How the Science of Radiation Biology Can Help Reduce the Crippling Fear of Low-level Radiation

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Abstract—**The fear of radiation has been present almost since the discovery of radiation, but has intensified since the "dawn of the atomic age" over 75 y ago. This fear has often served as an impediment to the safe and beneficial uses of radiation and radioactive material. The underlying causes of such fear are varied, can be complex, and are often not associated with any scientific knowledge or understanding. The authors believe that a clear understanding of the current scientific knowledge and understanding of the effects of radiation exposure may be useful in helping to allay some of the fear of radiation. This manuscript attempts to (1) address several scientific questions that we believe have contributed to the fear of radiation, (2) review the data derived from research that can be used to address these questions, and (3) summarize how the results of such scientific research can be used to help address the fear of low-dose and low-dose-rate radiation. Several examples of how fear of radiation has affected public perception of radiological events are discussed, as well as a brief history of the etiology of radiation fear. Actions needed to reduce the public fear of radiation and help fulfill the full societal benefits of radiation and radioactive materials are suggested.**

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Key words: public information; radiation, low-level; radiobiology; risk analysis

INTRODUCTION

The fear of radiation[5] to some extent has been present almost since the discovery of radiation in 1896. However, this fear, aka "radiophobia," has intensified since the "dawn of the atomic age" over 75 y ago. This fear has often served as an impediment to the safe and beneficial uses of radiation and radioactive material. The underlying causes of such fear are varied, can be complex, and are often not associated with any scientific knowledge or understanding. However, the authors do believe that a clear understanding of the current scientific knowledge and understanding of the effects of radiation exposure may be useful in helping to allay some of the fear of radiation. To that end, in this manuscript, the authors address several questions that we believe have contributed to the fear of radiation.[6]

BACKGROUND

In the years following the discovery of radioactivity and its resultant radiation, many radioactive materials were sold to the public in drinks and food supplements with claims that these materials could cure almost any ailment, including many types of diseases. Extensive studies were done using radiation to cure ring worm (Shore et al. 2002), inflammatory diseases like ankylosing spondylitis and arthritis (Kuhns and Morrison 1946; Calabrese 2018), and other inflammatory diseases (Calabrese 2018). At that time, there seemed to be beneficial effects from the radiation treatment. Many of these studies were not scientifically based and were carried out at high doses, which resulted in not only some evidence for curing the disease but also an increased cancer frequency. As time went on, it became apparent that the high doses of radiation used in some of these treatments were also increasing the frequency of cancer and other adverse effects. For example, radiation for ring worm increased the incidence of skin cancer (Shore et al. 2002), and children treated for enlarged thymus glands had a significant increase in thyroid cancers and increased cancers in the head and neck (Modan et al. 1974; Shore et al. 1993).

Data were also becoming available on the adverse effects of radiation from occupational and medical exposures.

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[5]All references in this manuscript to "radiation" should be inferred as ionizing radiation.

[6]It should be noted that the authors are working on a related manuscript that will more broadly address the history and underlying causes of the fear of radiation.

The classic case was the increase in bone cancer in radium dial painters, where the young women who were painting the dials of watches would lick the brush to provide a sharp point. During this process, they were taking in large amounts of radium. After very large doses to the bone, the frequency of bone cancers in this worker population rose dramatically. However, there was a well-documented (Evans 1966; Jee 1976; Rowland 1994) threshold dose below which no increase in bone cancers were observed.

Early studies were also conducted on radiation-induced mutations in fruit flies that demonstrated a linear response of mutations with dose, with no influence of sex or time of exposure (Muller 1927). Although these data were generated using only high radiation doses (millions of times higher than background doses), the linearity of the response was assumed to hold at low doses, even in the face of contrary data (Russell 1965), and were instrumental in forming the linear no threshold dose-response model (LNT) for the induction of mutations, which was later used as the general model for cancer induction due to radiation exposure.

Extensive research from the US Department of Energy (US DOE) Low Dose Radiation Research Program (LDRP) has demonstrated that at the cellular and molecular level, the responses due to low-dose radiation are very different than the responses due to high doses (Brooks 2018). Many of these low-dose responses are thought to be protective since cancer cells are killed by the process of apoptosis (Bauer 2007). Cell transformation following low doses of radiation has been shown to be lower than the control levels (Redpath et al. 2003), and many metabolic pathways are altered to produce chemical and biochemical species that are protective (Spitz et al. 2004). Several international studies (e.g., Tubiana 2005; Averbeck 2009) have suggested that the LNT model overestimated cancer risk. Still, with little human data on the late effects of radiation exposure, the LNT was generally thought to be conservative for establishing regulations for radiation exposure (NAS/NRC 2006; McClellan 2014).

The US DOE LDRP has raised some serious questions regarding the implied potential for detrimental late effects of low-dose and low-dose-rate exposures to radiation. Many of these questions concerned the effects of internally deposited radioactive materials, which not only resulted in a low dose rate but a non-uniform dose distribution in the body. Extensive research was initiated in several Department of Energy National Laboratories to address these questions and has been nicely summarized in two very extensive reviews (Stannard 1988; Thompson 1989).

SCIENTIFIC QUESTIONS CONTRIBUTING TO THE FEAR OF RADIATION

This manuscript attempts to (1) address several scientific questions that we believe have contributed to the fear of radiation, (2) review the data derived from research that can be used to address these questions, and (3) summarize how the results of such scientific research can be used to help address the fear of low-dose and low-dose-rate radiation.

We readily note that extensive studies on the generation of the fear of radiation have been conducted and show that the fear is, for the most part, not generated or modified in the face of scientific data (Slovic 1996; Flynn and McGregor 2003). Nevertheless, we feel that a necessary first step in easing public fear of low-dose radiation is to document the scientific data and knowledge that clearly reveal such fear to be unjustified. This belief is supported by a recent report issued by the Subcommittee on Physical Sciences for the Committee on Science of the National Science and Technology Council (USNSTC 2022). This report was written to support coordination among Federal agencies for radiation biology research with a goal of reducing the uncertainties in the health risks posed by low-dose radiation. This report further states that: "Resolving scientific uncertainties of the health impacts of low-dose radiation could also alleviate fears among some members of the public on the associated environmental, occupational, or sociological impacts, although this may be a significant challenge."

Of major importance, this unjustified fear of radiation has resulted in crippling impediments to the myriad public benefits provided by radiation. Hence, it is instructive to present this material in a historical manner to demonstrate how the fear developed over time and why. Much of this fear was based on misunderstanding or even misinformation associated with several major questions. Some of these major questions, a total of five, are given below. Many of these questions made front-page headlines in the news media at the time and stimulated the fear of radiation. We then review the scientific research that addresses these questions and finally discuss the results that demonstrate such fear is not founded in scientific knowledge and understanding.

- Question 1: Are the long-term risks for genetic damage and cancer from radiation almost equal? Does radiation exposure increase the genetic load and result in long-term genetic degeneration of the human genome?
- Question 2: What is the radiation-induced cancer risk as a function of dose following a single acute radiation exposure?
- Question 3: What are the risks from internally deposited radioactive material? This includes the risks from fallout produced by atmospheric nuclear weapons testing, the risk for bone cancer and other health effects induced by exposure to ^{90}Sr and its decay product ^{90}Y, the risk of thyroid cancer from exposure to ^{131}I, the risk of liver cancer, and the risk of ^{239}Pu. Is ^{239}Pu really the most hazardous substance known to man?
- Question 4: Can molecular markers be used to help predict the risk of cancer? What can research at the cellular and

molecular level tell us about the risk from cancer and the mechanisms involved in low-dose radiation-induced cancer?
- Question 5: What are the costs to society—driven in a great part by the fear of radiation—and how does fear of radiation impact the economy, health, and well-being of exposed populations?

SCIENTIFIC RESEARCH AND RESULTS

Question 1: Are the long-term risks for genetic disease and cancer from radiation almost equal? Does radiation exposure increase the genetic load and result in long term genetic degeneration of the human genome?

