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Strengthening Transparency in Regulatory Science
Docket ID No. EPA–HQ–OA–2018–0259

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Comments of Beyond Nuclear

EPA should abandon finalization of this rule. While in principle, we applaud transparency of the science on which regulations are based, for radiation protection this proposed rule fails to fully address the real regulatory challenges EPA faces. In the case of radiation damage models, and specifically the linear and no threshold models, transparency is not the overarching concern. The overarching concern is that EPA has failed to protect women, children and pregnancy. Making EPA's science more transparent while simultaneously allowing use of models even less protective than current ones, would risk their health further.

The linear, no threshold (LNT) model used to represent cancer risk from radiation exposure has been peer reviewed already and is supported by the majority of independent scientific research that exists to date. If anything, science also demonstrates that current EPA use of LNT underestimates cancer risk by averaging those more susceptible to radiation damage—females and children—with adult males. Further, EPA only uses the LNT model for *cancer*. EPA provides no comparable assessment for non-cancer impacts of radiation, basically ignoring major and unique developmental processes during pregnancy.

The trouble with EPA's radiation models is not a lack of transparency and peer review for the models it *does* use. EPA was provided peer review and transparency, to a degree, through a National Academy of Sciences committee process and report on these issues. The trouble is that EPA wields the model in a way that fails to protect a majority of the population by 1) underestimating cancer risk for women and children and 2) failing to provide *any* model for non-cancer impacts, leaving them completely unaccounted for. Further, EPA feels it can abandon even these insufficient protections in the face of nuclear catastrophe and other "incidents" (requiring consideration of remediation and response) as clearly shown by its adoption of the Protective Action Guides or PAGs.

A) EPA instituted a peer review for radiation models by tasking the National Academy of Sciences (NAS) to conduct a review of radiation science, including the linear, no threshold, and other models. The 2006 NAS BEIR VII report supported use of the LNT after examination of the data, but failed to point out the increased harm to women and children apparent in these data, even with use of LNT.

Proposed rule EPA–HQ–OA–2018–0259 states *"EPA shall conduct independent peer review on all pivotal regulatory science used to justify regulatory decisions, consistent with the requirements of the OMB Final Information Quality Bulletin for Peer Review (70 FR 2664) and the exemptions described therein. Because transparency in regulatory science includes addressing issues associated with assumptions used in models, EPA shall ask peer reviewers to articulate the strengths and weaknesses of EPA's justification for the assumptions applied and the implications of those assumptions for the results."*

OMB Rule 70 FR 2664, EPA's benchmark for peer review, determines that "highly influential scientific assessments" should be peer-reviewed but also leaves the agency a lot of discretion in doing so. OMB states that the agency

> …must ensure that the peer review process is transparent by making available to the public the written charge to the peer reviewers, the peer reviewers' names, the peer reviewers' report(s), and the agency's response to the peer reviewers' report(s). This Bulletin requires agencies to adopt or adapt the committee selection policies employed by the National Academy of Sciences (NAS)…

EPA's reliance on LNT has, in fact, already been peer reviewed as suggested by the OMB regulation. In 2006, the NAS Biological Effects of Ionizing Radiation VII committee released a report on the impact of low doses of radiation, *Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII Phase 2*. The committee was tasked with this charge by a number of U. S. Federal agencies, including EPA.

This report concluded that the LNT was the best model for protection against radiation: "A comprehensive review of the biology data led the committee to conclude that the risk would continue in a linear fashion at lower doses without a threshold and that the smallest dose has the potential to cause a small increase in risk to humans."[1] The public was included in the multi-year process, and provided written and verbal comments. At least two panelists were removed due to conflicts of interest that were pointed out by public comment.

Even so, members of the public feel the process fell short of fully accounting for health impacts, resulting in incomplete protection for members of the public, particularly females. One major component of this report was buried in the data rather than being highlighted by the committee. These data point to adult women suffering 50% more harm, and female children suffering nearly 10 times more harm, to radioactivity than the adult males on which U. S. protection standards are based (Figure 1). In fact, it was the public transparency of the NAS process, and the availability of the report and its references that allowed members of independent non-profit groups to bring this gender harm into the spotlight. U.S. agencies, however, have yet to protect against this harm.

