



**INSTITUTE FOR ENERGY AND
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**MULTIGENERATIONAL HEALTH IMPACTS OF NUCLEAR WEAPONS TESTING AND PRODUCTION:
A SURVEY**

Arjun Makhijani, Ph.D.

President, Institute for Energy and Environmental Research

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Preface and Acknowledgements

Despite the vast literature on the health impacts of radiation, the topic of multigenerational impacts is seriously understudied. This is especially the case for teratogenic impacts, that is impacts such as stillbirths and children born with severe malformations. For example, many women in the Marshall Islands, where the United States conducted 67 nuclear weapons tests totaling 108.5 megatons of explosive power, between 1946 and 1958, have described tragic pregnancy outcomes. These include stillbirths and children who died shortly after birth. The latter category included a child “missing the whole back of his skull”; a child who did not look human but rather “just like the inside of a giant clam; and a baby that “had two heads.” There were those who survived much longer but had enormous medical problems. There were “children born during this time [of nuclear weapons testing] did not have any noticeable deficiencies, and yet lacked the ability to understand anything. Others were incapable of any motor activity although they seemed to comprehend their surroundings.”¹ Despite such evidence, there is no official systematic scientific study of adverse pregnancy outcomes across the Marshall Islands that examines the relationship of testing, fallout, and radiation exposure of pregnant Marshallese after the start of testing.

Radiation exposures are, of course, not limited to nuclear testing fallout. There are exposures related to nuclear power plants, production and processing of nuclear materials for weapons and power, and diagnostic and curative medical exposures. In 1956, Alice Stewart and her colleagues found a relationship between exposure to x-rays during pregnancy and early childhood cancer.²

Despite the evidence, there is as yet no specific regulatory standard to protect pregnant women who are members of the public, that is, almost all women, or the embryo and fetus, from the variety of harms that exposure to ionizing radiation may inflict. The only limitation on exposure during pregnancy is in regulated nuclear workplaces when women declare their pregnancies. In that case, the usual limit of exposure for the duration of pregnancy is 1 mSv, the same as the annual limit for members of the general public. In the United States the limit is 5 mSv, five times greater than for members of the general public.

A note on terms. The term “radiation” in this report relates only to ionizing radiation. The term “multigenerational impacts” is used in this report to mean an impact on any generation other than that of the person who is directly exposed to internal and/or external ionizing radiation. A complex, even fraught, issue of terminology relates to sex-specific and gender-specific terms surrounding pregnancy. The use of gender-specific language when treating individuals who identify as men who are or may become pregnant is appropriate and important, as noted in Gribble et al. (2022), an article on “Effective Communication About Pregnancy, Birth, Lactation, Breastfeeding and Newborn Care.” Yet, they also point out that it is necessary to consider the consequences of removing sex-specific language for its “impact on accuracy or potential for other unintended consequences” including “a number of potentially deleterious consequences...”; the paper provides examples of such consequences.³ For this

¹ Testimonies as quoted in Barker 2013.

² Stewart et al. 1956.

³ Gribble et al. 2022. The authors’ affiliations were: School of Nursing and Midwifery, Western Sydney University, Parramatta, NSW, Australia and Department of Women, Children’s Health, King’s College London, London, United Kingdom; Mount Auburn Hospital, Cambridge, MA, United States; Department of Medicine, Harvard Medical School, Boston, MA, United States; Alive & Thrive Southeast Asia, FHI Solutions, Hanoi, Vietnam; Chelsea and

report, I have followed the guidance of the National Institute for Health Care Excellence of the UK Royal College of Obstetricians and Gynaecologists in regard to pregnancy: “The guideline uses the terms 'woman' or 'mother' throughout. These should be taken to include people who do not identify as women but are pregnant or have given birth.”⁴ This use of this sex-specific language in a general report on inter-generational issues, such as the present one, does not obviate the need to use gender-specific language in treating individuals who identify as men who are, or may become, pregnant.

This report was prepared for the Nuclear Truth Project, which “is an international initiative connecting Indigenous and First Nations Peoples, affected community members, international and civil society organizations, experts and governments working for nuclear abolition.”⁵ The specific context for the formation of the project and commissioning of this report was the coming into force of the Treaty on the Prohibition of Nuclear Weapons in January 2021, since the treaty is centered on humanitarian concerns and explicitly recognizes the disproportionate impact of radiation on women and children. It also provides for the parties to the treaty to provide assistance to impacted people.⁶ Some discussion impacts beyond nuclear weapons testing and use, including potential multigenerational impacts from uranium mining and production of nuclear weapons materials, is also provided in this paper.

I would like to thank Professor Timothy Mousseau for providing me with important research papers from his vast library as well as some review comments. This report was reviewed by Dr. Tilman Ruff and Dr. Cath Keaney. Their reviews helped me to improve this report. However, as the author, I remain fully responsible for its contents and conclusions and any errors that may remain. It was generously funded by an anonymous donor.

Arjun Makhijani
President, Institute for Energy and Environmental Research
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Westminster Hospital National Health Service (NHS) Foundation Trust, London, United Kingdom; School of Nursing and Midwifery, Griffith University, Brisbane, QLD, Australia; Centre for Health Care Research, Coventry University, Coventry, United Kingdom; Department of Women’s and Children’s Health, Karolinska Institutet, Stockholm, Sweden; Breastfeeding Promotion Network of India, New Delhi, India; and School of Nursing, Midwifery and Paramedicine, Australian Catholic University, Melbourne, VIC, Australia.

⁴ NICE 2021.

⁵ Nuclear Truth Project website at <https://nucleartruthproject.org/>

⁶ TPNW 2017; specifically see the Preamble and Article 6.

I. Introduction

This paper focuses on the multigenerational health impacts of exposure to ionizing radiation, using examples from nuclear weapons production, testing, and use. There is considerable overlap of these impacts with those associated with nuclear power, like the mining and milling of uranium and its processing to produce fuel for nuclear power reactors or nuclear weapons.

There was knowledge of multigenerational impacts during the period of atmospheric nuclear weapons testing. An example, which still has the power to shock, is provided by a 1960 editorial in the *California Engineer*, a University of California engineering alumni magazine. It estimated that nuclear testing would produce “an additional 6,000 babies born with major birth defects” throughout the world as a result of U.S. testing alone. But the editorial nonetheless concluded that “you must weigh this acknowledged risk with the demonstrated need of the United States for a nuclear arsenal.” The putatively “demonstrated need” was the first use of nuclear weapons in “brush-fire wars,” like the Korean War.

The assumption, supposedly based on lessons from the Korean War, was that the Soviet Union and China would exercise restraint if either side used “tactical” nuclear weapons. Specifically, the editors of the *California Engineer* believed that “there is no reason not to expect both sides in a future “brush fire” war to keep the nuclear weapons used within tactical limits.”⁷

The posture was the United States could inflict harm on the world’s children everywhere to support the development of its arsenal.⁸ The presumption that harm could be inflicted on the domestic and global public is a hallmark of the practices of nuclear-armed states. As documented in *Nuclear Wastelands*, produced by the International Physicians for the Prevention of Nuclear War and the Institute for Energy and Environmental Research, nuclear weapons states have inflicted harm without informed consent, including on their own people, as well as on others.⁹

Multigenerational impacts of ionizing radiation include:

1. **Teratogenic impacts and cancer risks:** Exposure to radiation can cause early miscarriages (in the first few weeks of pregnancy) and a variety of teratogenic impacts. Exposure may be to *external radiation*, such as gamma rays emitted by some fission products like cesium-137, neutron

⁷ An April 1960 editorial in the *California Engineer*, reprinted in *California Engineer*, Vol. 68, No. 3, March 1990, as quoted in IPPNW and IEER 2000, p. 18. It is useful to remember, for context, that the first U.S. meeting to consider the feasibility and design of nuclear weapons was held at the University of California Radiation Laboratory in Berkeley in the summer of 1942.

⁸ The specifics of the risk estimates are not at issue here; it is the presumption that harm could be inflicted without informed consent or any consent at all. While the risk estimates have since been revised, the particular once used in the *California Engineer* editorial seem to be based on the official scientific understanding of the time of germline mutations and their multigenerational impact. These were well summarized in a 1958 paper in *Science* by Linus Pauling: “700,000 embryonic and neonatal deaths”; “300,000 stillbirths and childhood deaths”; “80,000 children with gross physical or mental defect” over “many generations” assuming a 0.1 roentgen dose (about 1 mGy) average total dose over 30 years to the gonads of the world’s population due to atmospheric nuclear testing. The impact in the first generation immediately following was estimated as being an order of magnitude less than the multigenerational impacts for major birth defects and more in the case of the other outcomes. Pauling 1958.

⁹ IPPNW and IEER 2000, Chapter 1. See also IPPNW and IEER 1991, which describes pollution and cancer-related global impacts of atmospheric nuclear weapons testing.

radiation from the bomb explosions and in some workplaces where nuclear weapons are produced. In these cases, harm occurs when external radiation penetrates through to the embryo and fetus. *Internal exposure*: Internal exposures occur in utero radioactive materials are inhaled or ingested or when they are incorporated into the body via cuts and wounds. Internal exposure to iodine-131 (often via milk) is perhaps the best known example of fallout from nuclear weapons testing producing widespread internal exposure. Internal exposure also occurs in production-related activities, of which uranium mining and milling are the most widespread. There are also multigenerational cancer risks, which are briefly touched upon in this report. It is common in the case of radioactive fallout due to nuclear explosions and in nuclear weapons production for both internal and external exposures to be present. This is also the case for accidents related to nuclear weapons production and severe nuclear power accidents.

2. **Multigenerational impacts**: Multigenerational impacts can occur if gametes are mutated due to radiation exposure. Such mutations could occur via internal or external radiation exposure.
3. **Other multigenerational impacts**: A variety of other impacts may occur including developmental abnormalities due to thyroid gland exposures, and due to heritable epigenetic impacts.

The focus of most radiation research has been on cancer risk – both in terms of cancer incidence and cancer mortality. It is well established that nuclear weapons testing and use has caused exposures to children that have caused cancers among them. Cancer risk is also increased when a fetus is exposed to radiation during pregnancy.¹⁰ Cancer risk coefficients by age, starting at infancy, and sex can be found in a National Academies 2006 report (often called the “BEIR VII” report). Cancer risk per unit of radiation exposure decreases with age and is highest for infants. The risk for female infants is also double that of male infants.¹¹

Research on harm in the early stages of pregnancy is especially deficient, as is research on multigenerational impacts. The biggest gaps relate to non-cancer health harm, including pregnancy loss in the early stages of pregnancy and teratogenic impacts. Despite these gaps, what is known about impacts of radiation at the cellular level as well as epidemiological evidence clearly indicates serious impacts do occur and have occurred. There are also personal testimonies of severe adverse birth outcomes in nuclear testing fallout areas.¹²

Oxidative stress and mitochondrial damage are important mechanisms for harm during early stages of pregnancy. The impacts range far beyond cancer risk and can be multi-generational, as discussed in the body of this paper. Suffice it to note in this introduction that, among other things, oxidative stress as a result of radiation exposure to ionizing radiation,¹³ including at low doses to pregnant women, puts embryos and fetuses at risk.¹⁴

¹⁰ Stewart et al.1956.

¹¹ BEIR VII 2006.

¹² See for example Barker 2013.

¹³ BEIR VII 2006, p. 29 notes the particularly potent role of hydroxyl radicals in creating oxidative stress. This is also discussed in relation to intra-cellular tritium decay in Makhijani 2023.

¹⁴ Tharmalingam et al. 2017.

II. Teratogenic impacts

The causal relationship of cancer risk for a child who had exposure in utero is reasonably well-established. It was first shown by Alice Stewart and her colleagues in 1956.¹⁵ The United Nations Scientific Committee on the Effects of Atomic Radiation reported in 1996 that maternal exposure increased leukemia risk in children that were statistically significant and that the risk of solid cancers increased about 40 percent when mothers were exposed during pregnancy to doses in the 10 to 20 milligray (mGy) range.¹⁶ Exposures in this range and much higher exposures as well have occurred often in nuclear weapons testing, and the bombings of Hiroshima and Nagasaki, among others.

For instance, at least 544 women who were pregnant at the time of the atomic bombings of Hiroshima and Nagasaki who survived those bombings and their immediate deadly aftermath, were exposed to more than 10 mGy of radiation. Of them 169 are estimated to have received exposures between 100 mGy and 500 mGy and 19 had exposures more than 1,000 mGy.¹⁷

Exposures on Rongelap Atoll in the Marshall Islands from the BRAVO test fallout alone were hundreds times higher than the 10 to 20 mGy range. They were high enough to produce somatic effects. Women who were pregnant lost their pregnancies, among other impacts. Adults on Utrik Atoll, which is much farther from Bikini than is Rongelap, received an estimated 110 mGy of external dose and 76 mGy of internal effective dose equivalent (EDE) for a total of 186 mGy, according to U.S. government estimates of the impact there of the 15-megaton Bravo test on 1 March 1954. An independent estimate places the external dose at more than 500 mGy and the Effective Dose Equivalent (EDE) at 860 mGy, for a total of more than 1,360 mGy – more than seven times higher than the government estimate. Fallout-related thyroid doses due to the Bravo test were also very high. The official thyroid dose estimate for Utrik adults was 1,550 mGy; the independent estimate was over 17 times higher at 27,000 mGy.¹⁸

These high doses overall and the very high thyroid doses are significant not only for cancer risk for those exposed but also for multigenerational impacts. Exposures to people living near the Semipalatinsk Soviet test site were also high, An official estimate of average exposures to over 6,000 people in nearby villages was 400 mSv. An independent researcher estimated that 30,000 to 40,000 people averaged 1,600mSv, with even higher does for nearby villagers.¹⁹

The rest of this chapter will focus on teratogenic impacts – loss of pregnancy and risks of malformations during the first two trimesters for surviving fetuses.

