Shifting the radiation exposure paradigm from protecting the industry to protecting the public

Comments from Beyond Nuclear to the Nuclear Radiation and Studies Board of the National Academy of Sciences -- Committee to assess cancer risks around Nuclear Regulatory Commission licensed facilities, Phase I

September 23, 2011

Thank you for the opportunity to comment on the very important work of your committee. According to your charge, “Phase 1 scoping study will identify scientifically sound approaches for carrying out an epidemiological study of cancer risks...including methodological approaches for assessing off-site radiation dose...(and) methodological approaches for assessing cancer epidemiology ...”

This charge requires your committee to consider how or if current radiation dose reconstruction methods and estimates are applicable to health studies. These dose estimates are often derived from industry estimates of emissions. The industry has a vested interest in under-reporting its radioactive emissions because it is focused on not only maximizing profits from the current fleet of nuclear reactors, but also on establishing a new generation of reactors. Finding routine reactor operations cause cancer would further turn public opinion against this industry. Therefore, the public would likely be distrustful of any health assessments using industry-derived data. Although the industry provides some measurements of releases, they lack the ability to provide accurate assessment of all releases. Dose estimates often under-represent, or fail to represent, biological damage from radiation, especially damage across generations. Designing an epidemiological study that accurately represents health outcomes may be possible but would require your committee to set aside some dominant, and often incomplete or incorrect methodology.

Dose limits, dose assumptions, dose response

Jill Sutcliff from the Institute of Ecology and Environmental Management, Hampshire, UK, has written a paper delineating the history of radiation discovery, exposure, and health effects research in which she recognizes two forms of latency. One is the time lag between radiation exposure and disease manifestation; the other is the time it takes for policy to catch up to science. In the case of radiation, this policy lag time can be 40 years or more.

Industry-derived emissions numbers

The Nuclear Regulatory Commission (NRC) relies completely on industry for its regulatory function. At your committee’s February 2011 meeting, the NRC stated: “Our entire regulatory process is based on licensees providing us complete and accurate information.” Part of this “complete and accurate information” is a combination of industry-measured and industry-estimated emissions data for radioactivity. NRC collects these data primarily to assure regulatory compliance, NOT for use in health assessments. Yet, researchers use these data for health studies with the assumption that the doses are too low to create a detectable incidence of disease. When researchers do find disease increases, such as childhood leukemia, they fail to attribute these increases to radiation exposure because they have been constricted by their previous assumptions. This distorts the true disease risk picture by disallowing excess cancers near nuclear facilities to be associated with radiation exposures. “When excess cancer near nuclear facilities cannot be interpreted as evidence of an affect of releases, it is because the expected response from the estimated dose is too small to detect. (emphasis added)”
But, when investigated, estimated dose is low compared to actual dose. In a comparison between estimated dose and measured biomarkers, both of which were used to derive dose among Chernobyl survivors, it became clear that the estimated doses put forth by UNSCEAR (United Nations Scientific Committee on the effects of Atomic Radiation) had grossly underestimated the true exposure. The measured dose, derived by assessing the number of radiation exposure biomarkers, know as dicentric (a sensitive marker of radiation exposure, even at lower doses) and centric rings, showed a much higher radiation exposure.\(^5\)

These same authors have also used dicentrics to show that members of the German public had been exposed, repeatedly, to radiation levels from nearby reactors. These exposure levels were many times what was supposedly allowed by the regulatory limit. These biomarkers were corroborated by identification of artificially created radioactivity in the surrounding environs.\(^6\) This difference between released radioactivity (as measured by industry), and estimated versus measured dose, strongly suggests that dose estimates are often unreliable for health assessments and that industry cannot be relied upon for correct source term.

