

**Subjecting Radiological Imaging to the Linear No-Threshold Hypothesis:
A Non Sequitur of Non-Trivial Proportion**

Jeffrey A. Siegel, Ph.D.¹

Charles W. Pennington, MS, MBA²

Bill Sacks, Ph.D., M.D.³

¹President and CEO, Nuclear Physics Enterprises, Marlton, NJ

²NAC International, Norcross, GA, Retired; Executive Nuclear Energy Consultant,
Alpharetta, GA

³U.S. FDA, Medical Officer and Clinical Radiologist (Retired), Green Valley, AZ

Please address all correspondence to first author:

Jeffrey A. Siegel, Ph.D.

President and CEO, Nuclear Physics Enterprises

4 Wedgewood Drive, Marlton, NJ 08053

Phone: (856) 899-9767

E-mail: nukephysics@comcast.net

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ABSTRACT

Radiological imaging is claimed to carry iatrogenic risk of cancer, based upon an uninformed commitment to the 70-year old linear no-threshold hypothesis (LNTH). Credible evidence of imaging-related low-dose (<100 mGy) carcinogenic risk is nonexistent; it is a *hypothetical* risk derived from the demonstrably false LNTH. On the contrary, low-dose radiation does not *cause*, but more likely helps *prevent*, cancer. The LNTH and its offspring ALARA (as low as reasonably achievable) are fatally flawed, focusing only on molecular damage, while ignoring protective, organismal biological responses. While some grant the absence of low-dose harm, they, nevertheless, advocate the “prudence” of dose optimization (i.e., using ALARA doses); but this is a radiophobia-centered, not scientific, approach. Medical imaging studies achieve a diagnostic purpose and should be governed by the highest science-based principles and policies. The LNTH is an invalidated hypothesis, and its use, in the form of ALARA dosing, is responsible for misguided concerns promoting radiophobia, leading to actual risks far greater than the hypothetical, carcinogenic risk purportedly avoided. Further, the myriad of imaging’s benefits are ignored. The present work calls for ending the radiophobia caused by those asserting the need for dose “optimization” in imaging: medical imaging's low-dose radiation has no documented pathway to harm, while the LNTH and ALARA most assuredly do.

Key words: Radiological imaging, linear no-threshold; ALARA; hormesis; adaptive response; radiophobia

INTRODUCTION

The LNTH has been applied to low-dose/dose-rate ionizing radiation for more than 70 years, but remains a hypothesis, lacking valid scientific foundation. Nonetheless, this hypothesis is the orthodox foundation of radiation protection science, in turn forming the basis of regulations and public policy.

The LNTH derives from incomplete, early-20th-century genetic-experimental observations yielding inaccurate conclusions, heretofore undetected by other scientists (1). Hermann Muller, in his 1946 Nobel Lecture, asserted a no-harm threshold was nonexistent, since linearity had been demonstrated for doses down to 4,000 mGy, a stunning non sequitur. Nor has any evidence since validated carcinogenicity of low-doses. The LNTH extrapolation from evidence-supported, high-dose effects to putative low-dose responses claims *all* acute ionizing radiation exposure down to zero is harmful proportionally to dose, and that it yields *cumulative* harm throughout life, regardless of how low the *dose rate*. Both claims are demonstrably false and harmful, leading to LNTH-derived regulations and policies that are not protective (2,3); for example, more than 1600 deaths resulted from the LNTH-based evacuation policy for nearby residents following the Fukushima nuclear accident (3).

Throughout time, we have been bathed in low-dose radiation from land, sky, and our own bodies. Today's average annual natural background exposure ranges from 1 mSv to 260 mSv in some places on the planet. No associated adverse health effects have been documented anywhere (4). For comparison, typical computed tomography (CT) and combined whole-body ¹⁸F-FDG positron emission tomography (PET)/CT scan doses delivered acutely are 10 mSv and 14 mSv, respectively. This radiation-rich history implies extant life-forms must have developed adaptive, biological repair/removal responses to radiation damage. The primary LNTH fallacy is it *excludes* this evolutionary biology, ignoring the body's differing responses to high versus low radiation doses (5). Low doses stimulate protective responses; high doses overwhelm and inhibit such protections.