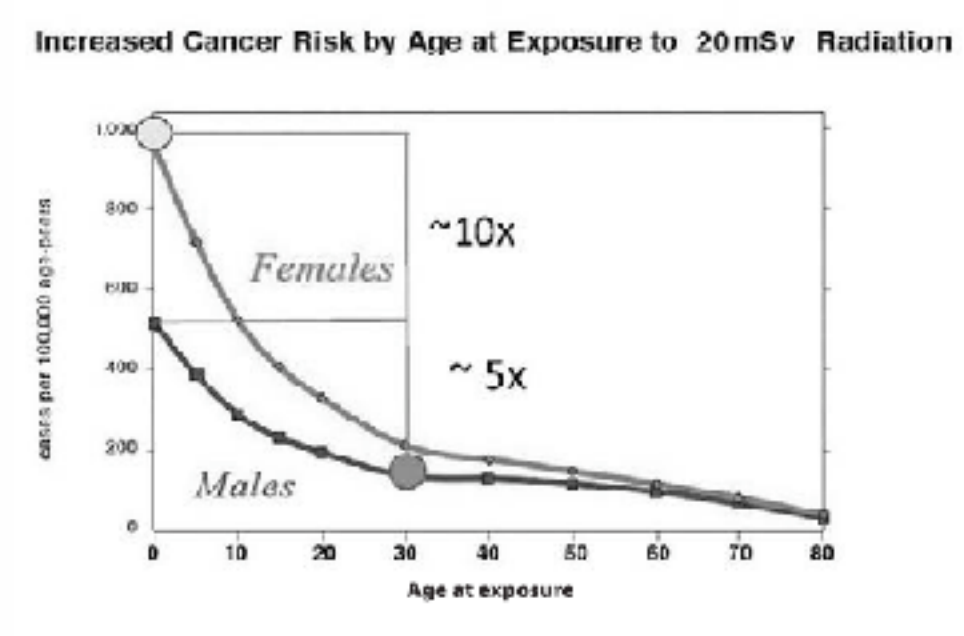

Figure 1.

In November 2015, members of the scientific community, NAS and several government agencies including EPA, hosted a conference to determine whether an update to BEIR VII was needed. Ultimately, a BEIR VIII has not yet been funded. BIER VII recommendations stand. Research since 2006 continues to reaffirm that very low doses of radiation are associated with cancer. (see partial list of studies below)

B) EPA has been very transparent about its review of linear, no threshold, and other cancer models and why it has chosen the LNT, as opposed to other models, for cancer risk from radiation. EPA's reliance on NAS conclusions, and its averaging of damage across genders and ages, leaves sensitive women and children less protected.

Proposed rule EPA–HQ–OA–2018–0259 states *"…there is growing empirical evidence of non-linearity in the concentration response function for specific pollutants and health effects. The use of default models, without consideration of alternatives or model uncertainty, can obscure the scientific justification for EPA actions." "To be even more transparent about these complex relationships, EPA should give appropriate consideration to high quality studies that explore [other models]"*

To be very clear, there is NOT *growing* empirical evidence of non-linearity for cancer causation in the direction of hormesis from radiation exposure. EPA has argued that whatever non-linearity exists has never been, and is not now, robust enough to form the basis of regulations.

Luckily, the EPA has already thoroughly considered alternatives to the linear and no threshold models for ionizing radiation carcinogenesis and the surrounding uncertainties. More than 50 years of peer reviewed, validated, and repeatable *in vivo*, not just laboratory studies, underlie the LNT model. To its credit, despite pressure from special

interests, EPA continues to recognize the model's pervasive scientific underpinnings, recently stating

> Of all the agents demonstrated to be carcinogenic, the evidence for LNT is particularly strong for ionizing radiation…[g]iven the continuing wide consensus on the use of LNT for regulatory purposes as well as the increasing scientific confirmation of the LNT model, it would be unacceptable to the EPA to ignore the recommendations of the NAS and other authoritative sources on this issue. The EPA cannot endorse basing radiation protection on poorly supported and highly speculative proposals for dose thresholds or doubtful notions concerning protective effects from low-level ionizing radiation.

EPA has relied on the NAS BEIR VII 2006 report that reviews already peer reviewed studies and synthesizes their conclusions to come up with its recommendations. (see above) The only reason to revisit the linear model is to make it more stringent because the current linear model does not fully protect women, children and pregnancy to the extent it does adult men, as indicated by data NAS examined. (see Figure 1)

And in as much as EPA uses a DDREF at low doses, this further risks the health of women and children and serves to make the cancer curve non-linear. For this reason, the DDREF should be abandoned, and the model should be reexamined to determine how steep the slope might be (in other words how much more damage is caused) for women and children at lower doses.