Allowable release limits

Throughout years of radiation regulation, regulators realized that smaller and smaller doses could carry greater and greater risk. Radiation dose limits of the International Commission on Radiation Protection (ICRP) are traditionally used by regulators such as the NRC to set exposure standards. Since 1934, the ICRP has revised its dose limit recommendations four times, each time reducing the permissible dose. Initially, these limits were issued without benefit of public input, relying only on input from a small number of scientists who were often not identifiable and were, therefore, not accountable. ICRP’s 1990 recommendations were considered a late response to the accruing scientific evidence of the time. This prompted the Lancet to ponder if ICRP’s reluctance to recommend more protective standards was due to concerns about potential financial consequences to the nuclear industry.\(^7\)

Ignoring evidence from the Environmental Protection Agency (EPA) and the National Academy of Sciences (which had produced a study requested by agencies including the NRC, but which the NRC subsequently ignored), the NRC is considering the increase of permissible concentrations for two-thirds of radionuclides. The NRC intends to achieve this increase through use of effective dose equivalent (EDE). This concept is apparently borrowed from the ICRP’s recommendations (including ICRP reports #62 26, 60, 103 which are extremely expensive for members of the public to purchase). The ICRP recommends that some cancers should count as partial cancers based on degree of pain and suffering, years of life lost, treatability, etc. Bladder, colon, kidney or thyroid cancer can be “discounted” in this way.\(^8\) The EPA estimated that a regulation limiting doses to 25 millirem whole body, 75 millirem thyroid, and 25 millirem to any other critical organ would be equal to 10 mrem “effective dose equivalent.” Based on the EPA’s assessment of EDE, it appears to underestimate actual whole body and organ doses substantially.\(^9\)

From Hirsch 2011:

Federal Guidance Report 13 gives dose to [cancer] risk as 8.46 X 10^-4 person-rem while BEIR VII, released after FGR 13, sets the risk even greater at 1.14 X 10^-3 cancers per person-rem. NRC’s primary permissible radiation dose level (10 CFR 20) for members of the public is 100 millirem per year over a lifetime. This officially equated to a risk of cancer of producing approximately 6.4 cancers per thousand people exposed, above and beyond
the number of cancers that would have occurred without the exposure or one out of every 156. Using the recent National Academy of Sciences BEIR VII study, the risk would be 8.6 per thousand, or approaching one cancer per hundred people exposed.

This means that NRC allows public exposures to radiation at risk levels one hundred to ten thousand times higher than the federal government permits for any other carcinogen. EPA has previously opposed NRC’s radiation standards for this reason, asking why radiation should be treated as a ‘privileged pollutant,’ permitted to expose people to cancer risks at levels far above that allowed for any other pollutant. 10

This “one in 156” is a rather simple calculation and, to lay-members of the public, appears to translate into real risk. The NRC should justify to members of the public why ionizing radiation from nuclear facilities is conferred this special status. The double-strand-break (DSB) damage, that is a hallmark of ionizing radiation, is notoriously difficult to repair because this damage often occurs in relatively close time and space.11 Compared to DNA damage from regular cell processes, radiation damage to DNA can be unique and strongly dominant, producing significant lesions. 12 Only a few other chemicals are capable of producing DSB; and the double-strand breaks they produce are more random in space and time, allowing more chance for correct repair. These chemicals, called radiomimetics, include streptonigrin, 8-Ethoxycaffeine, Bleomycin, m-AMSA. “…radiomimetic chemicals are well-known human clastogens and strictly regulated.” 13 Apparently, these chemicals are dangerous enough so that we severely limit their release to the environment. Why are releases of radionuclides, which can cause less easily repairable damage, not more stringently limited?

Further, the NRC can already grant a licensee’s request for a 500 millirem per year exposure to individual members of the public during a facility’s routine operations (NRC 10 CFR Part 20.1301(d)). There is no guarantee that, similar to post-Fukushima disaster Japan where officials arbitrarily dictated that a dose of 2 rem per year to a child will not affect their health,14 the US NRC will not act in a beneficial way toward industry over the public in the case of a dirty bomb or nuclear facility catastrophe.

**Both natural and background radiation doses increase risk**

**Just in the past year or two, the NRC has raised its estimate of the nation’s annual “background radiation” dose to 620 millirem.** 15 Until then, it had been 360 millirem and a still lower exposure level before that.

