**INSTITUTE FOR ENERGY AND
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*Democratizing science to protect
health and the environment*

MEMORANDUM

To: Committee on Developing a Long-Term Strategy for Low-Dose Radiation Research in the United States, National Academies of Sciences, Engineering, and Medicine (hereafter "Committee")
From: Arjun Makhijani, President, Institute for Energy and Environmental Research (IEER) [1]
Subject: Recommendations for the study on low-level radiation research
Date: January 7, 2022 (footnote 37 corrected on January 10, 2022)
cc: Ourania Kosti, Staff Officer

I focus this note on some of the key areas of study that need your attention, before making briefer comments on the linear-no-threshold hypothesis, radiation protection standards, and the review process for your report.

- Various radiation-induced effects can arise in the embryo and fetus. These include effects that can arise from damage to a single cell or damage to multiple cells, effects that arise directly from the irradiated cells and effects that arise in bystander cells, and effects that arise in the earliest phase of pregnancy or during organogenesis, which occurs for individual tissues and organs at various times, especially during the first two trimesters of pregnancy.

- Although these effects can arise following external irradiation of the pregnant woman (and this is the context in which they have been observed and studied to the extent that that has been feasible), assessing their likelihood of occurrence following intakes of radionuclides is a problem in its own right. It is much more complicated because consideration must be given to the degree of cross- placental transfer that occurs, and inhomogeneities in deposition and retention in the bodies of both the mother and fetus. The effectiveness of internal radiation, including high-LET radiation, following maternal-fetal radionuclide transfer in inducing teratogenic damage needs to be evaluated for specific endpoints.

- Tritium requires specific consideration because of its high bioavailability, mobility within the body, its low energy beta particles that deposit all their energy within a single cell, all pointing to the need for consideration of the appropriate relative biological effectiveness values to apply in evaluating its radiological significance. The import of these issues is heightened by its wide prevalence as a pollutant from nuclear power and nuclear weapons-related activities.

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[1] This memorandum was reviewed by several people including Dr. M.V. Ramana, Dr. Tilman Ruff, Cathie Sullivan, and Dr. Mike Thorne, who was Scientific Secretary of the International Commission on Radiological Protection (ICRP) when ICRP's Publication 49, was adopted. The reviews have helped me improve this memorandum. But as the author, I am solely responsible for the contents of this memorandum, including any errors and omissions that may remain as well as its recommendations.

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- The large number of early failed pregnancies – before the implantation of the blastocyst or in the days-to-weeks that immediately follow – makes it difficult to detect the impact of specific environmental factors, including ionizing radiation, especially since the event often occurs before a woman realizes she is pregnant. Nonetheless, it is important to recognize explicitly the potential for this impact and to attempt to devise ways to measure and minimize it.

I. Areas of study

a. Background to and focus of this memorandum

Major gaps in radiation research relate to impacts on the embryo and the fetus during the period from fertilization through the sensitive periods of organogenesis, to the second trimester and beyond that to the time of delivery. Critical adverse outcomes to be studied include failure of blastocyst formation, failure of blastocyst implantation, and a variety of possible teratogenic impacts, ranging from disability to death. In this memorandum, I refer to the first two of these outcomes as "early failed pregnancies." The relative neglect of these issues is illustrated by the absence of regulations aimed specifically at protecting female members of the public of child-bearing age, including those who are pregnant, and of the embryo and fetus during pregnancy.[2]

In September 1999, the Institute for Energy and Environmental Research sent a letter to the BEIR VII committee on radiation risks and health impacts; this letter was initiated by our then-Outreach Coordinator, Lisa Ledwidge, and myself. It was signed by more than 100 scientists, activists, physicians, and other concerned people throughout the United States and in a number of other countries, many with impeccable academic credentials. Among other things, it urged the committee to study the impacts of "[r]adionuclides that cross the placenta" and "effects of radiation on female fetuses."[3] The signatories also specifically asked the BEIR VII committee to consider malformations and miscarriages due to internal ionizing radiation exposure. Although the letter was submitted several years before the publication of the BEIR VII Phase 2 report, the only time the word "placenta" appears in the report was when it noted that we had raised the issue; it considered a part of the issue briefly and then made the following recommendation:

> The committee recommends that this issue be addressed as part of a larger review of maternal exposures in humans that may affect the fetus.[4]

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[2] Women in radiation-regulated workplaces who declare their pregnancies are somewhat protected in that fetal exposure in such cases in most industrialized countries is limited to 1 millisievert (100 millirem); the standard in the United States is more lax 5 millisievert (500 millirem). Even the lower limit is inadequate; see Section III.b below.

[3] Lisa Ledwidge and Arjun Makhijani, letter to Richard Monson, Chair, BEIR VII committee, September 3, 1999 with other signatories. IEER also wrote four follow up letters to the BEIR VII committee, including to inquire the progress of the research into the issues raised in the September 3, 1999 letter. The September 1999 letter and related materials can be downloaded at https://ieer.org/resource/energy-issues/beir-vii-report-raises-major-issues-for-radiation-protection/

[4] Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation, Board on Radiation Effects Research. *Health Risks from Exposure to Low Levels of Ionizing Radiation: BEIR VII – Phase 2*. National Research Council of the National Academies. Washington, DC: National Academies Press, 2006., Appendix C, p. 330; hereafter referred to as BEIR VII 2006.

The BEIR VII report has many strong points; a careful consideration of the impact of maternal exposures, especially internal exposures, on the embryo and the fetus is not one of them. The failure is all the more stark because there was an extensive literature on the topic at that time; the BEIR committee could have carried out a detailed scientific assessment. I cite the following three reports as examples of official literature on this topic:

1. International Commission on Radiological Protection: *Doses to the Embryo and Fetus from Intakes of Radionuclides by the Mother,* ICRP 88, Corrected Version 2002 at https://www.icrp.org/publication.asp?id=ICRP%20Publication%2088 (hereafter ICRP 88, 2002).
2. U.K. National Radiological Protection Board. *Doses to the embryo/fetus and neonate from intakes of radionuclides by the mother Part 1: Doses received in utero and from activity present at birth*, 2001 at https://www.hse.gov.uk/research/crr_pdf/2001/crr01397.pdf (hereafter NRPB 2001).
3. A report by the Pacific Northwest National Laboratory prepared for the Nuclear Regulatory Commission (1996), *Contribution of Maternal Radionuclide Burdens to Prenatal Radiation Doses*, 1996, at https://inis.iaea.org/collection/NCLCollectionStore/_Public/27/065/27065464.pdf (hereafter Pacific Northwest National Laboratory 1996).