Nor, as EPA and the NAS have already made clear, is there any level of radiation that is low enough not to cause cancer: no threshold. Maintaining that there is no safe dose is especially important for protection of pregnancy, since a single radiation hit can destroy a cell that can develop into an entire organ like the brain. This pregnancy sensitivity implies that there is *also* no threshold for *non-cancer effects*, for which EPA does not regulate.

In fact, this one-hit destruction is an example of stochastic impacts, defined by the Health Physics Society as occurring "by chance and … occur[ring] without a threshold level of dose, whose probability is proportional to the dose and whose severity is independent of the dose." If the severity of the effect is not based on dose, even low doses can result in severe consequences, something even LNT would fail to protect against.

C) EPA should do more to reveal its reasons for not establishing protections against the non-cancer impacts of radiation. While the non-cancer impacts of other substances are recognized, particularly for pregnancy and females, why are they not for radiation exposure? What are the EPA's underlying assumptions for NO dose model?

While EPA's justification and use of the LNT model (and its shortcomings) are quite transparent already, EPA's non-cancer models seem non-existent. EPA must have a reason for why they fail to protect the public from non-cancer impacts of radiation. EPA claims the function of this rulemaking is to "increase transparency of the assumptions underlying dose response models." What are the underlying assumptions for NO dose response model?

Increasing transparency would help make clear to the public why EPA has (apparently) failed to protect against:

- radiation damage past the second generation since data show a trajectory of increasing vulnerability across generations.
- developmental changes and damage to all the sites and stem cells responsible for haematopoietic formation.
- damage during pregnancy to the maternal exchange system and embryo organ tissues.[2]
- damage to the placenta, a temporary but immensely important structure that performs organ-like functions.[3]
- effects on the estrogen pathway.[4]
- cumulative biological damage from continuing exposure to low doses.
- synergistic health effects of radiation with other chemical and biological stressors.

EPA has constructed a hazard index (HI) for non-cancer toxic chemical impacts, but radionuclides are not included. The EPA Superfund program includes radionuclides in the Hazard Ranking System (HRS) if present with other toxins, but does not have an HRS exclusive for radionuclides and radiation impacts nor does the HRS include radioactive releases from currently operating facilities. Since EPA currently lacks regulations protecting against non-cancer impacts of radionuclides, a hazard index for radionuclides should be created.

Kirchner has suggested a number of properties be included in a hazard index for radionuclides:

• large releases to environment;
• widely used in society (industrial/military/research/medical uses);
• rapid nuclide transport, solubility and cycling in biosphere;
• global distribution and resulting large collective doses;
• many environmental pathways to humans;
• rapid molecular exchange rates (that is, fast uptake by humans);
• large uptake fractions to blood after intake;
• organic binding in biota;
• long biological half-life in humans;
• long radiological half-life;
• long nuclide decay chains with radiotoxic daughters;
• high radiotoxicity (the dose coefficient of the nuclide, that is, the radiation dose imparted from the disintegration of one atom of the nuclide in question).

Many radionuclides exhibit a number of these characteristics including tritium, polonium-210, carbon-14, iodine-129 and krypton-85, to list a few.

D) EPA should account for the true costs of exposure to radioactivity and be honest about who is paying the price. Internal costs to the nuclear industry are not the only consideration. Allowing more radiation exposure externalizes costs to other industries, to individual members of the public, and to the population experiencing collective effects of radiation from the nuclear power and weapons industries.

Proposed rule EPA–HQ–OA–2018–0259 states *"The proposed regulation provides that when EPA develops regulations, including regulations for which the public is likely to bear the cost of compliance" that the underlying science is available "for independent validation". "EPA also requests comment on whether the disclosure requirements… should be expanded to cover economic…impact…"*

Like non-cancer health impacts of radioactivity, EPA does not account for the economic impact of radiation exposures. In reality, any benefit to the nuclear industry of non-compliance or non-protection is a cost borne by the exposed public, other industries, and current and future inhabitants (human and non human species and gene pools) of contaminated environments.

Radioactivity places a disproportionate health burden on women and early life stages. The cost of this extra burden is not known because it has not been examined. Costs of this health burden must be researched and added to the burden on society of using nuclear technologies; EPA must not only recognize that this burden weighs disproportionately on women and children, but must focus on protecting them instead of more resistant males or some hybrid model of men, women and children.