¹⁵ Stewart et al. 1956.

¹⁶ As cited in BEIR VII, 2006, p. 173. In the case of gamma ray exposure, one gray (Gy) can be considered the equivalent of 1 sievert (Sv) in terms of cancer risk effectiveness. Grays measure the energy deposited in tissue. The value of exposure in sieverts is derived from grays by multiplying by an “relative biological effectiveness” (RBE) factor that represents relative harm of different types of radiation for the same amount of energy deposited. For instance, an RBE factor of 20 is used for alpha radiation to derive the exposure in Sv from the dose in Gy, generally in the context of cancer risk. International units are used throughout this paper. Conversion factors for a variety of units may be found on the “Classroom” webpage of the Institute for Energy and Environmental Research at <https://ieer.org/resource/classroom/converting-units/>

¹⁷ ICRP 49, 1986, Table 1, p. 21.

¹⁸ Franke 2002, p. 39. Makhijani 2022a is a summary of impacts of testing on the Marshall Islands. It summarizes the impacts as well as health and remediation issues faced by the Marshallese people.

¹⁹ Makhijani 2022b summarizes Semipalatinsk exposures.

a. Early failed pregnancies (early miscarriages)

When an ovum is initially fertilized to form a zygote, it divides rapidly for a few days, after which some cell differentiation occurs and a blastocyst is formed. The blastocyst must successfully implant itself on the uterine wall, which usually occurs six or seven days after fertilization; it takes a few days for implantation to be complete.²⁰ The initial embryonic stage of pregnancy lasts for first eight weeks of pregnancy.

An early failed pregnancy, or early miscarriage, sometimes also called a “chemical pregnancy,” belongs in the category of involuntary failed pregnancies known as “spontaneous abortions.” Many such failures often occur so early in the pregnancy that most women may not realize they are pregnant.²¹ There is general agreement that the number of such early “occult” miscarriages is large, but estimates of the proportion of pregnancies that fail in the first few weeks vary a great deal. The Cleveland Clinic estimates that about one fifth of pregnancies end early enough – in five weeks or less – to be called “chemical pregnancies”²²; this estimate includes both recognized and unrecognized pregnancies but covers only the first five weeks.

Genetic and chromosomal abnormalities are among the causes of early failed pregnancies; exposure to environmental toxins, including radiation, is one cause. The 1988 National Academies report on exposure to radiation (commonly known as the BEIR IV report) noted the following in regard to external radiation impact:

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.²³

The official estimate of Bravo fallout doses to adults on Utrik was 186 mGy, almost double the above threshold of 100 mGy (10 rad) estimated in the BEIR IV report. An independent estimate of 1,360 mGy is almost 14 times higher. The people of Rongelap suffered even higher doses. Therefore, according to this BEIR IV analysis, there would be a considerable risk of “preimplantation teratogenesis” – early failed pregnancies.

²⁰ The description of normal pregnancy development is from Artal-Mittelmark 2021.

²¹ BEIR IV 1988, pp. 381-382. In this paper, length of the pregnancy is counted from the time of fertilization (though it is also common to count it from the date of the last period).

²² Cleveland Clinic 2021. The Clinic estimates that about one-fourth of pregnancies end in miscarriages by about 20 weeks and that 80 percent of them are chemical pregnancies – that is miscarriages in the first five weeks.

²³ BEIR IV 1988, pp. 381-382, italics added. “Low-LET radiation” is radiation that has low “linear energy transfer.” Such radiation typically deposits energy over relatively long tracks spreading damage over many cells. “High-LET radiation,” in contrast, deposits energy in a relatively small volume, causing focused impacts. Among other things the rate of energy transfer affects the chances of repair of the damage caused by radiation; high-LET radiation damage typically has less chance of completely successful repair and greater chances of cell-death or mis-repaired damage.

As a non-stochastic effect, early pregnancy failure stands in contrast to cancer, which is a stochastic effect. The latter is a multi-stage process where the effect is delayed from the initial triggering event (like radiation exposure) and may require additional events or exposures to actualize the harm. The vast majority of cancers caused by radiation exposure have long latency periods – extending to years, even decades. This multi-stage, multi-year process makes it difficult, and in most cases, impossible to link a specific cancer with a particular exposure or event. In contrast, radiation exposure that results in preimplantation loss of pregnancy is a short-term impact that should, in principle, be able to be linked to the exposure; it is an adverse outcome that requires no other exposure or triggering event. However, it is also difficult to detect, though for different reasons.

The main difficulty with assessing early pregnancy failure risk due to radiation exposure is that many women who experience such pregnancy loss do not even know they are pregnant; in addition, there may be a complex of environmental and genetic factors contributing to an early failed pregnancy. Indeed, the two may interact.

There are known risk factors; there are also suspected risk factors where more research is needed. For instance, comparative studies could be done in places where some women in well-defined populations had much higher exposures than others – such as in the Marshall Islands. While people all over the Marshall Islands were exposed to radiation by U.S. testing, there was a significant variation in exposures across the country. Pregnancy loss has been a common complaint. A 1987 survey of Marshallese women, though not definitive, is indicative of considerable increase on adverse pregnancy outcomes (including miscarriages) after the start of U.S. thermonuclear bomb testing in their country.²⁴ The report of the Special Rapporteur for the United Nations Human Rights Council whose charge included investigating the impacts of U.S. nuclear weapons testing on the Marshall Islands summarized the experiences of women as follows:

30. The Special Rapporteur heard compelling testimony by women on their experience of returning from Rongelap Atoll, including on the alarmingly high rates of stillbirths, miscarriages, congenital birth defects and reproductive problems (such as changes in menstrual cycles and the subsequent inability to conceive, even in those who previously had no such difficulties). Some gave birth to babies that ultimately died from foetal disorders, and they still endured the shame and trauma they experienced as a result...²⁵

Similarly, radiation doses experienced by thousands to hundreds of thousands of people downwind from the Semipalatinsk test site in Kazakhstan were high enough to have caused early failed pregnancies.²⁶ Despite the documented doses in a range that has been acknowledged to cause early failed pregnancies, there is as yet no comprehensive account or estimate of still births, miscarriages, and other reproductive difficulties experienced by those directly impacted by nuclear weapon testing. The same is the case with uranium mining and the vast wastes that it has created scattered all over the

²⁴ Makhijani 2025, Table 5-1.

²⁵ Georgescu 2012, paragraph 30. See also Barker 2013 for many tragic examples provided by Marshallese testimonies.

²⁶ Various estimates of doses from atmospheric testing by the former Soviet Union at the Semipalatinsk test site are summarized in Makhijani 2022b.

world, and especially in indigenous lands, such as in the Navajo Nation in the United States (see Chapter VI below).

The high thyroid doses should especially be noted in the context of early failed pregnancies. Iodine-131, a major fission product, goes preferentially to the thyroid, which uses ordinary, non-radioactive iodine for hormone synthesis. Iodine-131 and other radioactive iodine isotopes damage and destroy thyroid cells. Hypothyroidism is one result; it is a cause of miscarriages.²⁷

Numerous studies have demonstrated a link between hypothyroidism and an increased risk of miscarriage, particularly in the first trimester. Thyroid hormones play a critical role in maintaining the delicate balance of hormones necessary to support pregnancy. If thyroid hormone levels are insufficient, implantation and early fetal development can be disrupted, increasing the risk of early pregnancy loss.

There is semi-quantitative data on failed pregnancies in the aftermath of the atomic bombings of Hiroshima and Nagasaki in the medical survey done by the United States Strategic Bombing Survey, published in March 1947. Essentially all women within 900 meters of the hypocenter “have had miscarriages.”²⁸ However, the vast majority of people within this radius died; any survivors would have had severe physical injuries and emotional shock. Hence, it is difficult to know what part of this loss may be attributable to radiation, though the doses were high – greater than 2 Gy.²⁹

Between 900 meters and 2 kilometers “they have had miscarriages or premature infants who died shortly after birth.”³⁰ The fraction of pregnancy loss is not mentioned. The casualties and injuries in this area were also severe; the radiation doses were between a few tens of milligray and about 1 gray.³¹ There were also injuries in this area from flash burns, flying debris, broken glass, and collapsed houses. Severe pregnancy losses were also found for those between two and three kilometers of the hypocenter:³²

In the group of pregnant women between 6,500 and 10,000 feet [from the hypocenter] who could be traced, about one-third have given birth to apparently normal children. The remainder had not reached the term of their pregnancy or their pregnancy had terminated prematurely. No definite effects attributable to the bomb have been seen in those women. Records of the Hiroshima Prefectural Health Department revealed that 2 months after the explosion of the bomb the incidence of miscarriages, abortions and premature births for the entire city, without consideration of whether the women were even in the city at the time of the bombing, was 27 percent as compared with a rate of about 6 percent prior to the bombing. Other factors such as malnutrition, emotional disturbances and poor living conditions may play a large part in this increase. As a matter of fact, there is no concrete evidence upon which one can say that radiation alone played any definite part.

²⁷ California Center for Reproductive Health 2025.

²⁸ U.S. Strategic Bombing Survey 1947, p. 53.

²⁹ Normile 2020, map on page 324.

³⁰ U.S. Strategic Bombing Survey 1947, p. 53.

³¹ Normile 2020, map on page 324.

³² U.S. Strategic Bombing Survey 1947, p. 53

This author is not aware of any detailed analysis of these severe pregnancy losses and premature births, including what may be attributable even partially to radiation exposure. Suffice it to note here that in the two-to-three kilometer radius band the rate of pregnancy loss was two-thirds, compared to just above one-fourth (27 percent) in the set of women “in the entire city”. Thus, there was a significant impact due to the bombings, but the part attributable to radiation has not been isolated.

b. Teratogenic impacts on the central nervous system

Central nervous system damage as a result of exposure to radiation by the embryo/fetus from about the eighth week of pregnancy to about the twenty-fifth week has been established by follow-up of women who were pregnant at the time of the atomic bombings of Hiroshima and Nagasaki in August 1945. The impacts were described in a 1986 report of the International Commission on Radiological Protection, 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.*³³

The term “severe mental retardation” was used in ICRP 49 in its clinical sense as described in the above quote to describe someone who was “unable to form simple sentences, to solve simple problems in arithmetic, to care for himself or herself, or is (was) unmanageable or institutionalized.”

ICRP 49 also concluded that “[w]ithin the period of maximum vulnerability [8 to 15 weeks], the simplest statistical model consistent with the data appears to be a linear one without threshold.”³⁴ This kind of risk curve is a straight line that has zero risk only when there is no exposure and is called the a “linear no-threshold” risk profile. For this particular level, the risk of occurrence was estimated at one case in a population per 2.5 Gy of cumulative population exposure.³⁵ This specific impact was observed for exposures that occurred in women who were between seven and 25 weeks pregnant at the time of the bombings, and not before or after that interval.

ICRP 90, published in 2003, reanalyzed the Hiroshima-Nagasaki data and concluded that there was a threshold of 0.3 Gy for severe mental retardation. However, the re-analysis raises a number of questions, including omission of previously included cases and exclusion of new known cases.³⁶ There

³³ ICRP 49, 1986, p. 20; italics added. International units added in square brackets.

³⁴ ICRP 49, p. 31.

³⁵ ICRP 49, 1986, p. 20, paragraph 52, which states the risk as being “about 0.4 Gy⁻¹ with an estimated standard error of about 0.09 Gy⁻¹.” The symbol “Gy⁻¹” means per gray of exposure.

³⁶ ICRP 90 Chapter 5. It reframed the data with some assumptions that appear questionable, at best, to this author. For example, it omitted a case of Down’s syndrome as “unrelated to -in-utero radiation” despite the evidence,

are, in addition, general issues with the Hiroshima-Nagasaki cohort that may particularly impact analysis of severe mental retardation due to in-utero radiation, potentially biasing it. The cohort was established in 1950 from survivors who were still living in the two cities. As a result, women who had children with severe teratogenic impacts (including severe mental retardation) but died before 1950, were not included in the cohort. The women who may have died post-delivery but before 1950 were also not included. No attempt to address this likely source of bias was made in either ICRP report.

The potential bias in the Hiroshima Nagasaki in utero cohort is discussed in Section II.e below. Suffice it to note here that (i) a more careful re-analysis is needed and (ii) there is evidence for reproductive harm at low- or no-threshold in experiments on laboratory animals, though it should also be noted that it is not specific to neurological harm (see Chapter V).