Early after the dropping of the atomic bombs on Japan, the national and international standard-setting bodies predicted risks for radiation-induced damage from genetic effects to be almost equal to the risk for the development of cancer. This prediction was in large part dependent on the early data on mutations in drosophila (fruit flies) where there was a proposed linear dose dependence for mutation induction with little dose-rate effect or other modifiers (Muller 1927). However, as research progressed (still continuing today) on the induction of mutations in the children of survivors following the atomic bomb (Shull and Neel 1981; Neel et al. 1990), it was determined that there is no evidence for low-level radiation-induced mutations in humans (Kodaira et al. 2010; Yeager et al. 2021). This result was bolstered by research in mice. A mega-mouse study to evaluate radiation induced mutations in a mammalian species was conducted at Oak Ridge National Laboratory. The experimental design is shown in Fig. 1. This figure demonstrates the large number of animals involved in the study and the low frequency of mutations observed.

This research in mice further demonstrated a marked dose-rate effect (Russell 1963) and revealed that there was a sex difference, with female mice more sensitive to radiation-induced mutations (Russell 1965; Hsu et al. 1991). These observations were instrumental in modifying the risks associated with genetic damage and became an important part of our current standards, where the risk from radiation-induced genetic effects is much less than the risk from radiation-induced cancer (NAS/NRC 2006).

Additional research found that there were mutations in clones of cells and small groups of animals, both in the control (unexposed) and exposed populations (Selby 1998). Unfortunately, the impact of these observations was not brought to the regulatory community. The bottom line is that genetic effects of radiation have been grossly overestimated in the past. This overestimation may play an important factor in the increased fear of radiation. Even the perceived risk of a child with mutations that limit their ability to have a full and happy life could cause fear. The research was definitely effective in demonstrating that radiation-induced mutations have not been detected in humans (Neel et al. 1980; Schull and Neel 1981). This finding should have decreased the public fear of genetic damage from radiation. Scientific evidence determined that radiation is a poor mutagen and represents little risk to humans.

Another of the early worries associated with the genetic effect of radiation was the concern over the integrity of the human genome. Each of us carry a number of genes that are detrimental. These "bad" genes are for the most part negated in our offspring by "good" genes from our spouse. Would radiation exposure increase the number of bad genes in the gene pool or increase what was called the "genetic load?" This genetic load was postulated to be carried from generation to generation, and with increased radiation exposure, would it continue to increase? Such a change would of course result in an increase in abnormalities in the human population, and it was postulated that radiation exposure

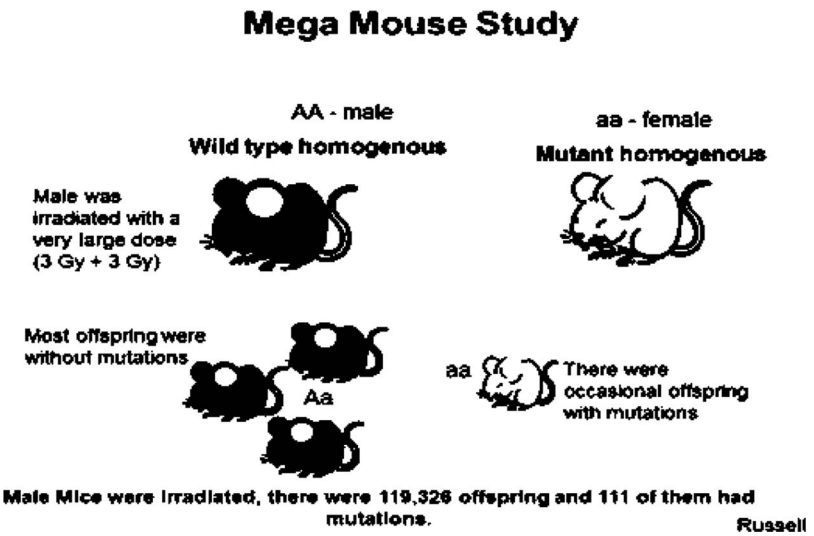

Fig. 1. Experimental design for Oak Ridge Nation Laboratory Mega Mouse Study.

would potentially result in a huge amount of genetic damage and human suffering in each successive generation.

This question was of great concern to the scientific community, and research was initiated to address this issue at the Los Alamos National Laboratory in New Mexico (Spalding et al. 1969). Spalding conducted a well-designed study in mice that was carefully conducted over many generations to determine if it would be possible to detect any change in the genetic load as a function of radiation-induced changes in each generation. The design of this study is shown in Fig. 2.

In this study, male mice were given doses close to the level that would result in sterilization—about 2.0 Gy. These irradiated males were bred to unexposed females, and genetic changes were monitored in the next generation. This process was repeated with the males from the offspring exposed to the same dose and mated to the offspring of the first-generation exposed females. This pattern was repeated for over 20 generations, and markers of genetic damage such as reduced litter size, fetal deaths, abnormal offspring, change in weight of the offspring, or any markers of genetic damage such as coat color, abnormal fetal development, or any other changes in offspring were monitored carefully in each generation. By the end of this long and vigorous study, there were no signs of any increase in the "genetic load." During the process of development, bad genes were carefully and automatically selected out so that they did not increase in the next generation.

What would have been a major reason for concern over the genetic effects of radiation with a potential for a serious outcome was shown scientifically not to exist. Thus, the fear associated with this hypothesis was not and is not currently justified. It is important to recognize that such a fear has no scientific basis and must be discounted.

Question 2: What is the radiation-induced cancer risk as a function of dose following a single acute whole body radiation exposure?

At the time the atomic bombs were dropped on Japan, there was almost no information on the long-term or late effects of radiation in terms of late-occurring diseases like cancer, genetic disease, heart disease, or stroke. This lack of information led directly to an increase in the fear of radiation, as the bombs killed a large number of people from blast, burns, and acute radiation exposures. The two bombs killed close to 200,000 people. Most members of this population had multiple insults generated by the bombs. The blast effects extended out almost 5 km from the center of the bomb, whereas the radiation doses were significant in areas less than 3 km. Thus, if an individual survived the burns and blast at distances closer to the bomb, they also were exposed to varied doses of radiation.

Additional information was badly needed on how the cancer frequency would change as a function of age at exposure and time after exposure. Since this population has now been followed for over 80 y (see https://www.rerf.or.jp/en/), most of the study populations have died. The results of these

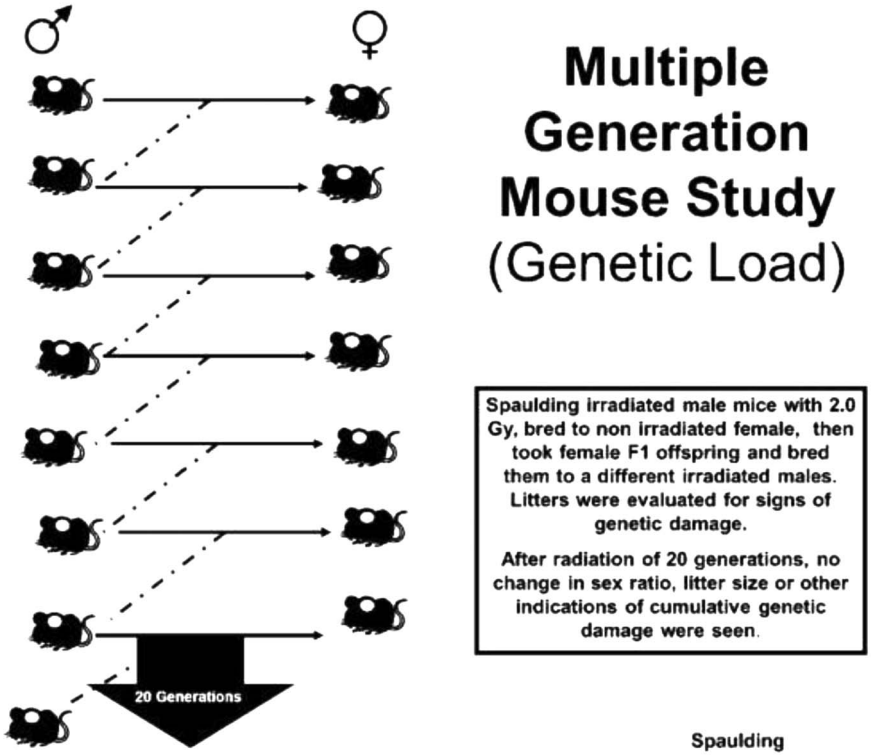

Fig. 2. Design of the Los Alamos National Laboratory Multiple Generation Mouse Study.

studies have been published many times over the years. The scientific community has for the most part accepted the fact that at high doses, there is a linear dose-response relationship, and the radiation-induced cancer frequency is approximately 5% per Sv. Those involved in such studies worked very hard to determine the radiation dose experienced by the survivors, and they have carefully determined the cancer frequency and mortality in the exposed and control populations (Preston et al. 2007).