The Office of Policy, National Center for Environmental Economics (NCEE) should be instrumental in helping to cost out the impacts of radioactivity released from the nuclear industry using the following research and guidelines. These guidelines are in no way comprehensive:

1) Women are more susceptible to exposure to radioactivity—a susceptibility that is not compensated—and this disproportionate impact should be accounted for in the public health costs.

2) Vulnerable life stages like pregnancy and childhood need to not only have disproportionate effects costed out for initial exposure, but also effects that may occur in adulthood due to a phenomenon known as intrauterine programming (also see Beyond Nuclear's comments to EPA dated July 31, 2014).

3) The following clinical and subclinical diseases have been associated in scientific, peer-reviewed literature, with exposure to low and very low levels of radioactivity. This list is NOT exhaustive. (supporting studies in previously-referenced documents or listed/ linked below):

- impaired neural development and decreased IQ (decreased lifetime earnings capacity)
- childhood cancers, particularly leukemia and central nervous system cancers (also treatment of secondary cancers caused by treatment of primary cancer)
- low birth weight (and accompanying health impacts)
- mental retardation and other birth defects
- placental impacts and resultant health issues
- delayed growth
- CFIDS (chronic fatigue and related)
- Female subfertility (inability to get pregnant and accompanying health care costs)
- Potential estrogenic impacts

4) Estimating costs of these health impacts from exposure to radioactivity can be informed by work already underway for cost estimates of other toxic exposures, although this work might have to adjusted for impacts unique to radionuclide exposures. While determining the cost of cancers to society seems less challenging, for costs of

subclinical and brain development impacts, Dr. Leonardo Trasande, Department of Pediatrics, New York University (NYU) School of Medicine, has provided good research to start.

5) For each child that is treated for a cancer caused in whole or in part by exposure to radioactivity, their risk of getting a secondary cancer later in life is increased from the cancer treatment that initially saved their life. So the cost of treating the initial cancer must be factored in, but so too must the cost of treating the secondary cancer.

E) EPA should use models that incorporate uncertainty and stochastic (random) impacts of radiation, including the probability that random impacts will increase with an increasingly radiologically contaminated environment.

Proposed rule EPA–HQ–OA–2018–0259 states *"EPA should also incorporate the concept of model uncertainty when needed as a default to optimize low dose risk estimation based on major competing models, including linear, threshold, and U-shaped, Jshaped, and bell-shaped models."*

One major recognized category of uncertainty in radiation exposure is the stochastic impact. The stochastic effect is random and therefore, difficult to predict in the context of radiation protection. According to the Health Physics Society, stochastic effects may occur "without a threshold level of dose, whose probability is proportional to the dose and whose severity is independent of the dose."

While the probability of an effect increases with dose, for a pregnancy where the fetus/ embryo is developing from single cells, one hit from radiation could damage or destroy the cell(s) meant to become the brain or spinal cord. Repeated hits, although considered very low doses to the whole body, could damage the bone marrow cells meant to make healthy blood. And as we increase our exposure, through contaminated food and water, air and soil, we increase the probability that we will be exposed, thus increasing the stochastic damage.

Therefore, any model that incorporates uncertainty, must incorporate our ever-increasing probability of exposure, in addition to the non-threshold nature of stochastic cancer and non-cancer impacts.

F) EPA needs to rely on radiation health studies that are independent and scientifically substantiated; and shun those that are not applicable to radiation protection, suffer from well-known and documented pseudo-scientific assumptions, or harbor conflicts of interest. Even report conclusions that are generally accepted, such as those found in NAS BEIR VII, can still reference, and be influenced by, these inappropriate studies. This is perhaps one reason why women, children and pregnancy are not afforded the protection they require.

Proposed rule EPA–HQ–OA–2018–0259 states *"EPA also solicits comments on whether and how the proposed rule should apply to dose response data and models underlying pivotal regulatory science if those data and models were developed prior to the effective date."*

Improper hypothesis formation and research design have been baked into, and continue to plague, studies on radiation's impact on health. This improper methodology has, unfortunately, evolved to be the only accepted way of examining health impacts. This rigid study design presupposes a conclusion of no impact before any health data are actually examined, a huge scientific faux pas. The design does this by relying on error-

ridden dose reconstruction, which almost always concludes that environmental exposure was too low to cause health impacts. Therefore, even though this research often finds increase in disease, associating it with radiation exposure becomes impossible, not because radiation isn't the cause, but because it was falsely vindicated from the beginning. See Section 1 after conclusion.