Background radiation is, of course, applicable to the charge of this committee in the context of any dose reconstruction and assumptions that are made regarding risk. The NRC’s estimate of background dose should not cause any minimization or dismissal of the risk of additional radiation doses, no matter how small those additional doses may appear in comparison. As BEIR VII dose-to-risk numbers, and mounting proof of epigenetic effects indicate, risk must still be assessed for doses even if below alleged background levels. The NRC does seem to make the assumption that adding an amount of radiation that is less than background to a person’s dose is all right. The agency is often quite dismissive of this additional risk by claiming that the cancers it causes will be undetectable compared to background disease levels. Undetectable does not mean non-existent and accounts neither for cumulative nor synergistic damage; the science to tease out the truth of these issues is just unfolding. 16 One idea does seem supported
by many research papers and reasoned arguments: adding more radiation from any sources will increase risk. Our ability to poison ourselves far outpaces our scientific ability to understand just how badly we are poisoning ourselves.

The NRC uses “background” to mean natural radiation from soil, cosmic rays, etc. along with radon, medical and industrial exposure. It appears that the NRC now presents these figures as exposures that everyone receives; that every American is exposed to the full 620 millirems per year. In reality exposure numbers vary by each individual depending on where they live, medical treatments they receive, etc. The use of this inflated 620 mrem annual dose is an inappropriate starting point for any health assumptions used to assess impacts. For example, not everyone undergoes diagnostic radiation examinations. Not everyone receives a radon dose, and certainly not the 200 millirems that the NRC claims. Further, the radon dose is specific to the lung, not to the whole body and, in some instances, can be mitigated by technological fixes. Radon that escapes from the ground and certain construction materials can be vented from indoor locations, reducing exposure. The NRC needs to justify why it thinks this potential radon dose should be counted in the total, unavoidable, whole body dose for every individual member of the public. In addition, the NRC needs to support that everyone receives the NRC’s full assumed radiation dose from medical procedures.

**Annual estimated average effective dose equivalent received by a member of the population of the United States before the recent NRC change in 2010.**

<table>
<thead>
<tr>
<th>Source</th>
<th>Dose (mrem)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhaled (Radon and Decay Products)</td>
<td>200</td>
</tr>
<tr>
<td>Other Internally Deposited Radionuclides</td>
<td>39</td>
</tr>
<tr>
<td>Terrestrial Radiation</td>
<td>28</td>
</tr>
<tr>
<td>Cosmic Radiation</td>
<td>27</td>
</tr>
<tr>
<td>Cosmogenic Radioactivity</td>
<td>1</td>
</tr>
<tr>
<td>Rounded total from natural source</td>
<td>300</td>
</tr>
<tr>
<td>Rounded total from artificial Sources</td>
<td>60</td>
</tr>
</tbody>
</table>
The above table is adapted from one posted by the University of Michigan’s Health Physics Society. The NRC claims that, in general, a yearly dose of 620 millirem from all radiation sources has not been shown to cause humans any harm. The NRC staff stressed this point at your committee’s first meeting in Washington, DC in February 2011. This is much the same reasoning industry and the NRC use to assume they will not find elevated disease incidence around nuclear facilities. On the contrary, BEIR VII concluded that no radiation dose could be considered safe. The NRC does not ensure that its standards keep the public safe, and instead claims that any harm will be undetectable. Is it possible that the NRC’s new 620-millirem “background” level is being used in order to justify and belittle the exposure of the public to ever-increasing amounts of artificially generated radioactivity?

Increasing numbers of individuals are now seeking to avoid excess radiation exposure by limiting medical radiation procedures whenever possible, mitigating for radon, opting for pat-downs at airports rather than walking into the security scanner, etc. They are making a conscious decision to assume their exposure. The nuclear industry and the NRC should not be allowed to rely on falsely inflated background radiation exposure levels to justify exposing an individual to additional radiation risk without that individual’s knowledge and consent. Furthermore, as the ability to detect lower levels of radioactivity continues to become more precise, and the ability to detect and understand damage to human health continues to improve, the public will no longer be vulnerable to false nuclear industry claims that radioactive releases, whether routine or accidental, cause no health damage.

Health risks of radiation exposure: Cancer in exposed and their offspring

Radiation exposure is linked not just to cancer, but also to cardiovascular problems, nerve damage, infertility, decreased fitness, accelerated aging, and genomic instability (GI), and the increased risk of genetic disease, greater radiosensitivity, and other health impacts.