Herein are offered dissenting views of subjecting medical imaging to the LNTH, as this hypothesis is characterized by its one-sided failure to incorporate experimental research findings and its support by erroneous mathematical and statistical maneuvers that merely confirm *a priori* assumptions through circular reasoning. Our heterodoxy maintains that this one-sidedness is two-fold: first, it focuses on unquestioned radiogenic cellular damage while ignoring the *organism's* proven biological responses to mitigate that damage plus the endogenous damage (due to normal metabolism) several orders of magnitude greater; second, it focuses only on imaging's hypothetical risks while ignoring its myriad benefits and the actual risks associated with imaging's alternatives (6-8). The *hypothetical* risks from medical imaging pale in comparison to these *actual* risks.

Unwarranted fear of low-dose radiation leads to the misguided doctrine of “prudence” in dosing – ALARA (as low as reasonably achievable) – that, by often diminishing image quality, increasingly produces suboptimal and even non-diagnostic CTs (9,10). Thus, today, 70 years after Muller's Nobel speech, another non sequitur advances, this time within the field of radiological imaging. This article provides a scientific rebuttal of the

key errors within the LNTH orthodoxy to rehabilitate and restore low-dose radiation's position of respect within science and medicine and to help undo needless public and professional radiophobia.

MATERIALS AND METHODS

The Failure of the LNTH Gold Standard

The Life Span Study's (LSS) atomic-bomb survivor cohort is the single most important dataset – the “gold standard” – for estimating radiation effects in humans (5,11). The 1958-1998 LSS data for acute exposure to low-dose, low linear-energy-transfer radiation, like x- and gamma-rays used in medical imaging, were reported by the Biological Effects of Ionizing Radiation (BEIR) VII Committee in 2006 (11) to be consistent with the LNTH dose-response relationship for development of solid cancers. The BEIR Committee operates under the auspices of the National Academy of Sciences, receiving significant financial support from various regulatory and other government agencies; thousands of government and private industry jobs depend upon the conclusions of the BEIR Report, which promotes acceptance of the LNTH. The 2005 French Academy of Sciences Report (12), however, reached very different conclusions. Providing evidence for protective adaptive responses and finding no valid evidence for harm below 100 mGy, the report questioned the LNTH's validity in that range.

The BEIR VII Committee, seeking putative low-dose cancer-risk (<100 mGy) reductions, but unwilling to forgo linearity, introduced the artifice of the “dose and dose-rate effectiveness factor.” A factor of 1.5 was chosen to reduce the slope of the LNTH-derived result, though the LSS data are not linear at doses <100 mGy (3,13). Rather, linearity is forced by the LNT model from high-dose extrapolation. Independent analyses of LSS data indicate an apparent threshold as high as 55 mGy, comparable to a threshold of 60 mSv reported by others (3).

Using updated LSS data, Ozasa et al. (14) reported that 0-180 mGy was the lowest dose range exhibiting no significant, acute-dose, excess relative risk (ERR) for all solid-cancer mortality. In this dose range, the ERR uncertainty (from their Figure 4) overwhelms its dose dependence, with the 95% confidence intervals including negative ERR values, consistent with a beneficial, as well as a harmful, effect. This uncertainty is not reflected in the linear dose response or its confidence range because that linear fit was estimated by extrapolation from dose levels of 1 Gy or higher (15).

Ozasa et al. used Poisson regression methods to mathematically derive background mortality rates at zero dose, which effectively enables the lowest dose cohorts to determine this rate by linear extrapolation to zero dose. Other studies show reduced mortality rates in low-dose cohorts (16), compared to cohorts experiencing no radiation above natural background; therefore, Poisson regression introduces negative bias in the background mortality rate, which artificially elevates the reported ERR values.

