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Radiation Protection Consequences of the Linear Non-Threshold Hypothesis

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Abstract

The linear non-threshold (LNT) hypothesis is based on the premise that even the smallest amount of ionizing radiation produces a biological detriment. It implies that exposure to low-dose radiation be minimized, which causes fear and anxiety regarding the beneficial use of radiation and radioactive materials. This paper examines the LNT premise and associated data, and notes that its intent of protecting the public likely causes physical and economic detriment in contrast with its intended purpose.

Keywords

Linear non-threshold hypothesis; LNT; radiation induced detriment; biological effects of ionizing radiation; radiological risk

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1.0 Introduction

The linear non-threshold (LNT) hypothesis is based on the assumption that any dose of ionizing radiation, no matter how small, produces a biological detriment. This hypothesis has been discussed in the literature and has been a controversial health physics topic since its introduction [1-153] with pro- and anti-LNT proponents having little common ground. This paper examines a representative portion of the data associated with the LNT hypothesis, its influence on the health physics field, and recommendations for its elimination.

A number of concepts are in conflict with and challenge the validity of the LNT hypothesis. Verifying the validity of any of these concepts would invalidate the LNT hypothesis. These concepts are addressed in this paper and include (1) hormesis, (2) the dose dependent response of the human immune system, (3) existence of a threshold for radiation detriment, and (4) modifications used by LNT proponents to justify its continued use (e.g., modifying factors such as the dose and dose rate effectiveness factor (DDREF)).

Current Radiation Protection Regulations are based on a safety paradigm derived from the LNT hypothesis for radiation-induced cancers [135,136] and the associated As Low As Reasonably Achievable (ALARA) concept. This approach was adopted in the 1950s by the various advisory bodies [1-3] following initial evaluations of Japanese atomic bomb survivor

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data. The LNT hypothesis increases public concerns regarding the use of radiation-generating devices and radioactive materials [1-153]. However, the LNT hypothesis is not based on any observed harm from low-dose radiation. Moreover, UNSCEAR 1958 [4] demonstrates a threshold for leukemia occurs at about 500 mSv [4, 112].

Acceptance of the LNT hypothesis occurred before there was a body of data to thoroughly validate its basis. That situation no longer exists and there is significant data available to better evaluate the adequacy of the LNT hypothesis. A representative sample of these data is addressed in this paper.

The LNT hypothesis has been repeatedly endorsed by most international and national advisory bodies. These organizations include the most influential advisory bodies (e.g., International Commission on Radiological Protection (ICRP) [10,11,22,23,26-28,54,60,125], National Academy of Sciences [1,2,6,16,116], National Council on Radiation Protection and Measurements (NCRP) [25,33,40,70], National Institutes of Health [17], National Research Council [13,20,56], Radiation Effects Research Foundation (RERF) [18,42,48,51], and United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) [4,19,36,110] and regulatory organizations (e.g., US Department of Health and Human Services [137-139], US Department of Energy (DOE) [136], and US Nuclear Regulatory Commission (NRC) [135]). It has also been accepted by most professional organizations, but there are exceptions that do not condone its universal application (e.g., American Nuclear Society, French Academy of Sciences, French Academy of Medicine, and US Health Physics Society) [38,55,78,147].

The LNT hypothesis has been debated vigorously and essential elements of this discussion are typified by a recent point-counterpoint paper by Doss and Little [114]. Calabrese [121] reviews the history of the LNT hypothesis and notes that it appears there was significant misunderstanding and misinterpretation of the scientific data, and in some research the promulgation of the model was deliberate scientific misconduct. Cuttler [104] notes abundant research on medical treatments utilizing low to moderate radiation doses present no observations of excess cancer incidence or genetic effects. These papers provide a viewpoint that is in sharp contrast with the LNT basis for contemporary radiation protection regulations.

An understanding of the various LNT positions is only possible by examining the research supporting each faction. This paper attempts that task and the results are summarized in subsequent discussion. The focus of this review is the impact of the LNT hypothesis on the radiation protection field and associated regulatory environment. This regulatory framework drives operational health physics activities including requirements for monitoring radiation dose, developing dose limits, and maintaining doses ALARA. Implications of the LNT approach also affect the acceptance of the use of radiation and radioactive materials and cause the ALARA concept to create harm rather than benefit. That is ALARA becomes *A Law against Radiation Applications*. The societal impact of the LNT hypothesis and ALARA concept is also examined. Some of the consequences of the LNT hypothesis are addressed in the next section.

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2.0 LNT Consequences

It has been argued that the use of the LNT model has affected the public in a negative manner and has caused more harm than benefit (See Section 9). Negative ramifications of the LNT hypothesis and associated ALARA concept include (1) limiting research using radiation and radioactive materials, (2) negatively impacting medical diagnoses, (3) limiting nuclear energy expansion in the US and Europe, (4) inhibiting the achievement of lower costs for radiation related services, (5) slowing recovery from the Fukushima Daiichi accident, and (6) contributing to the unwarranted public fear of radiation and radioactive materials.

Fear of radiation has inhibited research using low-dose radiation in the detection, prevention, and treatment of cancer and other diseases [69,92,142,144]. Unwarranted fears caused by belief in the LNT model has also effectively inhibited research involving unique applications of radiation and radioactive materials. Patients have refused to have computed tomography (CT) scans and physicians are not prescribing these procedures because the LNT hypothesis has created concern for the subsequent radiation detriment. As a compensatory measure, some CT scans are being performed with lower intensity radiation, resulting in poorer image quality which limits a radiologist's capability to diagnose diseases.

The expansion of nuclear energy in the US and Europe has been limited because the radioactive releases resulting from the Three Mile Island, Chernobyl, and Fukushima Daiichi reinforced unjustified fears regarding the effects of radiation. These effects include incorrect assumptions regarding the connection between cancer and hereditary effects and low-doses of ionizing radiation. The associated radiophobia promotes the utilization of higher cost and polluting generating sources that hamper economic growth and create a larger carbon footprint [75,76,89,90,94,103,104,114,123,141,142].

Increased regulations of radiation and radioactive materials and the associated costs to implement compliance further dampen the expansion and use of these tools. Regulations affect consumer, medical, industrial, healthcare, and research applications and result in significantly increased costs with very limited benefit. [75,76,89,90,94,103,104,114,123,141,142]

The mandatory evacuations of the Fukushima Prefecture and its prolonged duration created a pattern of social and societal disruption which was not justified by the actual radiation levels and concentrations of radioactive materials released during the accident. To this day, thousands of residents remain displaced and are unable to return to their homes, farms, and places of business. These disruptions remain because the Japanese government is basing decisions on the LNT hypothesis and the associated fear of low-level radiation exposure and radiation related litigation [89,90,104,114,123,141,142,150].

The evacuations could have been avoided or minimized by improved guidance that more credibly evaluated the radiological health effects, and provided a more credible assessment of the risks of evacuation and postulated radiation detriment. This will remain a difficult task as long as radiological organizations continue to utilize the linear non-threshold model for assessing the biological effects of ionizing radiation. Regulatory guidance based on the LNT hypothesis also

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impacts evacuees of the Fukushima Daiichi accident which continues to prevent their return to the evacuated areas.

It is well-known that the use of the linear non-threshold model has significant implications for nuclear regulations affecting routine operations. It is less obvious that these linear models affect emergencies by setting the criteria for implementation of protective actions including evacuation of the public during a severe reactor accident. By adopting the overly restrictive LNT hypothesis, optimum decisions may not be realized during emergencies. LNT usage increases costs during routine operations. It can also lead to a poor evacuation decision that affects the lives of the public directly impacted by the protective action. As such, the LNT hypothesis needs to be reviewed in terms of the harm it could potentially cause during an evacuation.

3.0 LNT Impacts on Radiation Protection

Radiation protection regulations are currently based on the linear non-threshold hypothesis, but this approach is not universally accepted and issues have been raised regarding its acceptability. One of the concerns with current regulatory models is their LNT basis derived from high-dose and dose rate data (e.g., atomic bomb and medical therapy) extrapolated in a linear manner to low-doses. Other data (e.g., occupational and environmental) are excluded even though the dosimetry is good and the exposed groups are large and well defined. In addition to the inclusion of all dosimetric data, the new regulations should consider a variety of dose response models including those that do not rely on the linear-non-threshold hypothesis. The use of validated data will eliminate the LNT approach and lead to a regulatory model that is based on physical evidence instead of unverified assumptions.

This section provides supporting information that forms a portion of the technical basis for current US radiation protection regulations [135,136]. The basis includes the linear-non-threshold approach and the selection and modeling of dose response models, risk models, excess risk functions, risk coefficients, biological detriments, and the dosimetry associated with these detriments. This paper also illustrates the influence of these models in assessing radiation risk to workers. In order to accomplish this goal, a number of issues and considerations are addressed in this section. These items include, (1) risk, (2) basic epidemiology, (3) dose response models, risk models, and biological effects, (4) BEIR VII uncertainties [56], (5) doubling dose, (6) probability of causation [16,17], (7) US Energy Employees Occupational Illness Compensation Program Act (EEOICPA) [137-139], and (8) future dose limits.

These eight items are either used to support or are affected by the LNT hypothesis. For example, BEIR VII [56] is used to support ICRP 103 [60] and associated regulations that are based on the LNT hypothesis. The LNT hypothesis affects assessments of risk and government programs including the EEOICPA as well as the promulgation of future dose limits. As such, these 8 items form a set of requisite information to address the LNT hypothesis and are addressed in subsequent discussion.

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3.1 Risk

Radiation is one of the most thoroughly studied agents associated with a biological detriment. These detriments are quantified in terms of stochastic and nonstochastic effects [10] and their associated health risks. The risk (R) is often quantified in terms of a risk coefficient (r) expressing excess radiation-induced effects per unit radiation dose (D) [62,68,75,142].

Accordingly, the risk of the radiation exposure is often determined from an assumed LNT relationship

$$\mathbf{R = r\,D} \qquad (1)$$

where the dose is the radiation exposure received and under evaluation. The risk coefficient is model dependent, depends on the data under evaluation, and the underlying modeling assumptions. Risk estimates are also influenced by the radiation characteristics (e.g., dose, dose rate, fractionalization, and radiation type), biological characteristics (e.g., age, sex, genetic background, and nature of the tissue or organ), and the approach to the analysis (e.g., dose-response model, projection model, and risk model).

In view of these factors, it is not surprising that there is considerable variance in risk estimates. For example, the ICRP 26 [10] risk coefficient is 2×10^{-2} radiation induced effects (RIE) / Sv, while BEIR V [20] with its 8×10^{-2} RIE / Sv coefficient yields a larger characterization of the risk. A summary of risk coefficients derived from major studies is provided in Table 1.

Table 1
Ionizing Radiation Risk Coefficient Summary[a]

Year	Report	Risk Coefficient ($\times 1.0 \times 10^{-2}$) (Radiation Induced Effects/Sv)
1972	BEIR I	1
1977	ICRP 26	2
1980	BEIR III	2
1985	EPA NESHAP	4
1988	NRC BRC Policy	5
1990	BEIR V	8
1991	ICRP 60	7

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2006	BEIR VII	5[b]
2007	ICRP 103	6

[a] Bevelacqua [75].

[b] Excess cancer deaths extracted by the author from BEIR VII data [56,75].

Eq, 1 is often applied carelessly. This equation is most valid for a large ensemble of subjects (10,000–100,000) who have each received at least 0.1 Gy of acute radiation exposure. [13,20,56]

The total risk coefficient (r) is the sum of the risk coefficients for the organs or tissues (T) composing the modeled human body:

$$\mathbf{r} = \sum_{T} \mathbf{r}_T \qquad (2)$$

Table 2 summarizes the various organs that are assumed in the ICRP 26 [10], UNSCEAR 88 [19], ICRP 60 [23], and ICRP 103 [60] formulations. The formulations do not contain the same organs or level of organ risk. Table 2 also provides the values of the tissue weighting factors (w_T) for these models:

$$\mathbf{w}_T = \frac{\mathbf{r}_T}{\mathbf{r}} \qquad (3)$$

where the weighting factor is a dimensionless number with a value between zero and unity.

Table 2

Tissue Weighting Factors for Various Models

Tissue	ICRP 26 (1977)	UNSCEAR (1988)	ICRP 60 (1991)	ICRP 103 (2007)
Gonads	0.25	-----	0.20	0.08
Breast	0.15	0.05	0.05	0.12
Bone Marrow (Red)	0.12	0.17	0.12	0.12
Lung	0.12	0.17	0.12	0.12
Thyroid	0.03	-----	0.05	0.04
Bone Surfaces	0.03	-----	0.01	0.01
Stomach	-----	0.18	0.12	0.12

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Colon	-----	0.09	0.12	0.12
Esophagus	-----	0.04	0.05	0.04
Bladder	-----	0.05	0.05	0.04
Ovary	-----	0.03	-----	-----
Skin	-----	-----	0.01	0.01
Liver	-----	-----	0.05	0.04
Multiple Myeloma	-----	0.03	-----	-----
Brain	-----	-----	-----	0.01
Salivary Glands	-----	-----	-----	0.01
Remainder	0.30	0.19	0.05[a]	0.12[b]

[a] The ICRP 60 remainder tissues are: adrenals, brain, small intestine, spleen, kidneys, muscle, pancreas, upper large intestine, thymus, and uterus.

[b] The ICRP 103 remainder tissues are: adrenals, extrathoracic (ET) region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate (♂), small intestine, spleen, thymus, and uterus/cervix (♀).

Tables 1 and 2 illustrate the model dependent variations encountered in the risk estimates. The ICRP 60 [23] and 103 [60] models include more organs with specified organ weighting factors than the ICRP 26 [10] formulation and also include a set of specified organs to be included in the remainder. An examination of Table 2 illustrates the model dependence (i.e., number of organs and assigned weighting factors) of the various ICRP internal dosimetry formulations. For example, the tissue weighting factor for the gonads changed significantly in the ICRP 26 (0.25) [10], ICRP 60 (0.20) [23], and ICRP 103 (0.08) [60] reports.

The risk coefficients summarized in Table 1 and the associated tissue weighting factors summarized in Table 2 are derived from an assessment of the number of radiation induced effects per unit dose. These assessments require that the source of the measured effect be determined and directly related to the radiation dose. This assessment utilizes basic epidemiological principles, which are briefly outlined in the next section of this paper.

3.2 Basic Epidemiology

Studies of radiation risk utilize epidemiological input that requires a sample size dependent on the magnitude of the radiation exposure. The sample size, required for statistically meaningful results, is 5×10^4, 5×10^8, and 5×10^{12} individuals for acute absorbed doses of 100 mSv, 1 mSv, and 0.01 mSv, respectively [24]. These sample sizes are based on LNT model

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based risk assumptions, and suggest that low-dose research is extremely difficult for typical occupational doses. This contention is another bias inherent in the LNT hypothesis that has been negated by more careful analysis [53, 106].

Contrary to LNT dogma, statistically meaningful results have been obtained with much smaller cohorts (e.g. the Taiwan data having an average dose of 50 mSv with a cohort of about 8,000 [53]). Other studies [29,41,52,101,114] demonstrate a statistically significant biological benefit from low-dose, low-dose rate radiation from sample sizes significantly smaller than the LNT required cohorts noted previously. These studies cast significant doubt on the validity of the LNT hypothesis, but this important consideration is omitted from BEIR VII.

The BEIR VII Committee [56] did not know whether dose rates of gamma or x-rays of about 1 mGy/year are detrimental to humans. This is a convenient omission because the data of Frigerio et al [7] shows a dose rate of 1 mGy/year reduced cancers. By ignoring such data, BEIR VII report could claim ignorance, and continue to justify use of the LNT hypothesis.

According to BEIR VII, somatic effects at these doses would be masked by environmental and other factors that produce the same types of health effects as ionizing radiation. Therefore, BEIR VII contends that assessments of the impact of doses on the order of magnitude of 1 mGy or less are not practical from a statistical perspective. Moreover, BEIR VII incorrectly applies the LNT hypothesis to doses approaching zero.

Epidemiological studies must also consider a number of factors including sex, age, time since exposure, and the age at exposure. Accurate studies are also of long duration since time is required to follow the exposed population and associated control group.

The number of cancers expected in a cohort (E) of exposed individuals is given by the sum

$$\mathbf{E} = \sum_{x} \mathbf{c(x)r(x)} \qquad (4)$$

where r(x) is the annual incidence (morbidity) per person at age x per year and c(x) is the sum of all years spent by cohort members at age x. Once the expected incidence is determined, the number of excess cancers (EC) is readily obtained:

$$\mathbf{EC = O - E} \qquad (5)$$

where O is the observed cancer incidence in the risk or exposed population.

The excess cancers per population year per incident exposure (Z) is given by

$$\mathbf{Z = (O - E)/N} \qquad (6)$$

The quantity N has the units of population year-Gy:

$$\mathbf{N = d_i\, y_i} \qquad (7)$$

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where d_i is the dose to the i^{th} group, and y_i is the number of years that the i^{th} group is observed. Therefore, Z is expressed in excess cancers per population year-Gy.

With these definitions, commonly utilized epidemiology terms can be defined. The relative risk (RR), standard mortality ratio (SMR), and excess relative risk (ERR) are defined as:

$$\mathbf{RR = O/E} \tag{8}$$

$$\mathbf{SMR = 100\ RR} \tag{9}$$

and

$$\mathbf{ERR = RR - 1} \tag{10}$$

These terms will be utilized in specific LNT applications in subsequent discussion.

3.3 Dose Response Models, Risk Models, and Biological Effects

The ICRP models should be viewed in their historical context. The models continue to evolve and incorporate available data regarding the biological effects of ionizing radiation. In subsequent discussion, the base ICRP report and its supporting publications are quoted. For example, ICRP 26 uses the lung model and gastrointestinal tract model of ICRP 30.

A portion of the scientific basis for ICRP 26/30 [10,11], ICRP 60/66/30 [11,23,26], and ICRP 103/66/100 [26,54,60] are summarized in Table 3. ICRP 26/30 are based in part on the Biological Effects of Ionizing Radiation (BEIR) III Report [13]. In BEIR III, the dose response relationships for both solid tumors and leukemia are defined to have a linear-quadratic (LQ) dose response relationship:

$$\mathbf{f(d) = ad + bd^2} \tag{11}$$

where f(d) is the effect of the radiation dose or dose response function, d is the effective dose equivalent, and a and b are risk coefficients. BEIR III based its preferred age-specific cancer model on the absolute (additive) risk model:

$$\mathbf{r(d) = r_o + f(d)g(\beta)} \tag{12}$$

where r(d) is the number of cancers of a specific type in the population group, r_o is the natural incidence of the specific cancer type, and g(β) is the excess risk function that contains the time dependence of these effects.

In order to derive its risk coefficients, BEIR III utilized the Temporary 1965 Dosimetry System (T65D) [5]. T65D was based on activation measurements at the Hiroshima and Nagasaki sites.