More studies have been carried out since then. But the job of seriously addressing the impacts of internal radiation exposures to pregnant women, and coming up with recommendations to protect them and the children they carry has yet to be done with the seriousness it deserves. I expect that you will not again give the issue short shift but rather give it your fullest consideration. Among other things, such consideration is necessary to remedy the failure of the BEIR VII committee on a matter about which society has expressed so much concern. You have also directly heard about this issue through concerns expressed by others who made presentations to you on October 28, 2021.

b. Teratogenic effects during pregnancy

Teratogenic impacts involve scientific issues that need deeper exploration as well as immediate actions for public health protection. These impacts were briefly addressed in the 1988 BEIR IV report of the National Academies in a section in Chapter 8 entitled "Fetal Effects, Teratogenesis, and Neonatal Effects of In Utero Exposure." I use that BEIR IV analysis as my point of departure. Let me first quote some relevant passages from BEIR IV:

In regard to "preimplantation loss" of the embryo, BEIR IV states:

> Only animal data are available, but they explain the observation that single doses of less than *10-rad of low-LET radiation* produce no detectable effects. It is speculated that the principal reason for the absence of human data showing preimplantation teratogenesis is that the losses occur before the mothers know that they are pregnant and therefore go unnoticed. Theoretical considerations strongly suggest that preimplantation loss must be a *nonstochastic effect*, with a definite threshold. Exceeding this threshold requires that some minimal number of cells be killed. [5]

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[5] Committee on the Health Risks of Ionizing Radiations, Board on Radiation Effects Research. *Health Risks from Exposure to Radon and Other Alpha Emitters*. National Research Council of the National Academies. Washington, DC: National Academies Press, 1988, pp. 381-382, italics added. Hereafter referred to as BEIR IV 1988.

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Regarding the teratogenic effects during organogenesis, BEIR IV expresses a similar conclusion:

> During the major organogenesis state, the *embryo appears to be sensitive to all the known teratogenic effects of radiation*….Windows of one to a few days are commonly observed during which a given developmental abnormality can be induced during the major organogenesis stage. Thresholds are expected theoretically and have been observed; single doses below about *10 rad of low-LET radiation* appear ineffective.[6]

Teratogenic impacts on the brain are an exception to the hypothesized threshold, according to BEIR IV:

> *One prominent effect on which there are human epidemiological data is characterized by a nonthreshold dose-effect curve.* Studies of children exposed in utero at Hiroshima and Nagasaki have shown that the incidence of microcephaly, often connected with mental retardation, was much higher among those exposed to doses greater than about 10 rad during weeks 10-19 of fetal development. In a recent study of 1,599 children exposed in utero at Hiroshima and Nagasaki, mental retardation was found to be apparently linearly related to dose during the sensitive period.[7]

BEIR IV's conclusions about these topics were as follows:

> Very recently, a task group of Committee 1 of the International Commission on Radiological Protection completed a study of the *effects of radiation on the development of the brain of the embryo and fetus*. The task group reported that, within the period of maximum vulnerability, *the data it reviewed appeared to be consistent with a linear non-threshold response*. This information was published after this report was prepared and therefore has not been examined by *this committee, which reached no conclusions concerning the effects of alpha dose on the developing brain*.

> ….For organs other than the brain, the concept of RBE can be used to translate estimates of the effects of acute low-LET exposures to the case of alpha particles. Virtually all other teratogenic effects of radiation are believed to be due to multiple cell killing, and *one can simply translate the accepted 10-rad threshold for single dose low-LET radiation by applying the RBE commonly observed for alpha particles in in vitro cell-killing experiments*. RBEs for cell killing by alpha particles are around 10, but could be higher for the very low dose rates expected from internal emitters. Sensitive time windows have been observed, particularly during the stage of major organogenesis, and much (if not all) of the total dose accumulates on either side of this window, which is apparently only a few days long even in man. Thus, most of the total dose accumulated during the 280-day gestation period would not be effective. It seems reasonable to conclude that except for brain tissues, high-LET alpha particle doses below about 1 rad will have no teratogenic effects.[8]

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[6] BEIR IV 1988, p. 383, italics added.
[7] BEIR IV 1988, p. 384, italics added.
[8] BEIR IV 1988, p. 392, italics added. The last quoted sentence says "1 rad" for the high-LET dose; it should be 1 rem, since an RBE has been applied to the low-LET dose. The referenced report appears to be International Commission on Radiological Protection, *Developmental Effects of the Irradiation on the Brain of the Embryo and*

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BEIR IV refers to ICRP 49, which was published in July 1986; and the first printing of BEIR IV was one-and-a-half years later, in January 1988; apparently the BEIR IV preparation and publication schedule did not allow detailed consideration of the findings of ICRP 49. Be that as it may, the ICRP 49 findings are stark; here is a key passage about specific teratogenic impacts from ICRP 49:

> First, 30 of the 1 599 pregnancies included in the revised clinical sample terminated in a child with severe mental retardation and, second, 18 of these, or 60%, had disproportionately small heads, that is, a head with a circumference more than two standard deviations below the mean observed among the 1,599. Of those pregnancies that terminated in a mentally retarded child…no fewer than 19 (and 17 of the 21 who received exposures of 0.01 Gy or more) were exposed in the 8th through the 15th week after fertilization. *This is many times the expectation based on the assumption of no effect of fetal age at exposure*. In this context, to reiterate, severe mental retardation implies an individual unable to form simple sentences, to solve simple problems in arithmetic, to care for himself or herself, or is (was) unmanageable or institutionalized.[9]

The term "severe mental retardation" is used in this memorandum in the clinical sense provided by ICRP 49, as quoted above. ICRP 49 also provides a dose-response estimate of 0.4 per gray – ranging from "one case per hundred individuals *exposed to less than 0.01 Gy* [1 rad] to approximately 40 cases per hundred at an exposure of 1 Gy [100 rad]."[10] Finally and importantly, while a substantial majority of the impacts observed occurred between the 8th and 15th weeks, an increase in "severe mental retardation" was also noted for exposures well beyond that time – up to 25 weeks after fertilization.[11]

The facts and analysis in ICRP 49 make very clear that teratogenic impacts should have become a principal element of the scientific and public health discussion of the impacts of low-levels of exposure decades ago – dating back at least to ICRP 49. It is imperative that you point that out and set in motion an urgent process for broad and deep scientific work on the impact of ionizing radiation on pregnant women, the embryo, and the fetus. Certain conclusions are clear from BEIR IV and ICRP 49:

1. Radiation exposure can result in early failed pregnancies; they are hard to detect because women may not realize that they are pregnant from the very fact of an early failure.
2. Radiation exposure can produce a variety of teratogenic effects, especially during the most sensitive period of organogenesis.