While the NRC has charged your committee to develop an assessment of cancer risk—a narrow charge -- other health impacts may also increase as radiation exposures increase. It would be appropriate for your committee report to at least list potential, additional health impacts.

Childhood leukemia from radiation exposure: study summary

In a synthesis examining childhood leukemia, Belson, et al., examine several factors that could be associated with childhood leukemia (CL). They conclude “Only one environmental risk factor (ionizing radiation) has been significantly linked to (ALL) [acute lymphocytic leukemia] or (AML) [acute myeloid leukemia].” Two comments were published on the Belson paper in the same journal. One claimed that EMF is also associated with leukemia. Another reviewer noted that the Belson paper does not present some of the strongest evidence supporting a link between ionizing radiation (IR) and CL. Belson does not seem to reference any of the nuclear power reactor studies showing the CL connection although some did exist at the time.
In their meta-analysis, Baker, et al. state: "Further, dose-response studies do not support excess rates found near nuclear facilities. However, it cannot be ignored that the majority of studies have found elevated rates, although not usually statistically significant. Baker conducted this meta-analysis before the KiKK study by Kaatsch et al. Baker has scant reference to nuclear power reactor studies.

Studies by Schmitz-Feuerhake and by Hoffmann on health impacts from German reactors, inspired an investigation that culminated in the 2009 KiKK health study that caused an uproar in the German press. The Schmitz-Feuerhake/Hoffmann studies show a link between CL and radiation exposure. They show parental chromosomal damage at lower, chronic IR doses. These researchers also theorize this damage comes from unreported releases of IR -- either from the Krummel power reactor or from the research reactors nearby, or both. The researchers found that attic dust in houses near these reactors contained radionuclides from those reactors.

Because both power and research reactor licensees in the US self-report their releases to the NRC, accidental and routine releases may not be detected or reported, making true exposures difficult to determine using conventional methods. However, Schmitz-Feuerhake and Hoffmann have also done research showing that one chromosome malformation, namely the dicentric or dic, is a sensitive indicator of radiation exposure (see below for details) and that these formations were elevated among the German population near these reactors.

The KiKK study shows elevated rates of CL around Germany’s nuclear power facilities. The study method for disease assessment only included examination of CL increases within concentric geographic rings radiating out from the reactors. Even though the study was geographic, not accounting for all weather patterns, etc, or any biomarker measurement, the researchers still found increased CL around the reactors.

In another paper investigating childhood leukemia, Kinlen contends that CL clusters around nuclear reactors are due to population mixing – that is people moving into a community for work, etc. He claims they introduce some sort of biological agent such as a virus, thus exposing the former more isolated population to new biological insults. However, researchers have not yet located such a responsible biological entity as they have for HPV-1, for instance. Without isolating any responsible biological entity, Kinlen’s theory is hard to support.

Taub theorizes that a large proportion of childhood ALL cases arise in utero. In several studies, a long latency period occurs between the initiation, in utero, of the original ALL clone and clinical diagnosis. This delay indicates that additional genetic events are required for the full development of the leukemia phenotype, such as postnatal exposures (e.g., infections).

Perhaps this points to a multi-insult model where an initial insult (from IR) may cause a child to be more susceptible to common biological infections. This is perhaps what Kinlen et al. are finding, rather than a single biological cause. For example, the Eden study also suggests that, for some leukemia victims, the first event may be enough to form a malignancy, but for other types of leukemia (such as ALL and AML), further changes may be required that are probably postnatal. Ionizing radiation has been confirmed causative for CL, and dysregulated responses to common infectious agents have been found to play a major role in converting pre-leukemia clones into overt precursor cells. Further, Eden states that little evidence exists to support viral transformation as a cause. “There is no single cause for childhood leukemia and for most individuals a combination of factors appears to be necessary...[t]o date few preventive measures have emerged, except the complete avoidance of first trimester X-rays...a healthy diet...early exposure of children to other children outside the home.”
Biomarkers

Biomarkers may help fill the void left by inadequate environmental radiation monitoring and may help clarify the “uncertainty” outcome of radiation damage at low doses.