Higher rates of microcephaly (small brain size) and neural tube defects have also been found in the areas that had higher contamination as a result of the 1986 Chernobyl nuclear power plant accident.³⁷

Radiation also impacts other organs, which form during the first trimester of pregnancy. Specifically, ionizing radiation is a teratogen^{38,39} – an agent that can cause malformations, especially but not only during the most sensitive periods of organ development. That certain levels of radiation exposure can cause malformations in the early part of pregnancy, as was recognized in the BEIR IV report of the National Academies based on animal experiments and theoretical considerations:

During the major organogenesis stage, 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 [100 milligray] of low-LET radiation* appear ineffective.⁴⁰

It is important to note three things about the BEIR IV conclusion:

- Malformations can occur as a result of exposure to radiation;

published in the Lancet in 1968, that maternal radiation exposure increase the risk (Uchida, Holunga, and Lawler 1968; Davidson 2013). It excluded four other cases as well. While reducing the ICRP 49 number from 30 to 25, it failed to include five known cases born to exposed women who were in Hiroshima and Nagasaki but were not living there in 1950, when the cohort was established. The establishment of the cohort with only those living in the two cities in 1950 poses a number of issues not easily addressed. But the presence of five acknowledged additional cases should at least have been discussed in terms of its potential impact on the revised conclusion that the new analysis indicated a threshold of 0.3 Gy for severe mental retardation. As a final example for this report, ICRP 90 acknowledges that uterine doses may be overestimated in the first 20 weeks of pregnancy. Rather than taking this factor into account (which could result in a higher risk factor for the cases included), ICRP 90 opines that the uncertainties involved were “probably much smaller than those associated with geographic locations and shielding configurations.” (p. 104) Finally, it should be noted that the children who had severe teratogenic impacts, including severe mental retardation (as defined in ICRP 49) but died before 1950 were not included in the cohort. No attempt to interview a sample surviving women or their physicians was described. See Section II.e for further discussion about the 1950 cohort.

³⁷ Wertelecki et al. 2014.

³⁸ BEIR IV 1998, p. 383

³⁹ Brent and Beckman 1990.

⁴⁰ BEIR IV 1988, p. 383, italics added.

- The hypothesis that there is a threshold for these impacts is based on external radiation experiments on animals and on theoretical considerations;
- The BEIR IV hypothesis is that observable teratogenic harm would occur only in a small window of a few days of organ development (with the exception of the central nervous system).

Organ development normally extends beyond the few days that the BEIR IV report considered to be the “sensitive period.” The Society for Birth Defects Research & Prevention (formerly known as the Teratology Society) recognizes that the stage of gestation at which exposure occurs is critically important; yet, impacts can occur over a longer period of the pregnancy outside the major period of from 14 to 50 days of organogenesis, discussed in the BEIR IV report.⁴¹ Specifically, exposure in the early and mid-embryonic period “often results in major structural anomalies”; disruption of development beyond that, including into the fetal period, can cause damage in the form of “abnormal organ differentiation” and other problems:⁴²

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.

c. Teratogenic impacts and thresholds

Internal radiation exposure occurs when radioactive materials are literally incorporated into our bodies by inhalation, ingestion, via cuts and wounds, and, in some cases, absorption from the skin. Some radioactive materials are only very briefly inside the body – tritium, radioactive hydrogen, when it is in the form of a gas, is an example. It is breathed in and breathed out with minimal radiation exposure. However, being hydrogen, tritium is oxidized into water; this radioactive water behaves like all other water in the body;⁴³ once in, it pervades the body. It also crosses the placenta and impacts the fetus. As another example, strontium-90, a fission product, behaves like calcium, and selectively impacts bones, including the bone marrow where among other things, stem cells of the immune system are formed.

⁴¹ BEIR IV 1988, pp. 383-384.

⁴² Bleyl and Schoenwolf 2018, italics added.

⁴³ There are some differences in chemistry since tritium atoms are three times the mass of ordinary hydrogen, but these are generally very small. This is because even at high concentrations of HTO in water, such as 1 mCi/ml (millicurie per milliliter), which is 5 million times the U.S. drinking water standard, there is only one tritium atom for every three million ordinary hydrogen atoms in the water (Dobson 1982, p. 4).

The thresholds postulated for early failed pregnancies and teratogenic impacts other than those to the central nervous system are based mainly on external radiation experiments. It is widely accepted that certain kinds of radiation, notably alpha radiation (which consists of an energetic nucleus of helium-4), are far more damaging per unit of radiation energy deposited in the body. The ratio of postulated harm, or health impact, per unit of energy deposited in the body for a specific type of radiation relative to high energy gamma radiation is called “relative biological effectiveness” (RBE) – with the term “effectiveness” referring to the ability to cause harm.

BEIR IV postulated, based on in-vitro cell-killing experiments with alpha radiation, that an RBE factor of 10 should be applied to the postulated threshold for teratogenic damage, including organ malformations and early failed pregnancies, if exposure was to alpha radiation. Thus, an internal exposure of 10 millisieverts would constitute the threshold for teratogenic impact when it concerned alpha radiation, such as that from plutonium-239 or the three naturally occurring uranium isotopes, U-234, U-235, and U-238.⁴⁴

Another important question relates to what the threshold for teratogenic impacts might be. Besides being based on external radiation experiments, the notion that there is a threshold for other teratogenic impacts also rests on the assumption, expressed explicitly in BEIR IV, that, unlike the central nervous system, the sensitive period for malformations for other organs is only “a few days.”⁴⁵

There are critical questions about the interpretation of laboratory data obtained from animal experiments as it concerns risk for human beings, including:

- How do laboratory experiments on animals relate to risks of harm to animals in the real life?
- What relative biological effectiveness factor should be used when converting external radiation experiment data to the internal exposures, notably those where radiation energy is deposited in a small volume? This is a characteristic of alpha radiation; it is called “high linear energy transfer radiation” (“high-LET radiation”).

In regard to the first question, real-life response of animals in the Chernobyl exclusion zone in terms of genetic harm to radiation has been shown to be more complex than that observed laboratory experiments.⁴⁶ Hence, there is need for caution in assuming that the thresholds observed in laboratory experiments would hold in real life.

In regard to the second question of high-LET radiation, a considerable amount of research indicates that when a large amount of radiation energy is deposited in a small volume, the relative biological

⁴⁴ 10 mSv is derived from the postulated 100 mGy threshold since a sievert is defined as a gray (which measures energy deposited) multiplied by the relative biological effectiveness, which in this case is assumed to be 10. U-234 is a decay product of U-238 and occurs in a fixed ratio to U-238 in natural uranium. When uranium is “enriched” in U-235, the lighter U-234 also enriches preferentially in the U-235 stream. This detail is important because U-234, though present in trace amounts in terms of mass, is much more radioactive per unit mass, than the other two naturally occurring uranium isotopes. U-238 delivers the main uranium dose when the uranium is depleted, while U-234 delivers the main dose when the uranium is enriched.

⁴⁵ BEIR IV 1988, p. 383

⁴⁶ Wildlife in the Chernobyl exclusion zone seems to be much more sensitive to ionizing radiation than estimated in laboratory experiments. Garnier-Laplace et al. 2013

effectiveness factor for certain types of genetic damage may be far higher than 10, assumed for cell death, or even 20, assumed for cancer risk.

For instance, Nagasawa and Little subjected Chinese hamster ovary cells to a rather high level of x-rays of 1 to 2 grays and examined genetic damage as indicated by the induction of sister chromatid exchange. They then examined the sister chromatid exchange frequency with internal alpha radiation from plutonium-238. Their conclusion was that only 0.31 milligray of internal alpha radiation produced the same level of genetic damage as 1 to 2 grays of external radiation.⁴⁷ In other words, it took 3,200 to 6,400 times less alpha radiation to produce this specific kind of genetic damage as external x-rays. Thus, for this particular genetic endpoint, the relative biological effectiveness factor would be in the range of 3,200 to 6,400 instead of 20 or 10 postulated for other impacts – cancer risk or cell-killing respectively (the latter in laboratory experiments).

The matter is even more complex, because the Pu-238 alpha radiation produced damage in cells that were not directly radiated but were near such cells. The Nagasawa-Little research found that more than 30 “bystander” cells showed increased sister chromatid exchange frequency for every cell that was actually traversed by an alpha particle.

Khadim et al. found even more dramatic results in their experiment with hematopoietic clonal cells. The cells selected were clonal descendants of cells that had “survived the passage of one or more radiation tracks before the initiation of clonal proliferation.” In other words, this endpoint was for genetic damage of the descendants of the cells that survived alpha radiation. Khadim et al. observed “a high frequency of non-clonal aberrations in the clonal descendants compatible with α -emitters inducing lesions in stem cells that result in the transmission of chromosomal instability to their progeny.” The non-clonal aberrations did not occur when x-rays were the source of the radiation dose.⁴⁸

It is difficult to extrapolate from these in vitro laboratory experiments to human beings or even mammals. However, they point to the potential for a much higher RBE due to high-LET radiation for specific types of genetic damage. In other words, while the cell-death endpoint may have a relative biological effectiveness factor of 10 for high-LET radiation, the RBE may be much higher for other endpoints, including for the progeny of surviving damaged cells. Further, the irradiation of cells in adults whose nuclear DNA has significant repair capabilities – a feature of homeostatic functioning – must be distinguished from the response of the embryo or fetus, whose cells are dividing rapidly and do not have that capacity for repair.

These observations do not resolve the question of thresholds for teratogenic impacts but they do indicate that, if there are thresholds, they may vary by radionuclide and by the specific type of impact under consideration and that they may be considerably lower for some impacts than indicated by laboratory experiments on animals exposed to external gamma radiation.

There is also evidence that a longer period of vulnerability than exposure between eight and 25 weeks should be considered, including specifically for nervous system harm. Serious teratogenic impacts can occur earlier, in the third and fourth weeks, if neural tubes fail to close,⁴⁹ resulting in problems such as a failure of the brain to develop or incomplete brain development. These malformations go under the

⁴⁷ Nagasawa and Little 1992, p. 970

⁴⁸ Khadim et al. 1992.

⁴⁹ ICRP 49 1986, p. 30.

rubric of “anencephaly”, resulting in miscarriages. In addition, if the fetuses survive to birth they “die very soon after birth.”⁵⁰ The brain continues to develop throughout the pregnancy. The prefrontal cortex, essential for cognition continues to develop after birth, by “differentiation of its neurons and development of synaptic connections.”⁵¹ Thus, a broader view of brain and neuronal vulnerability than that expressed in ICRP 49 is warranted.

d. Impact processes

Besides external radiation exposure, exemplified by gamma radiation exposure during the bombings of Hiroshima and Nagasaki, exposure during pregnancy also occurs due to internal radiation, which results when the mother has internal radionuclide burdens. The exposure of the embryo and the fetus depends on many factors, including the stage of pregnancy at which the exposure occurs, the specific radionuclide involved, which determines the type of radiation (alpha, consisting of energetic helium-4 nuclei); beta, electrons or positrons; and gamma, photons), and the specific element, which determines the ratio of the concentration of the radionuclide in the fetus to that in the mother.⁵² Exposure prior to pregnancy also impacts the fetus, though the transfer ratios may be different. Table III-1 shows the fetal to maternal radionuclide ratios for many elements that have radioisotopes.

Table III-1: Fetal to maternal radionuclide ratios for intakes before and during pregnancy

Element	Intakes prior to Pregnancy	Intakes during Pregnancy
H in HTO	1.6	1.6
H in OBT	1.6	1.6
Organic carbon	1.5	1.5
Phosphorus	0.5	10
Sulphur	1	2
Potassium	1	1
Cobalt	0.2	1
Zinc	2	2
Technetium	1	1
Ruthenium	0.01	0.2
Cesium	1	1
Lead	1	1
Bismuth	0.1	0.1

⁵⁰ Burke et al. 2009, p. 6.

⁵¹ Kolk and Rakic 2022.

⁵² Exposure to neutrons results once they are inside the body. Apart from rare spontaneous fission neutrons, emitted, for example by some plutonium-240 and uranium-238 decays, neutrons are created by fission events externally, penetrating the body, where they create indirect ionization events. BEIR VII,

Thorium (see note)	0.03	0.1;0.3;1
Uranium	0.1	1
Plutonium (see Note)	0.03	0.1;0.3;1
Americium	0.01	0.1

Source: NRPB 2001, Table 1, as compiled in Makhijani 2023.

Note: The three ratios shown for thorium and plutonium are for intakes during the first, second, and third trimesters.

The development of ovaries and the oocytes in female fetuses can also be affected by radionuclides that cross the placenta. For instance, tritiated water and organically bound tritium can become part of the oocytes, thus creating locations within those cells that will be impacted when the tritium decays and emits a beta particle. As shown in Table III-1 above, even radionuclides that are present in a woman's body before she becomes pregnant can affect the embryo and fetus after she becomes pregnant, with the caveat that pregnancy must be within 10 biological half-lives of the pre-pregnancy exposure. Fetal to maternal plutonium and thorium concentration ratios vary substantially during pregnancy. The same is true of iodine isotopes, which are higher in the fetus than in the mother throughout pregnancy.⁵³

Other radionuclides of importance, include short-lived fission products, notably iodine-131, for women exposed during pregnancy or for women exposed to fallout prior to pregnancy to an extent that their thyroids were compromised. In the case of marine environments, exposure to zinc-65, which is an activation product, is also important.⁵⁴

All people have radionuclides in them as a result of the dispersal and persistence of nuclear-weapons-related long-lived radionuclides, including carbon-14 (half-life: 5,730 years) and plutonium-239 (half-life: 24,110 years). The long half-lives mean that the problem of low individual exposures but large population exposures will persist for thousands of years.⁵⁵ Tritium from nuclear weapons testing will continue its impacts well into the 21st century, even presuming no further nuclear weapons testing or use; tritium impacts from nuclear power plants and tritium production for nuclear weapons will continue, so long as these activities continue.