Fetus, ICRP 49, 1986 at https://www.icrp.org/publication.asp?id=ICRP%20Publication%2049 – hereafter ICRP 49, 1986.

[9] ICRP 49, 1986, p. 20; italics added

[10] ICRP 49 1986, p. 31, italics added. The evidence of severe mental retardation at less than one rad and a linear dose-response curve leading to a conclusion that there is no threshold is consistent with other evidence for lack of thresholds. This includes the evidence for substantial increases in childhood leukemia and brain cancer at 1 rad presented to you on October 27, 2021 by Dr. Rebecca Smith-Bindman, Medical Perspectives, October 27, 2021, slide 11; hereafter Smith-Bindman 2021.

[11] ICRP 49, 1986, Table 1, p. 21. ICRP 49 found that "simplest statistical model consistent with the data appears to be a linear one without threshold." p. 31. The ICRP analysis also reported on a decline in IQ that was "consistent with the interpretation that there is a dose-related shift in IQ and that this could explain the increase in clinically classified cases of severe retardation. They do not exclude the possibility of two separate effects…. The statistical uncertainties in the data, and the known problems of obtaining a high consistency in intelligence testing, prevent quantitative statistical analysis of these data from refining these qualitative conclusions."

3. There is no threshold for "severe mental retardation" caused by radiation exposure between the 8th and 25th week of pregnancy. A linear no-threshold for impact means that exposures during this period would be expected to produce the severe disabilities, with the number being proportional to the population dose to pregnant women in the relevant period of pregnancy.

BEIR IV also makes statements that are tentative, narrow, and even questionable that need revisiting.

1. Thresholds and the time window(s): According to BEIR IV "[w]indows of one to a few days are commonly observed during which a given developmental abnormality can be induced during the major organogenesis stage."[12] This idea is repeated in the concluding section about teratogenesis in BEIR IV, which states that, except for the brain, the sensitive period of organogenesis is "apparently only a few days long even in man."

This is far too restrictive a view. The Teratology Society makes clear that organogenesis starts in the fourth week and extends throughout pregnancy, with different organ development risks being prominent in different periods. It summarizes possible adverse outcomes during pregnancy as follows:

> Teratogenic exposure during any period or phase of development can have dire consequences….In general, disruption of the earliest developmental stages (gametogenesis; fertilization, cleavage, and blastulation) results in the loss of the conceptus (that is, a miscarriage, often before the woman realizes she is pregnant). Disruption somewhat later during primary morphogenesis and organogenesis often results in major structural anomalies (a "birth defect" for example, a neural tube defect, such as spina bifida; a ventral body wall defect, such as gastroschisis; a heart defect, such as the formation of a single outflow tract; a limb anomaly, such as phocomelia; or a facial cleft, such as cleft lip or palate). Disruption during the late embryonic and fetal period generally results in abnormal organ differentiation, growth, and function (for example, cognitive impairment, hearing loss, neonatal hypoglycemia, lung immaturity). Thus, the timing of a particular teratogenic exposure can result in drastically different outcomes.[13]

Consideration of a much longer period than indicated by BEIR IV's conclusions is warranted since serious teratogenic impacts can occur well after the most sensitive primary morphogenic period of eight or nine weeks.

BEIR IV's statement of an "accepted" threshold of 10 rad (0.1 Gy) is based on laboratory experiments on animals. However, research on wildlife compared to laboratory experiments conducted since then puts this conclusion into considerable doubt. Specifically, a Chernobyl study compared laboratory experiments on a single species with the impact of radiation on wildlife seen in the field over wide range of dose rates. It concluded that wildlife is eight times more sensitive to radiation than indicated by laboratory experiments as measured by the median value of hazardous dose rate "suggesting that

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[12] BEIR IV 1988, p. 383

[13] **Steven B. Bleyl and Gary C. Schoenwolf, "**What Is the Timeline of Important Events During Pregnancy that May Be Disrupted by a Teratogenic Exposure?" in *Teratology Primer, 3rd Edition*, Teratology Society, at https://birthdefectsresearch.org/primer/Teratogenic-Exposure.asp

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organisms in their natural environmental [sic] were more sensitive to radiation."[14] This single study is suggestive rather than definitive. Still, the magnitude of the field and laboratory difference provides ample basis for caution in assuming that a threshold for most teratogenic effects, if it exists, is as high as 0.1 Gy (10 rad) of low-LET radiation.

2. Relative biological effectiveness: BEIR IV applies a relative biological effectiveness factor of 10, based on in-vitro cell killing data, to external low-LET radiation data to conclude that there is a threshold of 1 rem for internal high-LET radiation. Even assuming that an in-vitro relative biological effectiveness factor for cell-killing of 10 is correct for conditions in utero, cell death is not the only relevant endpoint. Other endpoints are also important. I discuss a few below including the failure of neural tubes to close, increased sister chromatid exchange frequency, the production of reactive oxygen species within the cytoplasm, and mutations of mitochondrial DNA.

Examining different endpoints greatly expands the range of relative biological effectiveness factors that should be applied. Consider the 1992 paper by Hatsumi Nagasawa and John B. Little, published in the journal *Cancer Research*.[15] They found that a rather high level of low-LET radiation – 1 to 2 grays (100 to 200 rads) in the form of x-rays produced the same level of genetic damage as indicated by sister chromatid exchange as just 0.31 milligray of high-LET radiation (plutonium-238 decay alpha particles), both being administered at high rates to Chinese hamster ovary cells. *This implies a relative biological effectiveness for the particular genetic endpoint under consideration of 3,200 to 6,400.* Such a range of factors applied to the BEIR IV indication of a threshold of 10 rad (0.1 gray) would translate into a high-LET alpha radiation *threshold of just 1.5 millirem to 3 millirem (0.015 to 0.03 mSv)*, which, given the uncertainties, is effectively a zero threshold.