Correcting this bias, ERR values become *negative* for doses below approximately 0.6 Gy, beneficially reducing cancer risk relative to background cancer rates (16). Another

LSS reanalysis (17) exhibits negative ERRs below a threshold at < 200 mSv, again consistent with radiation-induced benefit.

Contrary to the LNTH, Biology Responds Adaptively

The LSS data do not support the LNTH; rather, the observed thresholds and negative ERRs agree with experimental evidence for adaptive cancer protection following low-dose radiation exposure. These data are more consistent with a radiation hormetic (protective) model than with the LNT (harm at any dose) model. Yet, John Boice, President of the National Council on Radiation Protection and Measurements, continues to assert that the LNTH is the most plausible hypothesis (18) (this council is a congressionally-chartered, private corporation that receives financial support from Federal radiation regulators and other governmental agencies).

Whether or not low-dose damage is linear, the body's defensive response is nonlinear, leaving the net result nonlinear (19). The body deals with this damage through a set of proven mechanisms, collectively called the adaptive response (3,20,21), which offers cancer protection through DNA repair involving more than 150 genes, antioxidant production, apoptosis on the cellular level, bystander effects on the tissue level, and immune-system removal of surviving damaged cells on the organismal level. Double-strand-break repair occurs even after low-dose CT scans (22). Numerous studies demonstrate at least six mechanisms for reducing cancer rates and increasing longevity, stimulated by low-dose damage (23).

BEIR VII (11) grants the existence of “incomplete” repair, but because imperfect repair of initial DNA damage is assumed, the BEIR Committee dismisses a low-dose threshold for carcinogenicity, ignoring additional mechanisms of defense against radiation-caused damage when DNA repair fails. The report cites a paper by Rothkamm and Löbrich (24), but proceeds to misrepresent their findings (23). The paper provides evidence for mechanisms reducing both spontaneous and radiation-induced damage below spontaneous levels (a hormetic effect), by directly measuring the progression of double-strand-break foci at low doses. Post-irradiation counts of cultured cells with double-strand breaks were found to decrease to pre-irradiation counts, constituting evidence of repair and/or cell-destroying apoptosis - a finding not mentioned in the BEIR VII report.

The LNTH asserts radiation damage is cumulative, no matter the dose or dose rate. But this is directly contradicted by the practice of fractionation of high-dose radiation therapy, demonstrating that recovery occurs between treatments (25). More importantly, because low doses stimulate repair and/or removal of radiogenic damage *in excess of that immediate damage*, they provide enhanced protections against additional damage over time, including damage from subsequent higher radiation exposures, infections, endogenous production of reactive oxygen species, and other non-radiogenic damage. The net result is *reduction of damage below spontaneous levels* (21,26), likely contributing to a lifetime-cancer-risk reduction.

Further, spontaneous levels of DNA alteration resulting from a cell's normal metabolic processes dwarf those due to low-dose radiation (3,27). For example, the average annual

U.S. background of 3 mSv produces 3 to 30 DNA alterations per cell per year, and an acute-dose CT about 10 to 100, while mutation rates due to the body's normal metabolic chemistry are *a million times higher*. Thus, the LNTH extrapolation of high-dose levels, which are inhibitory of protective mechanisms, down to low-dose levels falsely predicts detrimental effects at low dose.

Another study involving radiation exposures to interventional cardiologists (median of 4 mSv/year) compared them to unexposed controls. Low-dose, chronic exposure was associated with two adaptive cellular responses: enhanced antioxidant defense and increased apoptotic response (28). These likely compensate for increased reactive oxygen species production and contribute to maintaining cellular homeostasis. An accompanying editorial noted these data confirm low-dose protective responses (29).