Ionizing radiation is known to cause a chromosome malformation called a dicentric (dic) ring. According to Hoffmann, this formation is “the single most extensively validated biomarker for radiation exposure... [S]tructural chromosome aberrations are presently the only marker with an established predictive value for increased cancer risk.” (emphasis added) Therefore, dic can serve both as a marker for effective dose and as an early response in epidemiological studies. However, predicting cancer dose response using specifically dicentric rings can be tricky or even misleading as can using these biomarkers for radiation protection. Many of the complex chromosome aberrations produced by high-LET would be non-transmissible because cell progeny would not survive. Low-LET produces more simple aberrations that can be passed on, and risk from this type may be underestimated. Other biomarkers exist which could better predict cancer outcomes – as opposed to initial damage -- like intra-chromosomal rearrangements or simple deletions. Goodhead’s research paper only includes cancer risks from targeted effects.

This biomarker is so highly sensitive that, in principle, its assay could apply to very low-dose range exposure, such as environmental exposures to cosmic radiation. For adults, dic assays could be used to analyze occupational exposures within permissible limits, or in the case of accidental overdose. Children apparently have fewer background dicentrics than adults.

The dic assay certainly seems to be applicable to assessing radiation exposure levels occurring as a result of the ongoing Fukushima disaster, and is better at assessing exposure than current, favored methods of estimating dose.

Independent laboratories could also perform dicentric biomarker examinations to determine the exposures to routine and accidental release of radioactivity from nuclear reactors, even though the cost of such assays could be high. Additionally, while biomarkers will probably not be able to completely replace epidemiological methods (generally more indicative of a whole population’s exposure) biomarkers could help with exposure assessments of individuals. A marker like a dicentric could also be used to spot check assumptions about dose estimate accuracy. No longer does a dose reconstruction or estimate need to be the primary method to assess disease risk from radiation exposure. Furthermore, biomarker assays could offset the shortcomings of current monitoring and exposure assessment techniques. By using biomarker assays, health investigations would not need to be relegated to just dose assessment but could also provide evidence of actual damage among a population since, these markers can represent an increased risk of cancer, although assessing how much increased risk there is could be tricky. This could also help resolve compensation controversies regarding worker and population exposures to radiation.

Transgenerational effects of radiation include cancer and other diseases

The charge of your committee is to assess cancer risks around NRC-licensed nuclear facilities. Therefore, you should not only be concerned about cancer experienced by the initially exposed generations, but also about transgenerational cancer that comes from this initial exposure.

There is mounting evidence that epigenetic/non-targeted effects, which were unrecognized as conveyers of radiation damage traditionally, can also cause disease. These are generally defined as effects that occur from radiation exposure and which are not attributable to direct genetic
damage—that is, direct damage to the primary DNA sequence, rather than damage or changes to or through methylation, chromatin, histone covalent modification and chromatin structure. For our purposes, a reasonable explanation of the use of epigenetic would be:

...heritable changes in gene expression that are not due to changes in DNA sequence... Diverse biological properties can be affected by epigenetic mechanisms... Epigenetic changes are crucial for the development and differentiation of the various cell types in an organism... epigenetic states can become disrupted by environmental influences or during ageing, and the importance of epigenetic changes in the development of cancer and other diseases is increasingly being appreciated.

These effects occur in addition to and/or outside of DNA damage as a consequence of the functional processes of a cell and are so fundamental that they promise to overturn a number of assumed radiation effects and may warrant the creation of new paradigms for health risk from radiation exposure. Epigenetic effects include transgenerational effects (TE), bystander effect (BE) and genomic instability (GI). TE, specifically, are defined as influences from an irradiated parent that are manifest in an unirradiated offspring. These could include: increased cancer predisposition, increased mutation rates, decreased fertility rates, and a radiation-induced increased sensitivity to further mutation from a variety of insults, not just radiation. These effects indicate a general destabilization of genomic integrity through several generations subsequent to the one initially exposed. Other TE may include cardiovascular, gastrointestinal and respiratory diseases.