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Table 3
Comparison of the Basis for Recent ICRP Models

ICRP Model	Basis	Dose Response Relationship[a]		Risk Model
		Solid Tumors	Leukemia	
26/30	BEIR III	LQ	LQ	Absolute
60/66/30	BEIR V	L	LQ	Relative
103/66/100	BEIR VII	L	LQ	Various[b]

[a] L = Linear and LQ = Linear-Quadratic

[b] See Table 5.

BEIR V [20] forms a portion of the basis for ICRP 60/66/30. In BEIR V, the dose response model is linear (L) for solid tumors:

$$\mathbf{f(d) = cd} \tag{13}$$

and linear-quadratic for leukemia. In Eq. 13, c is a risk coefficient. In contrast to BEIR III [13], BEIR V uses a relative (multiplicative) risk model:

$$\mathbf{r(d) = r_o[1 + f(d)g(\beta)]} \tag{14}$$

BEIR III, BEIR V, and BEIR VII assume the dose response models have no threshold (i.e., the non-threshold hypothesis). That is any dose no matter how small has an effect (detriment).

BEIR V is based on the 1986 Dosimetry System (DS86) analysis [18] of the Japanese atomic bomb survivors. DS86 expanded the T65D methodology [5] to include additional numerical analysis of the effects of the nuclear detonations. The DS86 dosimetry lowered the overall delivered dose results in comparison with T65D. This resulted in a larger BEIR V [20] risk coefficient in comparison to BEIR III [13].

There are significant differences between the BEIR III and BEIR V reports. Table 4 illustrates the variation in both leukemia and nonleukemia (solid tumor) cancer risk estimates. The solid tumors include respiratory, digestive, breast, and other cancer types. For leukemia, BEIR V leads to a factor of 4-5 greater risk. A similar increase of about 3-5 occurs for nonleukemia cancers if BEIR III and V relative risk models are compared.

Considerably larger factors of 11-19 occur for nonleukemia cancers if the BEIR III absolute risk model is compared to BEIR V's relative risk model. BEIR VII supports a combination of absolute and relative risk models, and it is compared to BEIR III and BEIR V in Table 5.

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Table 4

Lifetime Cancer Risk Estimates (Deaths per 100,000 persons) [a]

Cancer Type	Continuous Lifetime Exposure 1 mGy/year		Instantaneous Exposure 0.1 Gy	
	Male	Female	Male	Female
Leukemia				
BEIR III	15.9	12.1	27.4	18.6
BEIR V	70	60	110	80
BEIR V/BEIR III	4.4	5.0	4.0	4.3
Nonleukemia				
BEIR III (absolute)	24.6	42.4	42.1	66.5
BEIR III (relative)	92.9	118.5	192	213
BEIR V (relative)	450	540	660	730
BEIR V/BEIR III (relative)	4.8	4.6	3.4	3.4
BEIR V/BEIR III (absolute)	18.3	12.7	15.7	11.2

[a] Derived from Bevelacqua [68].

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The BEIR VII Report is consistent with BEIR V. The key elements of BEIR VII and their comparison with BEIR III and BEIR V are summarized in Table 5.

Table 5
BEIR III, V, and VII Comparison

Parameter/Quantity	BEIR III (1980)	BEIR V (1990)	BEIR VII (2006)
Dose Response Model - Solid Tumors	LQ[a]	L[a]	L
Dose Response Model – Leukemia	LQ	LQ	LQ
Preferred Risk Model	Absolute	Relative	Various [c,d]
Dosimetry System[b]	T65D	DS86	DS02
DDREF[e] (Range)	-----	2-10	1.1-2.3
DDREF (Adopted)	-----	------	1.5 for Linear Models

[a] L = linear LQ = linear-quadratic

[b] T65D = Tentative 1965 Dosimetry [5] DS86 = Dosimetry System 1986 [18]
DS02 = Dosimetry System 2002 [48,51]

[c] For solid cancers other than lung, breast, and thyroid, the preferred risk model is a weighted average (on a logarithmic scale) of relative and absolute risk models with relative risk given a weight of 0.7 and absolute risk a weight of 0.3. These weights are reversed for lung cancer. The preferred breast cancer model is based on the absolute risk model, and the preferred thyroid cancer model is based on the relative risk model.

[d] For leukemia the preferred risk model is a weighted average (on a logarithmic scale) of relative and absolute risk models with relative risk given a weight of 0.7 and absolute risk a weight of 0.3.

[e] Dose and dose rate effectiveness factor.

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The BEIR VII total cancer morality and leukemia risk estimates from radiation exposure have not changed significantly from BEIR V. BEIR VII's risk estimates are based on expanded epidemiological data including cancer incidence data and 15 years of additional mortality follow-up for the Japanese atomic bomb survivors. Studies involving occupational and environmental exposure were evaluated, but not utilized in BEIR VII.

In formulating its risk models, the BEIR VII Report used the revised Dosimetry System 2002 (DS02) for atomic bomb survivors as a portion of the basis for evaluation of the dependence of risk on dose [48, 51]. The risk models were developed from atomic bomb survivor and medical therapy patient data.

DS02 [48,51] superseded the T65D [5] and DS86 [18] analysis with additional activation measurements of long-lived radionuclides produced by the Hiroshima and Nagasaki nuclear detonations. The DS02 dose assessment was about 10% larger than the DS86 assessment which leads to a smaller BEIR VII risk coefficient compared to BEIR V.

BEIR VII also reviewed the dose response model and its functional dependence, the emergence of hormesis as a positive consequence of the radiation dose, and the existence of a threshold for radiation induced effects. Hormesis was proposed in 1980 [12] as a method of reducing cancers. The importance of hormesis and its relationship to the LNT issue are addressed in subsequent discussion.

According to BEIR VII, the updated molecular and cellular data from studies of radiation exposure do not support the postulate that low-doses of low-LET radiation are more harmful than predicted by the linear-non-threshold model. That is, the contention that the dose response curve exhibits supra-linearity is not supported. In addition, the updated molecular and cellular data from studies of radiation exposure do not support hormesis. BEIR VII re-affirms the LNT hypothesis and concludes there is cellular level evidence for the LNT approach. As noted previously, BEIR-VII fosters these conclusions by ignoring important data (e.g., the data summarized in Section 9).

Thresholds were considered in BEIR VII, but not endorsed as representing the best scientific view of low-dose risk. These conclusions are based on an incomplete consideration of the human physiological response to ionizing radiation. BEIR VII only examined the effect of radiation dose shortly after irradiation, and did not consider the time for activation and mobilization of body defense mechanisms. The human immune system and robust DNA repair mechanisms are important considerations ignored by proponents of the LNT hypothesis including BEIR VII. As noted in subsequent discussion, these omissions are a significant weakness that invalidates the LNT hypothesis and its threshold assumption.

BEIR VII also noted that other effects were observed to exist, but were too small to definitively quantify. In particular, BEIR VII concluded that the genetic risks of low-dose, low-LET radiation are very small compared to the baseline frequencies of genetic disease. In addition, a dose response for non-cancer mortality in atomic bomb survivors has been

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demonstrated, but data are not sufficient to determine if this effect exists at low-doses and dose rates. BEIR VII does not provide risk estimates for non-cancer mortality.

The BEIR VII analysis of non-cancer mortality is open to challenge. For example, non-cancer mortality during the period of about 1950-1965 was significantly lower for the cohort that had about 500 – 1000 mGy absorbed dose. This reduction is dismissed by RERF 13 [42] as a healthy survivor effect. Deviation from the LNT hypothesis is a more likely cause, but BEIR VII does not consider this possibility.

Reports such as BEIR VII are important because they are used to refine the internal dosimetry models and affect risk estimates. Consequently, the conclusions of BEIR VII carry significant weight and ideally are clear, unambiguous, and widely accepted. Unfortunately, BEIR VII is accepted because its aforementioned faults are not recognized. The BEIR and ICRP committees utilize the LNT hypothesis as an inherent assumption, and justify this by discounting relevant data. A portion of these data are addressed in Section 9.

3.3.1 Dose Response Relationships

Dose response relationships describe how the effect of an exposure to ionizing radiation varies with dose. Currently, the two most popular dose response relationships are the linear and linear quadratic models. These models are discussed in Section 3.2.

The dose response models utilized in the BEIR reports are zero threshold approaches. This hypothesis leads to the suggestion that detrimental health effects exist at very low-doses. The linear extrapolation from high-dose and dose rate data to low-doses is open to challenge and addressed in subsequent discussion.

Uncertainties associated with the extrapolation from high-dose and dose rate data to the low-dose region are minimized by utilizing the complete set of available radiation dosimetry data. The complete set of radiation data includes a wealth of information including occupational data from nuclear power reactors, DOE weapons complex facilities and national laboratories, universities, medical facilities, and commercial facilities utilizing radioactive materials. Environmental data are also tabulated from high-dose rate areas of the world. In addition, low-dose imaging data and other diagnostic medical data are available. Utilization of the complete set of dosimetry data could significantly improve the justification for the functional dependence of the dose response relationship. These relationships should consider thresholds and functions more diverse than the assumed linear or linear quadratic dose response models.

3.3.2 Risk Models and Biological Effects

There are two general types of models that are traditionally used in assessing risk. These are the absolute and the relative risk models that were outlined in Section 3.2.

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As noted in Table 6, the BEIR models have been applied to a variety of cancer types. An example of their application to leukemia and nonleukemia cancers illustrates the conclusions drawn by the BEIR Committees in attempting to assess radiation risk. This is illustrated by summarizing a portion of the BEIR V report [20] and its conclusions regarding the relative risk model.

These conclusions are specific and well defined, but are based on high-dose and dose rate data. As noted previously, their extrapolation to low-doses using linear-non-threshold models is open to challenge.

Table 6

BEIR V Preferred Relative Risk Model[a]

Cancer Type	Dose Response Model	Comments
Leukemia	Linear quadratic	Minimum latency of 2 years.
Breast	Linear	Highest risk in women under age 15 at the time of exposure. Risk is low for women if exposed after age 40.
Respiratory	Linear	Minimum latency of 10 years. Risk decreases with time after exposure. Relative risk for females is twice that for males.
Digestive	Linear	About seven times the risk if exposure occurs at age 30 or less. Risk does not change with time post exposure.
Other	Linear	Contributes significantly to total risk. No age or sex effects have been noted. Insufficient data to permit detailed modeling.

[a] BEIR V does not support the absolute risk model [20].

The assumed functional forms of the absolute and relative risk models and the typical exponential and step functions used in the excess risk function are not unique. Other functional relationships should be investigated with the utilization of complete dosimetry data sets.

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The large lifetime cancer risk uncertainties illustrated in Table 4 also suggest that an investigation of other functional forms for the dose response model, risk model, and excess risk function is warranted. This investigation should also include thresholds and all available dosimetry data.

3.4 BEIR VII Uncertainties

Although BEIR VII [56] does not provide an excess cancer risk coefficient, a public risk coefficient for excess cancer deaths per Sv can be developed from the report's Table 13-1 data. An illustration of the uncertainties involved in the BEIR VII analysis is provided by developing this risk coefficient.

Using BEIR VII data, the number of excess cancer deaths (ecd) from exposure to 0.1 Gy to males is 410 (200, 830) ecd in an exposed population of 100,000 individuals. The values in parenthesis are the 95% confidence intervals. For females, the number of excess deaths from exposure to 0.1 Gy is 610 (300, 1200) ecd in an exposed population of 100,000. These distributions are broad and indicate the uncertainties encountered in the BEIR VII analysis. When considered in the historical context of the BEIR III [13] and V [20] reports, summarized in Table 4, a view of data uncertainty is provided. This uncertainty suggests that many functional forms could be used to fit the available data. Limiting the analysis of evaluated data sets to linear and linear quadratic models with no thresholds is not unique and open to challenge. In addition, restricting the analysis to absolute and relative risk models or combinations of these models is also overly restrictive. Other functional forms, the existence of thresholds, and a variety of risk models should be evaluated to ensure that radiation protection regulations and evaluation of the radiation induced biological detriment are based on an unbiased analysis.

The public risk coefficient for all cancers is obtained by averaging over age and sex (410 ecd + 610 ecd)/2 which produces a value of 510 ecd. These data can be used to obtain a corresponding risk coefficient:

$$\mathbf{r} = \frac{\left(\frac{\mathbf{410ecd + 610ecd}}{\mathbf{2}}\right)}{\mathbf{(100{,}000persons)(0.1Gy/person)}} \frac{\mathbf{1Gy}}{\mathbf{1Sv}} = \mathbf{5x10^{-2}}\,\frac{\mathbf{ecd}}{\mathbf{Sv}} \qquad (15)$$

As a comparison, BEIR V [20] derived a value of 695 ecd/100,000 persons exposed to 0.1 Gy (no DDREF was utilized in BEIR V). This is again averaged over males and females [(660 ecd + 730 ecd)/2 = 695 ecd]. If the BEIR VII DDREF is applied to the BEIR V data, 695 ecd/1.5 provides a value of 463 ecd. Using the methodology illustrated by Eq. 15 and keeping one significant figure, lead to a public, excess cancer death risk coefficient for BEIR V of 5×10^{-2} ecd/Sv. Therefore, BEIR V, BEIR VII, and ICRP 60 have the same excess cancer risk coefficient of 5×10^{-2} ecd/Sv. This calculation illustrates the consistency of these reports.

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The LNT hypothesis is an expedient regulatory model, but it is not universally accepted. For example, a number of professional organizations including two French Academies [55] have challenged the BEIR VII Report's conclusion regarding the LNT hypothesis. Issues associated with the LNT hypothesis are addressed in subsequent discussion.

3.5 Doubling Dose

The qualitative relationship between radiation dose and the probability of a mutation is often described in terms of the doubling dose. The doubling dose is the radiation dose that would lead to a doubling of the natural mutation rate. Table 7 summarizes the doubling dose from the BEAR [2] and from BEIR I [6], III [13], V [20], and VII [56] Reports. A doubling dose of about 1.0 Sv appears to a consistent value from the reports summarized in Table 7.

Table 7
Doubling Dose

Report	Doubling Dose (Sv)
BEAR (1956)	0.05 – 1.0
BEIR I (1972)	0.20 – 1.0
BEIR III (1980)	0.50 – 2.5
BEIR V (1990)	< 1.0
BEIR VII (2006)	1.0

The doubling dose is an important consideration in an assessment of the LNT hypothesis because it suggests 1 Sv is required to produce a mutation rate corresponding to the natural rate. The natural rate of mutations is part of nature and man has adapted and evolved with this condition. In particular, the significant redundancy in DNA structure and its robust repair mechanisms adequately counter natural mutations. The influence of DNA repair and its effect on the LNT model are addressed in subsequent discussion.

3.6 Probability of Causation

A consequence of the LNT hypothesis is that its assumptions are replicated in derivative work. One of the LNT derivatives is the concept of Probability of Causation, which is used to assess if a disease or detriment is attributable to radiation exposure.

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US Public Law 97-414, the Orphan Drug Act of 1984, directed the Secretary of Health and Human Services to construct radioepidemiological tables providing the probability that certain cancers could result from prior exposure to ionizing radiation [68,75]. The probability of causation (PC) is defined as a number that represents the probability that a given cancer, in a specific tissue, has been caused by a previous exposure or series of exposures to a carcinogenic agent such as ionizing radiation.

The PC tables are based upon the BEIR III report. National Institutes of Health Publication No. 85-2748 [17] established the foundation for the PC concept for radiogenic tumors. The original PC tables are outdated because BEIR III has been superseded by BEIR V and VII.

Probability of Causation has the form

$$\mathbf{PC = R_e/(1 + R_e)} \quad (16)$$

where R_e is the relative excess. In the case of a single exposure of short duration to an individual representative of a population group, the relative excess is given by

$$\mathbf{R_e = F\ T\ K} \quad (17)$$

In this equation, F is the exposure factor that characterizes the dependence

of R_e on the radiation dose to the risk organ. The use of effective dose from an external dosimeter is not appropriate because absorbed dose in tissue is the desired quantity. The appropriate value of *F* is defined as a function of absorbed tissue dose (D), measured in cGy. The factors T and K are defined in the subsequent discussion.

The specific functional form for F depends on the radiation quality and cancer site. For example, a consideration of ^{224}Ra irradiating the bone and leading to bone cancer results in the simple relationship

$$\mathbf{F_{Bone} = D} \quad (18)$$

for high-LET alpha radiation. For low-LET radiation, the values of *F* for thyroid,

breast, and other cancers are

$$\mathbf{F_{thyroid} = D} \quad (19)$$

$$\mathbf{F_{Breast} = D} \quad (20)$$

and

$$\mathbf{F_{other} = D + (1/116)D^2} \quad (21)$$

The second factor (T) in the definition of relative excess (Eq. 17) represents the relative likelihood that a cancer induced at age A_1 will be diagnosed after Y years. For diagnosis times between Y and Y + 1 years, Y is utilized in the computation. Under the relative risk model, which is used for cancers other than leukemia and bone cancer, T depends only on Y and has a

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value that increases with Y. For Y = 0–4 years, T = 0 and it rises to a value of unity for Y ≥ 10 years. T values of 0.25, 0.5, and 0.75 occur at about Y = 6, 7, and 8 years, respectively.

The relative risk model has not been assumed to hold for bone cancer and leukemia. For these two cancer types, T is a conditional probability, which assumes that the cancer has been caused by an exposure at age A_1 and will be diagnosed Y years later. For these cases, T is calculated as the lognormal probability that a cancer is detected between years Y and Y + 1 after exposure at age A_1. The PC tables compile T for the various forms of cancer.

The final factor defining the relative excess is K, and it provides the dependence of R_e on age and baseline cancer incidence for persons of age A_2 and sex (S) for exposure at age A_1:

$$\mathbf{K = K(A_1, A_2 \text{ , and } S)} \quad (22)$$

The PC tables include both human and animal data. Smoking data is also included, but prior medical exposure is not included.

The reader has by now drawn the conclusion that the PC concept is not precise. A qualitative estimate of the uncertainties in the PC estimate is illustrated by a few examples. If the PC is calculated to be 2% or less, the true PC could be as large as 7% even if an accurate knowledge of all the input parameters is known. If the PC is within the 5–10% range, the true PC could lie within the 1–30% range. Finally, if the PC is calculated to be a least 20%, the true PC could be in the 5–40% range.

A final complication of the PC concept lies in its ties to the BEIR III methodology. The differences between BEIR III and BEIR V and VII suggest that a review of the current PC approach and its underlying assumptions is in order. This is addressed in the next section.

As noted earlier, the PC concept is a derivative of the LNT hypothesis. Assessing the probability of a radiation-induced detriment (i.e., cancer) based on the LNT hypothesis unnecessarily biases the likelihood of associating low-doses of ionizing radiation with cancer or another detrimental effect. This association overestimates the radiation risk and perpetuates the fear and bias regarding the use of radiation and radioactive materials for the benefit of society.

