From: Michel Lee
Sent: Tuesday, November 03, 2015 11:09 PM
To: Hanlon, Edward <Hanlon.Edward@epa.gov>
Subject: Comments to the Radiation Advisory Committee from the Council on Intelligent Energy & Conservation Policy and the Indian Point Safe Energy Coalition

Dear Mr. Hanlon,

The referenced Comments are attached and pasted below. We appreciate the opportunity to comment.

Michel Lee, Esq.
Steering Committee
Indian Point Safe Energy Coalition

Chairman
Council on Intelligent Energy & Conservation Policy (CIECP)

November 3, 2015

COUNCIL ON INTELLIGENT ENERGY & CONSERVATION POLICY (CIECP) AND INDIAN POINT SAFE ENERGY COALITION (IPSEC) COMMENTS TO THE SCIENCE ADVISORY BOARD RADIATION ADVISORY COMMITTEE (RAC)

RE: U.S. Environmental Protection Agency (EPA) Advance Notice of Proposed Rulemaking (ANPR) to Consider Revision of Environmental Radiation Protection Standards for Nuclear power Operations (40 CPR Part 190)

Dear RAC:

Preliminary Statement

The RAC has a genuine opportunity to significantly advance public health.

Current – or, more accurately, the currently operative 1977 – standards and the International Commission on Radiological Protection (ICRP) standards articulate the principle of public health protection, but deeply incorporate the assumptions and objectives of the nuclear industry.

We urge the RAC to advise the EPA to substantially strengthen public protections and take into consideration real world conditions with respect to nuclear power radiation release standards. A "safety margin" needs to be built into the regulations to accommodate the reality of other forms

of unavoidable or medically necessary radiation exposure as well as the inevitable continuing unplanned nuclear site releases.

We, as citizens, want our children to live in a world that is thriving, clean, safe, better. We hope you do too.

This is not going to happen if our regulatory schemas continue to effectively promote dirty, dangerous, and extractive forms of energy. It is not going to happen with radiation and countless other toxins poisoning the air and soil, and draining into our rivers, lakes, streams, and groundwater.

We are creating an ecosystem awash in toxins.

And we need to stop.

Recommendation Number One: Eliminate Industry Accommodation Bias

A certain cost-benefit analysis has long been embedded in acceptance of radioactive emissions from nuclear power. Indeed, federal documents have referred to radiation as a "privileged pollutant."

Arguments have been made that acceptance of contamination is a "necessary evil" with respect to activities for which there is no viable alternative.

However dramatic and rapidly evolving developments in 21st Century energy provision, distribution and use (*e.g*., renewable power generation, transmission, demand-side management, and efficiency technologies) utterly eradicate this argument with respect to nuclear power. There are available, cheaper, cleaner, safer alternatives, which are far less toxic.

The EPA mandate is protection of public health and the environment, not protection of humans and nature to the extent *feasible*, while enabling the commercial nuclear industry to maintain a desired level of profit.

Recommendation Number Two: Standards Should Give Consideration to Combined Effects From All Radiation Exposure Pathways

Another assumption which has long been embedded in the regulatory scheme, is the notion that radioactivity from nuclear power operations somehow impacts populations independently from other exposures. Protection of the public, however, mandates consideration of *all* radiation emission generators and pathways. These pathways include solar radiation, radon, medical imaging, and unmonitored and accidental effluent releases.

Your Committee is undoubtedly aware of the contributions to "background" radiation made by atomic bomb testing, Chernobyl and Fukushima. You may be less aware of the fact the Nuclear

Regulatory Commission (NRC) has acknowledged that every nuclear power plant site in the United States has had accidental radiation leaks.

Moreover, at numerous uranium mining and milling, nuclear waste, and reactor sites, accidental releases have occurred repeatedly and continue to seep into the environment. At Indian Point nuclear plant in New York, for example, cesium-137, strontium-90, and tritium have leaked into the Hudson River – and leaking continues from the site's aged spent fuel pool structure.