A central finding of the Nagasawa-Little paper was that 30 percent of the cells showed increased sister chromatid exchange frequency even though less than one percent of the cells were traversed by an alpha-particle. In other words, a very large part of the damage caused to the ovary cells was not only due to the high rate of linear energy transfer (high LET) in the directly affected cells. More than 30 "bystander" cells were impacted for every cell in which the alpha particle directly deposited energy. Very large relative biological effectiveness factors were also inferred by Khadim et al. in their experiment with hematopoietic clonal cells derived from stem cells that "survived the passage of one or more radiation tracks before the initiation of clonal proliferation" – that is, the experiment was for an endpoint of radiation that was not cell death. They observed "a high frequency of non-clonal aberrations in the clonal descendants compatible with α-emitters inducing lesions in stem

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[14] Field radioecological studies are critical. For instance, wildlife in the Chernobyl exclusion zone appears to be much more sensitive to ionizing radiation than indicated by laboratory experiments. J. Garnier-Laplace, S. Geras'kin, C. Della-Vedova , K. Beaugelin-Seiller, T.G. Hinton, A. Real, A. Oudalova, "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*, Vol. 121, 2013, pp. 12-21.

[15] Hatsumi Nagasawa and John B. Little. "Induction of Sister Chromatid Exchanges by Extremely Low Doses of α-Particles," *Cancer Research*, Vol. 52, November 15, 1992, p. 970. On the Web at https://cancerres.aacrjournals.org/content/canres/52/22/6394.full.pdf

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cells that result in the transmission of chromosomal instability to their progeny." The effect was not observed when the cells were subject to x-rays.[16]

It is important to note in this context that the concept of relative biological effectiveness is being applied to a non- stochastic, i.e., a somatic, effect. The concept has generally been applied to adjust stochastic risk for a single end point – cancer – for radiation with different rates of linear energy transfer.[17] Generally two or more adverse events separated in time characterize cancer risk; repair mechanisms also play a role.   Further, cancer risks for specific organs involve estimation of dose to the whole organ (integrated over time in the case of low dose-rates).[18] Teratogenic risks during organogenesis are quite different. They involve non-stochastic effects – like cell killing or genetic damage -- in rapidly dividing stem cells. In that context it is far more important to consider specific endpoints and specific cells to estimate particular teratogenic risks, especially in regard to maternal-fetal transfer of radionuclides.

A basic biological-ecological way of looking at the problem is that for fully grown people, ionizing radiation impacts generally occur in the context of day-to-day routine homeostatic functioning – the body is in dynamic equilibrium with its environment. That is not at all the case for the embryo and the fetus that are developing and growing rapidly. For instance, the drastic negative impact of cell-killing in the earliest stages of pregnancy contrasts starkly with an alpha particle killing a cell in an adult most of whose cells are, with critical exceptions (notably oocytes), replaced in the space of days or months. The Committee should recommend detailed research for different stages of organogenesis, for the specific organs involved, for the specific non-stochastic effects at issue, all for specific radionuclides and the radiation to which they subject embryonic and fetal cells.

One important issue to consider for research should be potential impacts on oogenesis during the relevant period of pregnancy and again in the period of post-puberty oocyte maturation especially if exposure continues, as for instance via drinking water or contaminated food (see also below on multi-generational impacts).

3. Vulnerability period for teratogenic impacts: BEIR IV considers failed pregnancies only in the earliest post-fertilization period and teratogenic impacts only in the balance of the embryonic period up to 50 days of the pregnancy for all but the central nervous system. In the latter case it considers a window up to 19 weeks.[19] This is very important, but far too limited. For instance, Table 4 of ICRP 49 mentions possible neural tube defects ("dysraphic abnormalities") during the third and fourth weeks of pregnancy.[20] As the Centers for Disease Control and Prevention has explained, failure of neural tubes to close can lead to severe teratogenic effects, including spina bifida and anencephaly (the failure of the brain to develop at all, or develop incompletely). The CDC states that "Pregnancies affected by anencephaly often result in miscarriages, and the infants who are born alive die very soon after birth."[21]

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[16] M. A. Kadhim, D. A. Macdonald, D. T. Goodhead, S. A. Lorimore, S. J. Marsden & E. G. Wright, "Transmission of chromosomal instability after plutonium α-particle irradiation," *Nature*, Vol. 355, 20 February 1992.

[17] See for example Section 2 in Office of Radiation and Indoor Air, *EPA Radiogenic Cancer Risk Models and Projections for the U.S. Population*, EPA 402-R-11-001 .Washington, D.C.: U.S. Environmental Protection Agency, April 2011; hereafter EPA 2011.

[18] See EPA 2011 for instance.

[19] BEIR IV 1986, pp. 383-384.

[20] ICRP 49 1986, p. 30.

[21] Beth Burke et al., Preventing Neural Tube Birth Defects: A Prevention Model and Resource Guide, Centers for Disease Control and Prevention, Sixth Printing, April 2009, p. 6.

Thus a broader view is needed even for miscarriages. This is also true of teratogenic impacts more generally.

For instance a 1979 synthesis study, "based exclusively on the abundant but dispersed data in the literature,"[22] covered the period of central nervous system development starting at 15 days after conception through the entire pregnancy and the post-natal period, when the pre-frontal cortex continues to develop. Thus, the vulnerability period for central nervous system teratogenic impacts extends through the entire pregnancy. That this applies to other teratogenic impacts is indicated by the view of the Teratology Society cited above. At least that should be the starting hypothesis for research, with the recognition that the types, severity, and frequency of impacts of ionizing radiation are likely to depend on the timing and type of exposure, among other factors.

In sum, research on the teratogenic effects radiation exposure needs to be greatly expanded, including notably for radionuclides that are transferred from the pregnant woman to the embryo and the fetus.

c.   Multigenerational Impacts

The previous section relates to teratogenic impacts on the embryo and fetus. Beyond that, consideration of multigenerational impacts is also essential. That there are intergenerational risks is clearly indicated by the established fact of maternal to fetal radionuclide transfers. [23] Radionuclide body burdens acquired prior to pregnancy also impact the embryo and fetus due to radionuclide transfer from the mother, if the biological half-life of the radionuclides is long-enough.