3.7 Energy Employees Occupational Illness Compensation Program Act

The probability of causation concept has been subsequently revised, and it is now used as the basis for determining the legal standard for resolving radiation related claims associated with the Energy Employees Occupational Illness Compensation Program Act of 2000 (EEOICPA) as outlined in 42CFR81, 42CFR82, and 42CFR83 [137-139]. EEOICPA models associated with the US Department of Energy Compensation Program incorporate relevant epidemiology, BEIR reports, and ICRP reports available at the time of its enactment.

The methods for calculating internal dose from the intake of radioactive material use the ICRP 66 Human Respiratory Tract Model [26] and the ICRP 30 ingestion model [11]. In addition, supporting radionuclide data (e.g., ICRP 56 [22], 67 [27], and 68 [28]) are utilized in

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the internal dose assessment. The EEOICPA permits calculational methods to be updated to reflect new reports and science as they become available. For example, ICRP 100 provides the Human Alimentary Tract Model [54] that updates the ICRP 30 ingestion model [11].

The EEOICPA established that a lump-sum payment and medical benefits can be awarded as compensation to covered employees suffering from designated illnesses (e.g., cancer resulting from radiation exposure) incurred as a result of their performance of duty for the Department of Energy and designated contractors. Under EEOICPA, an employee seeking compensation for cancer is eligible if the cancer has a 50% or greater probability of being caused by radiation doses incurred in the performance of duty or the employee is included in a specified cohort.

The risk models address a number of cancer types and most types of radiation exposure are relevant to employees covered by the EEOICPA. These models include the employee's cancer type, year of birth, year of cancer diagnosis, exposure information, and the dose received from gamma radiation, x-rays, alpha radiation, beta radiation, and neutrons. In addition, the risk model for lung cancer includes the worker's smoking history and radon exposure, and the risk model for skin cancer incorporates race and ethnicity. None of the risk models explicitly includes exposure to other occupational, environmental, or dietary carcinogens. Models incorporating chemical agents have not yet been developed.

Although it is appropriate for an organization to compensate workers for harm incurred through employment, the PC concept is also biased by the assumed LNT hypothesis. Accordingly, the PC results are inherently limited by the LNT issues that were noted previously.

In particular, the overestimation of cancer risk resulting from the LNT hypothesis further exacerbates the fears associated with the use of radiation and radioactive materials. The LNT bias perpetuates radiophobia and assigns the cause of a detriment to radiation without investigating its actual cause. The cause often should be attributed to environmental factors, natural causes, genetic effects, or toxic substances.

3.8 Dose Limits

The aforementioned discussion involves risk and its characterization. This characterization and the establishment of dose limits, risk coefficients, tissue weighting factors, and probability of causation tables are a direct consequence of the ICRP, NCRP, and other scientific organizations and their assessment of radiation and its associated detrimental health effects. These recommendations and the characterization of risk are reflected in national regulations that govern radiation protection activities. The regulations provide defined dose limits that are derived from the risk estimates.

Radiation protection regulations are currently based on the flawed LNT hypothesis. The current regulatory basis also assumes that detrimental effects occur in a linear, direct relationship with the dose delivered to an individual. The LNT hypothesis is examined in subsequent

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discussion. In particular, arguments and data supporting and challenging the LNT hypothesis are presented.

4.0 Overview of the LNT Hypothesis

The regulatory basis for radiation protection recommendations and limits assumes the validity of the linear-non-threshold hypothesis. Although there is considerable research that contradicts the LNT hypothesis, this model remains the basis for radiation protection regulations and assessments of radiation induced detriment.

The current radiation safety basis using the LNT hypothesis was introduced following the observation of linear dose dependence of leukemia in atomic bomb survivors, and the observation of linear dose dependence of mutations in drosophila. Both of these observations occurred at high doses, and these studies are not applicable for low-dose radiation. Linking the two high-dose radiation data sets and extrapolating these sets linearly to low-doses is an assumption that merits challenge since radiation protection regulations are based on their validity. Subsequent discussion will examine these issues in more detail.

A corollary to the LNT hypothesis is the introduction of the collective-dose assumption. Collective dose is the sum of individual doses in an exposed group, and is a method for quantifying dose in a population group. This assumption presumes that small doses to large populations can be summed to predict a set of calculated health effects that are representative of the population risk.

Collective dose often overstates the presumed risk and equivalent collective doses do not imply equivalent risk. For example, a large dose to members of a small group is not equivalent to a small dose to members of a large group, even if the collective doses are the same. For groups in which individual lifetime doses are less than 100 mSv above background, collective dose is a speculative and uncertain measure of risk [13]. It should not be used for estimating the health risks or radiation induced detriment to an exposed population. Unfortunately, this occurs routinely and adds to the public's radiophobia regarding the use of radiation and radioactive materials.

The previous discussion has provided an overview of the foundations for radiation protection regulations and its LNT foundation. Subsequent commentary proceeds to a review of the data supporting and refuting the LNT hypothesis. A review of this data is necessary because the subject is complex and often focuses on selective aspects of the LNT hypothesis. As will be noted in subsequent discussion, LNT supporters have not addressed this topic in a comprehensive manner that includes a range of radiobiological effects, human immune system response to low-dose ionizing radiation, hormesis, and threshold effects.

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4.1 Overview of Reports Supporting the LNT Hypothesis

The current radiation safety paradigm is based on the LNT hypothesis for radiation detriment. The LNT hypothesis was adopted in the 1950s by the various advisory bodies [1-4]. The adoption derived from concerns about the expanding use of radioactive materials and radiation-generating devices. It was based, in part, on atomic bomb survivor data that has subsequently been revised [98]. This high-dose and dose rate data was extrapolated to zero dose using a linear relationship that utilized no threshold. This presumed hypothesis was not based on low-dose and dose rate data, and relied heavily on the high-dose and dose rate survivor data from the atomic bomb attacks on Hiroshima and Nagasaki.

The LNT hypothesis continues to be recommended by most national and international advisory and regulatory bodies. Moreover, it is accepted by many professional organizations and remains the basis for contemporary radiation protection regulations. A summary of these organizations and their LNT supporting information is summarized in Table 8.

Although the references cited in Table 8 provide specific items to support the LNT hypothesis, their essential arguments are summarized by Brenner [39]. At low and intermediate doses (10 mGy to 1 Gy), Brenner notes that mutation and chromosome aberration induction data are consistent with a linear dose-response relation [39]. NCRP 136 [40] supports this view and notes: "*although other dose-response relationships for the mutagenic and carcinogenic effects of low-level radiation cannot be excluded, no alternate dose-response relationship appears to be more plausible than the linear-nonthreshold model on the basis of present scientific knowledge*".

As will be demonstrated in Section 9, these arguments do not include the effect of body defense mechanisms which are robust at low doses, and invalidate the conclusions of Brenner and the NCRP. Both the NCRP and Brenner ignore relevant data that yield a very different perspective than offered by the LNT hypothesis.

At lower doses, biophysical arguments are used by Brenner to justify the LNT hypothesis. These arguments include:

1. Tumors are largely of monoclonal origin;
2. Ionizing radiation produces sufficient damage in a cell to initiate oncogenesis; and
3. As the dose of ionizing radiation decreases, fewer cells are damaged by more than one radiation track. This results in a proportional decrease in the number of cells in which this damage occurs. The proportional decrease remains valid at very low-doses.

Following Brenner, the proportional decrease in the limit of zero dose forms the basis for the LNT hypothesis. If this proportionality can be unambiguously demonstrated as doses approach zero, the LNT hypothesis becomes the LNT Law.

Brenner fails to recognize that covert cancers exist in the body even in the absence of radiation exposure [115, 124]. In addition, adaptive response would reduce the mutations following low-dose radiation exposure [79,109]. By ignoring these data, Brenner draws the

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same flawed conclusions as other LNT proponents. Additional arguments against the LNT hypothesis and Brenner's contentions are provided in subsequent discussion.

Table 8
Representative Research and Associated Documents Supporting the LNT Hypothesis

Reference	LNT Support	Primary Conclusions
ICRP 26 [10]	Radiation protection recommendations are based on the LNT hypothesis.[a]	International consensus group of senior radiation protection experts support the LNT hypothesis in establishing radiation protection recommendations. BEIR III and T65D are supporting publications. ICRP 26 is based on data, a portion of which is challenged in Table 11.
BEIR III [13]	Evaluation of radiation detriment data by the BEIR III Committee supports the non- threshold hypothesis.[a]	Solid cancers and leukemia are modeled as a linear quadratic dose response function without threshold. BEIR III is based on data, a portion of which are challenged in Table 11.
BEIR V [20]	Evaluation of radiation detriment data by the BEIR V Committee supports the non- threshold hypothesis.[b]	Solid cancers are modeled as a linear dose response function without threshold. Leukemia is modeled as a linear quadratic dose response function without threshold. BEIR V is based on data, a portion of which are challenged in Table 11.
ICRP 60 [23]	Radiation protection recommendations are based on the LNT hypothesis.[b]	International consensus group of senior radiation protection experts support the LNT hypothesis in establishing radiation protection recommendations. BEIR V and DS86 are supporting publications. ICRP 60 is based on data, a portion of which are challenged in Table 11.
NCRP 116 [25]	Radiation protection recommendations are based on the LNT hypothesis.	US consensus group of senior radiation protection experts support the LNT hypothesis in establishing radiation protection recommendations. NCRP 116 is based on data, a portion of which are challenged in Table 11.

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Table 8

Representative Research and Associated Documents Supporting the LNT Hypothesis

Reference	LNT Support	Primary Conclusions
NCRP 136 [40]	Data evaluation by the NCRP 136 Committee supports the LNT hypothesis[c]	US consensus group of senior radiation protection experts evaluate available data and conclude that the LNT hypothesis is the appropriate vehicle for establishing radiation protection recommendations. NCRP 136 is based on data, a portion of which are challenged in Table 11.
Wakeford and Little [44]	Excess cancers are purported by Wakeford and Little to be observed at doses as low as 10 mSv.[d]	Wakeford and Little suggest that there is evidence of excess cancer incidence associated with radiation exposures of the order of 10– 20 mGy from diagnostic x-ray exposure in the Oxford Survey of Childhood Cancers and in various other groups exposed *in utero*. These authors also propose a consistency of the childhood cancer risk coefficients derived from the Oxford Survey and from the Japanese atomic bomb survivor cohort irradiated *in utero*. Rebuttal of the flawed study and conclusions of Wakeford and Little are provided in Table 11.
Preston et al [42]	The 2003 data set appear to be consistent with the LNT hypothesis.[e]	In Radiation Effects Research Foundation (RERF) Report 13, Preston et al continue the series of general reports on mortality in atomic bomb survivors. Preston et al conclude that the excess solid cancer risks appear to be linear even for doses approaching the origin. A counter argument to the conclusions of RERF Report 13 is provided in Table 11.
Cardis et al [50]	The original 2005 data show a statistically significant increase in cancers in radiation workers.[f]	Cardis et al (2005) was quoted by the BEIR VII [56] report to infer an increased cancer risk from low-dose radiation and to validate the radiation cancer risk factor. According to Cardis et al, the combined data from 15 countries show a statistically significant increase in cancers in radiation workers which led the authors to conclude that low-dose radiation increases the cancer risk. This result was driven by the Canadian data that suggested a much higher risk than data from other countries. A counter argument to the conclusions of Cardis et al is provided in Table 11.

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Table 8

Representative Research and Associated Documents Supporting the LNT Hypothesis

Reference	LNT Support	Primary Conclusions
BEIR VII [56]	Evaluation of radiation detriment data by the BEIR VII Committee supports the non-threshold hypothesis.[g]	Solid cancers are modeled as a linear dose response function without threshold. Leukemia is modeled as a linear quadratic dose response function without threshold. International consensus group of senior radiation protection experts support the LNT hypothesis in establishing radiation protection recommendations. BEIR VII is based on data, a portion of which are challenged in Table 11.
ICRP 103 [60]	Radiation protection recommendations are based on the LNT hypothesis.[h]	International consensus group of senior radiation protection experts support the LNT hypothesis in establishing radiation protection recommendations. BEIR VII [56] and DS02 [48,51] are supporting publications. ICRP 103 is based on data, a portion of which are challenged in Table 11.
Yablokov et al [74] and Levinger [148]	Quoted Chernobyl data confirms the LNT hypothesis.	In a review of the 1986 Chernobyl accident, Yablokov et al [74] presented a detailed analysis of the resulting radiation deaths. These authors suggest that the earlier estimate of 50,000 deaths should be doubled. Levinger suggests these data confirm the LNT hypothesis. The assertions of Yablokov et al and Levinger are challenged in Table 11.
Shimizu et al [82] and Levinger [148]	As quoted by Shimizu et al and Levinger, the atomic bomb data supports the LNT hypothesis between 0.1 and 2.5 Gy with no apparent threshold.	Levinger asserts that the data of Shimizu et al suggest a correlation of radiation exposure and circulatory disease risk from 1950 to 2003 for survivors of the Hiroshima and Nagasaki atomic bomb attacks. Levinger suggests that Figures 1 and 2 of Shimizu et al provide a linear fit of disease risk and radiation dose from 0.1 Gy to 2.5 Gy that indicates no indication of a threshold in either figure. The assertions of Shimizu et al and Levinger are challenged in Table 11.
Beyea [91]	The Soviet Techna River data, as interpreted by Beyea, support the LNT hypothesis.	Beyea evaluated radiation exposures from the radioactive contamination in the Techa River in the former Soviet Union. Beyea suggests a linear response for absorbed doses and that linearity holds at least to 100 mSv. The assertions of Beyea are challenged in Table 11.

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Table 8

Representative Research and Associated Documents Supporting the LNT Hypothesis

Reference	LNT Support	Primary Conclusions
Pearce et al [99]	Based on CT data, brain cancer risk increases with radiation dose.	Pearce et al observed an increased incidence of cancers following childhood CT brain scans and is routinely quoted as evidence for cancer risk from low-dose radiation (e.g., Leuraud et al [127]). The authors suggest that the brain cancer risk increases with radiation dose. The assertions of Pearce et al are challenged in Table 11.
Little et al [96] and Little [108]	Low-dose radiation has an associated detriment.[i]	In a systematic review and meta-analysis, Little and coworkers suggested an excess radiation risk at dose levels below 500 mSv. These authors also argue that there is accumulating evidence from the Japanese atomic bomb survivors and various moderate and low-dose exposed groups of excess risk of circulatory disease and cataracts. The assertions of Little and coworkers are challenged in Table 11.
BEIR VIII Planning Meeting [116]	Initial selection of relevant data continues to support the LNT hypothesis.	The BEIR VIII Planning Meeting continues to rely on data supporting the LNT hypothesis. The data and associated assumptions advocated during the BEIR VIII Planning Meeting are challenged in Table 11.
ICRP 131 [125]	Radiation protection recommendations are based on the LNT hypothesis.[j]	ICRP 131 notes the LNT model is used for the purpose of establishing radiation protection regulations. However, as noted on page 73 of the report, support for the LNT hypothesis is less assertive than previous ICRP publications. The LNT assertions of ICRP 131 are challenged in Table 11.
10CFR20 [135]	US Radiation Protection Regulations for Nuclear Regulatory Commission Licensees are based on the LNT hypothesis[a,h]	NRC Regulations are based on the recommendations of ICRP 26 which incorporates the LNT hypothesis. The LNT basis of 10CFR20 is challenged in Table 11.

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Table 8

Representative Research and Associated Documents Supporting the LNT Hypothesis

Reference	LNT Support	Primary Conclusions
10CFR 835 [136]	US Radiation Protection Regulations for Department of Energy Licensees are based on the LNT hypothesis[a,b,h]	DOE Regulations are based on the recommendations of ICRP 26 (dose limits) and ICRP 60 (computational and dosimetric assessments) which are based on the LNT hypothesis. The LNT basis of 10CFR835 is challenged in Table 11.
Alemayehu and Cochran [140]	The LNT hypothesis is valid because national and international organizations support its use.	Alemayehu and Cochran are a recent example of authors that support the LNT hypothesis because it is supported by national and international organizations. This argument relies on the authority of national and international organizations that presumes their correctness. The assertions of Alemayehu and Cochran are challenged in Table 11.

Additional discussion regarding the LNT hypothesis, as advocated by the noted reference, are provided in the following sections of this paper:

[a] Section 4.2.1.

[b] Section 4.2.2.

[c] Section 4.2.8.

[d] Section 4.2.5.

[e] Section 6.0.

[f] Section 4.2.6.

[g] Section 4.2.3 and 4.2.9.

[h] Section 4.2.3.

[i] Section 4.2.7.

[j] Section 4.2.10.

The sheer weight of the organizational support for the LNT hypothesis (e.g., BEIR, IAEA, ICRP, NCRP, RERF, UNSCEAR, USDOE, and USNRC) discourages attempts to

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challenge its basic assumptions and supporting data. Inertia and the power of established organizations are powerful obstacles, but subsequent discussion attempts to offer a counter argument to the LNT hypothesis.

4.2 Key References Supporting the LNT Hypothesis

The Table 8 references, particularly the NCRP, ICRP, and BEIR reports are used as justification for the LNT hypothesis and these reports have focused upon a subset of available data in their evaluations. These data primarily include information supporting the LNT hypothesis, but ignore opposing research.

Many of the Table 8 references are linked and provide mutual support. These supporting references and their relationship are summarized in Table 9 that provides the BEIR and associated dosimetry data and ICRP reports used by the regulatory agencies to justify their legal requirements. These self-supporting documents provide a concise package for justifying the LNT hypothesis. This linkage and the reputations of organizations promulgating their reports is a significant obstacle for arguments against the LNT hypothesis. However, there is considerable data that challenges the LNT hypothesis and these data are presented in Section 9.

Table 9
Linkage Between US Regulations and Publications Supporting the LNT Hypothesis

US Regulation (Regulator)	BEIR Report (date)	Dosimetry Report (date)	ICRP Report (date)
10CFR20 (Nuclear Regulatory Commission)	III (1980)	T65D (1968)	26 (1977)
10CFR835 (Department of Energy)	III (1980)-dose limits V (1990)-methodology	T65D (1968) – dose limits DS86 (1986) - methodology	26 (1977)-dose limits 60 (1991)-methodology
10CFR835 (Department of Energy) – Under consideration[a]	VII (2006)	DS02 (2004)	103 (2007)

[a] A similar revision to 10CFR20 was considered, but subsequently abandoned.

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4.2.1 ICRP 26, BEIR III, 10CFR20, and 10CFR835

As noted in Table 9, many of the references summarized in Table 8 are linked historically. ICRP 26 [10], BEIR III [13], and T65D [5] form the technical basis for the dose limits of 10CFR20 [135] and 10CFR835 [136]. 10CFR20 also utilizes ICRP26, BEIR III. and T65D as the basis for its calculational methodology. Most of the references summarized in Table 8 were published after ICRP 26, BEIR III, and T65D. Luckey's book *Hormesis with Ionizing Radiation* [12] was published in 1980, but did not have a significant influence on ICRP 26 and BEIR III or the original and subsequent revisions to 10CFR20, and 10CFR835. The ICRP 26, BEIR III, T65D, 10CFR20, and 10CFR835 series of publications and their supporting documents are based on the inherent validity of the LNT hypothesis. ALARA considerations and requirements follow from this presumption.