With an aging nuclear fleet and mounting levels of nuclear waste, such releases will undoubtedly continue and, in all likelihood, increase.

It is the reality of the *total* burden and the impact upon vulnerable persons that are the factors relevant to public health.

Recommendation Number Three: Standards Should Protect the Most Vulnerable Individuals and Populations

More specifically, the RAC must urge EPA to dispense with averaging risk models and focus attention on the protection of the most vulnerable individuals and populations.

A vast and growing body of scientific knowledge demonstrates that certain individuals and groups are disproportionately vulnerable to the effects of radiation, not to mention other environmental pollutants.

The highest imperative is to protect our young: prenatal and neonatal babies, toddlers, children, girls, and adolescents. It is beyond cavil at this point that there are developmental "windows" of heightened vulnerability.

Most alarmingly, rates of childhood cancers and neurodevelopmental disorders have been rising, as has incidence of autoimmune disease. We can debate the fine points of which toxin does what and with what synergistic effects for the rest of the century. But in the meantime, our children are sick.

It is also now well established – from findings of the National Academy of Sciences among others – that women are at significantly heightened risk to the effects of radiation compared to men. Given the fact that it is women who carry and nurse babies, the urgency of protecting women becomes even more clear.

Recommendation Number Four: Standards Should Further the Objective of Environmental Justice

Existing EPA and ICRP guidelines ignore environmental justice populations. Which environmental justice populations would fall within this ambit is a relatively unexplored question. But the lack of exploration, *in and of itself*, reflects the failure to give adequate consideration to such groups.

Federal regulatory actions have enabled nuclear power interests to impose enormous costs upon site communities which have little to no recourse to self-protection. It is beyond cavil that Native American, African American, Hispanic and impoverished communities have born – and continue to bear – a disproportionate share of the pollutant burden from every step of the nuclear power fuel cycle.

Two groups which existing evidence suggests would warrant particular focus are: African American women and Native Americans in Western states.

Recent data shows African American women are at particular risk of the most aggressive forms of breast cancer. This does not mean the cause is radiation exposure (in fact, prevailing hypotheses point to other reasons), but it does mean these women may be at heightened risk from radiation – a carcinogen associated with breast cancer.

Native Americans have most demonstrably been a group victimized and disregarded by those charged with protecting their welfare and environs. Native American reservations have been despoiled by uranium mining for decades. The impacts are heartbreaking. The failure of the federal government to conduct adequate health studies is quite simply egregious.

Recommendation Number Five: Standards Should Not Narrowly Focus on Cancer

The RAC must urge EPA to expand protection to include noncancer risks. Radiation is not just a carcinogen.

Cancer has long been the focus of study but the evidence suggests that cancer is the tip of the health impact iceberg.

The EPA needs to be pressed to recognize current developments in the understanding of disease processes relevant to radiation exposure. Most notably, seeing cancer and other illness as merely the consequence of DNA damage and mutations is an extremely obsolete view.

Radiation, like other neurotoxins, can cause substantial damage to the developing brain and central nervous system. Radiation, like many other chemicals, can affect hormone expression, which, in turn, can effect cognitive development, among other functions.

Significantly, current science shows that complex cellular interactions and mechanisms, including endothelial dysfunction, inflammation, mitochondrial dysfunction and oxidative stress clearly play a relevant role in radiation-induced negative health consequences.

Recommendation Number Six: Standards Need to Protect Future Generations

Uniquely among the poisonous stew we are spewing, radioactive isotopes emitted from every single stage of the nuclear power fuel cycle will remain lethal and dangerous for many centuries to come. Thus – even ignoring all the additional radiation exposures from nuclear accidents and leaks – the chronic so-called "low-levels" of radiation emitted during ordinary nuclear power operation will accumulate and long-lived isotopes will remain cycling in and out of biological organisms and the biosphere for tens of thousands of years – actually hundreds of thousands.

How many generations of children do we want to put at risk?