There are a number of areas to be researched if the goal is to protect women and future generations. The following are among the key questions for you to consider in recommending the low-level radiation research agenda; the list is not exhaustive:

- How long does each radionuclide transferred to the fetus stay in the child as it develops into an adult? What are impacts on subsequent generations of those body burdens? How does that vary between males and females?
- What is the impact on ova of internal radiation exposure during the oogenesis period of pregnancy and also due to exposure during the post-puberty maturation of ova? Specifically, what are the risks of germline mutations during oogenesis? What are the corresponding risks related to sperm, which are transient as well as to primary spermatocytes?
- What are the risks of mutations in mitochondrial DNA during oogenesis and the related implications for the female child and succeeding generations? How many generations can mitochondrial DNA mutations be expected to last?
- What are the intergenerational risks of germline mutations in oocytes due to ionizing radiation, including internal exposure, producing teratogenic outcomes in the children of a woman who was exposed as a fetus?
- Can mitochondrial DNA damage acquired during pregnancy create general health vulnerabilities for subsequent generations, for instance, via compromised intra-cellular metabolism?

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[22] Lucia Zamorano and Benedicto Chuaqui, "Teratogenic Periods for the Principal Malformations of the Central Nervous System", *Virchows Archiv A*, vol. 384, 1979, pdf p. 1.
[23] ICRP 88 2002, NRPB 2001, Pacific Northwest National Laboratory 1996.

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- What radionuclides are important for multigenerational impact, based on the following considerations?
  - Ubiquity of pollution – tritium is a prominent example in terms of the quantities routinely emitted and discharged from nuclear power plants, reprocessing plants, and other military and commercial facilities, whether as water, water vapor, organically bound, or other relevant chemical forms.
  - Specific radionuclide pollutants in areas of or as a result of specific activities such as uranium mining, uranium milling, fallout from nuclear weapon testing and nuclear power accidents, with Chernobyl and Fukushima being the most prominent examples;
  - Leaks and discharges from a variety of waste disposal and waste management activities as well as residual contamination of soils and waters at nuclear facilities. Uranium and radionuclides in the decay chain of uranium-238 are a common example.
- What pathways for fetal and multi-generational exposure have been understudied or even ignored?

A few examples related to nuclear weapons testing will illustrate some of the issues. Both pre-pregnancy exposure and intakes during pregnancy can result in fetal radionuclide burdens. Many women throughout the world, particularly in areas of high fallout during atmospheric weapons testing, had significant intakes of radionuclides. People who lived downwind from the Trinity test had direct inhalation doses. They also collected rainwater from their roofs in barrels and used it for drinking and cooking. This was, of course, typical of many areas in the world during atmospheric testing. They were also affected by various pathways due to fallout depositing on laundry hung out to dry. Collection of drinking water and outdoor laundry drying are frequently mentioned by the affected public in matters of exposure; yet, they have often not been given their due. Nor are the problems limited to the immediate vicinity of the test sites. For instance, the 1954 CASTLE test series in the Marshall Islands created significant cumulative fallout thousands of miles from the Enewetak test location.[24]

The Committee should also note that there have been many controversies over official estimates of fallout, and radiation doses resulting from testing, among other things. Most recently, an independent study of atmospheric testing in Polynesia that the number of people exposed to fallout was ten times higher than estimated by the French government, which carried out the tests. The exposures may also have been considerably higher than admitted by the French government.[25] The Committee should therefore emphasize the importance of independent studies as well as publication of health and environment-related documents and data to enable that independent research. This is especially important for the Committee given the strenuous protests it has heard regarding the Department of Energy, which oversees the U.S. nuclear weapons complex, funding of the study it is doing.

The problem of intergenerational impacts has too long been ignored, apart from the narrow and particular case of women who declare their pregnancy in regulated workplaces. I urge you to give the

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[24] Robert J. List, *Worldwide Fallout from Operation CASTLE*, U.S. Department of Commerce, May 17, 1955. See the map on p. 20 (pdf p. 26). On the Web at https://doi.org/10.2172/4279860

[25] Adrian Cho, "France grossly underestimated radioactive fallout from atom bomb tests, study finds**:** Declassified documents suggest 90% of French Polynesians received significant exposure, *Science*, March 21, 2021 at https://www.science.org/content/article/france-grossly-underestimated-radioactive-fallout-atom-bomb-tests-study-finds

broader issue relating to all women your detailed attention. More than one person testified at the public hearing on October 28, 2021 about the importance of considering exposure of pregnant women. I especially call your attention to the presentation Beata Tsosie-Peña of Tewa Women United in this regard. She called for radiation protection centered on pregnant women and a "Land-Worker-Mother" approach to the science and to radiation protection.[26] The concept, in my view, encompasses human health but also ecological health and thus extends, in technical terms, to radioecology.

d. Tritium

Tritium outside the body and even deposited on the skin, which has dead cells on the surface, should be considered differently from tritium inside the body (as tritiated water or as organically bound tritium). Once tritium crosses into a cell, its beta particle energy will generally be deposited entirely within that cell. The average and maximum stopping distances of tritium beta particles in water are 0.42 microns and 5.2 microns, respectively.[27] This is somewhat smaller than the range of diameters of cell nuclei and, therefore, obviously smaller than cell diameters; it is roughly two orders of magnitude smaller than a mature human ovum. The range of tritium beta particle energies (near-zero to 18.6 keV) and their stopping distances indicate that each tritium decay will typically result in hundreds of ionizations both in the nucleus and in the cytoplasm, with the most energetic beta particles creating well over a thousand ionizations.[28]

As noted in BEIR VII, exposure to beta particles can create the highly reactive hydroxyl radical, creating oxidative stress in the cell. Hydroxyl radicals are produced when the ions created by collisions between beta particles and water molecules go on to collide with other water molecules. The hydroxyl radical can create a very substantial amount of intracellular damage, including DNA damage:

> The relatively long-lived (about 10^{-5} s) OH• radical is believed to be the most effective of the reactive species; as an oxidizing agent, it can extract a hydrogen atom from the deoxyribose component of DNA, creating a DNA radical. *Early experiments demonstrated that about 70% of the DNA damage can be prevented by the addition of OH• scavengers*….Because OH• is so highly reactive, it has been estimated that only the radicals formed within about 3 nm of DNA can react with it….[29]

Within the cytoplasm, hydroxyl radicals can attack mitochondrial DNA, which is the core of the body's energy system. Mitochondrial dysfunction may cause genomic instability, possibly with heritable impacts;[30] it can also contribute to neurological diseases.[31] What would be the impact of such

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[26] Beata Tsosie-Peña, Tewa Women United, Video presentation to the Committee, October 28, 2021.