Most nuclear DNA mutations are somatic mutations – that is, mutations in cells other than germline cells (ova and sperm) that are the basis for reproduction. Somatic mutations can lead to diseases such as cancer. Exposure to diagnostic x-rays during pregnancy, for instance, has been shown to lead to an increase in cancer in childhood.⁵⁶

A special kind of risk of multigenerational may occur when radiation exposure of oocytes causes mutations when ovaries are being formed in female fetuses. Mutations can also occur in ova during a female's fertile lifetime and also in sperm, which are formed post-puberty in males. The resultant mutations are called *de novo germline mutations*. However, deleterious impact of such germline mutations on subsequent generations has not been established either in humans or in animals. The evidence is contradictory. For instance, transgenerational impacts due to germline mutations were not

⁵³ Sikov and Hui 1996.

⁵⁴ Franke 2002. Also see Chapters IV and VI below.

⁵⁵ IPPNW and IEER 1991, Table 3 (to the end of the 21st century) and Table 4 (for longer time frames), pp. 37-38.

⁵⁶ This was first discussed in Stewart et al. 1956.

found in the children of Chernobyl cleanup workers,⁵⁷ or in nematodes collected in the Chernobyl exclusion zone.⁵⁸ In contrast, mutations “partly of germline origin” appear to have contributed albinism in Chernobyl barn swallows, which had germline mutation rates “two- to tenfold higher than in birds from control areas in Ukraine and Italy.”^{59,60} In contrast, the harmful impact of somatic genetic changes has been repeatedly demonstrated in the aftermath of Chernobyl in various studies, including a major meta-analysis.⁶¹ Of course, excess cancer rates in the Hiroshima-Nagasaki cohort and much other evidence provides clear evidence of the deleterious impact of somatic mutations in nuclear DNA.

Mutations also occur in mitochondrial DNA; as is now well-recognized, mitochondrial DNA is a maternal inheritance; sperm have mitochondria that provide the energy for them to move and reach the ovum for possible fertilization; but they are eliminated after that. Mutations in mitochondrial DNA can also affect sperm and hence male fertility.⁶²

Further, mitochondria are coming to be recognized not only as “the powerhouse of cell[s]” but also as “the powerhouse of immunity.” regard to other diseases, mitochondrial DNA damage may not only harm the energy system of the body, but also the immune system.

Metabolic events in mitochondria are also proving to have profound effects on immunity. The major function of mitochondria is to generate ATP through the process of oxidative phosphorylation (OXPHOS)...Glycolysis, which converts glucose to pyruvate and then generates lactate, was historically shown to occur during hypoxia....Both glycolysis and mitochondrial metabolism have an effect on the immune response.⁶³

Damage to mitochondrial function, such as that caused by oxidative stress, can therefore harm both energy production and immune response (while also promoting other pathologies, such as neurodegenerative diseases⁶⁴).

A few examples of how radiation exposure might occur that could lead to the processes discussed above will illustrate the risks in practice. Many people throughout the world, particularly in areas of high fallout during atmospheric weapons testing, had significant intakes of radionuclides. Smaller intakes continue throughout the world. For example, 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 other pathways, such as 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

⁵⁷ Yeager et al. 2021. They have also not been found in the 1950 Hiroshima-Nagasaki cohort, which is discussed in Section II.e below.

⁵⁸ Tintori et al. 2024.

⁵⁹ Ellegren et al. 1997.

⁶⁰ There is still much to be understood regarding the multigenerational impact of germline mutations. One possible explanation is that healthy ova are preferentially retained and released for potential fertilization. About seven million oocytes are created during the formation of ovaries in the female fetus. The number declines during childhood. Only about 400, less than 0.01 percent of oocytes created, are ever released as mature ova for potential fertilization over a woman’s lifetime. *Developmental Biology* 2000.

⁶¹ Møller and Mousseau 2015.

⁶² Venkatesh et al. 2009

⁶³ Mills, Kelly, and O’Neill 2017.

⁶⁴ Wen et al. 2025.

exposure; yet, they have generally not been given their due in impact assessment. 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 hot spots thousands of miles from the Bikini and Enewetak test locations.⁶⁵ Medical exposures, exposures due to nuclear power plant accidents, and smaller routine radionuclide emissions and discharges from routine power plant operation also contribute to radiation doses. Whether the lower levels of doses result in multi-generation impacts is a matter of some controversy, a problem which has been compounded by the relative neglect of research on multigenerational issues, despite early recognition of the potential harm.

e. Hiroshima-Nagasaki survivor data⁶⁶

While the deaths and harm to health began at the moments of the bombings of Hiroshima and Nagasaki, a formal cohort for epidemiological study was not established until more than five years later, on 1 October 1950. Yet, both U.S. and Japanese medical personnel had visited the cities for the express purpose of assessing the various types of damage, including to health. The Japanese National Research Council started collecting clinical data soon after the bombings, but the data was “largely suppressed from publication by the [U.S.] Occupation.”⁶⁷

Starting in September 1945, the United States Strategic Bombing Survey made a detailed assessment of the medical situation that ranged from evaluating damage to medical facilities, to sanitary conditions, to the nature of atomic bomb casualties. The report was completed in March 1947. The U.S. Army and Navy also had their assessment teams in the two cities shortly after the bombings, as did the British government.⁶⁸ The Atomic Bomb Casualty Commission was established in late 1946; it was later named the Radiation Effects Research Foundation (RERF), the official joint U.S.-Japan body to gather and analyze the Hiroshima and Nagasaki data.

The RERF has described the selection of the initial cohort, called the “Life Span Study (LSS) Cohort”, as follows:⁶⁹

- Of the 284,000 survivors only about 200,000 people with permanent addresses in the two cities were considered eligible for inclusion. As a result, 84,000 people who were in the cities at the time of bombings were automatically excluded, apparently without a survey as to the representativeness of the eligible group.
- 99,382 survivors were selected from among the 200,000 with Hiroshima-Nagasaki addresses, including:

⁶⁵ For instance, there was a hot spot in Colombo, Sri Lanka and one in Mexico City. See the map in List 1955, pdf pp. 25 and 26

⁶⁶ It is clear, even from this brief account, that there are many unresolved issues relating to the cohort. This section does not cover many of them. It is oriented to those issues that appear to have a significant potential impact on the outcomes of the analyses of the data so far as multigenerational impacts are concerned. Even on that account, this analysis must be considered preliminary, especially as an exhaustive analysis of the issues associated with the cohort are well beyond the scope of this report.

⁶⁷ Putnam 1998.

⁶⁸ U.S. Strategic Bombing Survey 1947.

⁶⁹ RERF 2014, pp. 5-6.

- “(1) all survivors who were within 2,000 meters of either hypocenter at the time of the bombings (proximally exposed);”
- “(2) all survivors who were at 2,000–2,499 meters (semi-proximally exposed);”
- “(3) a sample of survivors at 2,500–9,999 meters, matched to group (1) by sex and age (distally exposed); and”
- “(4) a sample of persons, age- and sex-matched to group (1) who were at least 10,000 meters from the hypocenter.”

As a result of excluding those without permanent addresses in the two cities, “9,530 survivors located less than 2,500 meters from either hypocenter at the time of the bombings whose permanent domicile was not in either city of Hiroshima or Nagasaki” were excluded. They were retrospectively added in 1968. An additional “11,409 distally exposed survivors in Nagasaki” were added on 1980, making for an eventual total of 120,321 people.⁷⁰

The LSS cohort is well-known as one of the principal datasets for cancer risk estimation. For instance, the BEIR VII report states “The Life Span Study (LSS) cohort of survivors of the atomic bombings in Hiroshima and Nagasaki continues to serve as a major source of information for evaluating health risks from exposure to ionizing radiation and particularly for developing quantitative estimates of risk.” Further “...the LSS cohort of survivors of the atomic bombings in Hiroshima and Nagasaki plays a principal role in the committee’s development of cancer risk estimates.”⁷¹

Risks of genetically inherited diseases, including those thought to have fully genetic causes and those that are “multi-factorial”, also rely significantly on this dataset. As a result, the formation of the cohort and any assumptions about its composition are especially important to radiation risk estimation and to radiation protection. A central assumption was that the LSS cohort was representative in every respect, except radiation exposure. This assumption of the homogeneity of the cohort is important to all its findings. In the context of the present report, we note the following conclusion about the children of most exposed survivors:

[T]he results of the extensive genetic epidemiologic studies of A-bomb survivors in Japan have shown no adverse effects in the progeny that could be attributed to the radiation exposures (of the order of 0.4 Sv) sustained by most survivors.⁷²

In the 1980s and 1990s, Alice Stewart and George Kneale, examined “whether the people who survived, in spite of bomb-related injuries, are homogeneous in respect of variation of cancer risk with survivors without such injuries.”⁷³ The LSS cohort assumed age distribution homogeneity. The Stewart and Kneale statistical analysis of the data indicated it was heterogenous in ways that impacted the analysis of cancer risk. Specifically, Stewart and Kneale found that among those with high exposures (more than 0.5 Gy), those who were less than 10 years old or more than 50 years old at the time of the bombings were under-represented in the cohort. As a result, those who were between 10 to 50 years and highly exposed were overrepresented.

⁷⁰ RERF 2014, pp. 5-6.

⁷¹ BEIR VII 2006, p. 12 and p. 14.

⁷² BEIR VII, 2006, p. 252.

⁷³ Stewart and Kneale 2000.

This finding makes it reasonable to assume that, in the LSS cohort, there was under-representation of persons who (by virtue of their age in 1945 and their exposure positions) were most at risk of dying from radiogenic and nonradiogenic cancers during the next 20 or 30 y. Together with the earlier finding of a positive dose trend for noncancer deaths at high dose levels, the new finding makes it probable that some of the cancers currently ascribed to mutational effects of the radiation were actually the result of defective immune responses or cancer promotion effects of marrow damage.⁷⁴

They concluded that the “wide diversity in radiosensitivity” among large numbers of people may well be the result of “immune system control of radiosensitivity as well as [of] infection sensitivity.” In other words, some diseases involving immune system compromise might be incorrectly attributed to radiation exposure alone.

The Stewart and Kneale finding that the cohort was heterogenous in that highly exposed young (under 10 years) and old (over 50 years) people were under-represented raises the question whether the cohort may be similarly unrepresentative in other respects. If so, that would impact findings on teratogenic impacts of in utero exposure and other harm that might propagate across the generations. While a detailed analysis is beyond the scope of this report, available data indicate that this may well be the case.

The RERF also had an in utero cohort consisting of “children who were born during the period from the dates of the atomic bombings (August 6 in Hiroshima and August 9 in Nagasaki) to May 31, 1946.”⁷⁵ Stewart and Kneale noted that there was an underrepresentation in this cohort of those who had “exposures before 8 weeks of fetal age.”⁷⁶ This is a period when early failed pregnancies as well as many teratogenic impacts may be expected. Secondly, as noted above, there were very high rates of pregnancy loss, semi-quantitatively related to distance from the Hiroshima hypocenter. Pregnancy loss was near-total in the area up to 900 meters and severe in the 900-meter to 2-kilometer band, where there were “miscarriages or premature infants who died shortly after birth.”⁷⁷

Pregnancy loss was still very high in the 2- to 3-kilometer band, where about two-thirds of the pregnancies were lost, as compared to 27 percent in the post-bombing period in Hiroshima as a whole. There was an estimated 6 percent loss rate in the period prior to the bombings.⁷⁸ The city-wide post-bombing increase appears to be due to a variety of causes. For instance, the U.S. Strategic Bombing Survey reported “high mortality from diarrhea in children under 6 to 7 years of age.” As another example, “Doctor Akizuki, [a physician at Nagasaki’s Urakami hospital] believe[d] that many babies died because of maternal malnutrition during the period of pregnancy.”⁷⁹

Malnutrition and other general problems affected both those in the high-impact area within three kilometers of the hypocenter and those beyond it. Assuming that the general causes of pregnancy loss in

⁷⁴ Stewart and Kneale 1993; internal reference omitted.

⁷⁵ RERF 2016, p. 6.

⁷⁶ Stewart and Kneale 2000.

⁷⁷ U.S. Strategic Bombing Survey 1947, p. 53.

⁷⁸ U.S. Strategic Bombing Survey 1947, p. 53.

⁷⁹ U.S. Strategic Bombing Survey 1947, p. 74.

the city applied to the bomb-impacted area, an excess of 150 percent of pregnancy loss in the two-to-three-kilometer of the hypocenter can be roughly attributed to the various impacts of the bombing: flash burns, mechanical injuries (destruction of homes, flying debris) and radiation exposure. The excess loss was even greater within two kilometers of the hypocenter.

The data and information in the U.S. Strategic Bombing Survey medical assessment indicates that there may be a significant healthy survivor effect in the in utero cohort. For instance, premature babies within the cohort birth window but who died shortly after birth were not included. Children within that birth window were also likely among the ones who died of diarrhea. This author has not come across data on how many children with born with severe brain and neurological deficits, including severe mental retardation, but died before the cohort was formed or how such children were distributed across exposure groups.

There may also be a class-related bias. The severe shortage of medical facilities and personnel (most died or were injured in the bombings) and malnutrition would likely have disproportionately affected survivors with fewer financial means.

A third potential bias may be that the in utero cohort was limited to residents of Hiroshima and Nagasaki. Given the lack of food and medical facilities and medical professionals (presumed to include those who assisted in-home deliveries), many pregnant women may well have chosen to leave to deliver elsewhere, if they were able to do so. For instance, pregnant women who had family members in other parts of Japan may have moved in greater numbers than others. We do know that many people moved away in the aftermath of the bombings and that many also returned. How the complex migration patterns may have affected the cohort is unknown. We do know that the RERF found five cases of severe mental retardation among those who were not in the in utero cohort only because they were not living in Hiroshima or Nagasaki in 1950, when the cohort was created (Section II.b above).

