely on hard evidence and demonstration. Coherentism, an outcome resulting as if from entropy, represents a decayed state of more disorder when compared to empiricism. One might also recognize it as "consensus science," another way of saying non-science. The LNT has been cast out of science, and coherentism is now its home.

### Conclusion

The LNT is false. The imperial *imprimatur* ordained by its purveyors and advocates is not clothed in science. And because the LNT fails empirically against superior empiricism, it becomes misinformation and opinion, not knowledge. A contributor to the philosophy of knowledge, Blaise Pascal, once observed, "Opinion is the mistress of error; she cannot make us wise, only content." The LNT is still advocated and purveyed because it lends contentment to believers, whether to maintain bureaucracies, control, or a sense of conservatism.

The purpose of science is knowledge, hopefully applied with wisdom, to help accelerate human advancement. We can no longer tolerate universal application of LNT misinformation and opinion when, as people of science, we are about helping, not harming, humanity.

## References

1. Siegel JA, Pennington CW, Sacks B. Subjecting radiological imaging to the linear no-threshold hypothesis: a non sequitur of non-trivial proportion. *J Nucl Med.* 2017; 58(1):1-6.
2. Wenning CJ. Scientific epistemology: how scientists know what they know. Illinois State University, *J. Phys. Tchr. Educ. Online*; Autumn 2009; 5(2):1-15.  
<http://www2.phy.ilstu.edu/~wenning/jpteo/>. Accessed May 2, 2017
3. Siegel JA, Stabin MG. RADAR commentary: use of linear no-threshold hypothesis in radiation protection regulation in the United States. *Health Phys.* 2012; 102:90-99.
4. Pennington CW, Siegel JA. Nuclear energy's critical illness: continue with failed treatments or pursue the cure? *Public Utilities Fortnightly.* March 2017; 40-43, 74-75.
5. Brooks AL, Dauer LT. Advances in radiation biology: effect on nuclear medicine. *Semin Nucl Med.* 2014; 44:179-186.
6. Koana T, Tsujimura H. A U-shaped dose-response relationship between x radiation and sex-linked recessive lethal mutation in male germ cells of *Drosophila*. *Radiat Res.* 2010; 174:46-51.
7. Luckey TD Radiation hormesis. Boca Raton, FL: CRC Press; 1991.
8. Feinendegen LE, Pollycove M, Neumann RD. Low-dose cancer risk modeling must recognize up-regulation of protection. *Dose-Response.* 2010; 8:227–252.
9. Löbrich M, Rief N, Kühne M, et al. In vivo formation and repair of DNA double-strand breaks after computed tomography examinations. *Proc Natl Acad Sci.* 2005; 102(25):8984-8989.
10. Eberlein U, Scherthan H, Bluemel C, Peper M, Lapa C, Buck AK, Port M, Lassmann M. DNA damage in peripheral blood lymphocytes of thyroid cancer patients after radioiodine therapy. *J Nucl Med.* 2016; 57:173–179.
11. Eberlein U, Nowak C, Bluemel C, Buck AK, Werner RA, Scherthan H, Lassmann M. DNA damage in blood lymphocytes in patients after <sup>177</sup>Lu peptide receptor radionuclide therapy. *Eur J Nucl Med Mol Imaging.* 2015; 42:1739–1749.
12. Siegel JA, Silberstein EB. A closer look at the latest NRC patient release guidance. *J Nucl Med.* 2008; 49:17N-20N.
13. Sacks B, Meyerson G, Siegel JA. Epidemiology without biology: false paradigms, unfounded assumptions, and specious statistics in radiation science (with commentaries by Inge Schmitz-Feuerhake and Christopher Busby and a reply by the authors). *Biol Theory.* 2016;11:69–101.
14. Aurengo A, Averbeck D, Bonnin A, et al. Dose effect relationships and estimation of the carcinogenic effects of low doses of ionizing radiation. Paris: Académie des Sciences [Academy of Sciences] - Académie nationale de Médecine [National Academy of Medicine]; March 2005.
15. Feinendegen LE, Pollycove M, Neumann RD. Low-dose cancer risk modeling must recognize up-regulation of protection. *Dose-Response.* 2010; 8:227–252.
16. Dauer LT, Brooks AL, Hoel DG, et al. Review and evaluation of updated research on the health effects associated with low-dose ionising radiation. *Radiation Protection Dosimetry.* 2010; 140(2):103–136.
17. Grant EJ, Brenner A, Sugiyama H, et al. Solid cancer incidence among the Life Span Study of Atomic Bomb Survivors: 1958–2009. 2017; *Radiat. Res.* 187; <http://www.rerf.or.jp>. Accessed May 5, 2017