4.2.2 ICRP 60, BEIR V, and 10CFR835

The calculational methodology utilized in 10CFR835 [136] is based on BEIR V [20], DS86 [18], and ICRP 60 [23] which continued use of the LNT hypothesis in spite of the growing volume of references questioning its validity. As noted in Table 5, BEIR V did acknowledge the dose and dose rate effectiveness factor, but did not incorporate its use in developing recommendations or risk estimates.

The DDREF is a modifying factor that suggests an inherent weakness in the LNT hypothesis. The implications of utilizing a DDREF in BEIR VII (Section 4.2.3) should raise concerns regarding the validity of the LNT hypothesis. Adjustment factors such as the DDREF are accepted as part of the LNT dogma, and publications continue to support its justification and use. However, the DDREF and supporting factors (e.g., dose effectiveness factor and dose rate effectiveness factor) inherently partition the dose into regions that are distinguished by a biological or physics effect. This partition is an obvious challenge to the purity and inherent basis of the original LNT hypothesis.

4.2.3 ICRP 103, BEIR VII, and Revisions in Development for 10CFR20, and 10CFR835

Emerging regulations will likely incorporate BEIR VII [56], DS02 [48,51], and ICRP 103 [60] that continue the use of the LNT hypothesis. BEIR VII accepts the use of the dose and dose rate effectiveness factor, and establishes a range of values for low-linear energy transfer radiation for doses below 1 Sv. Therefore, there is a basis for application of the DDREF into two dose regions that is in logical conflict with a pure LNT model. The DDREF application is a tacit admission that the LNT model has been adjusted to account for other effects. These adjustments present a logical conflict to the proponents of the original LNT hypothesis. The author cannot reconcile the use of the DDREF and associated factors within the basis for the LNT hypothesis.

In 2015, proposals [128] were submitted to the NRC to reformulate 10CFR20 [135] and eliminate the use of the LNT hypothesis, but the regulator continues to promote the linear

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approach. Those proposals were reviewed, but rejected. No further modifications to 10CFR20 or 10CFR835 have been issued.

This LNT philosophy appears to be a continuing theme in the initial BEIR VIII discussions [116]. The LNT hypothesis is maintained as accepted dogma in spite of an expanding data set of research suggesting this approach is flawed. A body of research describing LNT weaknesses is addressed in subsequent discussion.

4.2.4 Atomic Bomb Survivors

Calabrese [121] provided an historical assessment of how prominent radiation geneticists in the United States during the 1940s and 1950s successfully used atomic bomb survivor and other epidemiological data to foster acceptance of the LNT hypothesis. These actions were instrumental in the development of the 1956 report by a Genetics Panel of the U.S. National Academy of Sciences (NAS) on Biological Effects of Atomic Radiation (BEAR) [1-2]. The 1956 report recommended that a linear dose response model be adopted for risk assessment. This recommendation was accepted, widely promulgated, and continued through a series of reports by the Radiation Effects Research Foundation (RERF).

In RERF Report 13, Preston et al [42] continues the series of general reports on mortality in the cohort of Japanese atomic bomb survivors. The noncancer data are consistent with some non-linearity in the dose response which is attributed to substantial uncertainties in the information. In addition, there is no direct evidence of radiation effects for doses less than about 500 mSv. In spite of these limitations, the data appear to be consistent with the LNT hypothesis [42]. Report 14 was the next generation of this RERF series and offered a different viewpoint that no longer supported linearity. RERF Report 14 is addressed in subsequent discussion.

4.2.5 Wakeford and Little

Wakeford and Little [44] refer to the Oxford Survey of Childhood Cancers, but that data remains controversial. The purported consistency of childhood cancer risk factors from Oxford and Japanese studies were suggested by Wakeford and Little as evidence for carcinogenicity of *in utero* low-dose radiation. In the Japanese study, leukemia was observed following high-dose radiation, and the risk coefficients were calculated using an LNT model. This approach created the perception of increased leukemia risk from low-dose radiation, but no effect was observed. Also, cohort studies by Brent [112] do not demonstrate an increased risk of leukemia risk. Further discussion of the weakness in the arguments of Wakeford and Little [44] are outlined by Doss [114] and provided in Section 9.

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4.2.6. Cardis et al

Cardis et al [50] was utilized in the BEIR VII report [56] to infer an increased cancer risk from low-dose radiation and to validate the radiation induced cancer risk factor. The combined data from 15 countries show a statistically significant increase in cancers in radiation workers which led the authors to conclude that low-dose radiation increases the cancer risk. This result was driven by the Canadian data that suggested a much higher risk than data from other countries.

The results of this study suggest that there is a small excess risk of cancer. This risk also occurs at the low-doses and dose rates typically received by nuclear workers. However, if the Canadian data were removed, the combined risk from the remaining countries would not be statistically significant. The validity of Cardis et al [50] depends on the viability of the Canadian nuclear worker data. However, problems were identified in the Canadian data by the Canadian Nuclear Safety Commission [84]. Subsequently, the CNSC withdrew the Canadian data from use. Removing this data from the 15 country study invalidates the low-dose radiation cancer risk conclusion of Cardis et al.

4.2.7 Little [108] and Little et al [96]

In a review and meta-analysis, Little and coworkers suggested an excess radiation risk at dose levels below 500 mSv. These authors also argue that there is accumulating evidence from the Japanese atomic bomb survivors and various other moderate and low-dose exposed groups of excess risk of cataracts and circulatory disease. However, the studies of Akiba [101] and Doss [114] noted in Section 9 casts doubt on these results.

4.2.8 NCRP 136

Although NCRP 136 [40] advocates the LNT hypothesis, it provides ample justification for questioning its viability. For example, the report notes that prior exposure to a small conditioning dose (e.g., 10 mSv) enhances the repair of chromosome aberrations. This statement suggests a differentiation in response to DNA repair mechanisms at high and low-doses which are indicative of the efficiency of these mechanisms at low-doses. A dose dependent effect is inconsistent with the LNT hypothesis and consistent with the data summarized in Section 9.

NCRP 136 also addresses the dose dependence for neoplasm induction. The dose response relationships for neoplasms vary with a variety of factors including the specific type of neoplasm; linear energy transfer and dose rate of the radiation type; and age, sex, and genetic background of the irradiated individual. Although the data are primarily derived at high-doses and dose rates, there is insufficient data to define the shape of the dose response relationship in the mSv dose range. These data do not definitively support the LNT relationship, but are consistent with the factors negating the LNT hypothesis summarized in Section 9.

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NCRP 136 [40] observed that there is no conclusive evidence to reject the assumption of a linear non-threshold dose response relationship. However, the report notes that additional data are needed to fully characterize the risks attributable to low-dose radiation. NCRP 136 notes that many, but not all, scientific data support this assumption. Following the LNT conjecture, the probability of effects at background levels is so small that it may never be possible to prove or disprove the validity of the LNT hypothesis as the delivered dose approaches zero. However, as noted in previous discussion and in Section 9, a growing set of data exist that disprove this assertion.

These statements embodied in NCRP 136 are not a strong statement of support for the LNT hypothesis. In general, the NCRP 136 caveats when combined with the data summarized in Section 9 negate the case for the LNT hypothesis.

4.2.9 BEIR VII

The BEIR VII Committee [56] reaffirmed the LNT hypothesis, but noted that a portion of the evaluated data suggested that this model overestimates the detrimental effects of low level ionizing radiation. However, the committee rejected contentions regarding risks that are lower than LNT predictions and the existence of hormesis. These contentions were rejected based on the BEIR Committee's assessment that the proposed data were either based on ecologic studies or cite findings that are not representative of the preponderance of applicable data.

The data summarized in Section 9 conflicts with the conclusions of BEIR VII. The author believes the Section 9 provides ample evidence for the inherent weakness of the LNT hypothesis, but advises the reader to examine the data from BEIR VII and Table 11 and draw their own conclusions.

BEIR VII concludes that both epidemiologic and biologic data are consistent with the linear model at doses where associations have been measured. The BEIR VII conclusions are based in part on atomic bomb survivor data. The committee argues that thresholds and beneficial health effects are not supported by this data. This conclusion is in stark opposition to the revised bomb data of Ozasa et al [98] that suggests a definitive nonlinear dose response relationship in the data below 2 Sv. This data [98] is addressed in more detail in Section 6. The weakness of bomb data in supporting the LNT hypothesis has been addressed in detail by Cuttler [113], Cuttler and Welch [122] and Doss [106, 114].

BEIR VII also notes that there is strong support for the linearity of cancer formation. The report suggests that radiation biology research demonstrates that a single radiation tract, resulting in the lowest credible dose, striking the cell nucleus has a small but finite probability of inducing damage. Included in these detrimental effects are ionization spurs that produce multiple damage sites in a short strand of DNA. This collection of damage may be difficult to repair or the repair may contain errors. BEIR VII concludes that no compelling evidence exists to indicate a dose threshold below which the risk for detrimental effects is zero. In formulating this conclusion, the

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BEIR VII Committee ignored adaptive response, the effectiveness of the various DNA repair mechanisms, and the human immune system. If these effects were included, the LNT conclusions of BEIR VII would have been invalidated.

The statements derived from BEIR VII in the preceding paragraphs are in conflict with the data summarized in Section 9. As noted in Section 5, DNA repair mechanisms are vigorous and dynamic. Given the large number of spontaneous mutations per cell that occur naturally [47], it is difficult to determine how doses near threshold disrupt this process since the human species has existed in a radiation environment for an extended period of time. Moreover, DNA repair mechanisms are efficient at low-doses.

BEIR VII adopted a DDREF of 1.5 for low-linear energy transfer radiation for doses below 1 Sv. These data effectively divide the radiation dose regime into two zones and stipulate that the behavior of radiation is inherently different above and below 1 Sv. In particular, the use of the BEIR VII DDREF creates a discontinuity at 1 Sv. The result of the BEIR VII approach is the use of two linear functions having different slopes that differ by a factor of the DDREF.

The use of the DDREF is inconsistent with the original LNT hypothesis derived from atomic bomb survivor data that treated the entire dose range as a simple linear function. This discontinuity at 1 Sv is an indication of the weakness and inherent flaws in the LNT hypothesis. Subsequent data summarized in Section 9 support this contention.

4.2.10 ICRP 131

ICRP Publication 131 [125] reviews emerging evidence from stem cell biology and its impact on the radiation protection field including the LNT hypothesis. Stem cell radiobiology impacts conclusions regarding the stochastic effects of ionizing radiation on health detriments including (1) defining the target cells for radiation carcinogenesis in various target tissues, (2) use of the LNT hypothesis and relative risk models, (3) establishing the relationship of high- and low-dose rate effectiveness, and (4) determining age dependency of the risk of radiation induced carcinogenesis. The third item was a key aspect associated with the validity of the LNT hypothesis.

There are a number of biological mechanisms that could contribute to the protection of stem cells from the accumulation of mutations. ICRP 131 notes that these processes may contribute to the differences in carcinogenic risk and may explain why rapidly replicating tissue (e.g., small intestine) is more prone to radiation risk. The processes also provide insight into the LNT model and the relative and absolute risk models.

ICRP 131 specifically addresses the LNT model and the relative risk model. According to ICRP 131, a single stem cell origin of radiation induced cancer mutation theory, and the requirement of multiple mutations are consistent with the LNT hypothesis for some tissues. The report notes that radiation induced cancer depends on three factors. These include the: (1) number and sensitivity of stem cells to mutations induced by radiation, (2) retention of these

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mutations in a tissue, and (3) population of stem cells with a sufficient number of predisposing mutations. ICRP 131 notes the LNT model is used for the purpose of establishing radiation protection regulations. However, the support for the LNT hypothesis is less assertive than noted in previous publications. Further commentary regarding the ICRP 131 and its support for the LNT hypothesis are provided in Section 9.

5.0 DNA Damage and Repair Basics

The LNT hypothesis assumes detrimental effects arise at the cellular level and are related to the associated radiation damage to DNA. However, the LNT hypothesis does not specifically address subsequent DNA repair mechanisms. In view of this situation, a brief review of DNA damage and basic repair mechanisms are provided.

Each cell in the human body suffers 1 – 10 DNA breaks per day [133]. Given this level of damage, repair mechanisms are required to preserve the body and maintain its various functions. These mechanisms are crucial to understanding the validity of the LNT hypothesis.

At a fundamental level, DNA consists of nucleotides with the bases adenine (A), guanine (G), cytosine (C), and thymine (T) [58]. Within the DNA double helix, A in one strand is always paired with T in the other, and C is always paired with G. These pairings are vulnerable to damage. For example, the C-G pairing can be disrupted such that cytosine loses an amino group. When this occurs, the damaged segments tend to pair with adenine which can produce a mutation if the defect is not properly repaired. This change can alter the genetic information encoded within the original macromolecular structure and can theoretically lead to a biological detriment.

Fortunately, there are robust mechanisms for repairing DNA. Cells contain several DNA repair systems that can correct alterations. These repair mechanisms fall into two general categories which include the repair of damaged bases and incorrectly paired bases during replication. In most cases, DNA repair is a multi-step process that includes detection of an abnormality in DNA structure, removal of the flawed DNA, and synthesizing normal DNA.

Genetic information is stored in the DNA helix and repair facilitating enzymes monitor the strands and replace damaged nucleotides. Most DNA repair mechanisms utilize the duplicate genetic information in each of the two DNA strands. Damage on one strand is repaired by an enzyme and a corrected section is produced using the duplicate coding in the undamaged strand. In a sense, the DNA strand is a computer program having multiple redundant paths with the capability to repair damaged sections of the code.

There are three fundamental mechanisms associated with DNA repair [58]. These are base excision repair (BER), nucleotide excision repair (NER), and mismatch repair. BER corrects a variety of defects that affect the bases A, C, G, and T without causing structural damage to the DNA strand. In base excision repair, the damaged base is removed, and this action is followed by excision of the resulting sugar phosphate.

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NER fixes various abnormalities that either interfere with the normal base pairings or distort the helical DNA structure. In nucleotide excision repair, the damaged portion of the DNA strand is removed from the double helix. In both cases, the gap left in the strand is filled by sequential action, and the undamaged DNA strand is utilized as the repair template. This is an example of the inherent redundancy associated with the DNA structure and its associated repair mechanisms.

Mismatch repair corrects defects when DNA is replicated, recombined, and mismatched. This repair method is strand-specific. During DNA synthesis, the new strand will include errors. The mismatch repair mechanism distinguishes the new strand from the original template, and corrections are made to ensure the new strand matches the original segment.

DNA damage induced by ionizing radiation is significantly less severe than the spontaneous damage that occurs from other causes. Most spontaneous changes in DNA are temporary and are immediately corrected by the collection of DNA repair mechanisms. Heat, metabolic transients, various sources of natural ionizing and nonionizing radiation, and exposure to chemicals in the environment create thousands of DNA random changes per day in a human cell. However, only a few survive as mutations in the DNA sequence. For example, less than one in 1000 base changes in DNA creates a permanent mutation [47]. Most are efficiently eliminated by the DNA repair mechanisms.

The number of natural mutations is significantly larger than those created by low-dose ionizing radiation. If low-dose radiation is a hazard, one would expect that the natural mutations would propagate cancer at a rate larger than observed. Since this does not occur, the DNA repair mechanisms and human immune system must function efficiently to remove both naturally occurring abnormalities and those caused by low-doses of ionizing radiation. Although this is a very qualitative argument, the rate of natural mutations suggests the LNT repair mechanisms should mitigate the detrimental effects of low-dose ionizing radiation.

DNA repair and other natural body processes, including the human immune system, provide a robust means to protect the body from a range of agents. These processes are also expected to facilitate the repair of damage caused by low-dose ionizing radiation. This subject is addressed in subsequent discussion.

6.0 The atomic bomb survivor data

Before addressing specific data that negates the LNT hypothesis, the BEIR VII contention regarding atomic bomb survivor data is addressed. According to BEIR VII, the most important information for determining health effects of low-dose radiation is the atomic bomb survivor data. Atomic bomb survivor data are frequently quoted to validate the LNT hypothesis and to establish the low-dose detriment.

RERF Report 14 by Ozasa et al [98] updated the RERF Report 13 [42] results and noted that formal dose-threshold analysis indicated no threshold; i.e., zero dose was the best estimate

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of the threshold. However, Ozasa et al note that: "*Although the linear model provided the best fit in the full dose range, statistically significant upward curvature was observed when the dose range was limited to 0–2 Gy (θ = 0.81, P = 0.02) (Tables 6 and 7). The curvature over the 0– 2-Gy range has become stronger over time, going from θ = 0.20 for the period 1950–1985 to 0.81 for 1950–2003, and has become significant with longer observation (Table 7)".* In the preceding quote θ is the curvature of the fit and P is the statistical significance (likelihood test). The reader should recall that RERF Report 13 [42] was a significant basis for establishing the credibility of the LNT hypothesis in the BEIR reports as well as a portion of the basis for radiation protection regulations (e.g., 10CFR20 and 10CFR835).

Ozasa et al [98] conclude that a linear non-threshold model fits the excess relative risk curve for solid cancers as a function of weighted colon dose for the full dose range. However, the authors suggest that a linear-quadratic (LQ) model provides the best if the data is restricted to a dose of 2 Gy.

A cursory examination of the published data in the 0 – 2 Gy range shows a definite depression in the curve that is an obvious deviation from linearity. This depression occurs at about 400 mGy [123]. In addition, the ERR is negative at low-dose values that suggests the need to correct the data for the bias in the baseline cancer rate. Doss [106] suggests this correction is 20% and reformulates the ERR [93].

Following Doss [93], the calculated ERR values can be corrected for such a bias using the following equation

$$\mathbf{ERR'} = \frac{(1+\mathbf{ERR})(100+\delta)}{100} - 1 \qquad (23)$$

where $\mathbf{ERR'}$ is the value of ERR corrected for the bias, and δ is the percentage bias in the baseline cancer mortality rate. Doss [93] uses a −20% bias which is based on the observed reduction in low-dose radiation cohorts in some population studies.

The correction as applied by Doss [93] shifts the ERRs to lower values resulting in negative ERR values for all the doses below about 600 mGy. Although there are fluctuations in the corrected ERR values for doses below about 300 mGy, the overall topology of negative ERR values for doses below about 600 mGy is suggestive of the a hormetic or cancer preventive effect of low-dose radiation that has been previously observed in animal and human studies [12,59,65,81,87,93,102,106,129,132].

Given this analysis, Doss [93] suggests that the qualitative shape of the dose response curve of the atomic bomb survivor data has a plausible explanation using a radiation hormesis model. This explanation results when the ERR data is corrected for the likely bias in the baseline cancer rate. However, there is no explanation for the observed reduction in ERR values in the 300 – 700 mGy dose range within the context of the LNT model.