Further, there are a number of studies regarded as reliable by international radiation committees, such as ICRP or NCRP, which find no health impact, or claim no *discernable* impact. Many of these studies were actually funded or influenced by industry and often used in court to counter claims of health damage from radiation. Worse still, these studies also received funding from Federal agencies, giving them the appearance of independence. Studies like these skew the overall balance of radiation damage assessment when committees, such as those from NAS, try to reivew the literature examining radiation's impact on health. Any conclusion relying on them is, therefore, skewed. See Section 2 after conclusion.

Conclusion

EPA claims that the purpose of Proposed rule EPA–HQ–OA–2018–0259 would be transparency in the science undergirding EPA's regulations. While transparency should be a good first step in more fully protecting the public from radiation exposure, the proposed rule mentions many damage models that are less protective than the current LNT. Use of *these* models would be unacceptable because EPA's current use of LNT for radiation cancer risk already leaves women, children without necessary protections.

A better use of transparency in process and science would be for the EPA to clearly state what its assumptions are for not protecting the public from non cancer and/or stochastic impacts of radiation exposure, particularly to pregnancy; and why EPA has failed to account for the costs of not complying with current exposure regulations for cancer. In the case of non-cancer impacts, why has the EPA not calculated the public health costs of having no regulations at all?

Section 1.

There are many primary, peer reviewed studies EPA may rely on that examine human and non-human health impacts that do not suffer from improper radiation dose assumptions or industry influence. Some of these studies are included in this partial list of studies, presented in alphabetical order by author's last name. This list includes cell as well as epidemiological studies.

We also include here basic studies on pregnancy and child development that, although not radiation specific, are reference material applicable to the type of damage radiation causes.

Almond, et al. Chernobyl's subclinical legacy: Prenatal exposure to radioactive fallout and school outcomes in Sweden. November 2008.

Barber RC et al.The offspring of irradiated parents, are they stable? Mutat Res. 2006 Jun 25;598(1-2):50-60. Epub 2006 Feb 28. Review.

Baverstock, K. Some important questions connected with non-targeted effects. Mutation Research 687 (2010) 84–88.

USEPA. Children's Health. Early Life Stages. http://www2.epa.gov/children/early-life-stages (accessed 3/22/15)

USEPA. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. http://www2.epa.gov/sites/production/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf

Fairlie, I. Comment on BASIS FOR DOSIMETRIC QUANTITIES USED IN RADIOLOGICAL PROTECTION. ICRP Committee 2. April 2005.

Fairlie. A 100 mSv threshold for radiation effects? November 27, 2012

Fairlie, I. US Nuclear Regulatory Commission (NRC): Consultation. August 2015.

Fairlie, Ian, A hypothesis to explain childhood cancers near nuclear power plants. J Environ Radioactivity 133 (2014): 10-17.

Gardner, MJ. Results of case-control study of leukaemia and lymphoma among young people near Sellafield nuclear plant in West Cumbria. BMJ 1990;300:423

Garnier-Laplace et al. Are radiosensitivity data derived from natural field conditions consistent with data from controlled exposures? A case study of Chernobyl wildlife chronically exposed to low dose rates. Journal of Environmental Radioactivity 121 (2013) 12-21.

Goncharova, et al. Results of Long-term Genetic Monitoring of Animal Populations Chronically Irradiated in the Radiocontaminated Areas. 1998.

Gude, et.al. Growth and function of the normal human placenta. Thromb Res. 2004;114(5-6):397-407

Kadhim M. A., et al. “Transmission of chromosomal instability after plutonium alpha particle irradiation.” *Nature*. 355:738 (1992). (Eric Wright is co-author)

Karam, et al. Changes in terrestrial natural radiation levels over the history of life. Radioactivity in the Environment. Volume 7. The Natural Radiation Environment VII: VIIth Int. Symp. On the NRE 2005. Pages 107–117.

Kendall, et al. A record-based case-control study of natural background radiation and the incidence of childhood leukaemia and other cancers in Great Britain during 1980-2006. Leukemia. 2013 Jan;27(1):3-9.