## AUTHORS' VERSION

18. Ozasa K. Epidemiological research on radiation-induced cancer in atomic bomb survivors. *J Radiat Res.* 57(Suppl 1), March 2016; doi: 10.1093/jrr/trw005
19. National Research Council of the National Academies. *Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII Phase 2.* Washington, DC: The National Academies Press; 2006.
20. Rothkamm K, Löbrich M. Evidence for a lack of DNA double-strand break repair in human cells exposed to very low x-ray doses. *Proc Natl Acad Sci USA.* 2003;100:5057-5062.
21. Taylor LS. Some nonscientific influences on radiation protection standards and practice: the 1980 Sievert lecture. *Health Phys.* 1980; 39:851-874.
22. Liu SZ. Cancer control related to stimulation of immunity by low-dose radiation. *Dose-Response.* 2007; 5(1):39-47.
23. Chen D, Wei L. Chromosome aberration, cancer mortality and hormetic phenomena among inhabitants in areas of high background radiation in China. *J Radiat Res.* 1991; Suppl; 2:46-53.
24. Siegel JA, Welsh JS. Does imaging technology cause cancer? Debunking the linear no-threshold model of radiation carcinogenesis. *Technol Cancer Res Treat.* 2016; 15(2):249-256
25. Billen D. Spontaneous DNA damage and its significance for the “negligible dose” controversy in radiation protection. *Radiat Res.* 1990; 124(2):242-45.
26. Russo GL, Tedesco I, Russo M, et al. Cellular adaptive response to chronic radiation exposure in interventional cardiologists. *Eur Heart J.* 2012; 33:408–414.
27. Scott BR, Sanders CL, Mitchel REJ, Boreham DR. CT scans may reduce rather than increase the risk of cancer. *J Am Phys Surg.* 2008; 13(1): 8-11.
28. Burian RM, Trout JD. Ontological progress in science. *Canadian J Philosophy.* 1995;25(2):177-201.
29. *The Japan Times.* 2016. “Cabinet OKs plan to lift Fukushima evacuation orders by end of fiscal 2016.” <http://bit.ly/1LbZuYe>. Accessed October 4, 2016.
30. Shimura T, Yamaguchi I, Terada H, Svendsen ER, Kunugita N. “Public health activities for mitigation of radiation exposures and risk communication challenges after the Fukushima nuclear accident.” *J Radiat Res.* 2015; 56:422-429.
31. Yajima K, Kurihara O, Ohmachi Y, et al. “Estimating annual individual doses for evacuees returning home to areas affected by the Fukushima nuclear accident.” *Health Phys.* 2015;109:122-133.
32. United Nations Information Service. 2013. *No Immediate Health Risks from Fukushima Nuclear Accident Says UN Expert Science Panel:* <http://bit.ly/1Tk3Bnf>. Accessed June 10, 2015.
33. World Health Organization (WHO). 2006. *Health Effects of the Chernobyl Accident and Special Care Programmes.* Report of the UN Chernobyl Forum Expert Group “Health.” Ed: Bennett, B., Repacholi, M., Carr, Z.; WHO Press, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland.
34. United Nations and International Atomic Energy Agency. 2006. *Chernobyl Forum: Chernobyl’s Legacy: Health, Environmental and Socio-Economic Impacts and Recommendations to the Governments of Belarus, the Russian Federation and Ukraine, 2003-2005.* Second Revised Version: <http://bit.ly/1FNP3dj>. Accessed on June 10, 2015.
35. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Annex D. 2011. Health Effects due to Radiation from the Chernobyl Accident, of Sources

## AUTHORS' VERSION

- and Effects of Ionizing Radiation, UNSCEAR 2008, Report to the General Assembly with Scientific Annexes; Sales Number E.11.IX.3. United Nations, Vienna, Austria.
36. Siegel JA, Welsh JS. Does imaging technology cause cancer? Debunking the linear no-threshold model of radiation carcinogenesis. *Technol Cancer Res Treat*. 2016; 15(2):249-256.
  37. Siegel JA, McCollough CH, Orton CG. Point/counterpoint: advocating for use of the ALARA principle in the context of medical imaging fails to recognize that the risk is hypothetical and so serves to reinforce patients' fears of radiation. *Med Phys*. 2017;44(1):3-6.
  38. Van Nostrand D. Prescribed activity of  $^{131}\text{I}$  therapy in differentiated thyroid cancer. *J Nucl Med*. 2017; DOI: 10.2967/jnumed.116.188862.