These are the genuine moral and ethical considerations that need to be factored into updating of the allowable radiation exposure and effluent release standards.

Recommendation Number Seven: Monitoring of At-Release-Point Radiation and Epidemiological Studies of "Exposure Zone" Populations Need to Be Conducted

There is a glaring dearth of data – and virtually no publically accessible, independently verifiable information – on actual radioactive emissions from nuclear sites. This is no accident. Lack of transparency suits the industry.

The EPA years ago effectively outsourced its responsibility to assess emissions to the NRC, which, in turn, effectively handed the job over to the industry to self-monitor and self-report.

Not surprisingly, monitoring and reporting has been done in a haphazard and unaudited fashion.

Shockingly, no ongoing epidemiological studies have been conducted of populations living near nuclear power plants by the federal government.

This is outrageous. At the very least, it should prevent any federal body from claiming nuclear power is "safe."

It is high time for (1) independent and verifiable monitoring of at-release-point radioactive effluents and (2) broad epidemiological studies of the health status (not just cancer incidence) of Exposure Zone populations; e.g., populations likely to be subject to chronic low level radioactivity exposure, both by virtue of proximity to site and downwind/downstream of site.

Conclusion

We leave it to your good intention, to consider the evidence, consider the trends, and consider the vital interests at stake.

Sincerely,

Michel Lee, Esq.
On behalf of the Council on Intelligent Energy & Conservation Policy (CIECP) and the Indian Point Safe Energy Coalition (IPSEC)
New York

Illustrative Studies & Analyses

NOTE: Bracketed synopses of specific points following citations have been the added for the purpose of advocacy and to support points of emphasis in the Comments, and are not intended to be summaries. Internal footnote references are excluded.

Multiple Exposures to Radiation

American Cancer Society, Radon: What is Radon? Accessed from web Jul 28, 2014. http://www.cancer.org/cancer/cancercauses/othercarcinogens/pollution/radon.

[Radon is a colorless, odorless, radioactive gas which forms naturally from the decay of radioactive elements, such as uranium. Radon gas from soil and rock in the ground can move into the air and into groundwater and surface water.

Radon decays into solid radioactive elements; and such radon progeny (eg, polonium-218, polonium-214, lead-214) can attach to dust and other particles and be inhaled into the lungs. As radon and radon progeny in the air break down, they give off high-energy radioactive alpha particles that can damage DNA inside the body's cells. According to the EPA, the average indoor radon level is about 1.3 picocuries per liter (pCi/L). People should take action to lower radon levels in the home if the level is 4.0 pCi/L or higher. The EPA estimates that nearly 1 out of every 15 homes in the United States may have elevated radon levels.

Scientists estimate radon exposure causes as many as 22,000 deaths in the US annually.]

Fazel R, Krumholz HM, Wang Y, Ross JS, Chen J, Ting HH, Shah ND, Nasir K, Einstein AJ, Nallamothu BK, Exposure to Low-Dose Ionizing Radiation from Medical Imaging Procedures, New England Journal of Medicine (2009); 361: 849-857. http://www.nejm.org/doi/full/10.1056/nejmoa0901249#t=articleBackground.

[Researchers are from Emory University School of Medicine; Yale University School of Medicine; Yale–New Haven Hospital; Mount Sinai School of Medicine; James J. Peters Veterans Affairs Medical Center; Columbia University Medical Center and New York Presbyterian Hospital; Mayo Clinic (MN); Johns Hopkins; Boston Medical Center; Veterans Affairs Ann Arbor Health Services Research and Development Center of Excellence; and the University of Michigan.

Analysis of data on a cohort of nearly 1 million adults ages 18 to 64 in the US enabled determination of estimated cumulative effective doses of radiation from medical imaging procedures. (Seniors were excluded, as they have a less significant long-term risk.) The investigation found that ~70% of the study population underwent at least one such procedure during the 3-year study period, resulting in mean effective doses that almost doubled what would be expected from natural sources. While most subjects received less than 3 mSv per year, effective doses of moderate, high, and very high intensity were observed in a sizable minority.