Mutations are necessary, but not sufficient, to produce clinically overt cancer. The immune system generally keeps cancers in check, and cancers develop mainly when the immune system is suppressed. The immune system's role in cancer development now replaces the outdated “one mutation = one cancer” model. Recent research shows the inaccuracy in mechanistic models of radiation-induced cancer suggesting that double-strand breaks lead to chromosome aberrations resulting in cancer. Low-dose radiation stimulates the immune system, causing a reduction in cancer rates (30). Furthermore, residents in higher background radiation areas (3.3 mSv/year) were found to have increased frequencies of chromosome aberrations compared to lower-background control populations (1.1 mSv/year), yet had *lower all-cancer mortality* (31).

The evidence for low-dose radiation's biological-response/cancer-reduction paradigm continues to mount. The 2015 Nobel Prize in Chemistry was awarded for research by Tomas Lindahl, Paul Modrich, and Aziz Sancar showing how cells safeguard genetic information, preventing it from disintegrating into chaos, through a host of molecular systems that continuously monitor and repair DNA.

The Absence of Acute, Low-Dose Radiation Carcinogenesis IS Evidence

As previously noted with the revised Ozasa et al. (14) data, most ERRs in the low-dose range have confidence intervals that include negative values. These negative values suggest imaging doses *reduce* cancer-risk compared to a valid baseline.

The most widely used estimate for the slope of the radiation-induced, cancer-mortality, dose-response relationship is about 5% *per* Gy for an all-age population. This estimate is primarily derived from LSS data at 1 Gy or higher using a linearity-preserving artifice, the dose and dose-rate effectiveness factor, with a value of 2 (15,32) and from the added *non-empirical* assumption that there is no threshold. This may be verified by a point-estimate calculation at 1 Gy (i.e., 5% at 1 Gy) based on the LSS data (14), but it is not a valid predictor of risk at lower doses, e.g., a 10 mGy CT dose would represent a hypothetical 0.05% risk estimate (corresponding to an ERR of 0.004). As can be seen from these same data, below about 200 mGy the dose-response relationship is not an extrapolated line from higher doses, but instead is roughly horizontal. Therefore, both the “L” and “NT” components of LNT are false. Since LNTH-derived, low-dose-risk

estimates have huge uncertainties and are not validated by observed LSS data (from which they are derived), these risk estimates are not merely notional, but flatly false.

Advocates excuse their inability to provide low-dose/dose-rate evidence for the LNTH, claiming this inability is because the ratio of radiogenic low-dose cancer risk (the “signal”) to the variation in spontaneous cancer risk (the “noise”) is too small to distinguish signal from noise. This explanation for radiogenic signal "invisibility" is a red herring for radiological imaging, including for children irradiated up to 200 mSv. Solid cancer incidence rates were examined among the Hiroshima and Nagasaki A-bomb survivors who were younger than 6 years when the bombings occurred (33). Their reported relative risk values and our analyses of their Table 3's raw data indicate no significant difference between the control group's adult-onset solid cancer incidence and that for those children with exposures up to 200 mSv; this agrees with Ozasa's adult results (14) and, therefore, suggests that children are not more radiosensitive to harmful effects at low doses.

Hundreds of studies have demonstrated the *benefits* of reduced cancer risk and increased longevity, not just absence of low-dose harm, e.g., reduced all-cause mortality (12,21,23). Since these demonstrated benefits are not rendered invisible by noise, LNTH advocates simply ignore or distort the evidence for benefit.

Nonetheless, these studies show *radiogenic* cancer-signal invisibility has some validity, but for a different reason. Adaptive responses likely negate a significant portion of the radiogenic signal, forcing the signal-to-noise ratio towards zero. But science cannot observe pure *radiogenic* signals separately from spontaneous cancer noise because they may be inseparable. According to Ozasa (15) it is difficult to estimate radiogenic risk at low-doses because acute, A-bomb low doses must be calculated on top of an uncertain background dose and these two values can overlap, becoming indistinguishable.