As noted above, the Hiroshima-Nagasaki 1950 LSS cohort is considered one of the foundations of ionizing radiation risk assessment so far as epidemiologic studies are concerned. It is therefore reasonable to suppose that a fundamental criticism about potential bias in the selection of the cohort, published in peer-reviewed literature by well-recognized authors would be taken into account in subsequent studies. But it is not mentioned in either the National Academies 2006 BEIR VII report or the ICRP 90 report in which prior ICRP estimates of teratogenic impacts were reviewed and revised (as discussed in Section II.b above). Both reports cited the work of Alice Stewart and George Kneale, but not the publications that analyzed the LSS cohort.

Specifically, the BEIR VII report reference list includes several works authored by Alice Stewart and George Kneale relating to occupational exposure or to pre-natal diagnostic x-ray exposure and the risk of childhood cancer.⁸⁰ But it does not cite their work relating to the Hiroshima-Nagasaki cohort raising the fundamental question of bias in its selection. ICRP 90 also cites their work many times in reference to diagnostic x-rays. None of their publications relating to the Hiroshima Nagasaki cohort are cited. The omission in the ICRP 90 report particularly stands out for the subject of this report, because Stewart and Kneale explicitly pointed out the deficit of children in the first 8 weeks of pregnancy in the in utero Hiroshima-Nagasaki cohort of pregnancies from the time of the bombings to 31 May 1946.

⁸⁰ BEIR VII 2006, reference list. Some of citations have other authors as well.

A more detailed analysis, clearly needed; it is, however, well beyond the scope of this report.

III. The role of oxidative stress

A variety of pollutants create harmful oxidative stress in the body. The focus in this report is on the oxidative stress created by radiation exposure, which could, of course, be compounded, by that created by other toxicants, like heavy metals.

Direct radiation damage occurs when the radiation directly creates DNA damage. For instance, a photon (x-ray or gamma ray) can knock an electron out of an atom (the “scattered” electron) and at the same time generate a secondary photon (“scattered photon”), a phenomenon known as the Compton effect, illustrated in Figure IV-1. The scattered electron “can then interact with the DNA molecule and create damage in the form of strand breaks or damaged bases.”⁸¹ The scattered photon can interact with further targets, creating more scattered electrons and photons.

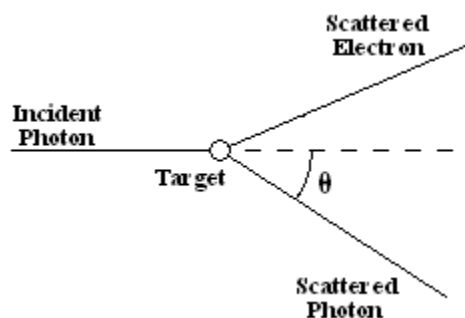


Figure IV-1: The Compton Effect. The electrons can do direct DNA damage.

Source: Tim314, August 30, 2005 from Wikimedia Commons at

https://commons.wikimedia.org/wiki/File:Compton_scattering_diagram.png

Direct impact also occurs with beta radiation, which consists of electrons or positrons, emitted by a radioisotope. Most beta particles outside the body do not penetrate far into it; very low energy electrons, like those from tritium decay, do not even get past the dead layer of the skin. Beta-emitting radioisotopes, like tritium, strontium-90, and iodine-131 do much more direct radiation damage when they are inside the body. Alpha radiation, consisting of relatively large helium-4 nuclei, also does not penetrate the dead layer of the skin. As a result, the harm from alpha particles occurs only when they are emitted when the radionuclide is inside the body.⁸²

Indirect impacts of radiation occur via the chemical changes induced by the deposition of the energy of the radioactive particles in the cell. For instance, they “can occur after a photon interacts with a water molecule. Water molecules make up 70% of human tissue. Ejection of an electron from a water molecule by an incoming photon produces an ionized water molecule, H_2O^+ .”⁸³ Such ionizations create oxidative stress – that is oxidization capacity inside the cell over and above the normal oxidation-reduction processes necessary for cell metabolism.

⁸¹ BEIR VII 2006, p. 29.

⁸² Many alpha-emitting radioisotopes also emit gamma radiation; for these radionuclides the impact is not limited to cases when they are inside the body.

⁸³ BEIR VII 2006, p. 29.

Hydroxyl radicals (OH•), produced when ionized water molecules, H₂O⁺, interact with normal water molecules, H₂O, are a particularly powerful source of oxidative stress:⁸⁴

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.

Low-dose ionizing radiation creates both kinds of impacts. For instance, Tharmalingam et al. (2017) have considered the impact of oxidative stress due low dose radiation during pregnancy. The dose was below 100 mGy, a level that is often postulated as a threshold for many non-cancer impacts (see Chapter II above). The specific context was women exposed to diagnostic external radiation in a medical context:⁸⁵

The intrauterine period of life provides a critical window during development in which the fetus is very sensitive to environmental perturbations. The epigenome of the developing fetus is vulnerable to oxidative stress, which can lead to aberrant epigenetic modifications that can persist into adulthood and induce numerous diseases. Exposure to radiation during pregnancy is often in the low-dose range and may potentially lead to long-term effects to the unborn child. Estimated fetal doses received during diagnostic treatments include 1.4 and 1.1 mGy for abdominal and pelvic X rays, respectively, and 8 and 25 mGy for abdominal and pelvic CT scans, respectively.

They note, in particular, that “[e]mbryonic organogenesis in particular is known to be a sensitive period for ionizing radiation-induced malformations” when cells are proliferating rapidly and organs are being formed. The impact of oxidative stress on DNA methylation is especially important. It can cause “hypermethylation”, which has specific teratogenic impacts:

Hypermethylation of the Pax3 gene provides a good example of oxidative stress-mediated developmental epigenetic changes due to intrauterine stress. Pax3 is a transcription factor and its expression controls neural tube closure during embryogenesis. Pax3 activity is inhibited during embryonic development in diabetic mice, resulting in developmental defects.⁸⁶

Failure of neural tubes to close produces devastating impacts, some of which are mentioned in Chapter II.

Epigenetic impacts, via DNA methylation, change can also cause a variety of immune-system related disorders. A 2021 review of the literature indicated that there was “converging evidence to suggest that maternal factors are associated with increased risk for developing autoimmune diseases, such as T1D [Type 1 diabetes], through epigenetic changes in fetal life.”⁸⁷

⁸⁴ BEIR VII 2006, p. 29.

⁸⁵ Tharmalingam et al. 2017, pdf p. 11; internal references omitted.

⁸⁶ Thamaralingam et al. 2017, pdf p. 11; internal references omitted.

⁸⁷ Barchetta et al. 2021.

Many epigenetic impacts, including those imprinted on the embryo and fetus, are heritable – that is, they produce multigenerational health impacts. They include:

- Diabetes, potentially exacerbated by forced changes in diet when people were moved due to nuclear testing and its impacts, as at Bikini, Enewetak, Rongelap, and Novaya Zemlya;⁸⁸
- Autoimmune disorders, such as Type 1 diabetes;⁸⁹
- Type 2 diabetes;⁹⁰
- Low birth weight, which can result a host of vulnerabilities throughout life, including cardiovascular disease.⁹¹

Some of these health risks are also discussed below in brief surveys of the impacts of specific radionuclides.

The disruption of mitochondrial function occurs through oxidative stress, which in turn increases the risks of a variety of adverse outcomes. It is important to note here that these impacts of radiation can be compounded by exposure to other toxics that cause oxidative stress and mitochondrial damage.⁹²

Exposure to environmental toxins, such as heavy metals, pesticides, and endocrine-disrupting chemicals (EDCs), can significantly impair mitochondrial function. Heavy metals like lead, mercury, and cadmium disrupt mitochondrial function by increasing oxidative stress and interfering with the electron transport chain. These metals can also induce mitochondrial DNA mutations, impairing energy production and leading to developmental abnormalities. Many pesticides act as mitochondrial toxicants, inhibiting key mitochondrial enzymes, disrupting membrane potential, and increasing the production of ROS [reactive oxygen species], leading to mitochondrial dysfunction and possibly negatively impacting fetal development.

Given this wide range of impacts, many occurring via oxidative stress on mitochondria located in the cytoplasm, the state of the science when it comes to protecting the public, especially pregnant women, is seriously lacking. Much remains undone and unknown, as indicated by a 2022 National Academies report on research needs in the field of low-level radiation:⁹³

Epigenetic changes also cause various other human diseases in addition to cancer such as protein aggregation diseases, metabolic diseases, neurological and psychiatric diseases, and imprinting disorders. Several recent studies *support a direct link between low-dose radiation and epigenetic changes*, and a comprehensive analysis in terms of the full range of epigenetic modifications and alterations to chromatin structure after low-dose exposures is clearly warranted. How much of low-dose effects are in fact mediated through reactive oxygen damage forms an intricate part of this equation and needs to be explored in detail. The *modulation of reactive oxygen species is likely directly or indirectly related to changes in mitochondrial function that have been observed after low-dose exposures*. Some studies suggest that reactive oxygen damage

⁸⁸ Makhijani 2022a and Makhijani 2022c

⁸⁹ Barchetta et al. 2021.

⁹⁰ Tharmalingam et al. 2017.

⁹¹ Tharmalingam et al. 2017.

⁹² Adelezzì et al. 2024

⁹³ National Research Council 2022, pp. 156-157; internal references within the quote are not shown; italics added.

from low-dose and repeated low-dose exposures may even contribute to the proliferation of pre-cancerous cells in tissues. *Furthermore, the role of radiation damage to other organelles (excluding the cell nucleus) has received little attention, but this too may have an impact on health outcomes and follow different dose response relationships.*

The scarcity of attention is particularly troublesome, since pregnancy-related exposure impacts have been noted in the literature since the 1950s, both in the context of medical x-rays and the exposures resulting from the bombings of Hiroshima and Nagasaki.

Having covered the general risks, we now turn to considering the risks associated with exposure to specific radionuclides.

IV. Iodine radioisotopes and thyroid-related multigenerational impacts

Atmospheric nuclear weapons testing has resulted in especially high doses to the thyroid. This is because iodine-131 and other short-lived iodine radioisotopes are highly radioactive and particularly target the thyroid gland. Pathways to ingestion include inhalation, drinking contaminated water, such as that collected from rooftops, and ingestion of food with iodine isotopes, especially milk. When fallout deposits on vegetation that is eaten by ruminants like cows and sheep, it gets concentrated in milk. When iodine is “applied to the skin, it appears in the blood soon after application and is taken up by the thyroid gland...”⁹⁴

Thyroid doses due to fallout from atmospheric testing at the Nevada Test Site and the attendant thyroid cancer risks were examined in detail in a notable 1997 study by the U.S. National Cancer Institute.⁹⁵ That study found that iodine-131, half-life 8.1 days, was the dominant radionuclide for thyroid dose; places where it rained out were disproportionately impacted. Milk was the dominant pathway; as a result farm families who drank fresh milk tended to have the highest doses, other things being equal. The combination of the factors resulted in the highest average doses being in rural counties in Idaho and Montana, which were 800 to 1,000 kilometers away from the test site.

Regarding multigenerational impacts, Dr. Tilman Ruff, of the International Physicians for the Prevention of Nuclear War, provided this overview statement indicating a wide variety of serious harms:

Thyroid hormones are crucial for fetal, infant and child development, and hypothyroidism during pregnancy and childhood can lead to stillbirth, growth retardation, reduced child survival, impaired brain function and development and permanent brain damage (cretinism at its most severe end). Hypothyroidism is certainly one of the sequelae of thyroid irradiation; if that occurs during pregnancy or childhood the consequences can be severe and permanent.⁹⁶

The California Center for Reproductive Health has noted the following conclusions regarding hypothyroidism and pregnancy loss:⁹⁷

Numerous studies have demonstrated a link between hypothyroidism and an increased risk of miscarriage, particularly in the first trimester. Thyroid hormones play a critical role in maintaining the delicate balance of hormones necessary to support pregnancy. If thyroid hormone levels are insufficient, implantation and early fetal development can be disrupted, increasing the risk of early pregnancy loss.

A meta-analysis cited by the California Center, concluded that even subclinical hypothyroidism⁹⁸ resulted in adverse pregnancy outcomes like preterm births and “placental abruption”.⁹⁹ The latter term describes a condition “when the placenta partly or completely separates from the inner wall of the

⁹⁴ NCI 1997, p. A6.10

⁹⁵ NCI 1997.

⁹⁶ Tilman Ruff, personal email communication with Arjun Makhijani, 26 June 2022

⁹⁷ California Center for Reproductive Health 2025.

⁹⁸ The term “subclinical hypothyroidism” means that they hypothyroidism is not evident in the patient’s clinical symptoms.

⁹⁹ Zhang et al. 2017.

uterus before delivery,” according to the Mayo Clinic, which goes on to state that “[t]his can decrease or block the baby's supply of oxygen and nutrients and cause heavy bleeding in the mother....Left untreated, it endangers both the mother and the baby.”¹⁰⁰

Further, the fetus depends largely on the mother for thyroid hormone supply even during the latter stages of pregnancy. As a result, maternal thyroid health is critical to fetal development. Neurological and brain development deficits are among the documented problems of maternal thyroid deficits.¹⁰¹

Atmospheric nuclear weapons testing has resulted in widespread thyroid exposures. For example, people all over the United States had thyroid exposures due to iodine-131 in fallout during atmospheric testing at the Nevada Test Site.¹⁰² Millions of people, including pregnant women were impacted, the more so in rural areas where people drank fresher milk. All other things being equal, fresher milk produces higher intakes, since the half-life of iodine-131 is only about eight days. Pregnant and lactating women in Polynesia were affected due to French nuclear weapons testing.¹⁰³ High whole body and even higher thyroid doses suffered by people, including women and children, in the Marshall Islands have also been documented.