The evidence indicates that current health care practices are exposing ~4 million nonelderly adult Americans to "worrisome" cumulative effective doses exceeding 20 mSv per year.

In medicine, there exists an inherent difficulty in balancing the immediate – frequently substantial – clinical need for medical exposures to radiation against long-term stochastic risks. Recommendations have focused on justifying the clinical need for a procedure and optimizing its use to ensure that exposure is "as low as reasonably achievable" without sacrificing quality of care. But health care providers must recognize and inform patients about the risks of radiation. (One study of US health care providers using CT in patients with abdominal and flank pain, found that less than 50% of radiologists and only 9% of emergency department physicians reported even being aware that CT was associated with an increased risk of cancer.) An improved understanding of the risks of radiation is clearly needed.

The findings here of moderate, high, and very high annual effective doses – and not simply the overall population average – are important because many of these procedures are frequently performed on multiple occasions in the same person.

Several findings deserve mention: While younger people were less likely to receive high cumulative effective doses, rates for high and very high doses were "not trivial" in younger adults. More than 30% of men and 40% of women in this study population who received doses exceeding 20 mSv per year were under age 50.

High cumulative effective doses were also observed more frequently in women. "Understanding the age and sex distribution of effective doses of radiation from imaging procedures is critical because the related risks accrue over a lifetime and cancer may be more likely to develop in women than in men after similar levels of exposure."

The effective dose is a calculated estimate designed to provide a sex-averaged dose for a reference subject in a given exposure situation, not a dose for a specific subject. The effective dose calculation relies on assumptions regarding the radiation sensitivity of organs and tissues, half-life, distribution, and elimination kinetics. These assumptions have raised controversy, but effective dose remains the only measure currently available that reflects the overall potential biologic detriment across various types of radiation exposure.

A limitation of effective dose is that it was originally designed for use in a population, but risks of stochastic effects of ionizing radiation are dependent on age and sex. Also, doses from these

procedures vary across, and even within, institutions and can differ substantially in terms of the equipment used, the protocols in place, and the duration of exposure to radiation.

The current pattern of use of medical imaging in the US among nonelderly patients is exposing many to substantial doses of ionizing radiation., findings which have important implications for public health.]

Redberg, Rita F and Rebecca Smith-Bindman, We Are Giving Ourselves Cancer, New York Times Op-Ed, Jan 31, 2013. http://www.nytimes.com/2014/01/31/opinion/we-are-giving-ourselves-cancer.html.

[Rita F. Redberg, MD, a cardiologist, and Rebecca Smith-Bindman, a radiologist at the University of California, San Francisco Medical Center argue that overuse and inadequate regulation of radiation in diagnostic medicine is causing substantial harm. The authors state: "We are silently irradiating ourselves to death."

"The relationship between radiation and the development of cancer is well understood." High-dose CT scans are of particular concern. Americans exposure to medical radiation has increased significantly in recent decades. CT scans deliver 100 to 1,000 times more radiation than conventional X-rays. One in 10 Americans undergo a CT scan every year, and many of them get more than one. A single CT scan exposes a patient to the risk of cancer.

A 2009 study from the National Cancer Institute estimates that CT scans conducted in 2007 will cause a projected 29,000 excess cancer cases and 14,500 excess deaths over the lifetime of those exposed. Given the number of scans performed in recent years, "a reasonable estimate of excess lifetime cancers would be in the hundreds of thousands."

Two large clinical studies in Britain and Australia add to the evidence. In the British study, children exposed to multiple CT scans were found to be 3 times more likely to develop leukemia and brain cancer. A 2011 report by the Institute of Medicine (sponsored by Susan G. Komen) concluded that radiation from medical imaging, and hormone therapy, the use of which has substantially declined in the last decade, were the leading environmental causes of breast cancer. In addition to test overuse, there is scant oversight on machines, and no specific or mandatory guidelines with respect to delivered doses. Considerable variation exists from machine to machine and among institutions. The dose at one location can be 50 times higher than the dose at another. A study of one New York hospital, for example, found that nearly a third of its patients undergoing multiple cardiac imaging tests were getting a cumulative effective dose of more than 100 mSvs of radiation — equivalent to 5,000 chest X-rays.]