However, the radiation exposure that is most frequently encountered in occupational and environmental doses is in the mSv range—in many cases comparable to the range in natural background radiation. Hence, even using a LNT extrapolation to these lower dose ranges, the ability to detect changes in cancer frequency at doses less than 0.1 Sv is limited by the population size. The Million Man studies are currently under way to study the potential for an increased cancer frequency in this low-dose region when the dose is delivered over a protracted length of time (Boice et al. 2019a). As of this writing, the studies to date indicate there is no increase in cancer frequency in the atomic veterans (Boice et al. 2020), and there is no observed sex difference (Boice et al. 2019b).

The take-home message from the early results of the Million Man studies is the apparent absence of a relationship between radiation dose and radiation-induced cancer in some important populations. These include the atomic veterans, nuclear navy, and nuclear power plant workers. With extensive and proper communication, the risk numbers generated from these studies could and should alter radiation standards. As such, modifications to standards based on these scientific data should be very important in decreasing the fear of low levels of radiation.

In contrast to these recent results, the atomic bomb data suggested that women were as much as three times more sensitive than men to radiation-induced cancer and there was a suggestion that there was an increase in cancers in the atomic veterans. Hence, regulations have been set using these old data and can now be brought up to date. This should decrease the concern associated with low doses of radiation.

Question 3: What are the risks from internally deposited radioactive material?

Related questions:

- Question 3a: Has the dose from internally deposited radionuclides from atmospheric nuclear weapons testing fallout caused an increase in cancer frequency in populations exposed to higher than natural background doses?
- Question 3b: What is the risk of bone cancer and other health effects induced by exposure to ^{90}Sr and its decay product ^{90}Y?
- Question 3c: What is the risk for thyroid cancer from exposure to ^{131}I?
- Question 3d: How does the risk of lung cancer change as a function of type and dose of radioactive material inhaled?
- Question 3e: Is ^{239}Pu the most hazardous substance known to humans?
- Question 3f: Does the exposure to small depositions (e.g., hot particles) of ^{239}Pu or other alpha-emitting radionuclides increase the risk as a function of dose to the cells near the deposition or as a function of the average dose to the organ?

3a: Has the dose from internally deposited radionuclides from atmospheric nuclear weapons testing fallout caused an increase in cancer frequency in populations exposed to higher than natural background dose? Even with extensive human data on the risk from a single external whole-body exposure to radiation, a major question remained for the scientific community. Was the cancer risk greater from the low dose-rate (i.e., non-uniform distribution internal exposure to nuclear testing fallout radionuclides or from the radionuclides produced by nuclear power production) than the risk calculated and extrapolated from acute, single high-dose-rate exposure, external, uniform, exposure experienced by those that survived the atomic bombs in Japan? The concerns and fears raised by these early data helped trigger an extensive research effort in all of the national laboratories and many specialty laboratories to determine how and if internally deposited radioactive materials resulted in cancer and if the frequency or risk was higher or lower than predicted from the atomic bomb data. Years of research on this major question have been nicely summarized (Stannard 1988; Thompson 1989; McClellan 2014). The results of all these studies can be summarized in a single word—**No**! There was not a single scientifically documented case where the internally deposited radioactive materials increased the cancer rate above the rate that was predicted and calculated using the single exposure data from the atomic bomb.

3b: What is the risk of bone cancer and other health effects induced by exposure to ^{90}Sr and its decay product ^{90}Y? Strontium-90 is a major long-lived radionuclide that was present in the nuclear testing fallout, some nuclear accidents, and in nuclear waste. Following the development of atomic weapons, many studies were conducted to follow the movement of ^{90}Sr through the environment and its ultimate deposition in the bones (Durbin et al. 1956; Durbin and Jones 1958; Durbin 1975). The presence of ^{90}Sr in humans as the result of the nuclear testing fallout triggered front page news, as many books and scientific papers reported the presence and the amount of activity in humans. The major pathway for the ^{90}Sr to move from the environment to humans is though milk.

Cows that ate contaminated feed would pass the radionuclide into their milk, which would be available for human consumption, particularly by children. Additionally, the milk of mothers who were breastfeeding could become contaminated with ^{90}Sr due to consumption of contaminated foodstuffs by the mother. These pathways could lead to an increased level of ^{90}Sr in the milk and body of newborns and children (Pendleton et al. 1963). These scientific data, along with significant media coverage, contributed to the public fear of ^{90}Sr.

Unfortunately, this fear was stimulated by misrepresentation of data by some self-appointed "experts." It was suggested that the contamination with ^{90}Sr was responsible for many deaths in newborn infants (Sternglass 1963). Sternglass observed that the death rate for newborn babies had decreased at a rather constant rate over a number of years. About the time of the first atomic bomb explosion (1945), there was a plateau with little decrease in the death rate in newborn babies. He postulated that the plateau was caused by the atomic bombs and that this lack of further decrease was related to ^{90}Sr in the human population. This speculation showed up on the front page in the news media touting that ^{90}Sr kills many thousands of babies. This open literature publication was carefully reviewed and refuted with many scientific publications and presentations clearly showing that the derived conclusions were not valid. Because of this, future Sternglass publications were not accepted in the scientific literature; rather, they were only accepted in popular journals.

These popular articles (e.g., Sternglass 1969) were designed to stimulate public fear and were carefully and completely refuted by the scientific community. Strontium-90 is concentrated in the bone with no evidence of dose or adverse effects during fetal development, and the doses from this radionuclide were lower than natural background in many US cities, refuting the data presented by Sternglass (1963). In these many scientific studies, there was no evidence that fetal development was altered by the intake of ^{90}Sr, and no infant mortality could be attributed to the presence of ^{90}Sr in the population.

The major concern associated with ^{90}Sr was the induction of bone cancer from the long-term deposition and dose to the bones. This concern was addressed via lifetime feeding studies using beagle dogs conducted at the University of California, Davis. Researchers fed the radionuclide to the females during pregnancy and continued with the feeding through the life of the animals, including following each animal over its life to determine the cause of death (Fig. 3). This figure shows that when plotted on log scales, for both injected activity and time, an increase in bone cancer was observed only for long times after injection and only for very large activities (i.e., for doses greater than 10 Gy) to the skeleton. Thus, a threshold was demonstrated in these lifespan dog studies, which supported the dose thresholds for radiation-induced bone cancer seen in the human radium dial painters (Evans 1966; Jee 1976).