As a further evaluation of the Ozasa et al results [98], the ERR solid cancer data was fit to the power series:

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$$\mathbf{ERR} = \sum_{i=0}^{N} \mathbf{a_i\, d^i} \qquad (24)$$

where N+1 is the number of terms in the expansion, a_i are coefficients determined from a fit to the RERF 14 solid cancer data below 2 Gy, and d is the weighted colon dose. The fit was limited to $N \leq 8$, but considers functional forms typically used to evaluate radiological data.

Using the functional form of Eq. 24 is not intended to be a rigorous mathematical exercise because the error bars on the data were not included. The intent of the fit is to only evaluate the functional form and to investigate the optimum mathematical relationship to the data. Although this is a somewhat sterile presentation, it does assess the departure of the data from the assumed functional form required by the LNT hypothesis.

The restriction to linear and linear quadratic forms has been the usual standard for radiological data analysis, but subsequent analysis generalizes that approach to consider other polynomial forms. In particular, the existence of a threshold and minima in the data below 2 Gy as suggested by Doss [93,106,123] is investigated in a more general manner. The results of the numerical analysis are summarized in subsequent discussion.

Although there are numerous approaches to compare data sets, this paper uses the PSI-Plot™ computational package and its associated analysis features [71]. In comparing data sets, the fit parameter (Ψ) is used:

$$\Psi = \sum_{i=1}^{m} (\mathbf{O_i} - \mathbf{C_i})^2 \qquad (25)$$

where m is the number of elements in the data set, O_i is the RERF 14 solid cancer ERR value [98], and C_i are the corresponding polynomial fit values from Eq. 24. The area under the polynomial fit curves is also presented in Table 10. The area is calculated over the range of Ozasa et al data below 2 Gy.

The simple-minded analysis summarized in Table 10 is not intended to be definitive and its only purpose is to determine if the basic LNT requirements including no thresholds and deviations from a linear fit are appropriate. The analysis includes the linear (N = 1), linear quadratic (N = 2), and higher order polynomial fits (N = 3 – 8). The fit parameter provides an indication of how well the various N values reproduce the data. The N = 1 and N = 2 cases are typically used in health physics applications. However, higher order polynomial fits have been excluded from previous analyses.

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Table 10
Polynomial Fit to the Excess Relative Risk for Solid Cancers as a Function of Weighted Colon Dose Data Below 2 Gy[a]

Polynomial Order (i in Eq. 24)	Minimum in Polynomial (mGy)			Threshold (mGy)	Ψ (Eq. 25)	Area under polynomial fit (Gy)
	First	Second	Third			
1	---	---	---	12	0.646	0.979
2	---	---	---	-26	0.640	0.961
3	187	---	---	---	0.558	0.975
4	553	---	---	30	0.367	0.981
5	49	718	---	---	0.288	1.02
6	352	1016	---	29	0.155	1.05
7	41	531	1290	---	0.0574	1.11
8	45	530	1282	---	0.0572	1.11

[a] Osaza et al [98].

The results of Table 10 offer the possibility of a threshold in the 10 – 30 mGy range for N = 1, 4, and 6. However, a threshold is not observed in all data fits. In fact, the N = 2 fit has a negative threshold (-26 mGy).

Minima are also predicted by the N = 3 – 8 data evaluations. The minima vary with the polynomial order used in the analysis, but are suggested by the data. The first minimum lies in the 40 – 550 mSv range for N = 3 – 8.

As expected, the fit improves as more parameters are included. This is illustrated by the decreasing value of the fit parameter in Table 10. Fig. 1 plots the N = 1 (solid curve), N=2, N=4, and N = 8 cases.

The author does not attempt to draw specific conclusions regarding the magnitude of the threshold or position of the minima. However, the polynomial fits do suggest the non-linearity in the data. These calculations are also supportive of the nonlinearity contentions provided by Doss [93,106,123] and acknowledged by Ozasa et al [98].

The area under the curve is a nontraditional approach for judging the goodness of the fit. However, as noted in Fig. 1, the N = 1 (linear) and N = 2 (linear-quadratic) fits clearly do not fit

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the data (without consideration of error bars) as well as the N > 2 curves. This issue is exaggerated because the data error bars were not included in the simple-minded analysis.

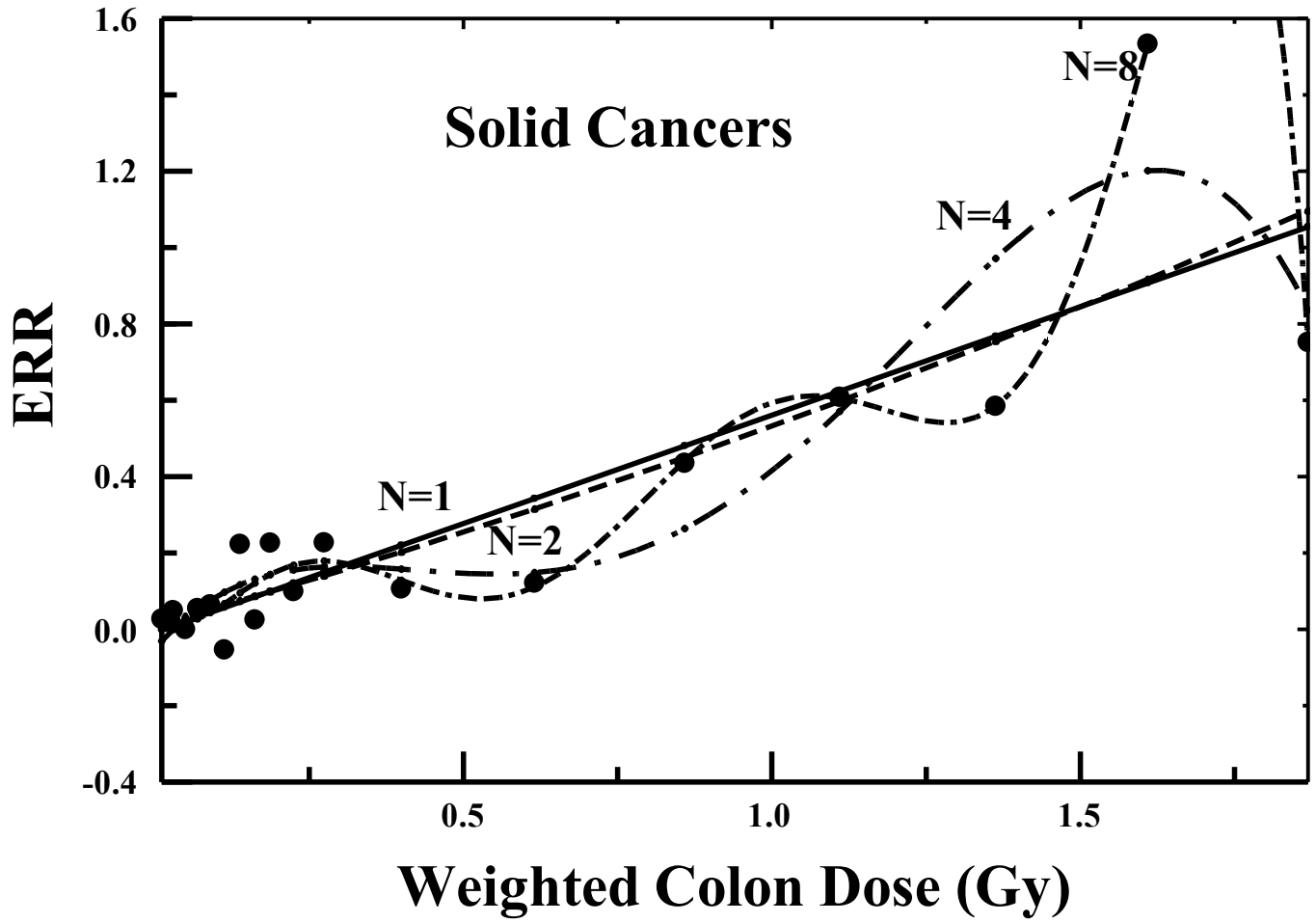

Fig. 1 Simple-minded fit of the excess relative risk (ERR) for all solid cancers as a function of the weighted colon dose [98]. The N = 1, 2, 4, and 8 polynomial fits are illustrated.

7.0 Biological Evidence against the LNT Hypothesis

DNA damage occurs within the body through a variety of mechanisms even in the absence of low-dose radiation. As noted in Section 5.0, the body has natural defense mechanisms to repair this damage and minimize its propagation. These mechanisms operate deterministically and below a dose threshold there is likely no propagation of DNA damage or biological detriment. This threshold suggests an inherent weakness in the LNT hypothesis. Above this

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threshold, the effects of ionizing radiation overwhelm the DNA repair processes and produces a net biological detriment, and this damage can be propagated beyond DNA to higher levels structures including cells, tissues, organs, and whole body [77].

As noted in Section 5.0, there are three repair mechanisms that inhibit damage propagation. These mechanisms can be recast in more qualitative terms as physical or metabolic processes. Physical defenses precede metabolic defenses. The physical defense mechanisms act immediately to scavenge toxic chemical species and free radicals produced by ionizing radiation interactions with tissue. Physical mechanisms also include molecular repair of cellular structures including DNA; removal of damaged cells by apoptosis, necrosis, and phagocytosis; cell differentiation and senescence; and response of the immune system to facilitate removal of damaged cells. Within the context of this paper, the immune system includes all body defense mechanisms. These mechanisms combat biological and other agents that damage cells or inhibit cellular repair processes, and other processes that return the body to its normal state when under attack by various agents.

Following these actions, base excision repair, nucleotide excision repair, and mismatch repair replace lost DNA elements. In addition, metabolic defense mechanisms arise from normal cellular processes that produce chemical agents that facilitate repair. Some of these repair mechanisms are effective for more than a year and all create temporary protection against radioactive and toxic materials. Following Feinendegen et al. [102] adaptive protections reach a maximum after single tissue absorbed doses in the range of 100 – 200 mSv, but are ineffective at higher doses. Low-dose rates initiate maximum protection if delivered repetitively at certain time intervals. Adaptive protection preventing about 2–3 % of lifetime cancer risk would fully balance a calculated, induced cancer risk at about 100 mSv which is in agreement with epidemiological data and consistent with an hormetic effect. To date, radiation protection regulations and low-dose risk assessments do not recognize hormesis and the positive aspects of low-dose radiation. A summary of the limitations of the LNT hypothesis associated with radiation protection regulations was provided by Doss [123].

8.0 General Arguments against the LNT Hypothesis

Support for the LNT hypothesis is not universal and numerous organizations including the American Nuclear Society [38], French Academy of Sciences [55], French National Academy of Medicine [55], and Health Physics Society [78,147] have expressed various degrees of opposition to the LNT approach. In addition to the arguments of these organizations, the LNT hypothesis can be reviewed in terms of its inherent assumptions from a physiological, cancer risk, dose threshold, radiation carcinogenesis, radiation biology, background radiation, and dose modeling perspectives.

This section provides an overview of the relevant data in terms of general data categories. Detailed data references are provided as warranted, but the specific details are provided in

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Section 9. The reader, overwhelmed by the mass of referenced data, should focus on Section 8 and review Section 9 after the initial data categories become more familiar.

8.1 Physiological Considerations

There are shortcomings in the LNT hypothesis that have not been fully evaluated. The LNT hypothesis does not incorporate a number of processes that are present in cellular repair and damage mitigation. For example, biological mechanisms involved in cellular repair are time and dose rate dependent. These mechanisms are not incorporated in the LNT approach since the hypothetical model does not consider the temporal dynamics of a DNA break or the various biological repair mechanisms.

The LNT hypothesis also does not include the evolutional development of a species and its adaptation to the natural radiation environment. An evolving species would minimize the low-dose radiation influence as a risk factor in its survival by developing an immune and repair system that was compatible with the natural background radiation environment.

In a similar manner, the LNT hypothesis does not account for DNA repair and its varied and effective mechanisms at low-doses. Ionizing radiation damage to DNA involves a double strand break that severs the double helix. These breaks are repaired or reconnected by the aggregation of cellular proteins. At low-doses, these cellular repair mechanisms are efficient. However, at high-doses, the more extensive DNA damage tends to form clusters. These damage clusters facilitate improper repairs that can lead to a biological detriment. Specific detriments include mutations (chromosome rearrangements) or cancer (malfunctioning cells). Since DNA repair is less effective at high-doses, it is problematic to extrapolate the high-dose results to low-doses when DNA repair effectiveness varies as a function of dose. This simple description also provides an explanation to the increased risk of cancer at high-doses, but it does not validate the LNT hypothesis.

Other mechanisms, including adaptive response, suggest that a biological insult (e.g., radiation exposure) enhances the body's ability to address further insults by activating its defense and repair mechanisms. Adaptive response suggests that a low-dose of radiation preconditions the body to withstand additional radiation exposure. This occurs because the initial exposure activates the collection of biological repair mechanisms (e.g., B and T cell response mechanisms).

There is also evidence to suggest that low-doses of ionizing radiation stimulate cellular defense mechanisms that protect the individual against disease. In addition, low-dose radiation can have a positive biological impact. This process is known as hormesis and has been observed experimentally in lower life forms. Hormesis and adaptive response present additional challenges to the LNT hypothesis.

A future system of radiation dose limits cannot ignore the specific differences in biological repair effectiveness at low- and high-doses. In addition, hormesis and adaptive

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response must be evaluated without regard to the historical LNT bias. The use of dose and dose rate effectiveness factors acknowledges the inherent difference between high- and low-dose exposures. However, a complete set of factors must be considered in establishing a valid model for radiation detriment.

For example, Doss [123] notes that autopsy studies have shown that the presence of cancer cells is not a decisive factor in the physical manifestation of clinical cancer. However, immune system suppression in organ transplant patients more than doubles the cancer risk. This supports an important immune system role in limiting occult cancers. Doss further notes that low-dose radiation elevates immune response, and so it may reduce rather than increase the risk of cancer [123]. The beneficial effects of low-dose radiation have been noted in numerous publications. However, the most recent BEIR VII report reviewed, but did not accept the role of hormesis and its challenge to the LNT hypothesis.

The LNT hypothesis focuses attention on DNA damage leading to further health detriments including cancer and hereditary effects. DNA damage is only one factor in assessing detriment and medical researchers suggest that it is not a decisive factor. By focusing on DNA damage, the LNT hypothesis ignores the response of the immune system, which is an important factor in determining the physical detriment. In addition, adaptive response appears to be a valid effect that stimulates the immune system and permits it to function at an optimum level to counter the ionizing radiation detriment.

From a physiological perspective there are three fundamental issues in the current radiation safety basis established using the LNT hypothesis. First, the LNT hypothesis focuses its attention on DNA damage and mutations which are not the only factors affecting the onset and propagation of cancer. Second, the LNT approach ignores the effect of the immune system response which is an important factor modulating the occurrence of cancer. The effect of radiation on immune system response is not linear, since low-dose radiation stimulates the immune system, and high-dose radiation suppresses it. Third, the LNT model ignores the large variability in cancer rates by specifying no threshold. Lifetime cancer risks are likely to have large errors arising from the variability in confounding factors. Moreover, cancer rates also vary from year to year.

These issues suggest a thorough review of the LNT radiation safety basis is warranted. Although it is the basis for current radiation protection regulations, there are numerous publications that suggest there is no justification for continuing the use of the current LNT radiation safety paradigm. The LNT hypothesis has contributed to an unjustified fear of low-dose radiation and has inhibited the study of potentially beneficial applications of low-dose radiation.

If the LNT hypothesis is discarded, what radiation protection approach would replace it? This issue is discussed in Section 10.

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8.2 Cancer-Risk Arguments

Raabe [100] presents cancer-risk arguments against the LNT hypothesis. He notes that the development of a radiation-induced malignant tumor is not the result of a single random interaction of the ionizing radiation with an isolated cell. Raabe offers the following arguments against the LNT hypothesis and suggests that major revisions of methodology and standards are needed:

1. The cancer risk associated with ionizing radiation exposure is a non-linear function of the lifetime average dose rate to the affected tissues;
2. Cancer risk exhibits a virtual threshold at low lifetime average dose rates;
3. Cumulative radiation dose is not an accurate or appropriate measure of cancer risk, but it is useful for describing the virtual threshold for various exposures.
4. High-dose rate atomic bomb survivor data from Hiroshima and Nagasaki cannot be used to estimate cancer risk from ionizing radiation exposures over long times and at low-dose rates.

Based on these considerations, currently accepted ionizing radiation detriment models should be reevaluated to assess the validity of LNT estimates of ionizing radiation cancer risk. Other arguments offered by the Health Physics Society [78,147] suggest that the LNT hypothesis is an oversimplification. The LNT approach can be rejected for specific cancer types (e.g., bone cancer and chronic lymphocytic leukemia). In addition, significant heritable genetic damage has not been observed in human studies. The effects of various biological mechanisms (e.g., DNA repair and adaptive response) on the induction of cancers and genetic mutations as a function of dose and dose rate have not been thoroughly investigated. These mechanisms do not appear to be credibly modeled by a linear-non-threshold model.

8.3 Threshold Dose limits

The credibility of the LNT hypothesis is further challenged by the observation that radiogenic health effects have not been consistently demonstrated below 100 mSv [78,147]. Primary cancers have been observed in humans only at doses exceeding about 100 mSv delivered at high-dose rates. Below this threshold, estimates of radiation detriment are speculative. As noted previously, risk estimates in exposed populations are based on epidemiological studies of well-defined groups (e.g., the Japanese atomic bomb survivors and medical therapy patients) exposed to relatively high-doses delivered at high-dose rates. Adverse health effects have not been observed in individuals exposed to chronic doses less than 100 mSv.

In its Radiation Risk in Perspective Position Statement, the Health Physics Society (HPS) concluded that risk estimates should be limited to individuals receiving a dose of 50 mSv in one year or a lifetime dose of 100 mSv [78,147]. This dose is in addition to natural background.

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Below these doses, risk estimates should not be performed. In addition, the HPS recommends that expressions of risk should only be qualitative and presented as a range of values based on uncertainties. This range of uncertainty values should include the inability to detect any increased health detriment, which acknowledges that zero health effects are a credible outcome.

8.4 Radiation Carcinogenesis

Raabe [80,86,100,117] notes that ionizing radiation carcinogenesis is not a linear function of cumulated dose. Moreover, it is not a stochastic single cell phenomenon. It is a whole organ process that is dependent on a variety of factors including the lifetime average dose to the sensitive organ cells. As a collective process, the arguments of Doss [123] suggest a whole body response including the importance of the human immune system. The elimination of a single cell effect and influence of collective body defense mechanisms suggest the LNT response model is an oversimplification of the onset and development of carcinogenesis.