Rehani MM, Challenges in Radiation Protection of Patients for the 21st Century, American Journal of Roentgenology (AJR) (2013); 200 (4): 762-764. http://www.ajronline.org/doi/full/10.2214/AJR.12.10244.

[Madan M. Rehani, a radiation protection specialist at the International Atomic Energy Agency (IAEA), Radiation Protection of Patients Unit (Vienna) discusses the challenges of radiation protection in an era with widespread use of radiation in medical diagnostics. The author notes, "for thousands of patients, the individual patient dose received through diagnostic imaging exceeded an effective dose of 100 mSv."

The National Commission on Radiation Protection and Measurements estimates (for 2006) the percentage contributions to the U.S. population's radiation dose for medical exposure from different imaging modalities as 49% from CT; 26% from nuclear medicine; 14% from interventional fluoroscopy; and 11% from conventional radiography and fluoroscopy. Globally, the United Nations Scientific Committee on Effects of Atomic Radiation (UNSCEAR) estimates CT as contributing to about 35% of collective dose. CT is also the major contributor at the individual patient dose level. Another significant contributor to individual patient radiation dose is hybrid imaging, where the combined dose from the radiopharmaceutical + CT scan reaches an effective single exam dose of 10 mSv or more.

The International Commission on Radiologic Protection, UNSCEAR, the National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation (BEIR), and the National Commission on Radiation Protection and Measurements endorse the linear nonthreshold theory.

Dr. Rehani argues the need to reduce confusion and complexities of cumulative dose evaluation by eliminating the use of multiple units of measurement for radiation dose; i.e, millisieverts for effective dose and dose equivalent, and milligrays for organ and surface dose. "A unit scheme should be designed such that each unit immediately brings to mind the corresponding dose to which it refers, somewhat like degrees Celsius for temperature, kilometer for distance, and so on." Development of methods to track history of patient exposure is also an imperative. Physicians need ready access to a computer system that facilitates proper risk-benefit evaluation for each particular individual. Built-in features of this system should be alerts and checks that warn doctors of inappropriate referrals or the need to consult imaging specialists. A helpful tracking tool could be a card that allows a patient access to their radiation exposure history, along with implications of the dose.

Rehani writes: "Medical physicists should have the ability to obtain the radiation exposure history of an individual patient at the click of a mouse and the tools to estimate cancer risk for organs, rather than the current whole body dose–based approach. The medical physicist should receive alerts remotely whenever a patient receives a radiation dose that is above a defined level and whenever an imaging machine is not performing within stipulated tolerance levels of exposure parameters. Finally, to assess the level of optimization, medical physicists should have access to daily statistics on dose performance for each imaging examination, indicating the dose (with patient weight accounted for) and, wherever available, the indication for the examination, measured against corresponding reference levels."

Diagnostic reference levels should be available for all examinations, not just for those frequently performed. An important metric that is often unknown and difficult to ascertain is radiation dose variation as a function of time; i.e., over decades.

Systems should provide alert messages whenever a patient is pregnant or there is a need to ascertain pregnancy.

"With reports emerging about the cancer incidence in children who have undergone several CT scans, there is the need to further establish an authentic rationale on the radiation effects resulting from diagnostic exposures."]

Robertson A, Allen J, Laney R, and Curnow A, The Cellular and Molecular Carcinogenic Effects of Radon Exposure: A Review, International Journal of Molecular Sciences (2013); 14 (7): 14,024-14,063. http://www.mdpi.com/1422-0067/14/7/14024/htm.