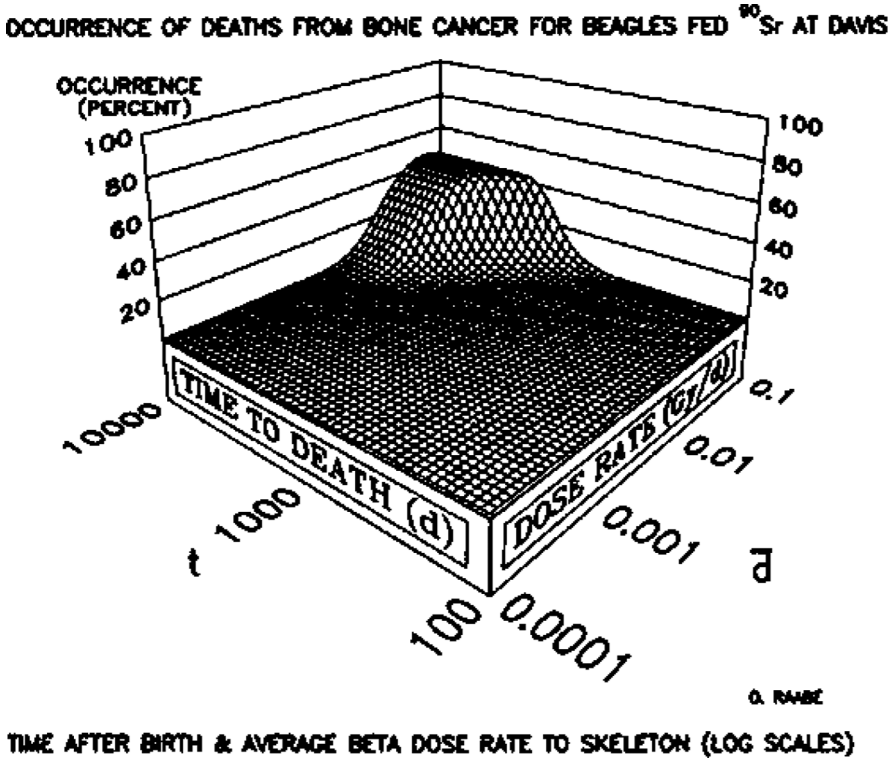

Fig. 3. Induction of bone cancer from ^{90}Sr–^{90}Y. Effective thresholds for time of exposure, dose, and dose rate.

These studies have been summarized and demonstrated that there were effective thresholds below which no increase in cancer rate could be observed. It required very high levels of ^{90}Sr to induce any increase in bone cancer (Raabe 1987, 2010, 2015). The dose to the bone can be calculated from the graph (Fig. 3) by multiplying the dose rate per day by the number of days. When we do this, we see that a dose rate of 0.001 Gy d^{-1} times 10,000 d results in 10 Gy dose, where no increased bone cancer was observed. A similar threshold at a very large dose can be obtained by multiplying 0.1 Gy d^{-1}by 1,000 d. The results of these extensive life-time studies in dogs were very helpful in demonstrating a threshold for bone cancer risk for ^{90}Sr. In fact, low doses of ^{90}Sr produced less bone cancer than was observed in the control animals (Raabe 2015). These observations, as well as observations of human populations that were exposed to ^{90}Sr at the Techa River in Russia (where the dose to red bone marrow was as high as 2 Gy) (Degteva et al. 2012), revealed a significant increase in solid cancers but no detectable increase in bone cancer, the target organ for ^{90}Sr (Napier 2014). Napier suggested that "The studies do confirm that radiation is a weak carcinogen." However, he then suggested, "A significant result of most of the studies is that internal doses protracted over many years seem to be just as important as instantaneous external doses" (Napier 2014). The latter statement would seem to support the LNT. However, these data are not supported by the controlled dog data that required very large doses ($\geq$10 Gy) to produce a significant increase in bone cancer. Other human populations that had radioactive materials in the bone, such as the radium dial painters, clearly demonstrated a threshold in the level of radiation to the bone at about 10 Gy, below which no increase in cancer of the bone could be observed (Rowland 1994; Evans 1996).

3c: What is the risk for thyroid cancer from exposure to ^{131}I?

Fallout from Nuclear Weapons tests: One of the major radionuclides associated with fallout from nuclear weapons or nuclear reactor accidents is ^{131}I. As a result of nuclear weapons testing, much of the world has been exposed to this radionuclide. It concentrates in the thyroid gland and results in a significant dose to this organ. For some people living in Utah who were exposed to fallout from the nuclear weapons testing at the Nevada Test Site, ^{131}I concentrations were measured in cow's milk and calculations were made to determine the thyroid dose in people. The highest doses measured were 0.84 Gy resulting from the ^{131}I deposited in the thyroid of children in some farm families who consumed milk from their cows within the fallout zones (Pendleton et al. 1963). These observations resulted in extensive news coverage and suggested that ^{131}I had the potential for the induction of thyroid cancer. However, studies of children exposed to the higher levels of ^{131}I in Utah were compared to children with low levels of ^{131}I in Arizona (Weiss et al. 1971; Lloyd et al. 1990), and the results showed no significant increase in leukemia (Lloyd et al. 1990), thyroid cancer, or thyroid nodules (Weiss et al. 1971) in the children with the higher levels of exposure to ^{131}I.

Medical use of ^{131}I: Iodine-131 is widely used in medicine as a tool to measure thyroid function as well as to detect and treat thyroid cancer. The widespread use of this radionuclide in medicine has confirmed the observation that the adult thyroid is very radiation resistant. International bodies have set a tissue weighting factor for the thyroid as 0.03 (US NRC 1992; ICRP 2003, 2007; NCRP 2008), suggesting that it is rather radiation resistant for cancer induction.

Whereas there is ample scientific data demonstrating that the thyroid is very radiation resistant in adults, there is complementary data revealing that ^{131}I can produce cancer in children following high radiation doses. Hence, care must be taken to avoid exposure of children to this radionuclide. However, fear in the adult population, particularly over 40 y of age (US FDA 2022), is not justified.

Controlled releases of ^{131}I: During the production of plutonium at Hanford, WA, there was a controlled release of ^{131}I into the environment. This was called the Green Study. This release was conducted to test instruments designed to detect trace amounts of ^{131}I released by secret nuclear weapons tests performed by other nations. Extensive follow-up and epidemiological studies of the exposed population showed no increase in thyroid cancer (Davis et al. 2004).

Reactor Accidents: There have been three major reactor accidents where substantial levels of ^{131}I were released to the environment: Three Mile Island, Chernobyl, and Fukushima. The levels of ^{131}I following Three Mile Island and Fukushima were low enough that no increase in thyroid cancer was predicted or found. Extensive follow-up and testing for thyroid cancer in the population following Chernobyl found that there was an increase in thyroid cancer in children but no increase in adults. The thyroids of children were found to be more sensitive to the induction of cancer than adults (Chernobyl at Twenty 2007). It was calculated that there was an increase of about 7,000 childhood thyroid cancers in populations exposed to the ^{131}I from the Chernobyl accident with no increase reported in adults (ICRP 2012). The radiation doses to these children were in some cases very high. The population in Pripyat, which is located very close to the Chernobyl reactor, was not evacuated for almost a week after the accident. This resulted in some extensive radiation doses to the thyroid. Fortunately, only seven deaths actually resulted among the calculated 7,000 children with radiation-induced thyroid cancer. Thyroid cancer is easily treated, and the survival rate is high. Still, the fear of developing thyroid cancer in adults remains high. For the general public, particularly children and adults up to age 40, the use of potassium-iodide (KI) pills to increase the level of normal

iodine and decrease the deposition of ^{131}I may be recommended following a nuclear release dependent upon the projected level of exposure.[7]

3d: How does the risk of lung cancer change as a function of level and duration of exposure for inhaled beta-gamma emitters? When ^{90}Y, ^{91} Y, ^{144}Ce, or ^{90}Sr are trapped in a fused clay matrix, inhaled, and deposited in the lungs, the biological half-life is very long so that the effective half-life for clearance from the lung is dependent on the physical half-life of the radionuclide. This set of radionuclides has a wide range of physical half-lives resulting in effective half-lives varying from 2.5 to 600 days. Extensive studies on the biological effects of these inhaled beta-gamma emitting radionuclides were conducted at the Lovelace Inhalation Toxicology Laboratory (McClellan 2014). The induction of radiation pneumonitis following the inhalation of beta-gamma emitters in fused clay particles required 1,000 times more radiation dose than was needed to produce the same disease following acute exposure to ^{60}Co gamma rays (Scott 1980). It was also determined that radiation doses from inhaled beta-gamma emitters in fused clay matrix must be greater than 10 Gy to induce an increase in the frequency of lung cancer (Puukila et al. 2017, 2018). If the radiation dose was below the level required to create an increase in lung disease, the frequency of radiation-induced cancer was not significantly different from the level seen in the controls. The induction of lung cancer by protracted radiation from inhaled beta-gamma emitting radionuclides was closely linked to cell killing, inflammatory disease, and tissue disorganization. Such data demonstrate that biological damage from inhaled beta-gamma emitting radionuclides requires a very large dose rate and dose distribution factors and are much less effective than single acute radiation exposures. As with beta-gamma emitting radiation, the risk from inhaled ^{239}Pu was found to be lower than implied by the radiation weighting factor of 20 currently most commonly used. Extensive research has been conducted on the biological changes and the risk from inhalation of ^{239}Pu. The results are briefly reviewed in the next section.