The profound impacts on the Marshallese people, including adverse pregnancy outcomes, were noted in the 2012 report of the rapporteur of the United Nations Human Rights Council who investigated the matter and to whom the people provided testimony. The impact on failed pregnancies and fetal disorders has been quoted above in Chapter II; the thyroid cancer impact was also noted in the report:

31. Several years after exposure, a high incidence of thyroid cancer was reported, as well as an unusually high prevalence of stunted growth among Marshallese children. The incidence of such cases was also supported by the number of claims before the Nuclear Claims Tribunal.¹⁰⁴

Official thyroid doses were estimated in the 7.6 to 12 Gy range on Rongelap Atoll, and in the 0.76 to 1.65 Gy range on Utrik Atoll. Independent expert assessments estimated doses to be much higher; for instance the average thyroid dose on Utrik Atoll was estimated to be 27 Sv – which is 16 to 36 times (rounded) higher than official estimates.¹⁰⁵ Testimony before a Congressional subcommittee indicated that the government’s lower estimates were due to omission of certain factors and data, including neglect of blood count data.¹⁰⁶

The thyroid impacts were felt throughout the Marshall Islands and not just on the atolls that received the highest fallout and medical follow up by the U.S. government.¹⁰⁷ Late effects on the thyroid, including hypothyroidism, thyroid nodules, excess thyroid cancers, and instances of “profound growth failure” in two boys “due to radiation related thyroid atrophy” have been noted.¹⁰⁸

¹⁰⁰ Mayo Clinic 2025.

¹⁰¹ Johns Hopkins. accessed 2022. Also see Utiger 1999.

¹⁰² NCI 1997

¹⁰³ Tilman 2022.

¹⁰⁴ Georgescu 2012

¹⁰⁵ Makhijani 2022a, where the references may also be found.

¹⁰⁶ Mauro 2005, pp. 88-89.

¹⁰⁷ Plasman 1985, p. 69

¹⁰⁸ Plasman 1985, pp. 70-71

V. Tritium

Tritium, (T), is the sole radioactive isotope of hydrogen. It is the most ubiquitous pollutant connected to nuclear weapons and nuclear power. It was produced in vast amounts during atmospheric testing and dispersed throughout the environment – 240 exabecquerels or almost 700 kilograms,¹⁰⁹ compared to a few kilograms of worldwide natural tritium inventory.

Nuclear power reactors routinely emit tritium to the atmosphere; they also discharge it into water bodies. Amounts vary; a typical pressurized water reactor of 1,000 megawatts-electrical capacity, a common design and size, emits and discharges several terabecquerels a year to the environment.¹¹⁰

Common chemical forms of tritium are HTO and T₂O, where tritium has replaced one or both the hydrogen atoms in H₂O. HTO and T₂O are radioactive forms of water, known as tritiated water. Once ingested, inhaled, or absorbed through the skin, tritiated water pervades the body. Like many other radionuclides, it also crosses the placenta; unlike most, its concentration in the fetus is greater than the that in the mother. When taken up by plants, it becomes part of the photosynthesis process in the same way as water. This organically bound tritium propagates throughout the environment, including as food eaten by animals.

Tritium is a beta-emitter; the electrons it emits in the course of radioactive decay have energies that range from very low (close to zero) to 18,600 electron-volts, with an average of 5,700 electron-volts. Water ionization energy is about 12.6 electron volts, meaning that, on average, each tritium decay could ionize about 450 water molecules. The bond energy of biological molecules is typically just a few electron volts per bond; as a result, on the order of a thousand biological molecules could be dissociated by a single tritium decay at average energy.

The relatively low energy of tritium beta particles means that their energy would be deposited entirely within a single cell because the stopping distance of even the most energetic beta particle from tritium decay is smaller than the typical cell diameter. Tritium beta particles therefore produce a large number of ionizations within the cellular volume. This is, in principle, similar to alpha particle high-LET radiation. However, alpha particles resulting from radioactive decay are much more energetic; their relative biological effectiveness varies by endpoint, but is generally assigned a value to 20 for cancer risk.

As noted earlier, ionization of water produces the highly reactive hydroxyl radicals in cells, creating oxidative stress. That can, in turn damage mitochondrial DNA (mtDNA) which has much more limited repair capacity than nuclear DNA. Mitochondria are present in essentially all eukaryotes, including plants, animals, and fungi; they are essential to cellular energy production. Mitochondrial dysfunction can cause genomic instability, possibly with heritable impacts.¹¹¹ It can also contribute to neurological diseases.¹¹²

Given these various characteristics of tritium, the issue of the relative biological effectiveness (RBE) is important: how much more biological damage does a tritium beta particle create to the same amount of

¹⁰⁹ Calculated from UNSCEAR 2000, Vol. I, p. 49.

¹¹⁰ Tables for one year for all U.S. power reactors can be found at <https://ieer.org/resource/tritium/tritium-releases-to-air-and-water-from-nuclear-power-plants-tables-of-data-from-2004-and-2005/>

¹¹¹ Kim, Chandrasekaran, and Morgan, 2006. abstract.

¹¹² Arun et al. 2016

energy deposited by a fission product gamma ray, such as that arising from cobalt-60 or cesium-137 decay. Research in Russia, reported in the U.S. journal *Health Physics*, found that the impact of tritium beta particles on the DNA of hematopoietic cells was two to six times greater than gamma radiation from cesium-137, indicating an RBE of 2 to 6 for this end point.¹¹³

Research at the Lawrence Livermore National Laboratory in California examined tritium RBE for a specific endpoint of loss of mammalian oocytes. It concluded the relative biological effectiveness (RBE) of tritium increased with decreasing dose from 1.5 at a dose rate of 4 rads per day to nearly 3 at very low levels of exposure.¹¹⁴ At a high level of tritium exposure – 1 rad per day – the “startling” result was that “the female germ-cell population would be completely (99.9%) destroyed in the fetus before birth.” Further, the data indicate that there was “no evidence of a “safe” threshold below which cells [oocytes] are not killed.”¹¹⁵

The Livermore experiments indicating no threshold of oocyte damage were conducted at rather high levels of exposure; even the lowest levels were far higher than the drinking water standard. Yet, the indication from this research that there was no threshold below which oocyte killing did not occur finds support in other experiments that included tritium contamination below the U.S. drinking water limit of 740 Bq/liter (20,000 picocuries per liter). One experiment with tritium-laced seawater found adverse impacts on goose barnacle molting at levels as low as about 15,000 picocuries per liter (about 560 Bq/L), a little about three-fourths of the U.S. drinking water limit.¹¹⁶ Tritiated water was found to damage or kill carp eggs at levels as low as 500 Bq/L, which is about two-thirds the U.S. drinking water limit.¹¹⁷ The damage to carp eggs was at a tritium concentration remarkably similar to the goose barnacle study.

Given that much of the impact of tritium (and other radionuclides) is via increase of oxidative stress, the reproductive health and ecological protection must take other environmental sources of oxidative stress into account. Mousseau and Todd have recently published a thorough survey of the literature on tritium and its impacts that can provide further insights into the issues discussed in this chapter.¹¹⁸

¹¹³ Balanov et al. 1993.

¹¹⁴ Dobson 1982, Figure 3.

¹¹⁵ Dobson 1982, p. 8 and p. 6.

¹¹⁶ Abbot and Mix, 1979.

¹¹⁷ Bondareva et al. 2022.

¹¹⁸ Mousseau and Todd 2023.

VI. Uranium

Natural uranium is the basis of the nuclear enterprise – weapons and power. Uranium-235 is the only fissile material that occurs in nature in usable amounts.¹¹⁹ Natural uranium contains three isotopes – uranium-238 (a little less than 99.3%), uranium-235 (0.7%), and uranium-234 (0.0054%). All three isotopes are alpha emitters. This means that they create health risks mainly when they are inside the body, though they also emit gamma rays (photons) of low energy (U-234 and U-238) and medium energy (U-235).¹²⁰ Thorium-232 is abundant, but it is not fissile.

The main components of natural uranium have very long half-lives (about 4.5 billion years for U-238 and 700 million years for U-235). U-234 (half-life 245,000 years) is a decay product of uranium-238 and is present only in trace amounts in natural uranium. However, since it has a much shorter half-life, it contributes almost half of specific activity of natural uranium.¹²¹ Plutonium-239, with a half-life of 24,110 years, is about 100,000 times more radioactive than natural uranium per unit mass. Their alpha particle energies are comparable; therefore it would take several orders of magnitude more uranium to create comparable radiotoxic impact as plutonium-239 in terms of energy deposited in the body.¹²²

But uranium is also a heavy metal and, as such, is toxic. It turns out that the amount that would deliver a radiation dose of the same order of magnitude as some regulatory limits can also result in heavy metal toxicity, notably to the kidneys. The simultaneous heavy metal toxicity and radiation impact created by uranium can have synergistic impacts regarding the risk of adverse health consequences.¹²³ This dual toxicity makes uranium unlike most other radioactive heavy metals associated with nuclear weapons and other nuclear industries. For instance plutonium-239, plutonium-238, and americium-241 have such a high specific activity the severe damage from radiation results from amounts far lower than those needed for heavy metal toxicity.

The radiation impact of an intake of a given mass of uranium depends on the level of “enrichment” – that is the degree to which the concentration of the fissile isotope, U-235, has been increased. Nuclear weapons and U.S. naval reactors use uranium enriched to more than 90% U-235. The radiation dose resulting from exposure to highly enriched uranium is mainly from U-234, which, being lighter, is enriched to an even greater degree than U-235. The process of enrichment also creates a waste stream – uranium that is depleted in U-235 (and U-234); it is known as “depleted uranium”, which has, in its metal form, been used for a variety purposes including making armor-penetrating shells for military use.

Research done at the U.S. Armed Forces Radiobiology Research Institute has contributed significantly to understanding of the toxicity impacts of uranium. Though the research was focused on depleted uranium, it is broadly applicable to the impacts of all uranium, though it should be noted that (i) the

¹¹⁹ Plutonium-239 occurs in trace amounts due to the presence of neutrons from the spontaneous fission of uranium-235 and uranium-238 nuclei.

¹²⁰ ANL 2007, pdf p. 58.

¹²¹ Radioactivity per unit mass, called specific activity, is (approximately) inversely proportional to half-life and mass number.

¹²² Their metabolic pathways also differ somewhat; their biological half-lives are also somewhat different, though both depend on the solubility of the chemical form that is inhaled or ingested. The radiation dose impact per becquerel of plutonium-239 is greater than any of the three natural uranium isotopes.

¹²³ Makhijani, Smith, and Thorne, Chapter 8.

amounts needed to produce a given radiation dose vary with enrichment, and (ii) the balance of radioactivity and chemical toxicity impacts also changes with the level of enrichment.¹²⁴

There are four aspects of uranium exposure that are relevant to the question of multigenerational impacts:

1. **Impact on sperm:** Uranium exposure has been found to increase its concentration in semen. A British report on veterans concluded that such increases “raise[d] the possibility of adverse effects on the sperm from either the alpha-particles emanating from DU [depleted uranium], chemical effects of uranium on the genetic material or the chemical toxicity of uranium.”¹²⁵ Such impacts are also indicated by laboratory experiments on animals, though these are generally done at high rates of exposure.
2. **Impact on the fetus:** Animal experiments indicate that uranium crosses the placenta to impact the fetus.¹²⁶ The concentration of uranium in the fetus is about one-tenth that in the mother for pre-pregnancy intakes and about the same as in the mother for intakes during pregnancy (see Table III-1 above).
3. **Impact on maternal kidneys:** Maternal kidney disease and malfunction can seriously impact the embryo and fetus in a variety of ways.
4. **Other impacts:** A number of other uranium impacts are indicated, such as on endocrine function and cardiovascular disease, though with low levels of confidence often due to insufficient research.

The third and fourth aspects are considered here in more detail, because they are rather different than the impacts of internal radiation considered so far in this paper.

Current research suggests that chemical toxicity from the intake of small quantities of uranium through contaminated drinking water may increase the risks of a range of diseases. A survey of the literature, focused in part on impacts of uranium water contamination on indigenous lands and people, cited evidence of many impacts, some of which are summarized in Table V-1 below. Impacts where statistical significance was not found are not included in Table V-1.

¹²⁴ It also depends on the chemical form of uranium. For instance, if insoluble uranium is inhaled it will stay in the lungs longer and be released only slowly into the blood stream and the rest of the body (including bones and kidneys). The dose to the rest of the body per unit time is relatively low but it endures for a long time (many years). Soluble uranium clears the lungs more rapidly, impacting other organs with larger doses in shorter periods of time. See Hon, Österreicher, and Navrátil 2015.

¹²⁵ As quoted in Makhijani, Smith, and Thorne 2006, p. 70.

¹²⁶ Makhijani, Smith, and Thorne, 2006, pp. 70-71.