8.5 Radiation Biology Considerations

Ionizing radiation can damage DNA through direct molecular events (e.g., ionization and excitation) or through indirect mechanisms including chemical reactions caused by reactive oxygen species produced by radiation induced reactions. Tubiana et al [72] observe that these species are also an abundant consequence of natural oxygen metabolic processes. Animal life would be unsustainable without natural defenses against reactive oxygen species. Accordingly, the human body has adapted to the effects of both direct and indirect radiation effects through prolonged exposure to natural background radiation and normal biological functions required for growth and sustaining life.

Natural defense mechanisms occur throughout the cell life cycle and accommodate DNA repair or apoptosis. These actions decrease the probability of chromosome aberrations and genomic instability in a manner that is most effective at low-doses [35,66]. Tubiana et al [72] note that the mutagenic effect per unit dose varies with dose rate and reaches a minimum in the range of 1–10 mGy/min [37,43]. This dose rate effect is approximately equal to the rate of reactive species–inducing DNA damage during oxidative stress [67]. In humans, chromosome aberrations are not produced by doses less than 100 mSv or at low-dose rates [45,46,57]. Large studies have not revealed an increased incidence of chromosomal aberrations at doses below 20 mSv [36].

If the LNT hypothesis is correct, damaged cells are created and their numbers increase with increasing dose. At low-doses, the human cellular response does not follow this assumed production sequence, and eliminates damaged or malfunctioning cells through death or terminates their proliferation. The elimination of cells with damaged DNA can occur through apoptosis (controlled death) shortly following irradiation in the range of a few mSv to about 200 mSv [15,34,72]. These mechanisms are less effective at higher doses.

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8.6 High Background Radiation

The population living in Kerala, India experiences background radiation levels up to 70 mSv a year [72]. This radiation level is much higher than other locations in India, but no increased cancer risk has been observed. In Yangjiang, China and the surrounding area, the population is exposed to two levels of annual background radiation (i.e., 6.4 and 2.4 mSv). In spite of this significant difference in annual dose, there was no increase in cancer incidence or mortality. The higher level of background radiation was confirmed by an increased incidence of chromosomal aberration, but no excess in cancer incidence was observed [72].

The observation of an increase in chromosome aberrations without a proportional increase in the incidence of cancer appears to negate the LNT contention regarding the causal relationship between a chromosomal aberration and cancers at low-doses. This observation contradicts the LNT hypothesis and is another example of its failure to account for established data. It is worth noting that proponents of the LNT hypothesis, as embodied by the BEIR Reports, do not utilize data from these high background areas to assess the validity of its assumed linear approach.

8.7 Use of Modifying Factors

Modifying factors were introduced in ICRP 26 [10] to relate the effective dose equivalent to the absorbed dose. The DDREF follows in the spirit of a modifying factor to account for a biological effect or modification of that effect. In particular, the DDREF attempts to overcome discrepancies between epidemiological data and LNT predictions. As such, the use of modifying factors illustrates an inherent weakness in the LNT hypothesis. If the LNT approach were absolutely valid, no modifications would be required and high-dose and dose rate data could be extrapolated linearly to zero dose. Clearly, the pure LNT hypothesis is invalid, but modifying factors have been used to justify a modified LNT approach. However, the literature does not make this distinction, and continues to refer to the LNT hypothesis without qualification.

The use of a DDREF implies that for low-doses and/or dose rates, the probability for DNA damage to be carcinogenic is reduced by a DDREF value. In the Case of BEIR VII, a DDREF value of 1.5 is judged to be appropriate for low LET radiation for effective doses below 1 Sv. However, the LNT proponents suggest the DDREF leaves unchanged the concept that even the smallest dose can induce cancer.

The high-dose data is extrapolated to zero dose, but the slope of the line below 1 Sv is reduced by the DDREF and subsequently extrapolated to zero dose. This approach leads to a discontinuity at 1 Sv which is clearly nonlinear.

The DDREF is composed of two component factors that are conceptually distinct from a biological perspective. These factors are dose effectiveness factor (DEF) that applies to low acute doses, and the dose rate effectiveness factor (DREF) that is affected by low protracted doses where long-term kinetics of target/stem cells in tissue may modify the dose response [125].

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These factors attempt to assign an *ad hoc* parameter to explain an inherent weakness in the LNT hypothesis. The DEF, DREF, and DDREF appear to be an attempt to rationalize a flawed concept. From the author's perspective, thresholds, hormesis, adaptive response, and immune system function are more credible concepts to explain the biological effects of low level radiation exposure than the aforementioned effectiveness factors.

9.0 Data Negating the LNT Hypothesis

This section summarizes specific data that contradicts the LNT hypothesis. The documentation provides direct as well as supporting data that suggests the LNT hypothesis is flawed and in need of revision. For each data reference, Table 11 provides its impact on the LNT hypothesis as well as the primary conclusions derived from the work. The research summarized in Table 11 not only supports the need for a revision of the LNT hypothesis, but also suggests that thresholds, hormesis, the immune system, and adaptive response are important considerations in determining the appropriate dose response relationship. In addition, the dose response characteristics of low-dose radiation are significantly different than the high-dose region, and the LNT hypothesis extrapolations from the high-dose region are not justified.

Table 11 is an analogue to Table 8 that provided justification for the LNT hypothesis. The reader should carefully review each table and supporting data to determine the validity of the LNT hypothesis. This review will clearly illustrate the reason that the pro and anti LNT supporters have found little common ground with each faction quoting data that supports their viewpoint.

The studies summarized in Table 11 vary in size and scope. Some studies are limited to a particular age group or population having specific characteristics. The reader is referred to the specific study for details regarding its purpose and scope. The content of Table 11 is significant in that there are numerous studies challenging the LNT hypothesis directly or specific aspects underlying its basic assumptions.

Entries in Table 11 are ordered chronologically. Much of the data has been available to the BEIR and ICRP committees, but have not been incorporated into their reports. Although it is understandable that there are differences in technical perspective in the initial studies, many studies provide strong evidence for LNT weaknesses. In particular, revisions to data incorporated into Cardis et al [50] and RERF Report 14 [98] have not been incorporated into BEIR and ICRP Reports (e.g., initial BEIR VIII [116] discussions and ICRP 131 [125]). These omissions are significant because data issues clearly challenge the LNT hypothesis.

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Table 11
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Reference	LNT Impact	Primary Conclusions[a]
Frigerio et al [7]	Evidence supports a trend of reduced cancers with increasing background radiation dose which is in conflict with the LNT hypothesis.	Frigerio et al observe a trend of lower US cancer mortality rates associated with higher background radiation levels.
Evans [8] and Rowland [31]	The dose response curve exhibits a well defined threshold.	Evans notes a threshold dose of ~10 Gy for induction of bone sarcomas in radium dial painters. There is no observed increase in cancers below this threshold. This threshold was affirmed by Rowland.
Chaffey et al [9]	Low-dose radiation has a positive biological impact in contrast with the predicted detriment resulting from the LNT hypothesis.	Chaffey et al investigated the survival of lymphosarcoma patients treated with whole body irradiation and chemotherapy. Low-dose radiation (150 mGy) applied 10 times during 5 weeks (Total dose 1.5 Gy) had a therapeutic effect in treating the cancers.

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Reference	LNT Impact	Primary Conclusions[a]
ICRP 26 [10], BEIR III [13], BEIR V [20], ICRP 60 [23], NCRP 116 [25], NCRP 136 [40], BEIR VII [56], ICRP 103 [60], 10CFR 20 [135], 10CFR835 [136], Alemayehu and Cochran [140], and Siegel et al [151,152]	The LNT hypothesis must be validated by data. Simply invoking it as valid approach because national and international organizations support its use is not a scientific justification.	Alemayehu and Cochran are a recent example of authors that support the LNT hypothesis because it is supported by national and international organizations. Siegel et al note the arguments of Alemayehu and Cochran do not justify the extrapolation from very high to zero dose. In general, international and national authoritative bodies have not utilized the vast array of data that suggests LNT issues have failed to adequately evaluate studies supporting hormesis, thresholds, and nonlinear trends in the data.
Bursch et al [15] and Chandra et al [34]	Following the LNT hypothesis, damaged cells are created and their numbers increase with increasing dose. This contention is in conflict with observations. Cellular mechanisms effectively eliminate damaged cells at low-doses.	At low-doses, Bursch et al and Chandra et al observe that the human cellular response does not follow the LNT hypothesis and eliminates damaged cells through death (e.g., apoptosis) or terminates their proliferation. The elimination of cells with damaged DNA can occur through apoptosis shortly following irradiation in the range of a few mSv to about 200 mSv. These mechanisms are less effective at higher doses.
Kostyuchenko and Krestinina [29]	The dose response curve exhibits a U-shaped minimum that occurs near 120 mSv. This minimum is in conflict with the LNT hypothesis.	Kostyuchenko and Krestinina investigated the long-term irradiation effects in the population evacuated from contaminated areas in the East-Urals. Significantly reduced cancer mortality rates relative to the control group were observed in the 120 mGy and 500 mGy cohorts from evacuated villages near the Mayak Chemical Combine (Chelyabinsk-65) which was a nuclear waste reprocessing and production facility similar to the Hanford Site in the US.

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Reference	LNT Impact	Primary Conclusions[a]
Cohen [30]	Reduced lung cancer mortality rates with increased residential radon levels are in conflict with the LNT prediction.	Cohen observed reduced lung cancer mortality rates with increased residential radon levels in US counties. This study finds that with or without corrections for variations in smoking prevalence, there is a strong tendency for lung cancer rates to decrease with increasing radon exposure which is in sharp contrast to the increase expected from the linear non-threshold theory.
Imaida et al [32] and Greaves [115]	The LNT hypothesis presumes that increased mutations mean increased cancers, but mutations do not imply cancer.	Imaida et al note that the cancer mortality rate increases drastically with age, but the percentage of patients with cancerous mutations is unchanged. Graves observes that almost all individuals have cancerous mutations, but everyone does not have cancer. Failure to link increasing mutations with cancer risk suggests the LNT hypothesis is flawed with respect to its inherent cancer induction assumption.
Dikomey and Brammer [35] and Shrivastav et al [66]	The dose dependence of DNA repair mechanisms is inconsistent with the LNT hypothesis. These repair mechanisms are most effective at low-doses.	Natural defense mechanisms occur throughout the cell life cycle and accommodate DNA repair or apoptosis. Dikomey and Brammer and Shrivastav et al observe that these actions decrease the probability of chromosome aberrations and genomic instability in a manner that is most effective at low-doses
Vilenchik and Knudson [37,43]	The dose dependence of mutations is inconsistent with the LNT hypothesis.	Vilenchik and Knudson observe that the mutations per unit dose vary with dose rate and reach a minimum in the range of 1–10 mGy/min.

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Reference	LNT Impact	Primary Conclusions[a]
UNSCEAR 2000 [36], Hooker et al [45], Loucas et al [46], and Zeng et al [57]	Radiation induced detriment as a function of dose is inconsistent with the LNT hypothesis.	Studies including those of Hooker et al, Loucas et al, and Zeng et al observe that human chromosome aberrations are not produced by doses less than 100 mSv or at low-dose rates. Large studies including UNSCEAR 2000 have not revealed an increased incidence of chromosomal aberrations at doses below 20 mSv.
Cuttler and Pollycove [41]	The shape of the dose response curve for breast cancer deaths as a function of breast dose is non-linear and exhibits a distinct minimum.	Cuttler and Pollycove note a reduction of breast cancer mortality in tuberculosis patients. The dose response curve for breast cancer deaths as a function of breast dose has a minimum at about 150 mGy. Cuttler and Pollycove observe that patients, receiving a total dose in the range from 50 to 300 mGy, had a breast cancer incidence up to one-third less than the background incidence. These authors also note that a hormetic model provides a better fit to the data than the LNT hypothesis
Preston et al [42], Ozasa et al [98], and Doss [106]	The 2003 data set of RERF 13 [42] has been superseded by the 2012 data of RERF 14 [98]. This revision is not consistent with the LNT hypothesis.	RERF 13 has been superseded by RERF 14 and this report no longer definitively supports the LNT hypothesis. Doss observes that the shape of the dose-response curve, with correction for bias in the baseline cancer rate, is consistent with the concept of radiation hormesis.
Wakeford and Little [44], Brent [112], and Doss and Little [114]	Assuming the applicability of the LNT model creates a bias that overestimates the observed leukemia risk.	In a point-counterpoint paper by Doss and Little, Doss provides a rebuttal to the arguments of Wakefield and Little. The occurrence of leukemia was observed only following high-dose radiation and risk coefficients were based on an LNT model that created the impression of increased risk at low-doses where no effect has been observed. As noted by Brent, cohort studies, that are generally a better approach than case controlled studies, yield no increased leukemia risk.

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Reference	LNT Impact	Primary Conclusions[a]
Sakamoto [49]	Low-dose radiation has a hormetic effect that is in conflict with the predictions of the LNT hypothesis	Sakamoto observed improved survival of non-Hodgkin's Lymphoma patients when subjected to 100 - 150 mGy total-body irradiations combined with radiation treatments directly to the tumor using a total dose of 1.5 Gy. Suppression of distant metastasis of tumor cells was also observed by Sakamoto following low-doses of total-body irradiation.
Cardis et al [50] and Canadian Nuclear Safety Commission (2011) [84]	The revised Canadian data no longer supports the LNT hypothesis.	Cardis et al [50] was quoted by the BEIR VII [56] Report and initial BEIR VIII efforts [116] to infer an increased cancer risk from low-dose radiation and to validate the radiation cancer risk. The combined data from 15 countries show a statistically significant increase in cancers in radiation workers which led the authors to conclude that low-dose radiation increases the cancer risk. This result was driven by the Canadian data that suggested a much higher risk than data from other countries. However, problems were identified in the Canadian data by the Canadian Nuclear Safety Commission. Subsequently, the CNSC withdrew the Canadian data from use. Removing this data from the 15 country study invalidates the low-dose radiation cancer risk conclusion of Cardis et al.
Sponsler and Cameron [52]	The LNT hypothesis, which predicts increasing detriment at higher doses, is in conflict with the observed results.	Sponsler and Cameron published a summary of their nuclear shipyard worker study (1980–1988) that involved a large cohort exposed to low-dose rate gamma radiation. The median cumulative dose for the main cohort of shipyard workers was 35.8 mGy (2.8 mGy x 12.8 years). The authors observed significantly reduced cancer mortality in the workers subjected to median cumulative radiation doses of 35.8 mGy in comparison to non-radiation workers. These higher-dose workers demonstrated significantly lower circulatory, respiratory, and all-cause mortality than did unexposed workers. Mortality from all cancers was also lower in the exposed cohort.

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Reference	LNT Impact	Primary Conclusions[a]
Hwang et al [53] and Doss [106]	Low-doses have a hormetic effect which is inconsistent with the LNT hypothesis. Smaller sample sizes than predicted by the LNT hypothesis lead to meaningful results for low-dose radiation.	Hwang et al assessed the cancer risks in a Taiwanese population that received prolonged low-dose rate γ-irradiation for about 10 years as a result of occupying buildings containing ^{60}Co-contaminated steel. As reported by Doss, a statistically significant reduction was observed for all cancers in the apartment residents receiving an average dose of about 50 mSv. This reduction continued in the 2008 follow-up report as described by Doss. The Taiwan data [53], having an average dose of 50 mSv with a cohort of about 8,000, provide statistically meaningful results with a much smaller cohorts than predicted by the LNT hypothesis. LNT sample size arguments suggest that low-dose research is extremely difficult for typical occupational doses. This contention is negated by more careful analyses [53, 106].
Tubiana and Aurengo [55]	A review of human and environmental data does not support the LNT hypothesis that increasing dose leads to an increased radiological detriment.	The French Academies of Science and Medicine reviewed the validity of the LNT dose response relationship. Tubiana and Aurengo conclude that the LNT hypothesis overestimates the radiological risk. In addition, use of the LNT hypothesis may discourage physicians and patients from utilizing radiological examinations because the risk is assumed to be large. The arguments of Tubiana and Aurengo against the validity of LNT hypothesis are based on various data including the following: (1) there is no epidemiological evidence for cancer excess in humans for doses below 100 mSv, (2) there is no experimental animal data for carcinogenic effects for doses below 100 mSv, (3) practical thresholds or hormetic effects have been observed in a large number of experimental studies, and (4) DNA repair and elimination by the death of cells with DNA damage varies with dose and dose rate.

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Reference	LNT Impact	Primary Conclusions[a]
Pollycove [61]	The LNT hypothesis ignores the human immune system that is an important factor in establishing the dose response function. Low-doses of radiation have a positive benefit in contrast to the LNT hypothesis.	The human immune system is a key factor in cancer development, but it is ignored by the LNT hypothesis. As described by Polycove, DNA alterations from background radiation produce about one additional mutation per 10 million cells/d. These values apply to a young adult, living in a low LET background of 1 mSv/y. As ageing progresses, mutations accumulate and gradually degrade the antimutagenic system, and mortality increases correspondingly. Cancer increases at about the fourth power of age. Pollycove notes that genomic, cellular, animal and human data have shown that low-dose ionizing radiation, including acute doses up to 300 mGy, stimulates the immune system. However, high-dose ionizing radiation suppresses the immune system. Studies of cancer in animals and clinical trials of patients with cancer also show, with high statistical confidence, the beneficial effects of low-dose radiation.
Orsini et al [64], Woods et al [73] and Fogarty et al [85]	An enhanced immune system response reduces the risk of cancers. The LNT hypothesis fails to consider the impact of the immune system.	Fogarty et al observe that high-intensity exercise produces free radicals that causes increased DNA damage. Woods et al determine that cardiovascular exercise training results in improved antibody responses to influenza vaccination by boosting the immune system response. Orsini et al. suggest that higher levels of physical activity and an active lifestyle are associated with reduced cancer incidence and mortality and increased cancer survival. These studies are consistent with an immune suppression model associated with cancer development.

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Reference	LNT Impact	Primary Conclusions[a]
Rithidech and Scott [65]	The observed hormetic effect is inconsistent with the LNT hypothesis.	Rithidech and Scott demonstrate gamma ray hormesis during low-dose neutron irradiation. The gamma rays are derived from (n, γ) neutron capture reactions in tissue. This protective effect may be responsible for the neutron RBE energy dependence when the total radiation dose is ≤100 mGy. The authors suggest that the hormetic effect is based on the gamma-ray activation of high-fidelity DNA repair and stimulation of apoptosis in aberrant cells. Therefore, the RBE for neutron induced stochastic radiobiological effects may depend on physical (e.g., LET and lineal energy spectra) as well as biological (DNA repair and apoptosis) effects. Stimulation of the immune system by low-doses of gamma rays could also impact the low-dose neutron RBE for *in vivo* radiobiological effects such as cancer.
Yablokov et al [74], Levinger [148], and Siegel et al [151,152]	Siegel et al suggest the data of Yablokov et al support a threshold when properly evaluated. The existence of a threshold is inconsistent with the LNT hypothesis.	Siegel et al note that the data of Yablokov et al [74] as interpreted by Levinger do not indicate a linear response. When properly interpreted, the data suggest the existence of a threshold.
Shimizu et al [82], Levinger [148], and Siegel et al [151,152]	Siegel et al suggest the data support a threshold when properly evaluated. The existence of a threshold is inconsistent with the LNT hypothesis.	Siegel et al note that Figures 1 and 2 of Shimizu et al do not indicate linear responses down to 0.1 Gy or 0.05 Gy as asserted by Levinger. However, if properly interpreted, these data suggest thresholds. Shimizu et al admit that the existence of risk below 0.5 Gy is "unclear." Levinger asserts without evidence that the LNT hypothesis is probably true.