3f: Is ^{239}Pu the most hazardous substance known to humans? In the early days of radiation biology, a common quote was that "plutonium is the most hazardous substance known to man." Relative to ^{239}Pu, the long physical half-life (24,000 years), the uptake and retention in the bone and liver, and its long biological half-life in these tissues (Durbin 1975) were the bases for this claim. These characteristics would result in a significant radiation dose. However, on closer examination of the data, the pathways from the environment to man are limited. Gut absorption of ^{239}Pu is very limited, 0.05% (Hamilton 1948), or expressed as the percent of the administered dose given depending on the compound: nitrate 0.003; chloride 0.007; oxide 0.0001 (Bair et al. 1974; Stannard 1988). Thus, ingestion is not an important pathway for incorporation of this radionuclide into the body. The primary pathway for movement of this alpha emitter from the environment to humans is when it is incorporated into the body by inhalation or wounds (Jee 1976; McClellan 2014). Thus, respiratory protection is required when handling ^{239}Pu and other alpha emitters.

The cancer hazard associated with ^{239}Pu is dependent on the radiation dose, dose rate and dose distribution (Jee 1976; Brooks et al. 1983; Park et al. 2012; McClellan 2014). This research has demonstrated that ^{239}Pu is not unique and that the biological response to the alpha particle from ^{239} Pu is the same as any other alpha emitter. Stated in a simple way, if a cell in the respiratory tract, bone, or liver gets "hit" by an alpha particle from ^{239}Pu or an alpha particle from almost any other alpha emitter, for example radon, the response is the same, and the damage produced and response of the body are the same.

Research suggested that the initial chromosome damage from alpha-emitting radionuclides per unit dose is about 20 times the frequency of chromosome aberrations as the same dose from a beta- or gamma-emitting radionuclide or external exposure to gamma rays (Brooks 1975). Such data was combined with other information by standard setting groups resulting in a radiation weighting factor of 20 being used when comparing the risks from alpha emitters to that from beta-gamma exposures (NCRP 1990; US NRC 1992). However, extensive studies in beagle dogs demonstrated that the important risk (i.e., for radiation-induced cancer) from ^{239}Pu is only about a factor of 6 to 10 times as effective as radiation exposure to low LET radiation (Muggenberg-et al. 2008; Park et al. 2012; McClellan 2014). These data have been compared to human data following exposure at the Mayak facility in Russia (Gilbert et al. 2007, 2013a and b) that occurred during development of atomic weapons. When these data were compared to those in the dog, there was rather good agreement between them (Wilson et al. 2010), suggesting that the dog is a rather good model for predicting cancer risks in humans. Restating, recent scientific data suggest that we have overestimated the health risk associated with the exposure to ^{239}Pu by a factor of at least 2 to 3.

All these data demonstrate that ^{239}Pu is not the most hazardous substance known to humans. Rather, the risk from exposure to it is the same as exposure to any other alpha-emitting radionuclides. There should be no unique concern regarding the well-established risk from inhaled ^{239}Pu.

3 g: Does the exposure to small depositions (e.g., hot particles) of ^{239}Pu or other alpha-emitting radionuclides

[7]US Food and Drug Administration. Frequently asked questions on potassium iodide (KI). Undated. [online]. Available at https://www.fda.gov/drugs/bioterrorism-and-drug-preparedness/frequentlyasked-questions-potassium-iodide-ki. Accessed 31 August 2022.

increase the risk as a function of dose to the cells near the deposition or as a function of the average dose to the organ? Small radioactive particles of ^{239}Pu are generated in many nuclear accidents and by atomic weapons detonations. When a plutonium particle is deposited in the lung, the cells close to this particle are exposed to the short-range alpha particles (about 40 μm in soft tissue) released by the ^{239}Pu. These cells receive very high radiation doses. These high cellular doses are present even when the average dose to the whole organ could be very low. It was postulated that this non-uniform radiation dose distribution from alpha-emitting particles could result in a very large cancer risk. But this is only true if there is a causal relationship between localized cellular dose and cancer risk. This was known as the "hot particle hypothesis" (Tamplin and Cochran 1974). If this hypothesis was scientifically verified and supported, the currently accepted risk for cancer from ^{239}Pu particles would be grossly underestimated, potentially making commercial nuclear power less acceptable.

Extensive research at both the cellular and whole animal level has been conducted and summarized (McClellan et al. 1986; Bair 1974). Fig. 4 is an autoradiograph of hamster liver injected with either plutonium citrate or particles of plutonium oxide. It shows the non-uniform distribution of dose in the liver. It was demonstrated that ^{239}Pu was equally effective in producing chromosome damage in the liver (Brooks 1975) regardless of the dose distribution from citrate or hot particles. For the induction of liver cancer (Brooks et al. 1983; Guilmette et al. 1989a and b), when ^{239}Pu was given in the citrate form where the distribution of the dose was uniform and where almost all the cells were "hit" by alpha particles, the latent period and the total incidence of liver cancer was higher than when ^{239}Pu was given as oxide particles of different particle sizes with no difference observed as a function of particle size. The number of cells "hit" by alpha particles was dependent on the particle size. Large particles of $^{239}PuO_2$ resulted in very few cells being "hit" compared with small particles or citrate where many or most of the cells had alpha particle interactions or "hits."

Such data were evaluated when the National Council on Radiation Protection & Measurements (NCRP) considered the influence of "hot particles" on radiation risk (NCRP 1999). With the discovery of the bystander effects (Barcellos-Hoff and Brooks 2001; Geard et al. 2002), it was demonstrated that organs respond as a whole to radiation insult and not as single cells; thus, bystander effects occur both in vivo and in vitro (Brooks 2004). These studies demonstrated that the "hot particle hypothesis" was not scientifically validated or accurate. The cancer risk from ^{239}Pu was derived using uniformly distributed ^{239}Pu and was an accurate representation of the cancer risk regardless of micro dose distribution (Brooks 2004). Thus, the current risk numbers used to regulate exposure to ^{239}Pu are conservative and acceptable and do not need to be revised to account for nonuniform distribution of the alpha emitters. Scientific data were used to show that hot particles are not a major concern and again provided data that should decrease the level of fear associated with the use of nuclear energy.

Question #4: Can cellular and molecular markers be used to predict the risk of cancer? What can research at the cellular and molecular level tell us about the risk from cancer and the mechanisms involved in low-dose radiation-induced cancer?

Ever since the discovery of radiation there has been an attempt to find the cellular and molecular basis for the interaction of radiation with biological materials.

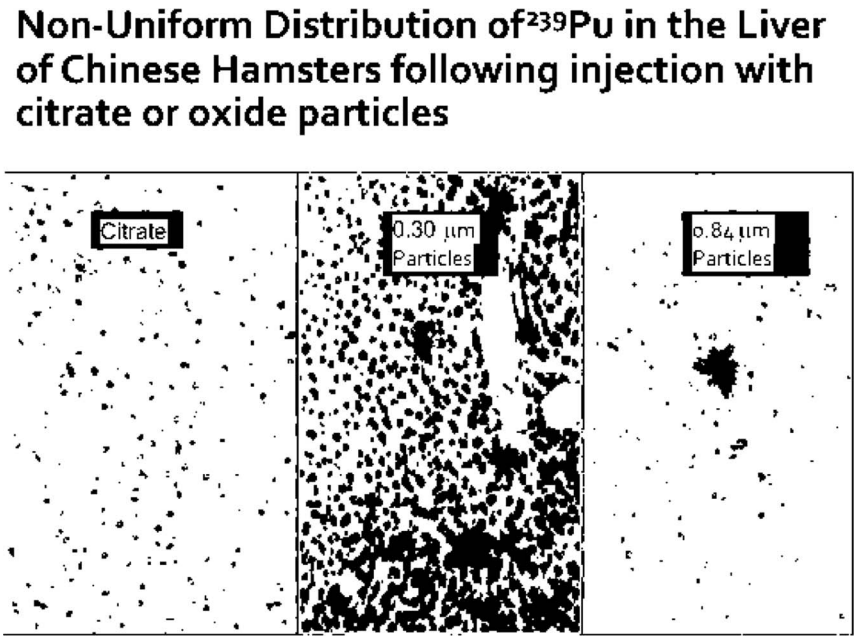

Fig. 4. Dose distribution of plutonium in the liver of Chinese hamsters.