Table V-1: Some health impacts of uranium in drinking water detected at levels well below the U.S. drinking water standard

Health effect	Confidence level for health effect	Statistically significant?	Minimum drinking water concentration associated with health effect	Current U.S. drinking water standard for uranium
Various cancers (kidney, lung, leukemia, liver)	Low	Yes	1 to 10 micrograms/liter	30 micrograms/liter (EPA 2000 at 40 CFR 141.66(e))
Cardiovascular hypertension	Low	Yes	> 1 microgram/liter	
Renal Nephrotoxicity	High	Yes	>2 micrograms/liter	
Endocrine urinary glucose	Moderate	Yes	>3 micrograms/liter	
Thyroid function	Low	Yes	>2 micrograms/liter	

Source: Redevers et al. 2021

Note: References and notes in the source paper are not reproduced here. The U.S. uranium drinking water standard is provided for comparison with the minimum drinking water concentrations at which the cited effects were detected with statistical significance (even if low). Impacts where statistical significance was not found are omitted in this table.

Impacts on the mother can translate directly into impacts on the offspring. For instance, a 2021 review by Weiner and Wolfe summarized the impact of maternal cardiovascular disease on the offspring as follows:¹²⁷

Maternal cardiovascular disease (CVD) during pregnancy is on the rise worldwide, as both more women with congenital heart disease are reaching childbearing age, and conditions such as diabetes, hypertension, and obesity are becoming more prevalent. However, the extent to which maternal CVD influences offspring health, as a neonate and later in childhood and adolescence, remains to be fully understood....This review demonstrates that maternal CVD leads to higher rates of complications among neonates. Ultimately, our review supports the hypothesis that maternal CVD leads to intrauterine growth restriction (IUGR), which...can have health repercussions, including an impact on CVD risk, both in the immediate newborn period as well as later throughout the life of the offspring. Further research remains crucial in elucidating the mechanism of maternal CVD long-term effects on offspring....

Similarly, maternal kidney disease leads to negative impacts on the health of the newborn, including pre-term birth, increased admission to neonatal intensive care units, and low birth weight.¹²⁸

There are, of course, many causes of cardiovascular and kidney disease. Many factors, including poverty, pre-existing conditions like diabetes, may exacerbate the impacts of pollutants like uranium. Nonetheless, indications are that uranium contamination of drinking water may aggravate or even cause such diseases. This raises the question of the extent to which there have been multigenerational impacts of uranium mining, milling, processing (including enrichment) on children born to exposed women.

¹²⁷ Weiner and Wolfe 2021, from the abstract.

¹²⁸ Kendrick et al. 2015.

Being a natural primordial element, there are varying levels of uranium that occur naturally in rocks, soil, and water. Natural levels in both soil and water vary a great deal and can be above the drinking water standard.

Mining and mine wastes in and around abandoned mines can contaminate drinking water. However, sufficient research to attribute contamination, including on indigenous lands, to uranium mining and its aftermath has still not been done, despite the fact of intensive uranium mining in the United States, including on indigenous lands like those of the Navajo Nation, goes back to the 1950s. There are more than 500 abandoned uranium mines on the Navajo Nation's lands alone.¹²⁹ Given that uranium mining has disproportionately impacted colonized and indigenous populations, there are evident and obvious serious environmental justice implications. Redvers et al. (2021) summarized the issues and concerns relating to the impacts on indigenous people, the vast amount of work that remains to be done, and the protective measures that need to be taken as follows:

Uranium contamination of drinking-water sources on AI [American Indian] reservations in the United States is a largely ignored and underfunded public health crisis. With an already marginalized population, we feel that the potential for adverse health effects and outcomes are multiplied.¹³⁰

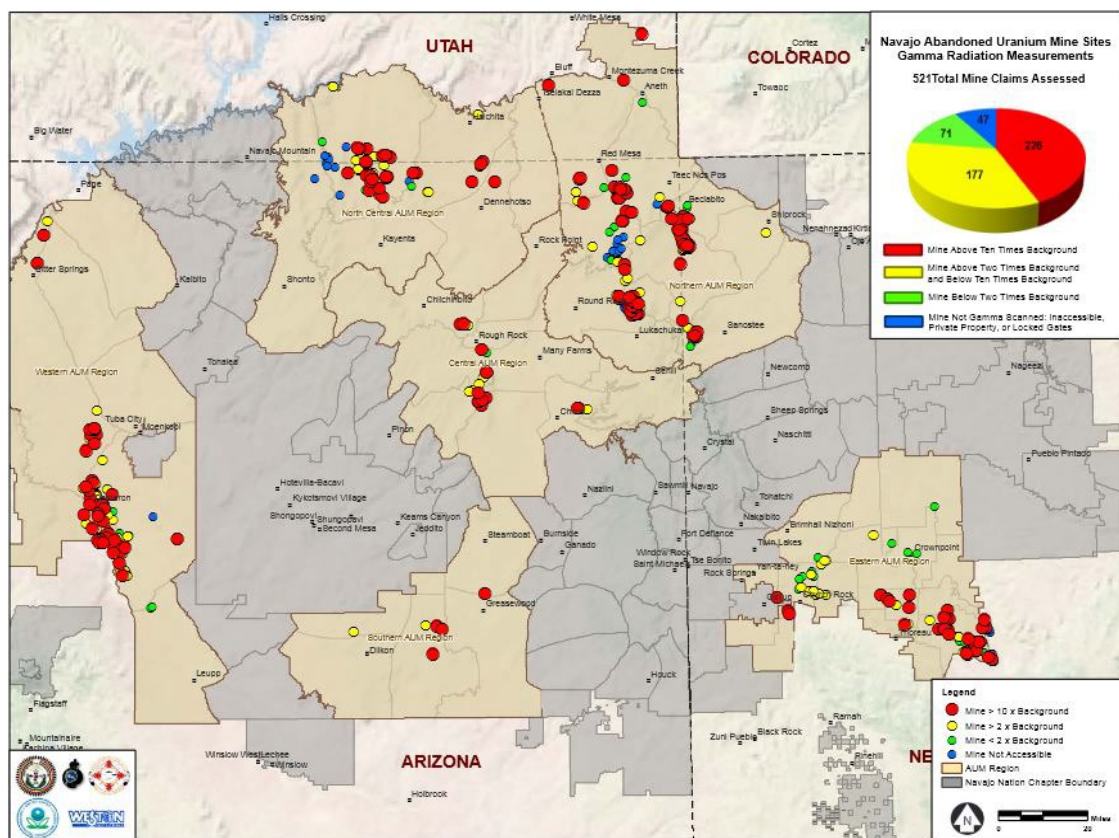


Figure VII-1: Abandoned uranium mines on Navajo Nation lands. Source: EPA 2016.

¹²⁹ Redvers et al. 2021.

¹³⁰ Redvers et al. 2021

Figure VII-1 above shows an Environmental Protection Agency map of abandoned uranium mines on Navajo Nation lands, with a categorization according to gamma radiation rates. These would generally be indicative of uranium contamination (including decay products, notably radium-226).

While the Redvers et al. paper's conclusions were about indigenous people and lands in the United States, the same could also be said of the uranium mining legacy on indigenous and colonized lands across the world. Uranium mines exist in or have existed in Australia, Canada, China, Democratic Republic of Congo, the former Czechoslovakia (now Chechia and Slovakia), France, India, Kazakhstan, Namibia, Niger, Russia, Ukraine, and Uzbekistan, among others.¹³¹

Given the above, the history of uranium drinking water standards and guidelines on how the understanding of risk has been translated into guidelines and standards is interesting and instructive. The United States set limits on drinking water contamination by radionuclides in the late 1970s but the limit for uranium was not included and deferred to a later time. It was set at 30 micrograms per liter in 2000.¹³² The World Health Organization relaxed its guideline from 2 micrograms per liter in 1998 to 15 µg/L in 2004 and finally to 30 µg/L in 2011; at that point it matched the U.S. standard, which is more lax than in several other industrialized countries.¹³³ Coincidentally or not, in 1991 the United States Environmental Protection Agency had proposed a range of uranium drinking water limits from 5 to 80 µg/L, with an intent to focus on either 20 or 30 µg/L in the final rule; the rule finalized the limit at 30 µg/L in the year 2000.¹³⁴

¹³¹ IPPNW and IEER 2000 has a chapter on uranium mining that list the principal countries and uranium mining locations up to the early 1990s.

¹³² EPA 2000.

¹³³ Redvers et. al 2021.

¹³⁴ EPA 2000.

VII. Notes on some other radionuclides

Besides the specific radionuclides discussed above, a number of other radionuclides that have been widely dispersed or caused severe pollution due to accidents and testing are listed here. These radionuclides, if present in the mother can adversely impact the health and development of the embryo and fetus, including the various negative outcomes that have been discussed in this report. Here is a partial list of radionuclides that cross the placenta with brief mention of the reasons for concern:^{135,136}

- **Carbon-14:** Like tritium, carbon-14 is made naturally by the interaction of cosmic rays with the atmosphere. It is created both by nuclear explosions and in nuclear power plants. In some designs, notably those that use graphite as a moderator or to encapsulate fuel, nuclear reactors have generated very large amounts of carbon-14. Unlike tritium, it has a very long half-life – 5,730 years. Carbon-14 is chemically like non-radioactive carbon and enters ecosystems via photosynthesis. Almost all the carbon-14 created by nuclear weapons testing remains in the environment. Unlike tritium, the natural inventory is significantly larger than the man-made one. It decays by emitting relatively low-energy beta particle (49 keV). Both tritium and carbon-14 become part of the very structure of all living beings.
- **Cesium-137:** Cesium belongs to the same group of elements as potassium, which is an essential trace element in living beings, and involved in muscular and neurological functioning and health. It emits a high energy beta particle as well as high energy gamma rays. Soils, such as those on Rongelap Atoll, which are deficient in potassium and contaminated with cesium-137 will preferentially take up cesium-137, thereby bioconcentrating it. This results in radiation exposure via ingestion, in addition to the exposures from residual radiation in the soil, with resultant impact on all who consume those plants, including pregnant women. Cesium-137 has a half-life of about 30 years. Cesium is relatively volatile compared to strontium-90; it has been present in far larger amounts in the plumes from nuclear power reactor accidents, notably Chernobyl and Fukushima.
- **Strontium-90:** Strontium belongs in the same group of elements as calcium and therefore selectively concentrates in bony tissue and teeth. It impacts the formation of bones and teeth in utero. Strontium-90 decays by emitting a beta particle (200 keV); it decays into yttrium-90, which is also a beta emitter (940 keV). Sr-90 has a half-life of 28.8 years, only slightly lower than cesium-137 (30.1 years). Since the stem cells of the human immune system are made in the bone, strontium-90 can have an impact on immune system functioning, in addition to the cancer risks it creates. Strontium is much less volatile than cesium, but has been emitted into the atmosphere in large amounts during atmospheric and shallow underwater nuclear weapons testing and some accidents, notably the 1957 explosion of a tank containing highly radioactive waste at the Mayak nuclear weapons site in the former Soviet Union.¹³⁷ Strontium-90 is relatively more mobile in soil and water than cesium-137 and can have wide ecosystem impacts

¹³⁵ Unless otherwise mentioned, the properties of these radionuclides mentioned here can be found in ANL 2007. Half-lives differ slightly in various publications. NRPB 2001 provides fetal to maternal radionuclide concentration ratios for a number of radionuclides. See Table 1, pdf p. 18.

¹³⁶ Measurements residual strontium-90 and cesium-137 measurements in sediment cores with comparisons across test sites can be found in Rapaport, Hughes, and Hughes 2022. A notable features of the paper is a comparison of strontium-90 to cesium-137 ratios, with higher ratios found in the Northern Marshall Islands.

¹³⁷ IPPNW and IEER 1992, Chapter 4.

in aquatic environments. Strontium-90 contamination of shellfish is a notable problem in island countries like the Marshall Islands,¹³⁸ where nuclear tests have been done.

- **Plutonium:** While some of the plutonium-239 in nuclear bombs undergoes fission and provides some of their explosive power, most remains unfissioned. Thousands of kilograms of plutonium-239 remain in oceans and soils around the world. They will impact people and ecosystems essentially forever, since the half-life of plutonium-239 is over 24,000 years. While the individual dose is very small (except possibly in directly impacted areas), the population dose over tens of thousands of years due to testing is significant.¹³⁹ There are much larger amounts of plutonium in spent fuel – the used, highly radioactive waste from nuclear power production; this plutonium presents both proliferation and environmental risks. Americium-241 is both a gamma and alpha-emitting decay product of plutonium-241, which is a beta-emitter with a half-life of 14.4 years. Am-241 has a half-life of 432 years and has a radiological impact similar to plutonium-239.
- **Short-lived radionuclides:** A number of short-lived radionuclides (defined here as less than 5 years half-life) are created in the process of fission. Among the more important for health include cesium-134, strontium-89, ruthenium-106, and zirconium-95.
- **Very long-lived fission products:** Very long-lived radionuclides tend to produce low individual doses because their radioactivity per unit mass is relatively low; but since they are extremely persistent, they produce high cumulative population doses as they repeatedly cycle through ecosystems. The assessment of their impact depends a great deal on the understanding and assessment of the impact of low-levels of radiation exposure not only for cancer but for a host of other health risks, including during pregnancy. There also risks for ecosystem health. Three very long-lived fission products of concern (half-lives more than 100,000 years) are:
 - **Technetium-99**, half-life, 210,000 years: It distributes itself in various part of the body, concentrating in the thyroid and stomach wall. While it has a long physical half-life, it tends to stay in the body for relatively short periods of time.
 - **Cesium-135**, half-life 2.3 million years: Its impact per unit of radiation dose is similar to cesium-137.
 - **Iodine-129**, half-life about 16 million years: Its impact per unit of radiation dose is similar to the short-lived iodine isotopes discussed above.