While this discussion applies to acute doses (like CT imaging), the total dose from nuclear medicine procedures is protracted, which is known to reduce risk compared to acute exposure of the same total dose (34). Studies involving thousands of children younger than 20 who received ¹³¹I for diagnostic purposes (< 3.7 MBq, small children < 0.37 MBq) have been reported by Siegel and Silberstein (35). These children, some followed for 40 years, received mean thyroid doses of about 1 Gy. No evidence of increased risk of thyroid cancer due to childhood intake of ¹³¹I was found.

RESULTS

The evidence presented shows a reduced, not increased, cancer risk at radiological imaging doses, and the LSS data show the LNTH-predicted, low-dose carcinogenicity is invalid up to approximately 200 mGy. Thus, medical imaging's much lower doses for children or adults should not be feared or avoided for radiophobic reasons. A typical CT scan effective dose is about 10 mSv; a PET/CT brain scan, 5-7 mSv; and a routine whole-body ¹⁸F-FDG PET/CT scan, 12-15 mSv (36). In general, epidemiological studies focused on providing direct low-dose LNTH-consistent risk estimates fail to address the basic sciences (e.g., biology and chemistry), and employ often-hidden circular reasoning

(assuming that which must be demonstrated empirically), thereby rendering their conclusions false and indefensible (23).

While recent large epidemiological studies - Pearce et al. (37) and Mathews et al. (38) - suggested increased low-dose cancer risk associated with pediatric CT scans, these results have been effectively rebutted. Major flaws are their willingness to draw causal conclusions from mere association and their failure to consider the association is likely due to reverse causation (i.e., cancer/illness give rise to CT, not the reverse). Additionally, inaccurate dosimetry and implausible risk estimates are apparent. Other recent large-scaled cohort studies examining pediatric CT cancer risk - Journy et al. (39) and Krille et al. (40) - concluded that confounding by indication and reverse causation must be ruled out completely, or observed excess cancer risk may be falsely, and facilely, attributed to CT exposure.

Radiological imaging, nuclear medicine procedures, and, therefore, cumulative public radiation doses have increased dramatically over several decades, but their contributions to reduced morbidity and improved longevity have also. Concomitantly, concerns have arisen that radiation produces higher radiogenic cancer risk. Pediatric imaging's dose-optimization movement led to the “Image Gently” campaign, which seeks to *lower* doses. Consensus guidelines for administered activity for pediatric nuclear medicine studies have been developed (41) that advise lower doses, based on the LNTH: “A reasonable assumption is to apply the linear no-threshold hypothesis for radiation-induced carcinogenesis when making judgments about the relative radiation-associated risks of different imaging studies” (42). Yet, as demonstrated herein, assuming the LNTH accurately assesses risk, in the face of voluminous evidence to the contrary, can never be called “reasonable.”

The usual justification for this assumption is that it errs on the side of caution – the precautionary principle, which may be useful if action to control the feared agent has no, or less-harmful, side effects. However, for radiological imaging, significant, collateral, negative consequences of lowering dose arise. Reducing patient doses to mitigate *purely hypothetical* cancer risks increases other well-known risks resulting from fear of imaging (7). These include imaging avoidance, non-diagnostic image quality, and use of alternative imaging procedures, e.g., a longer-duration MRI study, requiring risk-incurring sedation for young children (6-8). The risks of misdiagnoses from inadequate dose could be much higher than the cancer risks that the LNTH falsely predicts and that are putatively avoided by ALARA-based dose-reduction strategies (9).

DISCUSSION

A Non Sequitur: Medical Imaging Should Be Influenced By the LNTH

Discussing potential risks and ignoring corresponding benefits is improper and even harmful (43); unfortunately, quantitative estimates demonstrating relative and absolute benefits of diagnostic imaging are uncommon (6,8). Further, comparing long-term cancer risks with the present benefit from an imaging study is not a like-to-like comparison. For example, in a CT study in young adults, underlying medical morbidity, rather than CT-

induced cancer, was shown as the much greater driver of adverse patient outcomes; the observed risk of a patient dying within 5 years from his/her underlying disease was at least one to two orders of magnitude greater than the hypothetical LNTH-derived risk of dying from a CT-induced cancer (44).