Kitchin, A critique of the use of hormesis in risk assessment. *Human & Experimental Toxicology*. 2005. 24. 249- 253.

Korblein. Strontium fallout from Chernobyl and perinatal mortality in Ukraine and Belarus. Radiats Biol Radioecol. Mar-Apr;43(2):197-202. 2003.

Korblein. Perinatal Mortality in West Germany Following Atmospheric Nuclear Weapons Tests. Archives of Environmental Health. Nov. 2004.

Little, M. et al. Leukaemia and myeloid malignancy among people exposed to low doses (<100 mSv) of ionizing radiation during childhood: a pooled analysis of nine historical cohort studies. *The Lancet: Haematology*. Volume 5, ISSUE 8, Pe346-e358, August 01, 2018.

Loganovsky et al. Intelligence and Brain Damage in Children Acutely Irradiated in Utero As a Result of the Chernobyl Accident.

Lorimore S. A., et. al. “Chromosomal Instability in the descendants of unirradiated surviving cells after alpha particle irradiation.” *Proc. Natl. Acad. Sci. USA*. 95: 5730-5733 (1998).

Møller et al. The effects of natural variation in background radioactivity on humans, animals and other organisms. Biol Rev Camb Philos Soc. 2013 Feb. 88(1):226-54.

Møller. Strong effects of ionizing radiation from Chernobyl on mutation rates. Scientific Report. Nature. 10 February 2015.

Morgenstern, H., et al. “Epidemiologic Study to Determine Possible Adverse Effects to Rocketdyne/Atomic International Workers from Exposure to Ionizing Radiation” Report by the UCLA School of Public Health. September, 1997.

Noshchenko, et al. Radiation-induced leukemia among children aged 0–5 years at the time of the Chernobyl accident. International Journal of Cancer. 127, 412–426 (2010).

Schmitz-Feuerhake. The 100 Millisievert Threshold Lie: Accepted Knowledge about Radiation Effects after Chronical Low-Dose Exposure and Remaining Issues. Citizens and Scientist International Conference on Radiation Protection, Fukushima, June 2012.

Salomaa, et al. Editorial—Non-DNA targeted effects. Mutation Research 687 (2010) 1–2

Spycher, et al. Background Ionizing Radiation and the Risk of Childhood Cancer: A Census-Based Nationwide Cohort Study. Environ Health Perspect. 2015 Feb 23. [Epub ahead of print]

Stewart, A.M., et al. ”Radiation Exposures of Hanford Workers Dying from Cancer and Other Causes.” *Health Physics*. Nov (1977).

Stewart, A.M, et al. “Delayed Effects of A-bomb radiation: a review of recent mortality rates and risk estimates for five-year survivors.” *Journal Epidemiology and Community Health*. 36(2):80-6 (1982).

Sutcliffe, J. Review: Radiation a new paradigm. . .Societal impacts. Mutation Research 687 (2010) 67–72.

Svendsen E. et al. 137Cesium exposure and spirometry measures in Ukrainian children affected by the Chernobyl nuclear incident. Environ Health Perspect. 2010 May;118(5): 720-5

Svendsen, E. et al. "Reduced Lung Function in Children Associated with Cesium 137 Body Burden". Annals of the American Thoracic Society. Vol. 12 No. 7 (2015) pp. 1050-1057.

Wertelecki , W. Blastopathies and microcephaly in a Chornobyl impacted region of Ukraine. Congenital Anomalies 2014; 54, 125–149.

Wing S., et al. “Mortality Among Workers at Oak Ridge National Laboratory.” *JAMA*, 26 (11):1397 (1991)

Wing, S. A reevaluation of cancer incidence near the Three Mile Island nuclear plant: the collision of evidence and assumptions. Environ Health Perspect. 1997 Jan; 105(1): 52–57.

Wright, EG. Manifestations and mechanisms of non-targeted effects of ionizing radiation. Mutation Research 687 (2010) 28–33.

For a good review of radiation studies since BEIRVII was issued, see this video: https://www.youtube.com/watch?v=XTijIRsxTSE&feature=youtu.be

Section 2. The following categories of studies, should be disallowed for reasons of inapplicability or lack of independence.

Proclamations and studies from the Health Physics Society, or the journal they publish, *Health Physics*, should not be used to set radiation exposure standards. While it is true that the HPS is a non-profit, it is anything but independent. HPS is a 501c6 non-profit and therefore operates for the benefit of the businesses they represent, according to the IRS code definition. For too long HPS has been relied upon almost to the exclusion of other more independent experts.