Funding for the US DOE Low Dose Radiation Research Program (Brooks 2018) provided the first serious allocations of funds focused on the cellular and molecular changes induced by low doses of radiation. In this program, the research was required to include biological changes induced at or below 0.1 Gy. Much of the previous research for measuring changes in cellular and molecular biology was conducted at much higher doses. With this research money as a stimulus, extensive research and new findings were possible in the low-dose region using newly developed molecular and cellular techniques as well as the development of many different techniques and equipment. All this made it possible for measurements in the low-dose region where it was not possible in the past (Brooks 2018). As the result of this focused research program in the low-dose region, many of the current paradigms in radiation biology were challenged (Brooks 2005, 2018; Dauer et al. 2010; Tharmalingam et al. 2019). The shape of the cell-killing curves and the foundation of the "hit theory" was not the same as was previously predicted. It was noted that following very low doses of radiation, cells exhibited a low-dose hypersensitivity with excess cell killing in the low-dose region that had not been appreciated in developing the "hit theory" (Marples and Collins 2008). This observation, along with several other major scientific observations such as bystander effects and the response of tissues (not individual cells), resulted in a major challenge of the "hit theory" (Schwartz 2004).

The research funded by the US DOE Low Dose Radiation Program resulted in three major changes and observations that directly impacted current radiation paradigms. These are bystander effects, genomic instability, and adaptive response. All three of these changes are influenced by the genetic background of the system under investigation. This is true at all levels of biological organization from the molecular to humans. These unique observations are summarized in Fig. 5 and are discussed below.

Bystander effects The development of microbeams made it possible to expose selected individual cells to known numbers of alpha particles or other forms of radiation. With this tool, it became possible to measure biological responses in the cells traversed by an alpha particle or other forms of radiation as well as the "bystander" cells that were not "hit." These studies were critical in establishing the bystander effect.

There were two unique types of bystander effects: (1) where the cells are in direct contact with each other with direct communication (Azzam and Little 2004; Belyakov et al. 2005), and (2) where a substance is released into an organism (or culture dish) and has impact on cells located at distances from the "hit" cells (Morgan 2003). The dose response relationships for the induction of bystander effects are nonlinear and can result in thresholds and plateau effects. Again, one of the bottom lines from this research is that tissues respond as a whole and not as single cells.

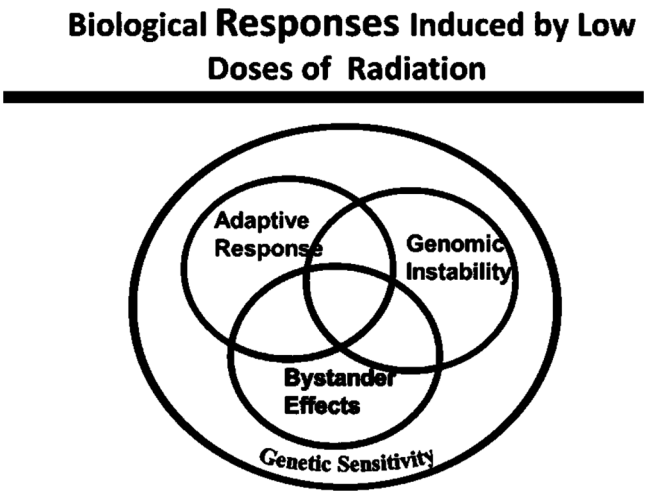

Fig. 5. New observations from the US DOE Low Dose Research Program that must result in paradigm shifts.

Genomic instability There are many direct and immediate responses from cells and tissues following exposure to radiation such as cell killing, chromosome aberrations, mutations, and DNA damage (Lavin et al. 2005; Huang et al. 2007). These have been extensively studied and related to radiation conditions such as dose, dose rate, and radiation type. However, the induction of cancer is a delayed response, with years between the initial exposure and the development of cancer. All the initial responses appear to be repaired and are no longer measurable or evident when the cancers develop. Thus, it has been hard to link initial events to cancer outcome.

It has been observed that multiple genetic changes occur during cancer development, and these seem to be related to the loss of genetic control or genetic stability of the cell. Genomic instability is one of the hallmarks of the cancer process (Hanahan and Weinberg 2011). Radiation-induced genomic instability was first observed and defined as an increase in the acquisition of genetic alterations including chromosome aberrations (Morgan et al. 1996).

Adaptive response Early in radiation research, it was suggested that low doses of radiation may be protective. This protective effect was called "hormesis" (Luckey 1990), where low doses of radiation result in less damage than observed in the normal background. Further research demonstrated that when a small "tickle" dose was given before a large "challenge" dose, the frequency of chromosome aberrations induced in response to the challenge dose was reduced by almost a factor of two (Wolff 1995). This response was termed to be an "adaptive response" and opened a whole new area of research.

As the result of this and other research, it has been demonstrated in many cellular and molecular studies that low doses of radiation may be protective and could reduce the risk of cancer to levels below that seen without radiation and result in cancer frequency below the background cancer rate (Calabrese et al. 1999; Calabrese and Baldwin 2003a and b; Redpath et al. 2003). Such research suggested that small radiation doses result in unique biological changes that may be protective for induction of apoptosis to damaged cells (Bauer 2007), mutations (Sykes et al. 2006), chromosome aberrations (Azzam et al. 1994), and cell transformation (Azzam et al. 1994; Redpath et al. 2003). Studies on the induction of epigenetic effects by low doses of radiation also suggested the potential for a protective effect (Bernall et al. 2013).

Studies have also been performed at near zero radiation, i.e., doses well below any background radiation occurring anywhere on earth. These studies (e.g., Croute et al. 1986; Planel et al. 1987; Thome et al. 2017) compared cellular response to near-zero radiation by encapsulating samples of living tissue in heavily leaded shielding containers, placing them in deep underground caves to reduce incident radiation to very near absolute zero, and then comparing the response of such tissues to identical tissues at ground level (i.e., normal background radiation). The samples at near zero radiation deteriorated with time, in contrast to the thriving identical samples that received normal background radiation—clearly revealing that living tissues need some radiation to prosper. Such observations are consistent with the adaptive response mechanisms described above and clearly reveal that low-level radiation is essential for life to survive (Waltar and Feinendegen 2020).

With continued research, two key factors are becoming clear: (1) we have not underestimated the risk from exposure to radiation, and (2) in the low dose region ($\leq$100 mSv), we have overestimated the radiation risk. These two well-established scientific observations support the serious need to revise regulatory actions. This revision must include all the impacts of the regulatory action and not focus only on the radiation dose. The use of "optimization" in regulatory action rather than ALARA will result in regulations that not only protect the individual, populations, and the environment but also help reduce the fear of very low doses of radiation in the range of natural background. Such actions will be a great benefit to nuclear medicine, nuclear power production, and nuclear waste disposal and will be critical in decreasing the fear of low doses of radiation.

Genetic sensitivity The role of genetic sensitivity and susceptibility has been carefully reviewed and provides additional information on this important subject. In these extensive reviews (NCRP 2010), it was found that the range of genetic susceptibility in humans, with the exceptions of a few serious genetic diseases, is rather narrow and that setting standards for a population is acceptable (NCRP 2010).

All of the above insights strongly suggest the need for a systematic approach to understanding the biological effects of low-dose radiation (Dainiak et al. 2017).

Question 5: What are the costs to society that are driven in a great part by the fear of radiation, and how does fear of radiation impact the economy, health, and well-being in exposed populations?