The very concept of dose “optimization” (ALARA-dosing), is one-sided and, therefore, flawed, ignoring much greater, fear-driven risks, along with imaging's likely dual benefits: first, the diagnostic information provided, including more accurate and rapid diagnoses, lives saved, quality-of-life improvements, reduced hospital stays, and cost reduction (8) - e.g., we know CTs strengthen confidence in prior diagnoses, often leading to better treatments and/or more accurate diagnoses (45); and second, the far more likely lifetime-cancer-risk reduction resulting from the radiation itself (21,23,26,30).

A recent study demonstrated a substantial benefit from PET/CT scans to assess response to chemoradiotherapy for primary treatment of patients with squamous-cell, head and neck carcinoma with advanced nodal disease (46). The trial assessed the noninferiority of PET/CT-guided surveillance of planned neck dissection, performed only if imaging showed an incomplete or equivocal response. The primary end-point was overall survival. Survival was similar between patients undergoing PET/CT-guided surveillance or neck dissection, but surveillance resulted in considerably fewer operations (approximately 80% of patients avoided neck dissection), which was additionally more cost-effective. Dissection is generally a three-hour operation, involving both considerable morbidity and potentially long hospital stays. Medical imaging's early and accurate diagnosis reduces mortality, the need for treatment and costs.

Brenner et al. (47) perhaps started the frenzy over CT dose and cancer risk. Based on an LNTH calculation involving unsupported parameters with significant uncertainties, they projected approximately 500 children under the age of 15 years would die of cancer attributable to CT radiation. The irresponsibility of this projection was underscored by the International Commission on Radiological Protection Publication 103 (48) and others stressing that the LNTH's low-dose risk uncertainties show it *should not be used* to calculate hypothetical cancers from small radiation doses received by large populations. According to Lauriston Taylor (25), this type of calculation is based on a literal application of the LNTH, treating it as fact, even though there is no statistical or other verification of this calculation. Such claims, he said, are “deeply immoral uses of our scientific knowledge.” Estimating future CT-caused cancers based on the LNT model's unsupportable assumptions (e.g., the fallacious 5% per Sv cancer risk) results in a purely fictitious prediction, serving only to generate fear-based negative consequences.

The goal of dose management should be aimed at achieving diagnostic-quality images, *not* dose reduction in the hormetic imaging-dose ranges. It has been suggested that 1 in 20 pediatric abdominal CT scans may be inadequate for diagnostic purposes due to radiation-dose-reduction efforts. This will negatively influence the care of some patients from misguided treatment (7). Importantly, doses cited for nuclear medicine and CT examinations do not even represent patient-specific doses, but rather doses resulting

from various models (49). In nuclear medicine, dose-optimizers look to lower administered activities, and dosing guidelines for diagnostic quality images, based on body weight, are available that propose to positively impact uniform pediatric dosing. But approaches based solely on administered activity are insufficient, as they ignore inter-patient biokinetics, which are highly variable, significantly affecting dose estimates and image quality. For example, in some patients, radiopharmaceutical clearance is quicker than average, and the result may be a suboptimal image resulting in lower counts and increased image noise from inappropriately-reduced, administered activity. Recently, a methodology incorporating adjustment for body morphometry, use of age-specific biokinetics, and more detailed phantom modeling has been described as a first step in reducing pediatric absorbed dose while maintaining image quality; but while image quality has objective, measurable properties, its subjective properties (i.e., radiologist or nuclear physician interpretability) are not easily quantified (50).