[27] NRC ML 2034, Attachment A, p. E-38. On the Web at https://www.nrc.gov/docs/ML2034/ML20343A210.pdf

[28] For instance, the ionization energy of water is about 12.6 eV; at that rate, the average beta particle energy of 5.7 keV would produce about 450 ionizations of H2O molecules. Source: NIST Chemistry WebBook, Water, National Institute of Science and Technology, at https://webbook.nist.gov/cgi/cbook.cgi?ID=C7732185&Units=SI&Mask=20 The maximum beta particle energy would result in over 1,470 ionizations of water molecules. Of course, the ionizations from a decay would not be confined to a single type of molecule (though water is the most abundant).

[29] BEIR VII 2006, p. 29.

[30] Grace J.Kim, Krish Chandrasekaran, and William F. Morgan, "Mitochondrial dysfunction, persistently elevated levels of reactive oxygen species and radiation-induced genomic instability: a review," *Mutagenesis*, Vol. 21, No. 6, October 2006 at doi:10.1093/mutage/gel048; hereafter Kim et al. 2006.

[31] Siddharth Arun, Lei Liu, and Gizem Donmez, "Mitochondrial Biology and Neurological Diseases," *Current Neuropharmacology*, Vol. 14, 2016 at https://pubmed.ncbi.nlm.nih.gov/26903445/

mitochondrial dysfunction produced by the excess reactive oxygen species created by ionizations in the cytoplasm in the embryo and fetus? That is one of the vital questions for research on the impact of ionizing radiation during pregnancy. Its intracellular impact should be of concern at all ages and for both females and males. One specific concern regarding post-puberty males is that sperm formation could be affected by intakes of tritiated water and organically bound tritium.

It is important for the Committee to recommend a research program and structure that recognizes that tritium has very different impacts within the human cell as compared to when it is externally deposited on the skin. ***Intra-cellular tritium should not be considered as low-LET radiation*** since it generally deposits its entire energy within a cell and, among other things, creates excess reactive oxygen species that can disrupt cellular metabolism and create mitochondrial dysfunction.[32] ***These considerations relating to tritium internal dose (as water and as organically bound tritium) should shape the recommendations of the Committee for research design on this topic for the entire public, including of course pregnant women, as well as for radioecology.***

Tritium in the form of tritiated water is the most common routine radioactive pollutant resulting from nuclear power plant operations, from some nuclear weapons plants, such as the Savannah River Site, and commercial reprocessing plants. Tritiated water is chemically essentially identical to water and so can penetrate every cell of the embryo and the fetus; and, once in a cell, deposits its energy generally within it. In light of these facts, tritium should be a research priority, notably, but not only, in the context of teratogenic impacts during pregnancy.

e. Recommendations summary for areas of study

The major thrust of this memorandum to the Committee concerns women who are pregnant, the embryo, and the fetus, and more generally women of child-bearing age as such. They should be central to your considerations and recommendations about radiation research and protection. Within that broad area, research on the teratogenic effects of radionuclides that cross the placenta as well as their multi-generational impacts deserve your special attention. In that light, the Committee should:

i. Acknowledge the relative neglect of research relevant to the protection of female members of the public who are pregnant or who may want to become pregnant, especially in regard to teratogenic effects of internal radiation and multi-generational impacts. High-LET radiation and tritium (including tritiated water and organically bound tritium) should be a principal part of the development of a detailed research agenda on teratogenic impacts during pregnancy.

ii. Recommend a broad and intensive program of research that puts pregnant women, the embryo and the fetus at the center of the design. This program should include detailed and special attention to the teratogenic effects of radiation, notably internal radiation, and to multi-generational impacts. Though it has not been discussed in this memorandum, post-natal cancer risk, a recognized impact of fetal in-utero radiation, should also be part of the overall research agenda both on scientific and health protection grounds.

iii. Recommend a research program to evaluate mutations in mitochondrial DNA caused by internal high-LET radiation and by intracellular tritium.

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[32] Figure 2 in Kim et al. 2006 provides a simple illustration of the problem (including feedback effects).

iv. Study the possible impact of maternal-fetal radionuclide transfer on formation of the gonads, the creation of ova, induction of mutations in oocyte mitochondrial DNA and make that a critical part of your recommendations on ionizing radiation research.

v. Evaluate study designs, including following women who intend to become pregnant, for elucidating effects of ionizing radiation that are difficult to detect, including preimplantation losses and early miscarriages due to certain neural tube defects and the neural tube defects themselves. Examples of places that could be studied include places with high fallout from atmospheric testing such as the Marshall Islands, the Nevada Test Site, and Maralinga. Other important areas include those with high environmental contamination due to nuclear activities such as uranium mining and areas that have been contaminated as a result of major accidental releases. Tritium pollution occurs in many places, such as the waters of the Savannah River downstream from the Savannah River Site in South Carolina. Of course, informed consent and other processes that are respectful of the women, their privacy and their health should be essential parts of the process.

vi. Estimate the range of relative biological effectiveness factors for various endpoints and different types of radiation (internal tritium, internal high-LET radiation, and external low-LET radiation) and their implications for pregnancy outcomes with specific reference teratogenic impacts to the embryo and fetus. The end points should include (but not be limited to) stem cell damage without cell killing, increased sister chromatid exchange frequency, neural tube defects, genomic instability, and bystander effects.

vii. Evaluate approaches that would allow the cancer risks of external low-LET radiation to children illuminated by Dr. Rebecca Smith-Bindman's research to be extrapolated to internal high-LET and tritium exposure, including what relative biological effectiveness factors might be used in case of pediatric internal exposure.

viii. Revisit the hypothesis that there is a threshold for teratogenic impacts for all organs except the central nervous system. In view of the evidence and analysis in Section I.b above, the Committee should recommend a no-threshold hypothesis be used for estimating these impacts both on scientific and health protection grounds, pending further research, including that enumerated in this list.

ix. Analyze and recommend research on the intracellular impacts of high-LET radionuclides and tritium, including intra-cellular metabolism, ion and electron transport, disruption of mitochondrial DNA communication with nuclear DNA, the impact of excess reactive oxygen species on mitochondrial dysfunction and genomic instability, and intergenerational impacts of mitochondrial DNA damage during oogenesis.

x. Recommend a radioecological research agenda that could shed light on the issues discussed in this memorandum. This should include but not be limited to more detailed, independent studies of accident contaminated zones (Fukushima, Chernobyl) and processing and mining areas with significant radioactive contamination. As noted above, Chernobyl field research indicates that radiation sensitivity measured in the field appears to be much greater than that seen in laboratory experiments. In formulating radioecological research program recommendations, the Committee should consult with indigenous leaders, including the ones who gave presentations to it on October 28, 2021, and give due consideration to the consensus

statements of radioecologists arising from a symposium held in November 2015.[33] The Committee should also give due attention to the fact that mitochondria are the basis for the energy system of all multi-cellular beings (among others) whose disruption or dysfunction can have profound impacts. A sound program of radioecological research holds promise for helping all species, including *homo sapiens*.

xi. Set forth a research program based on the "Land-Worker-Mother" concept, "centered on indigenous pregnant people as the standard"[34] in its own right and also as the basis for comparison with other approaches to radiation protection, including for long-term impacts of residual radioactivity after remediation, waste management, and disposal. In developing this program, the Committee should consult closely with indigenous communities, including the leaders who were invited to make presentations to it on October 28, 2021.