**TE in cells, animals and humans**

Yuri E. Dubrova, whose research has centered on TE in cells, mice, and humans, has presented the following observations: irradiated male mice produce germline and somatic mutations in their unirradiated offspring that can affect the frequency of gene mutations and chromosome aberrations. This instability results from a persistent subset of endogenous DNA lesions and can result not only in an increased risk of cancer, but decreased fertility and viability of offspring. Dubrova comments: "[c]an transgenerational instability be regarded as a significant component of the long-term genetic risk of human exposure to ionising radiation and other mutagens? Most probably – yes, but it is going to be a tough job to prove it." His rhetorical question and response illustrates the tangled nature of the current science on epigenetics.

There are studies that show transgenerational effects in humans at Semipalatinsk, Chernobyl, and on the Techa River, but not in the Japanese A-bomb survivors (Data currently under reexamination; see below) or certain other studies, indicating a supposed inconsistency of radiation TE among humans. Gardner found an increase in leukemia among the unexposed children of male nuclear facility workers from Sellafield, a nuclear waste reprocessing facility in the U.K. Despite the criticism levied on his research fifteen years ago, his data could certainly point to TE as a mechanism for these health impacts and also seem to support the additional evidence that TE can occur in humans. It is worth noting that, as our environment becomes increasingly contaminated by radionuclides, we will no longer have offspring who have lived in an environment of just natural radiation. This contamination can travel great distances by the weather and food chain. Areas of the US West Coast are being contaminated by radioactivity from the catastrophe at Fukushima that is still on-going and is expected to continue. We must
recognize that radiation exposure can increase radiosensitivity in cells, mice, and humans, and decrease adaptive response. In a world increasingly contaminated by human-generated radioactivity, increasing rates of disease could beset an increasingly radiosensitive population.

Not only is the science of radiation exposure amazingly complex, but the history of human research on the genetic effects caused by radiation exposure is fraught with dubious assumptions and scientific rigidity that helped to delay important discoveries. It started with a damaging assumption regarding the atomic bomb survivor cohort:

“In 1946...it became very likely...that even a very major effort would not yield a statistically significant difference between the children of survivors receiving increased radiation at the time of the bombings and the children of suitable controls.” Thus the famous negative results were built-in before the study began.

This is the “assumption methodology” Dr. Steve Wing rightly criticized both before your committee and in publication. Many researchers make the point that, unless humans differ significantly from every other plant and animal species studied for heritable, damaging mutations from radiation exposure, there is no logical reason to assume humans are somehow immune to these effects -- even though such damage has not been proven as yet, nor a definitive mechanism shown. In fact, a reevaluation of the A-bomb data is currently under way. Baverstock contends that socio-political opposition to new discoveries have distracted from research efforts that could have resulted in uncovering GI and other transgenerational effects decades sooner than 1992, when Kadhim’s study showed these effects. Had these effects been discovered and integrated sooner into the total knowledge of radiation health effects, we could very well have more protective exposure levels now.

**How does TE fit into conventional radiation paradigms and risk?**

While there is widespread concern that TE may require potentially major adjustment to current radiation paradigms (see references above), making definitive statements about whether, or how, all epigenetic effects, TE included, might impact health risks probably requires more data. However, we can start assessing implications for human health, despite needing more human data. This cloud of uncertainty is the perfect condition for the implementation of precautionary measures. (See the Precaution section below)

Current research on these effects has not yielded a conventional dose-response curve at the lowest doses tested. This is not a surprise, since the term “low dose” may carry a very different meaning at the cell level.

Cells that are traversed by a single [alpha] particle will receive a substantial dose (~0.3–0.5 Gy) and may sustain damage that is much greater and more complex than would be received from low doses of X- or gamma rays where a single radiation track would deliver a dose of the order of 1 mGy.

Wright adds that these interactions become even more complex because they vary by cell type and other factors. Damaging a given cell beyond repair might actually be less detrimental to the cells and the system at large, since it increases the chance of apoptosis (planned cell death). In contrast, a less damaging dose could cause preservation of a cell initially, only to result in a
mutation if the cell fails to properly repair itself. Likewise, cell killing can be decreased in a 3D model (a model considered more representative of reaction to damage) as opposed to 2D, but this may ultimately prove detrimental if the cell that is not killed will mutate and cause disease later. Additionally, research by Salomaa, et al., suggests that, while GI may be a non-linear response to radiation dose, it is maximally induced at the lowest doses studied. In the context of targeted (non-epigenetic effects): "even at lowest doses, a single electron has the capability of producing complex clustered damage in DNA, that such damage stands out as different from the much larger quantity of endogenous DNA damage, and that it can lead to a full range of stochastic cellular effects." And since x- and gamma rays deliver their damage by liberation of high-speed electrons (beta particles), then lower-energy beta emitter impacts should also be accounted for in the data on cell damage versus death.