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Reference	LNT Impact	Primary Conclusions[a]
Koana and Tsujimura [79]	The dose response curve is not linear as proposed by the LNT hypothesis.	Koana and Tsujimura determined a U-shaped dose-response relationship for mutation frequency in Drosophila as a function of absorbed dose. These data suggest that DNA repair was responsible for the U-shaped dose-response relationship in Drosophila.
American Association of Physicists in Medicine Position Statement PP 25-A [83]	The predictions of the LNT hypothesis are inappropriate for decisions involving the use of low-dose medical imaging. Risks for effective doses below 50 mSv for single procedures or 100 mSv for multiple procedures over short time periods are too low to be detectable and may be nonexistent.	The American Association of Physicists in Medicine (AAPM) Position Statement PP 25-A "*acknowledges that medical imaging procedures should be appropriate and conducted at the lowest radiation dose consistent with acquisition of the desired information. Discussion of risks related to radiation dose from medical imaging procedures should be accompanied by acknowledgement of the benefits of the procedures. Risks of medical imaging at effective doses below 50 mSv for single procedures or 100 mSv for multiple procedures over short time periods are too low to be detectable and may be nonexistent. Predictions of hypothetical cancer incidence and deaths in patient populations exposed to such low-doses are highly speculative and should be discouraged. These predictions are harmful because they lead to sensationalistic articles in the public media that cause some patients and parents to refuse medical imaging procedures, placing them at substantial risk by not receiving the clinical benefits of the prescribed procedures.*" This AAPM declaration directly contradicts the LNT hypothesis. Patient radiophobia of low doses of ionizing radiation are an unfortunate consequence of the LNT hypothesis and ALARA concept.

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Table 11

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Reference	LNT Impact	Primary Conclusions[a]
Tubiana et al [88]	Second cancers decreased with increasing absorbed dose up to about 200 mGy which is in conflict with the LNT hypothesis.	Tubiana et al investigate a new method of assessing the dose-carcinogenic effect relationship in radiotherapy patients exposed to ionizing radiation. A reduction of second cancers per kg of tissue was noted in regions of the body receiving an absorbed dose of about 200 mGy when compared to regions not subjected to any radiation dose
Beyea [91] and Siegel et al [151,152]	Siegel et al suggest the Soviet Techna River data analysis is flawed and does not support the LNT hypothesis.	Siegel et al note that the data analysis of Beyea is flawed and promulgates an illegitimate statistical ploy. A proper analysis does not support the LNT hypothesis.
Ozasa et al [98]	A basic tenant of the LNT hypothesis is invalidated by the most recent Japanese atomic bomb survivor data evaluation.	The latest update (RERF Report 14) of the Japanese atomic bomb survivor data no longer supports the LNT model. The dose-response data are not linear and have a significant curvature. Bomb survivor data is the gold-standard for the presumed basis for the LNT hypothesis. Observation of curvature in the dose response data, undermines the LNT approach to radiation protection and the associated radiological risk.
Levin [95], Oliveira- Cobucci et al [97], and Yang et al [118]	The LNT hypothesis fails to consider the immune system which is an important consideration in determining cancer risk. Suppression of the immune system enhances cancer progression, but is not included in the LNT cancer assertions.	Oliveira-Cobucci et al note that suppression of the immune system increases the cancer risk in transplant and HIV patients by a factor of about 3. This demonstrates the importance of the immune system for minimizing cancer progression. In investigating T-cell-mediated immunity, Levin observes that the immune system response declines rapidly with age. These data qualitatively explain the age-related increase in cancers. Yang et al observe that low-doses of ionizing radiation induce a direct expansion and activation of the defense system, which provides a potential mechanism for stimulation to enhance adaptive cellular immunity. These data suggest that low-dose radiation boosts the immune system.

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Reference	LNT Impact	Primary Conclusions[a]
Pearce et al [99], UNSCEAR 2013 [110], Boice [120], and Journy et al [126]	The study used to justify the LNT hypothesis has been challenged for containing significant flaws in its design.	Pearce et al observed an increased incidence of cancers following childhood CT brain scans, and is routinely quoted as evidence for cancer risk from low-dose radiation (e.g., Leuraud et al [127]). The authors suggest that the brain cancer risk increases with radiation dose. Boyce (2015) noted the Pearce et al study must be interpreted with caution. The reasons for performing the CT exams were not known, and the dosimetric approaches did not include individual dose reconstructions or account for the possibility for missed examinations. UNSCEAR 2013 concluded that the associations may have resulted from confounding factors, and not radiation exposure. The reported cancer associations may have been related to the patients' underlying health conditions that prompted the examinations. The study design contains weaknesses that cast doubt on its conclusions. Other studies considered the reason for performing CT scans and noted no increase in cancer risk with CT radiation dose, (e.g., (Journy et al). Journy et al suggest that the indication for examinations, whether suspected cancer or cancer-predisposing factors, should be considered to avoid overestimation of the cancer risks associated with CT scans.

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Reference	LNT Impact	Primary Conclusions[a]
Levin [95] and DeGregori [105]	Although mutations are necessary for causing cancers, they are not the only cause. Suppression of the immune system is a key factor that causes cancers and this consideration is not included within the scope of the LNT hypothesis.	In a study of young animals, DeGregori observed that cells dividing at the highest rates are most susceptible to mutations which is the expected result. In addition, the accumulation of mutations also occurs at the highest rates. However, Levin notes that the immune system response is at its highest level at a young age. Levin's work suggests that cancer rates would be at the lowest levels at young age which is supported by the immune suppression model of cancer. The low cancer rates observed in the young is consistent with the immune suppression model of cancer, but is in conflict with the mutation model.
Little et al [96], Little [108], Akiba [101], and Doss [114].	Arguments by Akiba and Doss negate the contentions of Little et al and Little. The existence of a threshold negates the LNT hypothesis.	Little and coworkers suggest an excess radiation risk at dose levels below 500 mSv, and also argue that there is accumulating evidence from the Japanese atomic bomb survivors and various other moderate and low-dose exposed groups of an excess risk of cataracts. However, Akiba performed an extensive review of the contentions of Little et al and Little summarized in Table 8. A variety of radiation and associated detriment information are evaluated by Akiba including data from the Mayak Production Association workers, Electricite de France workers, Chernobyl emergency workers, and Japanese atomic bomb survivors. Akiba notes that the heart disease meta-analysis combined low-dose rate and high-dose rate data. This combination transferred the high-dose radiation risk to the low-dose region. Doss notes that the Chernobyl and atomic bomb survivor data do show a threshold dose for cataracts requiring surgery. The arguments of Akiba and Doss negate the contentions of Little and coworkers.

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Reference	LNT Impact	Primary Conclusions[a]
Feinendegen et al [102]	Low-doses have a hormetic effect which is inconsistent with the LNT hypothesis.	Feinendegen et al note that low-dose radiation activates defense mechanisms. This adaptive response results in protective measures including antioxidants, DNA repair enzymes, and apoptosis. These mechanisms reduce the damage that would have occurred in the absence of the low-dose radiation.
Ferlay et al [107] and World Nuclear Association [134]	Evidence supports a trend of reduced cancers with increasing background radiation dose which is in conflict with the LNT hypothesis.	The data of Ferlay et al and the World Nuclear Association (2015) support reduced cancer rates in European countries with the highest background radiation levels.
Osipov et al [109]	The dose response curve is not linear which is in conflict with the LNT hypothesis.	The data of Osipov et al indirectly indicate that low level ionizing radiation *in vivo* may trigger repair of DNA double strand breaks. There is a dose threshold for this defense mechanism. These molecular level *in vivo* data suggest that the dose-response for DNA double strand breaks at very low-doses and dose rates is not linear.
BEIR VIII Planning Meeting [116]	Initial selection of relevant data for BEIR VIII continues to ignore significant data refuting the LNT hypothesis.	The BEIR VIII Planning Meeting continues to rely on data supporting the LNT hypothesis. The initial effort did not recognize the major change in the nature of Atomic Bomb Survivor data (Ozasa et al [98], Doss [94], and Cuttler [113]). BEIR VIII quoted the 15-country study of radiation workers as evidence for low-dose radiation cancer risk (Cardis et al [50]), in spite of withdrawal of Canadian data [84]. The initial discussions also ignore data illustrating a cancer reduction from low-dose radiation including the Nuclear Shipyard Worker Study (Sponsler and Cameron [52]) and the study of second malignant neoplasms in radiation therapy patients (Tubiana et al [88]).

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Reference	LNT Impact	Primary Conclusions[a]
Cuttler [113]	The dose response curve exhibits a threshold which is in conflict with the LNT hypothesis.	Cuttler notes that the leukemia incidence and associated dose response curve for 96,000 Hiroshima atomic bomb survivors is inconsistent with the LNT model. The dose response curve exhibits a leukemia threshold of about 500 mGy for Hiroshima atomic bomb survivors
Allison [119]	Low-dose radiation stimulates the immune system and suppresses cancer incidence. This effect is in conflict with the LNT hypothesis.	Allison observes that the initial effect of physical exercise and low-dose radiation on cells includes chemical action that increases the production of reactive oxidant species. These two stimuli elicit the same protective and adaptive responses. Moreover, a history of exercise and low-dose radiation exposure are both effective at stimulating adaptation. Doses of ionizing radiation at low rates suppress cancer incidence just as exercise does.
Cuttler and Welsh [122]	An error in atomic bomb survivor analysis of leukemia data invalidates the use of the LNT model. Thresholds noted by Cuttler and Welsh are inconsistent with the LNT hypothesis.	Cuttler and Welsh describe an error in the analysis of leukemia incidence among the 195,000 Japanese atomic bomb survivors. Based on this work, the threshold acute dose for radiation-induced leukemia is about 500 mSv. These authors note that it is reasonable to expect that the thresholds for other cancer types are higher than this level.

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Reference	LNT Impact	Primary Conclusions[a]
ICRP 131 [125]	In ICRP 131, support for the LNT hypothesis is less assertive than noted in previous ICRP publications. The report also notes that other models can be applied to the data. Key references supporting the LNT basis for ICRP 131 (e.g., RERF 13 [42] atomic bomb survivor data and Cardis et al [50]) have been shown to be invalid.	ICRP 131 notes that the LNT model is generally consistent with human epidemiological data of cancer induction in human populations exposed to ionizing radiation. The atomic bomb survivor data is judged by ICRP 131 to be the gold standard of human data supporting the LNT hypothesis. This statement fails to consider that the latest update of the atomic bomb survivor data of Ozasa et al [98] that no longer supports the LNT model. The dose-response data are not linear and have a significant curvature. ICRP 131 also notes that "there are a few clear tissue-specific exceptions to the general rule and that other models can be equally applied in some cases". As noted in ICRP 131, the general rule is the use of the LNT hypothesis.
Oakley [129]	Hormesis and immune system stimulation provide a cancer therapy approach that is inconsistent with the LNT hypothesis.	Oakley reports that low-dose total-body irradiation (TBI) therapy offers an additional radiation treatment to cancer patients. TBI is based upon the concept of radiation hormesis and provides very good success rates. The TBI treatment modality has been reported as an abscopal effect where the localized treatment of a tumor causes not only a shrinking of the treated tumor, but also a shrinking of tumors outside the targeted treatment volume. Compared to the contemporary treatments, such as immunotherapy drugs and localized high-dose radiation, Oakley notes that low-dose radiation stimulates the immune system, seems logical, and may prove to be superior.

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Reference	LNT Impact	Primary Conclusions[a]
Pateras et al [130]	Ionizing radiation triggers an immune system response that is not considered as an integral aspect of the LNT hypothesis.	Pateras et al provide evidence that the DNA damage response and repair (DDR/R) and immune response (ImmR) work together to enhance the function of cellular organisms. For example, DNA and RNA viruses directly and indirectly activate the DDR/R mechanisms in host cells. The DDR/R activation favors the immunogenicity of the incipient cell. Pateras et al suggest stimulation of DDR/R by cellular insults, including ionizing radiation, triggers innate and adaptive ImmR. Ionizing radiation is a DDR/R inducing agent and is an example of how DDR/R stimulation induces host immunity. The emerging DDR/R–ImmR concept opens up a new avenue of therapeutic options including ionizing radiation to stimulate the immune system response.
Rudant et al [131]	The LNT hypothesis does not fully consider the effects of the human immune system	Rudant et al study the effects of boosting the human immune system and its impact on the incidence of childhood leukemia. The immune system in children is enhanced with an increased rate of breastfeeding and earlier childcare attendance in daycare which subjects children to increased rate of infections. Both of these conditions stimulate the immune system and reduce the risk of childhood leukemia.

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Reference	LNT Impact	Primary Conclusions[a]
Tang and Loke [132]	The LNT hypothesis does not consider a variety of positive molecular mechanisms and effects attributed to low-dose ionizing radiation.	Tang and Loke review the molecular mechanisms of low-dose ionizing radiation (LDIR)-induced hormesis, adaptive responses, radioresistance, bystander effects, and genomic instability. LDIR has been reported to induce hormesis, adaptive response, radioresistance, bystander effect and genomic instability in living cells, tissues, organs, and the whole body. These radiation-induced responses are affected by an individual's genetic composition. The adaptive response may be considered as a special hormetic response or a manifestation of radioresistance. It may protect against bystander damage, but the bystander effect may induce genomic instability. The interrelationship among different responses suggests that they may have shared signal transduction pathways. Since many different signal transduction pathways are involved in LDIR-induced responses, the same pathways may be shared by different responses. Activation of some of these pathways may induce defensive or beneficial responses such as immunity, detoxification of reactive oxygen species, repair of DNA damage, and stem cell proliferation. However, activation of the same signal transduction pathways may also induce harmful effects such as genomic instability. Tang and Loke suggest that further studies are needed to determine the particular signal transduction pathways that can produce positive effects while preventing LDIR negative effects.

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Reference	LNT Impact	Primary Conclusions[a]
Cohen [144]	The LNT hypothesis is based on flawed and incomplete assumptions. LNT implementation in using low-dose CT procedures has had a negative impact on radiologists and associated patient diagnosis.	Cohen observes that there is no definitive scientific proof that low-doses of radiation from computed tomography (CT) imaging increase cancer risk. From a physician's perspective, Cohen notes that the ALARA and Image Gently Philosophy have caused harm to the profession of radiology and to patients. Accordingly, ALARA and Image Gently as they now exist should be terminated. Patient cancer risk from CT is nonexistent or minimal. The risk is equivalent to the normal risks of daily living.
Cuttler et al [145]	Low level radiation exposure appears to trigger adaptive response mechanisms to improve the condition of a patient with Alzheimer disease.	In a case report, Cuttler et al describe the improvement in a patient with advanced Alzheimer disease. The individual received 5 computed tomography brain scans of about 40 mGy each over a period of 3 months. Patient improvement appears to be radiation-induced stimulation of the adaptive protection systems. The treatment appears to have partially restored cognition, memory, speech, movement, and appetite. Although a single study, the case study is another example of the positive effects of low-dose radiation in treating disease.
Grass et al [126]	The LNT hypothesis does not fully consider the immune system that is an important consideration in cancer progression.	Therapeutic effects of radiation therapy apart from those observed at the treatment target (i.e., abscopal effect) have been observed for several decades. However, the underlying mechanisms regulating this phenomenon have not been clearly defined [132]. Grass et al observe that the immune system is a major determinant in regulating the abscopal effect, and that radiation therapy may enhance immunologic responses to tumors. Harnessing the immune system to target tumors in conjunction with radiation therapy is an emerging field with much promise. To optimize this approach, the host immune system, immunotherapy, and radiation therapy should be evaluated in a comprehensive manner.

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Reference	LNT Impact	Primary Conclusions[a]
Sacks et al [150]	LNT epidemiological studies are often based on weak or misleading data analysis. The LNT hypothesis fails to consider biological repair mechanisms and the validity of hormesis.	Sacks et al note that epidemiological studies that claim to confirm the LNT hypothesis either neglect experimental and/or observational discoveries at the cellular, tissue, and organismal levels, or mention them only to distort or dismiss them. Studies that claim to validate the LNT hypothesis rely on circular reasoning, biased data selection, faulty experimental design, and misleading inferences from weak statistical evidence. Sacks et al further observe that studies confirming hormesis are firmly based on biological discoveries. In particular, these biological studies demonstrate the validity of hormesis, and confirm the stimulation of biological responses that defend the organism against damage from environmental agents. Failure of the LNT hypothesis is also suggested from understanding of normal metabolic processes that are far more damaging than all but the most extreme exposures to radiation. However, Sacks et al note that evolution has provided plants and animals with defense mechanisms that repair such damage or remove the damaged cells. These repair mechanisms confer on the organism even greater ability to defend against subsequent damage. Sacks et al summarize the extent of damage caused by the LNT hypothesis in the practice of radiology, radiation regulatory policies, and the popular media culture. The result is mass radiophobia and harmful outcomes, including forced relocations of populations near nuclear power plant accidents (e.g., the 2011 Fukushima Daiichi accident in Japan), reluctance to avail oneself of needed medical imaging studies, and aversion to nuclear energy. All of these actions are unwarranted and harmful to humanity.

[a]See Section 9.1 - 10 regarding the primary conclusions and the lack of scientific consensus in data used to support and refute the LNT hypothesis as summarized in Table 8 and 11.

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A number of conclusions are suggested by the data summarized in Table 11. These conclusions include the: (1) importance of the immune system in the suppression of cancers, (2) immune system enhancement following low-dose radiation, (3) positive effects attributed to hormesis in populations exposed to low levels of ionizing radiation, (4) updated atomic bomb survivor data no longer support the LNT hypothesis, (5) key LNT references (e.g., Cardis et al [50]) being invalidated by updated data, (6) existence of thresholds, (7) use of modifying data (e.g., DEF, DREF, and DDREF)) to support inconsistencies between data and LNT predictions, (8) curative effects of low-dose radiation, (9) importance of DNA and natural repair mechanisms, (10) importance of signal transduction pathways, and (11) inconsistency between the LNT hypothesis and variations in background radiation levels. Any of these conclusions raise questions regarding the LNT hypothesis. Moreover, the reference data, noted in Table 11, suggest that the LNT hypothesis is unsustainable and an inappropriate basis for existing radiation protection regulations.