II. The Linear No-Threshold Hypothesis for cancer risk

The linear no-threshold hypothesis for cancer risk does not need to be and should not be revisited as such. There are already many studies that have rejected alternative hypotheses both as a scientific basis for risk assessment and for public health protection. These include the extensive study by the National Academies, BEIR VII (Phase 2) and the follow-up study done by the Environmental Protection Agency (EPA 2011). The evidence for it has become stronger over the years. Recent evidence includes research by teams that included one of the Committee's members, Dr. David Richardson, which I do not need to cite explicitly. The presentation of Dr. Smith-Bindman is also compelling in this regard. It provides direct epidemiological evidence for large excesses of pediatric leukemia (27%) and brain cancer (9%) at a dose of 10 milligray (1 rad). A large Australian study showed excess pediatric cancer risk from CT scans averaging 4.5 millisievert per scan (0.45 rem).[35]

The Committee should make a definitive statement that no further expenditure of public funds should be made to re-examine the linear no-threshold approach to cancer risk estimation for scientific, public health, or regulatory purposes. While research such as that by Dr. Richardson and his colleagues and by Dr. Smith Bindman (and other similar research) may lead to a revision of the risk coefficients, as has occurred in the past with the BEIR series of the National Academies and the work of the U.S. Environmental Protection Agency, the Committee would do the public a great service by stating clearly that public funds should not be expended on exploring hormesis or thresholds for cancer risk and that

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[33] François Brechignac, Deborah Oughton, Claire Mays, Lawrence Barnthouse, James C. Beasley, Andrea Bonisoli-Alquati, Clare Bradshaw, Justin Brown, Stephane Dray, Stanislav Geras'kin, Travis Glenn, Kathy Higley, Ken Ishida, Lawrence Kapustka, Ulrik Kautsky, Wendy Kuhne, Michael Lynch, Tapio Mappes, Steve Mihok, Anders P. Møller, Carmel Mothersill, Timothy A. Mousseau, Joji Otaki, Evgeny Pryakhin, Olin E. Rhodes Jr., Brit Salbu, Per Strand, Hirofumi Tsukada, Addressing ecological effects of radiation on populations and ecosystems to improve protection of the environment against radiation: Agreed statements from a Consensus Symposium, *Journal of Environmental Radioactivity*, Vol. 158-159, 2016, pp. 21-29.

[34] Beata Tsosie-Peña 2021, op cit.

[35] John D. Mathews, Anna V Forsythe, Zoe Brady, Martin W Butler, Stacy K Goergen, Graham B Byrnes, Graham G Giles, Anthony B Wallace, Philip R Anderson, Tenniel A Guiver, Paul McGale, Timothy M Cain, James G Dowty, Adrian C Bickerstaffe, and Sarrah C Darby, "Cancer risk in 680 000 people exposed to computed tomography scans in childhood or adolescence: data linkage study of 11 million Australians, *British Medical Journal*, Vol. 346, May 21, 2013 at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3660619/

such expenditures are an unwarranted waste of public resources. If the nuclear industry wants to undertake this research, it should finance it out of its profits and so inform the affected shareholders.

III. Standards

a. Interim tightening of drinking water standards

Remediation of research deficiencies and gaps, including those identified above, is going to take time. The potential for teratogenic damage, the established conclusion that there is no threshold for central nervous system teratogenic effects, and at least plausible arguments that there is no threshold for other teratogenic impacts point to the necessity and urgency of interim protective measures.

In particular, a tightening of drinking water standards is urgently needed, especially for tritium, which is the most common routine radiological pollutant. For the reasons I discuss below, the Committee should recommend a tightening to 400 picocuries per liter (from the current 20,000 picocuries per liter), as a much more protective standard that is all the more needed in view of the long neglect of protection of pregnant women and the embryo and fetus.

There is already a substantial official history pointing to the need to tighten drinking water standards for tritium because tritium in drinking presents risks to very large populations. For instance, the specific public health criterion used by the official Ontario Drinking Water Advisory Council, was a lifetime fatal cancer risk of one in a million people for tritium-contaminated drinking water; tritium is emitted from the province's heavy water-moderated nuclear power plants. The recommended limit was 20 Bq/liter.[36] Department of Energy remediation guideline for tritium in runoff water during the decommissioning of its Rocky Flats Plant in Colorado provides another example where the same risk criterion was used and the limit was set at 500 picocuries per liter, which is 40 times more stringent than the U.S. drinking water standard. [37] The concern in that case was the nearby drinking water reservoir that supplies the Denver area.

The Ontario and Colorado calculations were done using somewhat dated dose conversion factors to calculate the lifetime risk. The updated values in Federal Guidance Report 13 show that the tritium concentration limit corresponding to the same a one-in-a-million life time fatal cancer risk should be 400 picocuries/liter, whence my recommendation. The State of California has a drinking water guideline of 400 picocuries per liter (15 Bq/L) of tritium.

While tritium is the most common man-made pollutant, it is not the only one. The same criterion should also apply to other man-made radionuclides that cross the placenta. My calculations for drinking water limits at the one-in a million risk level based on Federal Guidance Report 13 are reproduced below in Table 1; they were presented to the New Mexico Water Quality Commission. I hope you will recommend that these limits be adopted on an urgent basis.

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[36] Ontario Drinking Water Advisory Council, Report and Advice on the Ontario Drinking Water Standard for Tritium, 2009, hereafter ODWAC 2009. The Council's recommendation was rejected in favor of a much more lax standard (7,000 Bq/L) even though the Council noted that the province's power plants could meet the 20 Bq/L limit if they did not exceed their discharge limit – ODWAC 2009, p. 5.