**Uncertainty shows the need for Precaution**

At the February 24th meeting of your committee, the requesting agency, NRC, stated that they would accept a concluding report that may contain uncertainties and gray areas. Such a vaguely defined outcome would be convenient for the nuclear industry -- findings of uncertainty could, of course, allow NRC to maintain the regulatory status quo. That is, the agency could retain industry-derived and industry-measured data.

Such an outcome would maintain NRC’s current regulatory limits, which are set to be “as low as reasonably achievable” (ALARA), not mandated to provide full protection of human, animal, or environmental health. The “Reasonably achievable” component of ALARA could quite probably be defined as that contaminant level the nuclear industry says it can afford to achieve. This is an example of the inherent conflict between profit and public protection. Even a well-intentioned implementation of the concept of ALARA is built on numerous unspoken assumptions of the status quo that do not include protection from deleterious epigenetic impacts. The concept of ALARA is not equal to the concept of adequate protection in any case. Further, the NRC has based its regulatory regime on these permissible release limits failing to fully acknowledge that permissible does not mean safe and implying that an increased disease level that is below detectable limits is all right. With proper study design, some research does show evidence of health impacts at current regulatory levels, meaning these diseases are detectable. Moreover, the NRC has not considered the full range of damage from TE or other epigenetic effects.

Enough high-and low-dose studies show that cancer association cannot be ruled out, even at the lowest possible doses and/or across generations. Acknowledging these data and conclusions, it becomes the burden of the nuclear industry to provide contrary evidence if available, that their technology is safe. **It should not be the burden of the public to prove harm.** The preponderance of the evidence on radiation exposure clearly indicates potential health damage.

If your study outcome is uncertain because of government, industry or scientific shortcomings (whether intentional or not is irrelevant), recommending a precautionary approach is prudent, perfectly applicable, and by many estimates, long overdue.

In a nutshell, precaution epitomizes a paradigmatic shift...precaution means that the absence of scientific certainty as to the existence or the extent (emphasis added) of risk should henceforth no longer delay the adoption of preventative measures to protect the environment.

Environmental policy has also evolved sufficiently to demonstrate that many of its principles
warrant inclusion in radiation policy -- including precaution, transparency, and public participation, and sustainable development. To date, these important concepts have not been well-integrated into the radiation risk paradigm and, health challenges identified in more recent research, such as epigenetic effects, have not yet been assessed.  

Practical implementation of the precautionary approach can occur in stages. Tickner and Geiser have set forth some appealing solutions in their 2004 paper. They focus on alternatives assessment and the necessary role it plays in full implementation of the precautionary principle. But several of their suggested steps can move policy-makers closer to making precautionary decisions without complete replacement of technology immediately. These include mandating greater public participation and representation, shifting the burden of proving harm from the public to the risk makers, recognizing and implementing protection from multiple, synergistically acting substances, and requiring alternatives assessment in environmental decision making. Government policies should shift the focus from evaluating how bad things are to seeking safer, less harmful alternatives to risk-laden activities.

Additionally, several authors present case studies, in a volume edited by de Sadeleer in 2007, outlining precautionary approaches in Europe and the United States. It was the purpose of this book to explore, through various analyses of regulatory settings, some of the key issues arising in applying precaution to risk assessment as well as to risk management.

According to the European Environment Agency, precautionary action for radiation exposure is needed because the governmental or institutional bodies that design, regulate, and or recommend the permissible levels of radiation have always been slow to react to “mounting incontrovertible evidence... where precaution has sometimes been lacking despite the clear warnings given...” Considering TE, and other emerging health concerns, we can not afford to wait for further proof before we take protective action. We are tempting fate and potentially ushering in a wave of irreversible disease. It could take decades to see and attribute the damage to the likely causes; by then, protective action could be too late.

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