Societal costs due to the fear of radiation have been manifest in a wide range of activities. This fear has resulted in detrimental impacts in both economic and human health outcomes. Unfortunately, succumbing to the unfounded fear of low-level radiation creates severe consequences to our health and prosperity (Waltar et al. 2016). Discussed below are four examples of how fear of radiation has been detrimental to human health and prosperity.

Nuclear power The growth of nuclear power, starting with the first employment of commercial nuclear power in the 1960s, now provides approximately 10% of the world's electricity and 20% of US electrical needs. The principal attributes of nuclear power are: (1) emission free (i.e., no CO_2 emitted to the atmosphere during operation); (2) reliability (about 93% on line, compared to 54% for natural gas-combined cycle, 49% for coal, 37% for hydro, 35% for wind, 25% for solar photovoltaic, and 20% for solar thermal) (Alves 2022); and (3) long-term, essentially inexhaustible (i.e., renewable) supply when considering fast spectrum reactors, combined with a potentially inexhaustible supply of uranium and thorium for fuel in the oceans where recent research on their extraction has been successful (Conca 2021).

It is generally recognized by responsible long-term planners that the global needs for electricity will continue to climb, especially as the push for the electrification of the transportation industry matures. A balanced source of supply is clearly the safest and most effective way to meet these growing needs, and nuclear power is positioned to be the most efficient and reliable ingredient in the energy mix, especially with the advent of small modular reactors now nearing the licensing stage to fill grid needs in a size- and cost-effective manner.

But the fear of anything nuclear continues to hamper this needed growth. This fear results in unnecessary safety measures, redundancies, and large evacuation zones that add significantly to the cost and acceptability of this technology.

Nuclear waste cleanup One of the issues that concern many about the acceptability of nuclear power is the question of what to do with the nuclear waste. First, we need to recognize that about 97% of this "waste" is not waste at all. It can be converted to useful nuclear fuel, since it is comprised mostly of uranium or plutonium, which can be extracted

from the used fuel and recycled back into power reactors—particularly fast-spectrum reactors.

Some dread the very long half-lives of the residue of used fuel, not recognizing that the longer the half-life, the less dangerous it becomes. It is the short half-life fission products (about 3% of the used fuel) that are dangerous, and they will have largely decayed after about 300 y. We certainly have the technology to properly sequester the real waste for that period of time. The long-lived radionuclides ^{129}I (half-life 1.57 × 107 y) and ^{99}Tc (half-life 2.13 × 105 y) and their low specific activity do not create a biological hazard. Rats were fed pure ^{129}I for their life span, and this did not result in a significant dose to the thyroid or thyroid cancer (Book 1983). Technicium-99 has a long physical half-life but has no known biological function and has a short biological half-life, limiting its dose and biological hazard (Strom 2003).

Unfortunately, the current regulations for the design of an underground nuclear waste repository in the United States require the radiation level at the surface of such a repository at the end of the design life of the repository to be 20 times less than natural background! This absurd restriction, based on the LNT model and the unnecessary fear that it engenders, makes it almost impossible to design and build a nuclear waste repository, thereby greatly restricting the growth and acceptability of commercial nuclear power.

Myriad of non-power benefits Another factor generally unknown to the general public relates to the myriad of services and products that radiation technology has provided to modern life. The book "Radiation and Modern Life: Fulfilling Marie Curie's Dream" (Waltar 2004) summarized the enormous contributions that radiation technology is already providing in the fields of medicine, agriculture, modern industry, transportation, space exploration, combating terrorism and crime, arts and sciences, and environmental protection. This use of radiation technology has been expanded considerably and documented in the recent Elsevier encyclopedia on nuclear energy (Greenspan 2021), which contains 26 chapters in the section titled "The Medical, Agricultural, and Industrial Applications of Nuclear Technology" and focuses on these highly beneficial contributions to our global society. Already, the value of these contributions well exceeds that of the commercial nuclear power industry—both in terms of jobs and the economy. Yet, there are still people who will not submit to a CAT scan because of radiation fear, even though scientific data clearly show that the benefits far outweigh the risks. There appears to be no end in sight for the further advancement of such radiation technologies if the unsubstantiated fear of low-level radiation can be curbed.

COVID-19 A current on-going medical example where the fear of radiation has hindered the use of potentially life-saving therapy is associated with the Sars CoV-2 pandemic. For decades, we have known that low levels of radiation are an effective treatment (70% to 90% effective) for viral pneumonia like that caused by SARS CoV-2 (Wilson et al. 2020). In this type of viral pneumonia, the overwhelming cause of death is the overreaction of the body's immune system that results in a cytokine release syndrome, also known as a cytokine storm. Such a storm is a deadly uncontrolled systemic inflammatory response of the body's immune system resulting from the release of great amounts of pro-inflammatory cytokines, which act as a major factor in producing acute respiratory distress syndrome, which is what kills (Hojyo et al. 2020).

It is the anti-inflammatory effects of radiation, not its antiviral action, that addresses COVID-19. Using a dose of about 0.5 Gy (50 rad) targeted to the lungs, approximately 100 times lower than those used for cancer treatments, repolarizes certain immune cells, such as macrophages, changing them from the proinflammatory M1 phenotype to the anti-inflammatory M2 phenotype. These doses carry little to no risk and have never caused any adverse reactions when targeted to the lungs from multiple sources (Dunlap et al. 2021).

Almost every hospital or cancer center is completely set up for these radiation treatments—no new preparation, additional equipment, or training is needed. Unfortunately, 70 y of irrational and unfounded fear of low doses of radiation has prevented these treatments from being given or even from being evaluated in large trials, even though these patients are at great risk of death. Small human trials have shown 90% effectiveness with COVID-19 (Hess et al. 2020), similar to results from the last 80 y. It is most unfortunate that the unsubstantiated fear of the word "radiation" has prevented a valid consideration of exploring this kind of treatment, which has the potential of preventing an untold number of deaths.

In addition to these examples, the impact of the fear of radiation may be seen in the public perception and response to the three major nuclear plant accidents in recent history, i.e., Three Mile Island, Chernobyl, and Fukushima, as discussed below.

Three Mile Island The Three Mile Island Nuclear accident on 28 March 1979 near Harrisburg, PA, occurred close to the time that a movie was released called the "China Syndrome," in which a fictional reactor accident occurred, and the potential consequences of the accident were dramatized to be catastrophic. The combination of the movie and the actual nuclear event were very important in stimulating the fear of nuclear power, in spite of the scientific evidence that the exposures were in the range of the normal background and should have had no detectable effect on human health or the environment.

Chernobyl The Chernobyl nuclear reactor accident that occurred on 26 April 1986 in Ukraine (part of the former Soviet

Union) was the worst nuclear accident in world history. There were about 30 deaths from acute high-level radiation exposure. These resulted from the high radiation doses received by many of the workers and firemen trying to contain the radiation (Chernobyl at Twenty 2007; NAS/NRC 2016; Samet et al. 2018). The whole world was exposed to some degree of radioactive fallout as the result of this event. Large populations of animals and humans who lived in the region around the reactor received what were considered large radiation exposures, resulting in doses greater than 1 Gy.

It should be noted that the design of the Chernobyl reactors was completely different from nuclear power reactors in the western world, such that they could never have been licensed outside the former Soviet Union. The basic physics parameters controlling the behavior of the Chernobyl reactors actually contributed to the severity of the accident, a situation which is completely unacceptable to those reactors licensed and operating in the rest of the world. Among other fundamental differences, the Chernobyl reactors had no containment systems. Hence, any radiation release from such an accident was released directly into the atmosphere with no safety barrier. The impact of exposures to ^{131}I from the Chernobyl accident were previously discussed. An increase in treatable childhood thyroid cancer was documented but with no increase in thyroid cancer in adults.

Studies on the animals in the "exclusion zones" where the doses were the highest have been published, and it has been carefully documented that in spite of having large doses from ^{90}Sr and ^{137}Cs (Chesser et al. 2001) there seemed to be little or no significant adverse effects from the radiation (Rogers and Baker 2000; Wickliffe et al. 2003a and b). In fact, since all humans were evacuated from the exclusion zones and only a few returned in the years following, the animal life has thrived. Record numbers of different species and individual animals now live in this higher radioactive environment with little evidence for adverse effects.