The goal of the Image Gently Alliance (51) is lowering the potential risk of CT-caused cancer in children, but this risk is hypothetical, lacking credible evidence. Furthermore, based on the LSS data, children are not more radiosensitive than adults in the imaging dose range. The Alliance mainly addresses pediatric, ALARA-based CT “optimization,” but without knowledge of actual patient doses and without demonstrated harm at diagnostic imaging doses, this unintentionally misleads and frightens the public.

All medical procedures require justification in the form of medical indication, but radiation exposure levels have no place in that process. There is no excuse for policies and warnings leading to non-diagnostic scans, fear-driven avoidance of medically-indicated imaging, and/or selection of less optimal alternative procedures. The problem is *radiophobia*, not radiation. Optimization – using doses that are ALARA – is, thus, without justification, only multiplying illnesses, injuries, and deaths. Therefore, the International Commission on Radiological Protection-recommended fundamental principles of radiation protection – justification and optimization – are mutually contradictory and without merit *for radiological imaging* and other sources of low-dose radiation exposure as well.

Many grant the absence of low-dose harm, yet, nevertheless, advocate lower imaging dose as a “prudent” approach; but this conflates actual prudence, restricting medical procedures to those clinically indicated, with the prejudice-based false prudence of limiting clinically-indicated imaging doses. This unjustified, radiophobia-centered approach falsely vilifies beneficial imaging without confirmatory data and entails extremely harmful consequences. The declaration that the LNTH provides “known” cancer risks due to imaging must stop. The use of the LNTH and the advocacy for ALARA dosing by various groups (e.g., Image Wisely and Image Gently) are misguided and not science- or evidence-based. These groups serve only to frighten rather than to educate, further enhancing the probability of negative outcomes; we, therefore, recommend that the imaging community come together to decide whether the activities of such groups should be terminated.

CONCLUSION

Medical Imaging and the Failure of LNTH Orthodoxy

Medical imaging is said to carry iatrogenic risk of cancer from radiation exposure. But credible evidence of cancer risk from imaging, particularly CT and PET/CT scans, is nonexistent; this risk is an *imaginary* prediction derived from the demonstrably false LNTH. Low-dose radiation from these scans does not *cause*, but more likely helps *prevent*, cancer. *Actual* risk arises from radiophobia through patients' fear-driven imaging avoidance and physician-recommended substitution of alternative procedures. Furthermore, *true* iatrogenic risk arises, not only from such alternative procedures, but also from *misdiagnoses* that are secondary either to patient *refusal* of medically-indicated imaging or to *non-diagnostic* scans resulting from insufficient exposure. Obtaining correct diagnoses and avoiding more-risky alternatives should be paramount; medical imaging is intended to achieve a diagnostic purpose and, thus, exposure cannot be reduced below the required level achieving this purpose.

Imaging is a medical procedure that should be governed by the highest, science-based principles and policies (use of proper procedures, appropriately calibrated equipment, etc.). Yet, many believe imaging should be managed by LNTH principles. Herein is the logical and medical fallacy of this conclusion: the LNTH is an invalidated hypothesis, spawning the ALARA principle. It is responsible for misguided concerns promoting dose "optimization," leading to risks far greater than even the imaginary low-dose carcinogenic risk it purports to avoid while ignoring medical imaging's benefits.

With no evidence supporting the LNTH, and much evidence to support hormesis at imaging doses, LNTH advocates are blindly responsible for promoting radiophobia with all its negative consequences. The LNTH and its offspring, ALARA, *do not err on the side of caution*. Radiophobia can no longer be ignored: proper low-dose radiation exposure has no documented pathway to harm, while the LNTH and ALARA most assuredly do.

The only rational and public-health-protective conclusion is that subjecting the life-saving practice of medical imaging to the LNTH is a non sequitur. Medical imaging must no longer suffer in the longstanding thrall of the LNTH. It is incumbent on the medical imaging community to finally and unambiguously denounce the LNTH and, unencumbered by false beliefs, act as advocates for the safety and life-saving benefits of medical imaging.

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