No hormesis: a model based on hormesis would allow that a little radiation exposure is beneficial. But much of the research on hormesis is based on bad science or assumptions and is often not applicable to real circumstances. Beyond Nuclear provided comments to the NRC on the LNT and the implications of replacing it with a hormesis-based model, as did Dr. Ian Fairlie and many others.

The studies below received industry funding or were influenced by industry in other ways, and were often used in court cases against plaintiffs who were claiming exposure to industry radioactivity. This is not a complete list but meant to be representative of the poor methods and industry conflicts that permeate and compromise study of radiation's impact on health. It is not in any order.

Hatch, M. Cancer near the Three Mile Island nuclear plant: radiation emissions.

Hatch, M. Cancer rates after the Three Mile Island nuclear accident and proximity of residence to the plant.

Talbott, Cancer incidence among residents of the Three Mile Island accident area: 1982-1995.

Talbott, Long-term follow-up of the residents of the Three Mile Island accident area: 1979-1998.

Talbott, Mortality among the residents of the Three Mile Island accident area: 1979-1992.

Intra-individual variation in G2 chromosomal radiosensitivity. | Curwen GB, Cadwell KK, Tawn EJ, Winther JF, Boice JD Jr. | 2012

A study of DNA damage recognition and repair gene polymorphisms in relation to cancer predisposition and G2 chromosomal radiosensitivity. | Curwen GB, Murphy S, Tawn EJ, Winther JF, Boice JD Jr. | 2011 | done at Westlakes w/ NIH funding

Germline minisatellite mutations in survivors of childhood and young adult cancer treated with radiation. | Tawn EJ, Rees GS, Leith C, Winther JF, Curwen GB, Stovall M, Olsen JH, Rechnitzer C, Schroeder H, Guldberg P, Boice JD. | 2011 | done at Westlakes, NIH funding

G2 checkpoint control and G2 chromosomal radiosensitivity in cancer survivors and their families. | Cadwell KK, Curwen GB, Tawn EJ, Winther JF, Boice JD Jr. | 2011 | done at Westlakes, NIH funding

Updated mortality analysis of radiation workers at Rocketdyne (Atomics International), 1948-2008. | John D Boice; Sarah S Cohen; Michael T Mumma; Elizabeth Dupree Ellis;
Keith F Eckerman; Richard W Leggett; Bruce B Boecker; A Bertrand Brill; Brian E Henderson | 2011

The heritability of G2 chromosomal radiosensitivity and its association with cancer in Danish cancer survivors and their offspring. | Curwen GB, Cadwell KK, Winther JF, Tawn EJ, Rees GS, Olsen JH, Rechnitzer C, Schroeder H, Guldberg P, Cordell HJ, Boice JD Jr. | 2010

Stillbirth and neonatal death in relation to radiation exposure before conception: a retrospective cohort study. | Signorello LB, Mulvihill JJ, Green DM, Munro HM, Stovall M, Weathers RE, Mertens AC, Whitton JA, Robison LL, Boice JD Jr. | 2010

Influence of polymorphisms at loci encoding DNA repair proteins on cancer susceptibility and G2 chromosomal radiosensitivity. Wilding CS, Curwen GB, Tawn EJ, Sheng X, Winther JF, Chakraborty R, Boice JD Jr. 2007

A pilot study examining germline minisatellite mutations in the offspring of Danish childhood and adolescent cancer survivors treated with radiotherapy. Rees GS, Trikic MZ, Winther JF, Tawn EJ, Stovall M, Olsen JH, Rechnitzer C, Schrøder H, Guldberg P, Boice JD Jr.2006

G(2) chromosomal radiosensitivity in Danish survivors of childhood and adolescent cancer and their offspring. Curwen GB, Winther JF, Tawn EJ, Smart V, Whitehouse CA, Rees GS, Olsen
JH, Guldberg P, Rechnitzer C, Schrøder H, Bryant PE, Sheng X, Lee HS, Chakraborty R, Boice JD. 2005

Chromosome analysis in childhood cancer survivors and their offspring--no evidence for radiotherapy-induced persistent genomic instability.Tawn EJ, Whitehouse CA, Winther JF, Curwen GB, Rees GS, Stovall M, Olsen JH, Guldberg P, Rechnitzer C, Schrøder H, Boice JD Jr. 2005