Fukushima The Fukushima accident, occurring on 11 March 2011 in Japan, was triggered by an extremely large earthquake (9.0 on the Richter scale), which created a tsunami that hit the eastern coast of Japan killing more than 19,000 people. This major tsunami also damaged the nuclear power plants at Fukushima, resulting in the release of radioactive material into the environment and destroying much of the infrastructure, power production, and communications, as well as the food and water supplies.

A most unfortunate result of this tragedy was that some 16,000 Japanese citizens were forced to evacuate their homes and hospital beds to avoid radiation exposure, and about 1,600 deaths have subsequently occurred among these evacuees. However, none of the deaths can be attributed to radiation health effects. Rather, the excess deaths were due to the trauma of the extended evacuation situations, including suicide, heart attacks, depression, lack of medications, etc. This conclusion was documented by the leading international agencies on radiation health effects (ICRP 2012; UNSCEAR 2014; WHO 2013).

By way of perspective, a recent paper vividly revealed the striking contrast in the results between two different responses to a radiation release: Fukushima and St. George, Utah (Church and Brooks 2020). It was determined that Southern Utah had about three times more external radiation dose than was measured in Fukushima. In addition, the fallout in Southern Utah had many radionuclides that deposited in the body that were not present at Fukushima. These included, to mention just a few, ^{90}Sr,^{239}Pu, and ^{144}Ce, which resulted in an increased dose not discussed in this referenced manuscript (Church and Brooks 2020). The doses in both events were low enough that a measurable increase in cancer frequency was not predicted or measured (Lloyd et al. 1990; UNSCEAR 2014; WHO 2013). However, evacuations were ordered in Japan (with no threshold considered), and serious damage was done to the population and the economy. No evacuations were carried out in St. George (where a threshold was recognized and honored by the authorities), and no damage to the people or the economy has been documented.

The results of the different regulatory actions can be summarized as follows: Fukushima suffered about 1,600 deaths due to the unnecessarily prolonged evacuation process, and the whole region is suffering a devastating economic aftermath. St. George, on the other hand, had no impact on everyday life and experienced a population growth from about 5,000 during the nuclear fallout test period to approximately 150,000 today. St. George now enjoys the status of a major vacation destination site. The contrast is stunning—all the result of employing a radiation threshold model at St. George, rather than the no-threshold presumption embedded in the very restrictive evacuation criteria employed at Fukushima.

ETIOLOGY OF FEAR OF RADIATION

What has caused the widespread public concern associated with low doses of radiation? Spencer R. Weart, Director Emeritus of the Center for History of Physics of the American Institute of Physics, in his book "The Rise of Nuclear Fear" (Weart 2012) provides an extensive discussion on how the imagery and symbolism, often originating from much different ancient roots, have been engrained in our perception of radiation. This imagery and symbolism have too often allowed fear, rather than scientific knowledge, to shape both public perception and public policy regarding beneficial uses of radiation.

In this manuscript, we have tried to describe some of the questions that have fueled this fear and the role of scientific research in addressing these questions. But even beyond the scientific questions, several additional factors have "fanned the unfounded flames"—namely, unjustified fears caused primarily by desires for funding, groups with agendas against nuclear power, and media outlets such as newspapers and movies. It is important to recognize that each individual's fear is the result of the sum of our experiences and that in many cases this fear has no basis in scientific knowledge or understanding.

We are almost all afraid of something, and we have generated names to describe these unfounded fears. For example, many of us are afraid of snakes. It doesn't matter if it is a large or small snake, a poisonous or benign snake, or just a stick or piece of hose that looks like a snake. Thus, fear has no threshold. Our reaction to such experiences can be an immediate fight-or-flight response based strongly on fear. Recognizing this deeply ingrained fear instinct is important as we discuss the sources of the fear of radiation. Radiation cannot be detected by the common senses (sight, smell, touch, taste, or hearing). Hence, radiation is often believed to be an "invisible" scientific phenomenon that can cause cancer or even kill you. All these factors are part of the reason why we may have a fear of radiation.

So, what are some of the characteristics of this fear of radiation? Here are some:

- Fear has no threshold. If you are afraid of something, it doesn't matter if there is a lot of it or a small amount. Unfortunately, this seems to be the case for radiation. Many treat background radiation, normal everyday exposures, low level medical exposures, or very small releases of radioactive material into the environment with the same response and fear as large lethal doses.
- Fear sells newspapers, magazines, and now social media hits, so that when an event occurs with potential health effects, the headlines will often show the potential negative side of the story. When research or other stories are published that show that the event did not have a negative health effect, the story often doesn't even make the media correct the article—or if it does, it is back page–not frontpage—news.
- Fear provides research grants. It is well established that fear equals funding. If a scientist can come up with a hypothesis that suggests that the impact of radiation has been underestimated, it makes for a good grant and funding follows. Often when the research is completed and the hypothesis is proven to be false, no one ever hears about it except the scientist and his few "friends."
- Fear sells books with or without the inclusion of facts. Many books have been written on radiation-induced cancers with little factual basis.
- Fear brings people to the movies. This is the source of much of the fear of radiation. When you see people in a movie about radiation, there is always death, cancer, and gross genetic changes.
- Fear is an emotional reaction that can be helpful if real and present danger occurs but can be very debilitating and detrimental if it has no basis in reality. Radiation, if delivered at high doses, can indeed be dangerous. But at low doses, the kind we experience in almost all facets of life, including major nuclear accidents, such exposure is simply not to be feared.

FINAL THOUGHTS

It has been our attempt in this manuscript to demonstrate that the current level of scientific knowledge and understanding associated with low doses and dose rates of radiation is sufficient to help allay fear of low-level radiation. Years of research on radiation exposure at every level of biological organization has established that the fear of damage from radiation is much greater than the reality. Further, this fear is becoming increasingly detrimental to the further development of nuclear power that is critically needed in a power-starved world. It is likewise thwarting further developments in using radiation technology to improve everyday life, e.g., in fields of medicine, agriculture, and modern industry.

So, what can we do? Some thoughts:

- It is critical to understand that cancer is actually a very complex set of diseases, and that the induction of a single mutation is not sufficient to induce one of these terrible diseases. Radiation is a very poor mutagen since almost all the human carcinogens are not mutagens. This has been shown by research and adopted by the responsible regulatory bodies. It is critical to challenge the LNT model in the light of the current scientific knowledge that demonstrates cancer can be produced by many factors associated with high doses of radiation but not by exposures to low doses.
- We need to insist on regulatory changes that both reflect current scientific knowledge and understanding and also help the public to understand the impact of radiation on their daily life. Regulatory changes are needed to reflect and establish a useful dose, dose rate, and effectiveness factor, which reflect a decrease in risk when the radiation is delivered at a low dose rate or in small fractions. This has been used in medicine for many years. The concept of ALARA (As Low As Reasonably Achievable) should be replaced with Reasonableness in Optimization of Protection. This requires consideration of all factors involved in standards, not just the radiation dose.

- It is also essential to modify some of the basic regulatory standards, such as the current annual limit of exposure of the public to 1 mSv, which is much less than natural background radiation. This limit, based on the LNT model, assumes any amount of radiation can be hazardous. It is totally unrealistic and leads directly to the unnecessary and unproductive public fears of radiation.
- We need to recognize that a further problem is the implementation of the radiation standards where companies, cities, states, and local governments regulate to levels even below the acceptable standards (which are already overly stringent). These "conservative" actions are expensive and provide no beneficial impact to public health or safety.
- Lastly, be engaged. As health professionals we have a responsibility to not only minimize any potential detriments associated with the use of radioactivity and radiation but also to help maximize its beneficial uses. We believe that to not avail society of the beneficial uses of radioactivity and radiation is a detriment itself, as we have tried to demonstrate in this manuscript. So if confronted with a situation where you believe a fear of radiation may be an obstacle to a beneficial use of radioactivity or radiation, use such a situation as an opportunity to help educate or at least to provide scientific information to rebut such fears.

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