¹³⁸ See Johnston and Barker 2008 for discussion of food-related issues, including contamination of shellfish, connected to nuclear testing in the Marshall Islands.

¹³⁹ IPPNW and IEER 1991, Chapter 3.

VIII. Findings and Recommendations

a. Findings

1. Different lines of evidence point to the conclusion that exposure to ionizing radiation creates adverse multigenerational impacts.

Their frequency and severity depend on a variety of factors, including stage of pregnancy at which exposure occurs, the specific radionuclide involved, the dose, and the specific endpoint. Impacts include the following:

- **Involuntary loss of pregnancy:** Such losses include early miscarriages, often so early that a woman may not realize she was pregnant. They may occur before implantation of the blastocyst on the uterine wall or in the weeks following implantation.
- **Central nervous system impacts:** Radiation exposure can lead to a variety of nervous system impacts, such as failure of neural tubes to close and severe mental retardation.
- **Teratogenic impacts:** Organ malformations, other than the central nervous system, can occur during organogenesis; every organ is vulnerable.
- **Nuclear DNA mutations:** Ionizing radiation causes both germline and somatic mutations.
- **Oxidative stress:** Ionizing radiation creates intracellular oxidative stress. Ionization of cytoplasmic water by radiation (such as the by the electron emitted when tritium decays) produces highly reactive hydroxyl radicals, “believed to be the most effective of the reactive [oxygen] species.”¹⁴⁰ The risks include damage to mitochondria and mitochondrial DNA, hypermethylation-related epigenetic damage with attendant teratogenic impacts, and other epigenetic impacts, such as those which increase the risk of Type 1 diabetes and other autoimmune diseases. Some epigenetic changes are heritable.
- **Synergistic oxidative stress harm:** Chemical pollutants also produce oxidative stress, creating the risk of additive or synergistic harm with oxidative stress created by ionizing radiation.
- **Increased cancer after birth:** In utero exposure has been demonstrated to cause increased post-natal cancer risk.

2. Thresholds for reproductive harm likely vary by endpoint and may be zero or very low in some cases, especially in the case of internally deposited radionuclides.

There are questions as to whether there is a threshold below which teratogenic harm does not occur, and if so what that threshold might be for any specific endpoint. While a threshold may exist for some endpoints, it may be much lower than that commonly postulated when internal radiation is involved.

3. There has been a lack of protection for pregnant members of the public, embryos and fetuses, notably but not only in regard to harm in early stages of pregnancy.

¹⁴⁰ BEIR VII 2006, p. 29.

Knowledge of harm to pregnant women, embryos, and fetuses as well as children dates back at least to the very earliest days after the bombings of Hiroshima and Nagasaki. It was documented early by Japanese physicians as well as by U.S. personnel, including by a medical assessment done by the United States Strategic Bombing Survey. The extensive fallout due to atmospheric testing, especially after the heavy exposure of Japanese fishermen after the 1 March 1954 15-megaton Bravo test at Bikini, extended that awareness to the world and sparked calls for an end to testing. The American Academy of Pediatrics formed a committee in 1957 to study the impact of radiation on malformations, as noted in a 2006 IEER report:¹⁴¹

...[O]ne of the most important turning points in the entire field of pediatric environmental health was the formation of the Committee on Radiation Hazards and Epidemiology of Malformations by the American Academy of Pediatrics. This committee was set up in 1957 as a result of a growing awareness that the impacts of nuclear weapons testing were disproportionately affecting children due to iodine-131 in fallout.

The American Academy of Pediatrics has reinforced that message more recently, in 2003:

Children have a number of vulnerabilities that place them at greater risk of harm after radiation exposure....Radioactive iodine is transmitted to human breast milk, contaminating this valuable source of nutrition to infants....*In utero* exposure to radiation also has important clinical effects, depending on the dose and form of the radiation; transmission of radionuclides across the placenta may occur, depending on the agent....¹⁴²

Despite the long history of scientific and medical concern, there is no radiation protection standard that explicitly aims to protect women in the general public against early failed pregnancies, teratogenic harm, including that which might have no thresholds (giving the benefit of the doubt in case of uncertainties), and other impacts due to in utero exposure.¹⁴³

4. Despite the early knowledge of a variety of harms from in utero exposure, large gaps in understanding remain.

The focus of most ionizing radiation research has been on cancer risk. At the cellular level, it has been on the nuclear DNA. These are no doubt critical subjects for scientific study. But other aspects that impact health, reproduction, and ecosystem integrity, such as mitochondrial DNA damage, reproduction-related topics, including teratogenic impacts, and oxidative stress are just as important; they have been relatively neglected.

For instance, as recently as 2022, a National Research Council report noted that “[t]he modulation of reactive oxygen species is likely directly or indirectly related to changes in mitochondrial function that have been observed after low-dose exposures.” Yet, the study also noted that “*the role of radiation damage to other organelles (excluding the cell nucleus) has*

¹⁴¹ Makhijani, Smith, and Thorne 2006, p. 35.

¹⁴² As quoted in Makhijani, Smith, and Thorne 2006, p. 36

¹⁴³ Only women who work in radiation regulated work places, like nuclear power or nuclear weapons plants, are protected if they declare their pregnancies; in that case the usual limit is 1 mSv of exposure, the same as the annual limit for members of the public. In the United States it is 5 mSv.

received little attention, but this too may have an impact on health outcomes and follow different dose response relationships.”¹⁴⁴

This relative lack of attention to organelle damage, including mitochondrial damage, is all the more remarkable in light of the fact that in the last several decades an entire branch of medicine has grown up around the study and cure of mitochondrial diseases. In this context, it is important to note that all animals, plants, and fungi have essentially the same mitochondria-based energy system. It is therefore reasonable to expect harms across ecosystems when it comes to oxidative stress and mitochondrial damage.

5. There is evidence of reproductive risk for other living beings at low levels of internal radiation exposure.

Goose barnacle molting was found to be adversely impacted by a concentration of tritium in seawater at a concentration less than the U.S. drinking water standard. A comparable concentration, also less than the drinking water standard, in freshwater was found to damage and kill carp eggs at statistically significant levels. It is important to note that the U.S. drinking water standard is stricter than that in some countries. The U.S. drinking water limit of 740 Bq/L would produce a relatively low annual dose in adults of 10 microsieverts, assuming an intake of two liters per day. Yet, reproductive harm in other living beings has been shown below this level.

6. The 1950 Life Span Study (LSS) and in utero Hiroshima-Nagasaki cohorts may have selection biases that could impact the conclusions that have been drawn regarding the risks of ionizing radiation exposure.

Though the potential selection bias in the 1950 Hiroshima-Nagasaki Life Span Study cohort in the form of a healthy survivor effect was pointed out in the peer-reviewed literature in the 1990s, relevant official literature has failed to address it.

Much of the evidence about teratogenic harm comes from the epidemiological analysis of the in utero and LSS Hiroshima-Nagasaki cohorts. It is therefore critical to reexamine the official assumption that the cohort is homogenous. The heterogeneity of the cohorts could be due to

- The deficit of people below 10 years and more than 50 years in the LSS cohort;
- The stage of pregnancy at the time of the bombings;
- Potential differential access to food and medical care due to differing financial means of survivors (i.e., a potential class bias);
- Mobility bias, in that pregnant women whose children would have been in the cohort may have selectively moved away but the cohort was limited to those who had permanent addresses in the two cities.

These potential selection biases could impact the epidemiologic findings both in regard to the various risk coefficients derived from LSS and in utero cohorts. That possibility has been known in regard to both

¹⁴⁴ National Research Council 2022, pp. 156-157, italics added; internal references omitted.

cohorts at least since the 1990s, when Alice Stewart and George Kneale published papers in the peer reviewed literature. It is noteworthy that the re-analysis they recommended does not appear to have been carried out. Indeed, official publications, notably the BEIR VII report and ICRP 90 which have examined reproductive issues related to ionizing radiation, did not even refer to this work despite several references to the work of the same authors in other areas of ionizing radiation-related risks.

7. Independent assessments of radiation doses suffered due to fallout from atmospheric testing have tended to be far higher than official dose estimates.

Besides the U.S. and Soviet examples cited above, independent dose estimates due to fallout from French atmospheric nuclear testing in Polynesia are 2 to 10 times the official ones.¹⁴⁵ Accurate, independently estimated doses are critical for many reasons, including:

- More accurate estimation of risks due to ionizing radiation, including multigenerational risks and direct risks for a variety of diseases to those who are exposed.
- Nuclear weapons states that have done atmospheric testing have inflicted harm on people across the world, and disproportionately impacted colonial and indigenous populations. Accurate dose estimates are critical for understanding who was put at risk as well as the types of risks.
- Lower dose estimates allow for claims of lower harm and can lower compensation by governments, where compensation programs have been put in place. This was explicitly pointed out by the researchers who produced the independent estimates for French atmospheric testing in Polynesia.¹⁴⁶

b. Recommendations

1. Radiation protection standards should be tightened to account for multigenerational risks and ecosystem harms, and make allowance for the large gaps in knowledge by adopting the precautionary principle.

Given the demonstrated multigenerational harm from ionizing radiation, the large gaps in knowledge, and the evidence of reproductive harm in ecosystems, a precautionary tightening of exposure to ionizing radiation, especially in regard to food and water, should be adopted.

For instance, toxic effects of various kinds are indicated for uranium – with high confidence at more than 2 micrograms per liter (in the case of nephrotoxicity) and low confidence at 1 microgram per liter (cardiovascular hypertension), and 1 to 10 micrograms per liter for various cancers (see Table V-1, in Chapter V). The current WHO guideline is 30 micrograms per liter. Since cardiovascular disease and nephrotoxicity in a pregnant woman can impact the fetus, there is a good argument to tighten the WHO

¹⁴⁵ Phillipe, Schoenberger, and Ahmed 2022.

¹⁴⁶ Phillipe, Schoenberger, and Ahmed 2022

guideline to 2 micrograms per liter, which was the pre-1998 level. Relatively weaker evidence points to a 1 microgram per liter guideline.

Tightening of drinking water standards relative to present levels is also indicated. For instance, the California drinking water guideline is 400 picocuries (14.8 Bq) per liter, compared to the U.S. standard of 20,000 picocuries (740 Bq) per liter and much more lax values in other places.¹⁴⁷

Overall, the precautionary principle should be coupled with existing knowledge to set tighter standards for ionizing radiation exposure, as illustrated by the two examples provided here.

2. A re-analysis of the LSS and in utero Hiroshima-Nagasaki cohorts in light of the potential selection biases, such as age inhomogeneity, is needed.

Hiroshima-Nagasaki data have been principal sources for radiation risk assessment and, hence, to the creation of radiation protection regulations. The neglect of potential biases created by heterogeneities in the in utero and Life Span Study cohorts should be remedied to the extent possible. A thorough re-assessment should include early Japanese and U.S. data, such as the early Japanese clinical data and the 1945-1947 U.S. Strategic Bombing Survey data cited in this report.

The many questions that remain as to the interpretation of laboratory studies should also be addressed. Among them are the following:

- How should laboratory animal studies be translated into radiation exposure in the context of real-life? For instance, Chernobyl studies showed animals in the wild in the exclusion zone had much more complex genetic outcomes than indicated by laboratory experiments.¹⁴⁸ As another example, most experiments are done on mice. But tritium experiments at Lawrence Livermore National Laboratory showed much greater impacts on squirrel monkeys than mice.¹⁴⁹
- Are animal studies using external exposure that found evidence of a threshold below which no effect was observed applicable to internal exposure?
- If there is a threshold for a specific impact, is it different for internal and external exposures and for different types of internal exposure?
- What are the specific ways in which the biological impact of different types of internal exposure should be evaluated for non-stochastic effects, given that relative biological effectiveness factors were developed primarily for cancer risk, which is a stochastic multi-stage effect?

3. A thorough re-assessment of the multigenerational risks of ionizing radiation is needed. It would be appropriate if it is done in the context of the Treaty on the Prohibition of Nuclear Weapons.

¹⁴⁷ Examples of official and non-governmental bodies that have recommended far stricter drinking water standards are discussed in Makhijani 2009.

¹⁴⁸ Garnier-Laplace et al. 2013

¹⁴⁹ Dobson 1982.

Nuclear weapons have been produced and tested by only nine countries. But uranium has been mined in a large number of countries that have neither nuclear weapons nor nuclear power. Most of the uranium that went into the Hiroshima bomb and that was used to make then plutonium for the Nagasaki bomb came from the Congo, which was subject to Belgium's brutal rule at the time.¹⁵⁰ Atmospheric testing, which has inflicted global harm,¹⁵¹ was conducted by all five permanent members of the United Nations Security Council. No thorough assessment has as yet been done.

Independent assessments are particularly important, especially given the fact that official dose estimates have tended to be systematically lower than independent ones.

A thorough accounting in the context of the Treaty on the Prohibition of Nuclear Weapons (TPNW) would be appropriate because the treaty is centered on humanitarian law and makes special reference to women, children, and indigenous people who have been disproportionately affected.¹⁵² Multigenerational impacts of should be a high priority of any such effort.

¹⁵⁰ Hochschild 1998

¹⁵¹ IPPNW and IEER 1991, Chapter 3.

¹⁵² TPNW 2017.

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