Congenital anomalies in the children of cancer survivors: a report from the childhood cancer survivor study. Signorello LB, Mulvihill JJ, Green DM, Munro HM, Stovall M, Weathers RE, Mertens AC, Whitton JA, Robison LL, Boice JD Jr. 2012

Cancer incidence and mortality in populations living near uranium milling and mining operations in grants, New Mexico, 1950-2004. Boice JD Jr, Mumma MT, Blot WJ. 2010

Low-dose-rate epidemiology of high background radiation areas. Boice JD Jr, Hendry JH, Nakamura N, Niwa O, Nakamura S, Yoshida K. 2010

County mortality and cancer incidence in relation to living near two former nuclear materials processing facilities in Pennsylvania--an update. Boice JD Jr, Bigbee WL, Mumma MT, Tarone RE, Blot WJ. 2009

Cancer incidence in municipalities near two former nuclear materials processing facilities in Pennsylvania--an update. Boice JD Jr, Bigbee WL, Mumma MT, Heath CW Jr, Blot WJ. 2009

A cohort study of uranium millers and miners of Grants, New Mexico, 1979-2005. Boice JD Jr, Cohen SS, Mumma MT, Chadda B, Blot WJ. 2008

Mortality among residents of Uravan, Colorado who lived near a uranium mill, 1936-84. Boice JD Jr, Cohen SS, Mumma MT, Chadda B, Blot WJ. 2007

Cancer and noncancer mortality in populations living near uranium and vanadium mining and milling operations in Montrose County, Colorado, 1950-2000. Boice JD Jr, Mumma MT, Blot WJ. 2007

Female survivors of childhood cancer: preterm birth and low birth weight among their children. Signorello LB, Cohen SS, Bosetti C, Stovall M, Kasper CE, Weathers RE, Whitton JA, Green DM, Donaldson SS, Mertens AC, Robison LL, Boice JD Jr. 2006

Mortality among radiation workers at Rocketdyne (Atomics International), 1948-1999. Boice JD, Cohen SS, Mumma MT, Dupree Ellis E, Eckerman KF, Leggett RW, Boecker BB, Brill AB, Henderson BE. 2006

Cancer mortality among populations residing in counties near the Hanford site, 1950-2000. Boice JD Jr, Mumma MT, Blot WJ. 2006

A comprehensive dose reconstruction methodology for former rocketdyne/atomics international radiation workers. Boice JD Jr, Leggett RW, Ellis ED, Wallace PW, Mumma M, Cohen SS, Brill AB, Chadda B, Boecker BB, Yoder RC, Eckerman KF. 2006

Childhood cancer mortality in relation to the St Lucie nuclear power station. 2005: John D Boice; Michael T Mumma; William J Blot; Clark W Heath

Cancer mortality in counties near two former nuclear materials processing facilities in Pennsylvania, 1950-1995. Boice JD Jr, Bigbee WL, Mumma MT, Blot WJ. 2003

Cancer incidence in municipalities near two former nuclear materials processing facilities in Pennsylvania. Boice JD Jr, Bigbee WL, Mumma MT, Blot WJ. 2003

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[1] Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII Phase 2. https://books.google.com/books?id=-bV9OrS9vZEC&pg=PA10&lpg=PA10&dq=that+it+is+unlikely+that+a+threshold+exists+for+the+induction+of+cancers&source=bl&ots=Mr46YtbfQU&sig=vRxeuGiKhBYS2WNYcxcFQVVoVdU&hl=en&sa=X&ved=0ahUKEwiW5LzThIXZAhXkc98KHZ6ZBNIQ6AEIKTAB#v=onepage&q=that%20it%20is%20unlikely%20that%20a%20threshold%20exists%20for%20the%20induction%20of%20cancers&f=false

[2] Embryo organ tissues form the heart, spinal cord and brain, major blood vessels and the beginning of bones and muscles.

[3] It supplies oxygen, removes metabolic products and provides a limited barrier against some toxins and drugs; it is active endocrinologically to support the ongoing pregnancy. Improper placental formation or function can cause a high or low birth weight, which in turn seems to be connected to disease later in adult life.

[4] In 2011, a medical hypothesis was published highlighting this interaction: “The impact of estrogen and estrogen receptors on the response of living organisms, including humans, after exposure to ionizing radiation should be included in future in radiation safety regulations...”