[37] Canada uses metric units; Ontario's *advisory level was* 20 becquerels/liter (Bq/L), which is the rounded value corresponding to the one-in-a-million life time risk; it is equal to 540 picocuries per liter. (Corrected footnote; corrected part in italics.)

Table 1: Recommended drinking water standards for certain man-made radionuclides in pCi/L (or Bq/L)

Radionuclide	pCi/L	Bq/L
Americium-241	0.19	0.007
Cesium-137	0.64	0.024
Plutonium239/240	0.15	0.006
Strontium-90	0.35	0.013
Tritium	400	15

Source: Witness Statement of Arjun Makhijani to the New Mexico Water Quality Commission on behalf of Amigos Bravos, August 27, 2009, p. 8. The statement and the transcript of the oral testimony are at https://ieer.org/resource/press-releases/mexico-strengthens-limits-public/

The U.S. drinking water standard applies only to public water systems. As a result, private wells are exempt, sparing them the expense of monitoring and other compliance-related expenses. However, this also allows nuclear power and weapons plants to emit and discharge tritiated water that can adversely impact private water supply. The Committee should consider what interim restrictions on such emissions and discharges would be needed to protect drinking water sources not covered EPA regulations to the equivalent standard of 400 picocuries per liter (15 Bq/L). Further, in the US and elsewhere there are groundwater protection standards to ensure the continuing quality of aquifers irrespective of whether water is drawn by public or private users. This places a requirement on polluters not to pollute aquifers irrespective of actual or potential use.

Evidently, this approach to strengthening the drinking water standard does not directly address the issue of teratogenic effects. It has official precedent and is recommended here as a precautionary measure while a detailed research program is created and implemented.

b.   Declared pregnancy in radiation-regulated workplaces

Fetal exposure of up to 5 mSv (500 mrem) is allowed for women in radiation-regulated workplaces in the United States; it should have been tightened decades ago, when the standard for the general public was changed to 1 mSv (100 mrem) per year. The latter is the level in other industrialized countries. The Committee should recommend the immediate adoption of 1 mSv (100 mrem) permanently for the United States. Considerations relating to severe mental retardation and, potentially, other teratogenic impacts indicate that a more stringent interim limit should be adopted until a more public regulatory process can be completed.

c.   Imaging radiation

Guidelines in medical imaging and means for ensuring that radiological practice is keeping radiation doses as low as reasonably achievable are urgently needed, in view Dr. Smith-Bindman's research and findings. Her presentation to you on October 27, 2021 also contains compelling evidence that the Committee should not only rule out the hormesis hypothesis but also explicitly warn against using it for rationalizing exposure, especially in public health settings. The 2015 statement of the head of the Board of Chancellors of the American College of Radiology, whom she quotes,[38] includes a suggestion of

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[38] The 2015 statement by the head of the Board of Chancellors, as quoted, was: "Based on what is known, a CT study could carry a small risk for cancer, have no detectable cancer risk, or carry a potential benefit of cancer prevention" and further going on to say "If we were to impose a requirement of informed consent a practitioner

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hormesis; this is appalling in light of compelling evidence that children are contracting cancer as a result of CT scans carried out in many or most cases without due regard to the principle of keeping doses as low as reasonably achievable. The Committee should be clear that such statements in the public health arena are highly inappropriate, besides being scientifically unsound. Given the presence of medical professionals on the Committee, it should also consider the causes of the lax practices that have led to excess doses and cancers and recommend approaches for better protecting patients, especially children.

IV.   Review process

The Committee should open up its review process to a larger range of stakeholders. Specifically, the Committee should set an example and formally consider members of affected communities who have evaluated the issues as peers and include them in its review process. You already have had presentations from some such community members; I recommend that a significant part of your review panel be drawn from among them. They have expertise, evidence, and experience that many in academic settings lack. It is high time that the National Academies broadened their concept of "peers."

Peer-reviewed literature is hardly a uniform gold standard. It spans the range from poor to indifferent to good to Einstein's utterly brilliant work in his 1905 papers. It is all-too-often rife with conflicts of interest. For instance, a 2016 review noted that 1960s research done at Harvard and published in the New England Journal of Medicine "singled out fat and cholesterol as the dietary causes of CHD [coronary heart disease] and downplayed evidence that sucrose consumption was also a risk factor."[39] The study was funded by the Sugar Research Foundation. While conflicts are usually declared today, they are still pervasive. For instance, the food researcher Marion Nestle (no relation to the company) publishes a blog (at FoodPolitics.com) that is full of examples of research funded by one branch or another of the food industry that almost always comes to the expected conclusions; she calls the series "Conflicted Study of the Week."

My advice is simple: it is long past time for you to open up the review process, especially on matters of such great import in a context where public trust in official science is low. I am not going to elaborate on the very good reasons for that low level of trust in this memorandum; I would be happy to supply you with my reasons should you desire to know them, along with the documentation for my conclusions.

Let me give you a simple, useful example from my own experience of being open to expert comment. One of the participants in IEER's technical training workshops several years ago, Jessica Azulay,[40] asked why the term "dose" was used to refer to both deposited energy per unit mass, and also when that dose was multiplied by an empirical relative biological effectiveness factor that seeks to capture the relative harm of different types of radiation. She has a liberal arts educational background and has intensively studied environmental issues related to energy, including nuclear energy. She said that dose multiplied

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would have to state that there is an unproven possibility that the CT study could increase the risk for cancer and then state that there is an unproven possibility that it may decrease the risk for cancer." Smith-Bindman 2021, Slide 15.

[39] Cristin Kearns et al., "Sugar Industry and Coronary Heart Disease Research: A Historical Analysis of Internal Industry Documents," JAMA Internal Medicine, November 2016, at https://jamanetwork.com/journals/jamainternalmedicine/article-abstract/2548255

[40] Name and example cited with permission.

by relative biological effectiveness was meant to indicate biological radiation harm; therefore it was no longer a simple, physical measure. Using the same word "dose" for both was misleading and confusing, she asserted. I agreed with her brilliant observation and still do; evidence for that can be found in the mix-up of rem and rad in BEIR IV cited above, where the report used the term "rad" instead of "rem" after a relative biological effectiveness factor of 10 was applied. I recommend that what is measured in rem (or sieverts) be referred to as "radiation harm" and what is measured in rad (or gray) as radiation dose. The term "radiation harm" will also be a good reminder that relative biological effectiveness factors should be specific to the type of harm being considered.

Finally, community experts should be compensated for their time at rates comparable to the compensation of senior academic researchers on radiation issues.