It is generally accepted that a primary reason for cancer is the transformation of a normal cell into a cancer cell through mutations. These mutations occur following DNA damage that causes the cell to malfunction. Greaves [115,124] notes that cancer cells exist in most human bodies, but everyone does not develop cancer. Imaida et al [32] performed an autopsy study and studied the existence of cancer cells as a function of age. Although the percentage of patients with cancer cells was relatively unchanged from ages 50 to 80, the cancer mortality rate increased by more than an order of magnitude between these ages [153]. This increase with a constant concentration of cancer cells indicates there is another cause for the incidence of the aforementioned cancers. In fact, the primary cause of these cancers is the suppression of the immune system as the body ages [95]. As noted previously, the immune system is not fully incorporated into the LNT hypothesis. Omitting an obvious and important aspect of cancer suppression is a major failing of the LNT hypothesis.

The naturally occurring mutations that exist in most bodies are influenced by ionizing radiation. Feinendegen et al. [102] notes that low level radiation exposure stimulates the immune system, and these increased defenses reduce the number of mutations that would have occurred naturally. This results in fewer mutations overall, and this effect has been observed in animal studies [109].

The effects expected from hormesis are observed in populations exposed to low levels of ionizing radiation. Studies of Frigerio et al [7] observed a trend of lower US cancer mortality rates associated with higher background radiation levels. Cohen [30] noted a strong tendency for lung cancer rates to decrease with increasing radon exposure, in sharp contrast to the increase expected from the linear non-threshold theory. Ferlay et al [107] with the background radiation estimates from World Nuclear Association [134] have published data supporting reduced cancer rates in European countries with the highest background radiation levels.

Ozasa et al [98], Cuttler [113], and Cuttler and Welsh [122] observe that the dose-response data for the Japanese atomic bomb survivors is not linear and has a significant

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curvature. Doss [106] notes the data appears to have a distinct dip at about 500 mSv and this feature was also noted by Cuttler [113] and Cuttler and Welsh [122].

9.1 Fundamental Conflicts

The reports summarized in Table 11 were available to the ICRP, NCRP, BEIR, DOE, NRC, and other advisory and regulatory organizations. However, these organizations do not share the author's conclusions which are an important consideration for the reader. Tables 8 and 11 illustrate the fundamental issues associated with the LNT hypothesis. These issues involve various interpretations of data and the complete lack of consensus among knowledgeable professionals. However, is this consensus a result of scientific data or a vested interest in the status quo and its associated benefits?

A comparison of Tables 8 and 11 reveals an interesting comparison. The LNT proponents and organizations base their arguments only on presumed damage, and neglect data regarding the biological response of human repair mechanisms and the immune system. Table 11 also provides evidence for hormesis and disease treatment potential as a byproduct of low-dose radiation exposure. These positive benefits are rejected by LNT proponents in spite of growing evidence for their existence in a variety of studies.

There is a growing tide of evidence that is in conflict with the LNT hypothesis. This accumulating research clearly illustrates the inherent weakness of the LNT approach.

9.2 LNT Resolutions

Although the collection of results summarized previously offer evidence that favors rejection of the LNT hypothesis, its proponents ultimately resort to one final argument when presented with the quality and abundance of data summarized in Table 11. This argument relies on the observation that no epidemiological study, with an appropriate unirradiated control group, has *definitely* demonstrated either the detrimental or beneficial effects of ionizing radiation doses less than 100 mSv in humans. As noted in Section 3.2, this argument is incorrect and a number of studies demonstrate a beneficial effect. The LNT approach suggests that assessing the risks of low-doses of ionizing radiation would require large scale epidemiological studies with long-term follow-up activities to accurately assess the associated detriment or benefit of the exposure. The discussion in Section 3.2 demonstrates that these conclusions are an artifact of the LNT hypothesis and are also incorrect. Since credible results can be obtained with much smaller sample sizes than suggested by the LNT hypothesis, research that further demonstrates the positive benefit of low-dose radiation should be actively supported. These studies would provide additional data to supplement the research noted in Table 11.

Issues associated with sample size are not new and have been discussed in a number of reports including BEIR III [13], V [20], and VII [56]. Table 12 further supports the discussion in Section 3.2 and illustrates the cohort sizes required for statistically meaningful results [24]. As

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noted in Table 12, the LNT based sample sizes are clearly challenging as the doses of interest decrease in magnitude.

The sample size arguments summarized in Table 12 are based on LNT model estimates. Previous discussion summarized in Section 3.2 and Table 11, demonstrate that credible results are obtained with significantly smaller sample sizes than required by the LNT hypothesis. Therefore, sample size arguments utilized by LNT hypothesis proponents do not have merit and are not justified by data.

Table 12
Required Epidemiological Sample Size for Various Doses of Low LET Radiation[a]

Effective dose (mSv)	Required Number of Individuals in the Exposed Group
100	$5x10^4$
10	$5x10^6$
1	$5x10^8$
0.1	$5x10^{10}$
0.01	$5x10^{12}$

[a] Based on Ref. 24 that used the LNT hypothesis.

LNT supporters suggest the only regulatory option is to accept the assumed detriment proposed by their flawed hypothesis. This argument is logically inconsistent. Since the LNT hypothesis excludes thresholds, hormesis, impact of the human immune system, and any nonlinear effect, validation of any of these items voids this approach. The wealth of reference data of Table 11 provides ample evidence for nonlinear effects, hormesis, immune system impact, and the existence of thresholds. Therefore, reasonable arguments support the abandonment of the LNT hypothesis and its replacement. If replaced, what is the appropriate model that will serve as the successor to the LNT hypothesis?

9.3 New Physical Interpretations

The radiation dose delivered to tissue is typically characterized by the energy absorbed per unit mass or absorbed dose. Absorbed dose is evaluated over a specified volume that is typically characterized by a length scale much larger than a few nm. However, the physical interpretation of the energy deposition mechanism is evaluated at the nm scale.

Ostrikov et al [149] observe that distinct physical phenomena (e.g., plasma production) arise following the localization of energy densities at the microscale and nanoscale realm. These

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effects can be achieved following the concentration of radiation into small volumes that lead to extreme energy densities. For example, depositing 1 MeV ($1.6x10^{-13}$ J) into a volume of 1000 nm^3 during 100 fs could lead to a power density of 10^{24} W/m^3. The physical effects of DNA damage at these densities have not been rigorously investigated and may provide additional insight into the failure of the LNT hypothesis.

A very preliminary review of these high localized densities suggests that DNA damage would be limited to the immediate reaction volume [149]. It is likely that the redundant, undamaged DNA would facilitate repair of the damaged volume. The inherent redundancy is a key aspect of DNA repair even at these extreme power densities. This localized damage is readily managed by the repair mechanisms summarized in Section 5.0 which would support the contentions of the Table 11 references.

These high power densities would also minimize the probability of damaged replication because all matter in the volume of these extreme power densities would be obliterated. This approach to radiation damage has yet to be rigorously investigated and at this stage of development remain speculative.

10.0 Future Regulations

Credible approaches to eliminate the LNT hypothesis and ALARA from US regulations have been proposed [128], but have not been positively received. In view of this situation, this paper proposes an initial step toward the goals embodied in Ref. 128. This first step is not the final goal, but it may be necessary in view the current US regulatory climate.

Viable regulatory bases for radiation protection regulations should investigate thresholds and utilize alternative dose response and risk models. To evaluate future regulatory proposals, all radiation data must be assessed and radiation protection regulations should not be based solely on high-dose and high-dose rate data linearly extrapolated to low-doses. The new regulatory basis should also investigate the need to incorporate dose and dose rate effectiveness factors, thresholds, adaptive response, positive impact of the human immune system, and hormesis.

10.1 New Regulatory Options

Any new regulatory approach must involve both pro- and anti-LNT advocates. Although the previous discussion offers strong evidence against the LNT hypothesis, this argument will not be accepted by all groups involved in the regulatory process. If future radiation protection regulations are to be forthcoming, they must be accepted by the radiation protection community. Accordingly, the following discussion is written from that perspective.

It has been over 60 years since the last significant change in the basis for radiation protection regulations. Prior to the 1950s, skin erythema was a major concern with radiation use, and physicians treated common diseases and conditions with radiation. In the 1950s, the

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observation of the increased incidence of leukemia in atomic bomb survivors shifted the radiation protection regulatory basis. Following the observation of detrimental effects from high-doses of ionizing radiation in studies of atomic bomb survivors, genetic effects became the dominant concern that led to the adoption of the current regulatory basis. With the adoption of the LNT hypothesis, advisory bodies such as NCRP and ICRP reduced the radiation dose limits. Since a number of studies suggest the validity of adaptive response, thresholds, impact of the human immune system, and radiation hormesis, it is time for a new radiation protection basis, and to abandon the LNT hypothesis.

Based on the data summarized in Table 11, a revised radiation protection basis should recognize adaptive response, the existence of thresholds for radiation detriment, non-linear effects, the human immune system, and the potential for the beneficial effects of low-dose radiation. Making this change will be challenging since it is contrary to the recommendations of most advisory bodies, current government regulations, and public perception regarding the effects of low-dose radiation. Attempts to change the current regulations will be viewed with suspicion by the public because of the widespread fear of radiation fostered by media coverage of significant radiological events (e.g., the power reactor accidents at Three Mile Island, Chernobyl, and Fukushima Daiichi) and general fear of anything related to radiation and radioactive materials. In addition, most members of the public have a limited knowledge of radiation and its associated health effects.

These challenges are significant and should be addressed in new regulations and their associated justification. New regulations should incorporate all available radiation data and not rely solely on high-dose data. Specific effects including adaptive response, hormesis, the positive impact of the human immune system, and thresholds must be thoroughly evaluated in terms of data sets that include the traditional high-dose data from the Japanese atomic bomb survivors and high-dose therapy patients as well as data sets that have not been thoroughly incorporated in the past. These data sets include occupational radiation protection dosimetry from power reactors, medical facilities, universities, fuel cycle facilities and government employees including the military; environmental data from areas of the world having elevated background radiation levels; and low-dose medical imaging data.

In addition to the inclusion of all data, the new radiation protection rules should consider a variety of dose response models and not solely rely on linear-non-threshold models. Risk models should be expanded to include other approaches that go beyond the historical absolute and relative risk approaches. Excess risk functions should also be expanded and utilize contemporary methods to fit data and not rely on traditional models utilizing step functions. Epidemiologists, health physicists, medical researchers, and radiation biologists must also perform rigorous evaluations to ascertain the radiation induced effects and the associated doses leading to these effects.

The scientific community should form a diverse group of professionals and interested public groups to develop the new regulatory approach. Adopting the proposed approach will

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present significant difficulties since the pro and anti LNT groups are firmly entrenched in their respective positions.

All participants must provide defensible data. This will be a challenge to the LNT proponents because the data summarized in Table 11 invalidate the LNT model. Participants should be asked to justify their positions based on valid, peer reviewed data. If they cannot, the individuals should be excluded from the new regulatory approach discussions.

In addition, an educational outreach campaign should be conducted to correct the current misconceptions in the scientific community, public, and government regarding the pathogenesis of clinical cancer and the biological effects of low-dose radiation. Research to demonstrate the beneficial health effects of low-dose radiation is essential to reduce the fear of radiation, and facilitate an understanding of its benefits. Finally, scientific accountability is needed. Statements and pronouncements of new regulatory approaches must be presented in terms of complete, peer reviewed methods, and data analysis. Communicating the new regulatory format and its basis should involve scientific organizations; industrial users of radiation and radioactive materials; medical professionals; researchers; local, state, and federal governments; labor unions; and public interest groups.

Alternatives for new regulatory approaches are numerous, but must conform to experimental observations. For example, inclusion of a threshold dose would provide additional credibility to radiation protection regulations. The threshold represents a dose below which no biological effect or detriment would occur. The threshold value would require careful evaluation, but a number of options exist. For example, the lowest dose where acute radiation effects are observed would provide an upper bound for a threshold. Large studies have not revealed an increased incidence of chromosomal aberrations at doses below about 20 mSv [36].

Another possibility would set the threshold at the level of a typical background or environmental dose (e.g., 3 mSv in the US) [70]. Using the environmental threshold level is supported by the large variability in the earth's background radiation level and the lack of observed radiation related health effects in high-dose areas of the world (e.g., India and Iran). Higher thresholds are justified, but the 3 mSv value would be an initial value subject to upward revisions as the scientific community and public became more comfortable with abandoning the LNT philosophy and research supported higher levels.

An additional regulatory format could include a threshold and then a data based extrapolation from the threshold dose to higher dose data. This approach creates a *de minimis* dose that would be exempt from regulatory control with dose limits based on values above this threshold.

If the 3 mSv exemption were adopted, it would have a significant impact on the practice of radiation protection particularly for power reactor and fuel cycle facilities. For example, application of this approach to power reactors would significantly reduce the radiation protection requirements. Since many workers do not exceed 3 mSv/y, radiation protection programs could focus on the more hazardous activities and not be burdened by regulatory concerns regarding

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minor skin contamination events, low level intakes of radioactive material, and excessive ALARA reviews for worker doses below the *de minimis* threshold.

Higher *de minimis* levels merit consideration, particularly for medical imaging and therapy applications. Recent work clearly demonstrates the need for reinforcing the benefits of using radiation and radioactive materials in medical imaging.

As applied to medical imaging, Siegel [154] and Siegel and coworkers [155] succinctly outline the fallacy of the LNT hypothesis and its illegitimate ALARA progeny. These authors note that credible evidence of imaging-related carcinogenic risk at low absorbed dose (<100 mGy) is nonexistent. Any perceived risk is a hypothetical consequence of the presumed validity of the scientifically unjustified LNT hypothesis. Low-dose radiation does not cause, but more likely helps prevent, cancer. Siegel et al [155] observe that the LNT hypothesis and associated ALARA concepts are fatally flawed and focus only on molecular damage while ignoring protective, organismal biologic responses. Table 11 and Refs 154-156 summarize the societal harm caused by the LNT hypothesis and ALARA.

The LNT hypothesis also affects acceptance of the use of radiation and radioactive materials and causes the ALARA concept to create harm rather than the presumed benefit. These concepts create a world in which ALARA created radiophobia is continually reinforced.

Radiophobia has inhibited research using low-dose radiation in the detection, prevention, and treatment of cancer and other diseases. Unwarranted fears caused by belief in the LNT hypothesis have also effectively inhibited research involving unique applications of radiation and radioactive materials. These applications include the use of low-dose radiation as a diagnostic protocol [154-156].

Patients have refused computed tomography scans and physicians are not prescribing these procedures because the LNT hypothesis/ALARA dogma has created concern for the subsequent radiation detriment. This fear could result in missed diagnoses because imaging doses are too low to produce adequate tissue resolution [144].

10.2 Conclusions and Recommendations

The reports and associated data summarized in Tables 8 and 11 present information used to support and challenge the LNT hypothesis, respectively. Both sides present arguments backed by data of varying quality, but data strongly favor rejection of the LNT hypothesis. Moreover, the observed positive benefits of low-dose radiation noted in Table 11 provide a strong basis for elimination of the LNT hypothesis and its associated ALARA principle.

Based on the results of Tables 8 and 11 and the references cited in this paper, the following conclusions and recommendations are offered:

1. High-dose radiation is useful and has a positive effect in treating cancer. The use of radiopharmaceuticals and external beams has a proven record in treating cancer.

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2. The biological responses to low-dose and high-dose radiation are fundamentally different. One of the key differences is activation of the human immune system following low-dose radiation exposure.

3. The biological response to ionizing radiation is an important consideration in mitigating the detriment of low-dose radiation. As a matter of design, the human immune system is activated by low-dose radiation to counter biological detriments produced by the ionizing radiation. Moreover, a growing body of research supports a net positive benefit from low-dose radiation.

4. Low-dose radiation has been shown in numerous cases to have a positive effect to lower the risk of cancer and to alleviate other medical conditions.

5. Low-dose radiation does not imply risk. In fact, the low-dose radiation has a positive or hormetic effect.

6. Although research involving the effects of low-dose radiation should be encouraged, it should be focused on improving the early medical approaches for utilizing low-dose radiation to treat illness, lower cancer risk, and improve longevity. Investigation of signal pathways [132] will be an important consideration in optimizing low-dose treatment approaches. This research will justify replacing the flawed ALARA concept with the radiation induced disease eradication and suppression (RIDES) approach. The RIDES approach will permit low-dose ionizing radiation to become a powerful medical treatment protocol.

7. Physicians should be free to select treatment methods without being influenced by concerns for the radiation dose delivered to a patient. The physician should act in the best interest of a patient and not be influenced by reports such as NCRP 160 [70] that has been used to encourage minimal dose delivery for imaging procedures. Medical personnel should be free of such influences and take measures deemed to be in the best interest of patients. The RIDES approach should be implemented when appropriate and replace the ALARA philosophy.

8. *De minimis* dose (DMD) levels should be established below which personnel do not require radiation monitoring or control. Establishing a DMD has several options. The DMD could be based on the annual background dose (e.g., 3 mSv in the US). Given the variability of background levels throughout the world and the lack of increased cancer incidence at locations of higher dose levels, this approach is justified and could be set at a higher value. Different DMD values for occupational exposure and medical exposures, including patients receiving radiation or radioactive materials prescribed by a physician, should be considered. For medical exposures, physicians should have maximum flexibility to treat their patients to ensure their health and well-being.

9. Radiation protection regulations based on the DMD and RIDES approaches could be further strengthened by research that investigates variations in genetic susceptibility in radiation workers. Workers found to have a genetic composition that increases the risk of radiological work should be excluded from high dose radiation environments based on improving their quality of life.

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Since, genetic susceptibility has been observed for high dose radiation only [13,20,56], studies should be performed to determine the effect of low-dose radiation on genetically susceptible individuals. Based on Table 11, low dose radiation may improve their health. This research should include physicians, scientists, regulators, and the public to ensure that human rights and opportunities are considered in developing a regulatory format that considers genetic factors.

These considerations are not unique and can be improved by other authors challenging this work, further expanding its content, and offering additional alternatives. There is no magic solution to moving radiation protection regulations from the traditional LNT and ALARA paradigms to the DMD and RIDES approaches. However, there are significant benefits to this approach and these efforts will foster a better allocation of resources, promote worker and patient health and safety, and expand the beneficial uses of radiation and radioactive materials.

As a supplement to the quantitative recommendations noted previously, risk assessments, such as those proposed by the Health Physics Society [78,147], can be used as a regulatory basis to select from a group of options associated with work involving radiation exposure. This risk assessment approach can be applied to a variety of radiological work activities including the selection of methods to remediate sites contaminated with radioactive material, disposition of low activity radioactive material, recovery options following a reactor accident, transport of radioactive material, and selection of decontamination end state criteria.

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12.0 Conflict of Interest

The author notes that he was a member of the Board of Directors and Treasurer of the XLNT Foundation (www.x-lnt.org). The mission of the XLNT Foundation is “To inform the public on the observed beneficial effects of low-dose ionizing radiation, and to campaign for eliminating use of the linear no-threshold (LNT) model in order to enhance public health.” The author’s membership in the SARI group which is opposed to the LNT hypothesis is also acknowledged. Any bias expressed in this paper is unintentional and solely the responsibility of the author.

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