[Authors from Royal Cornwall Hospital (UK) observe that chronic exposure to radon and its decay products is estimated to be the second leading cause of lung cancer after smoking.

Biologically damaging cytogenetic effects from alpha (α) particles produced by radon and its progeny – which include α particles, beta (β) particles and gamma (γ) radiation of various energies – could result from a variety of mechanisms. These include: mutations; chromosome aberrations; generation of reactive oxygen species (ROS); apoptosis; modification of the cell cycle; up or down regulation of cytokines; and the increased production of proteins associated with cell cycle regulation and carcinogenesis. There is also substantial evidence of bystander effects (cell signaling) which complicates calculation of risk estimates. At low doses cellular responses often appear to deviate (both beneficially and detrimentally) from the linear, no threshold model. At low doses, effects may also depend on cellular conditions, not just dose.

In sum, the cellular and molecular carcinogenic effects of radon are both numerous and complex.]

Vulnerable Populations: Developing Babies, Children, Adolescents, Women

Biro FM and Deardorff JD, Identifying Opportunities for Cancer Prevention During Preadolescence and Adolescence, Journal of Adolescent Health (2013); 52 (5): S15-S20. http://www.jahonline.org/article/S1054-139X(12)00414-4/fulltext.

["Early life exposures during times of rapid growth and development are recognized increasingly to impact later life." Epidemiologic studies document an association between exposures at critical windows of susceptibility. There is increasing awareness that early life events may shape developmental trajectories and impact later health. Breast cancer, for example, is believed to be linked to events during the early stages of life. This includes puberty.

"Puberty represents an important developmental window of vulnerability to environmental exposures." It is a time of rapid and profound change. In girls, "there is rapid expansion and differentiation of breast stem cells…which occurs contemporaneously with reactivation of the

hypothalamic-pituitary-ovarian axis, the onset of pubertal growth spurt, and the time of maximal accrual of bone mineral content."]

Braverman ER, Blum K, Loeffke B, Baker R, Kreuk F, Yang SP, and Hurley JR, Managing Terrorism or Accidental Nuclear Errors, Preparing for Iodine-131 Emergencies: A Comprehensive Review, International Journal of Environmental Research and Public Health (2014): 11: 4158-4200. http://www.mdpi.com/1660-4601/11/4/4158/htm.

[Article by scientists from the College of Medicine, University of Florida and McKnight Brain Institute; the PATH Foundation; Weill-Cornell Medical college; Texas Tech University; and the National University Hospital of Singapore.

The nuclear reactor incidents at Fukushima, Chernobyl and Three Mile Island "provide a sense of urgency and concern over the possible hazards of ionizing radiation to the thyroid from ^{131}I." (p 4159)

"Young children and developing fetuses are particularly vulnerable." (p 4184) The neonatal thyroid starts to concentrate iodine at about 12 weeks gestation. Due to its small size radiation doses to babies in utero are much higher than in children or adults. "Gaseous molecular iodine and gaseous particulate iodine are completely absorbed through the respiratory and upper digestive tract and occurs rapidly." (p 4170) The I-131 isotope is then transported in the bloodstream to the thyroid, where it concentrates. I-131 emits beta rays which have both mass and a negative electrical charge, they interact with surrounding tissue and transfer all of their energy within 1-2mm, potentially causing cellular damage or mutation.

Small doses increase thyroid cancer risk. Large radiation doses can destroy the neonate thyroid. (p 4178) Large quantities of radioactive iodine (I-131), can be released during the course of a nuclear accident. Actual events have shown that the plume of radioactivity can travel over 300 miles. Fukushima showed that the radioactive plume was able to migrate up to 50 miles daily.

Plume is defined as the initial cloud of concentrated radioactive particles released during an incident. "Plume size is directly related to wind speed and direction, wind turbulence resulting from solar heating, humidity and temperature, and the method of release." (p 4169) Plumes can thus spread considerable distances depending on meteorological conditions. An analysis of annual average wind speeds for regions of the US provides a rough estimate of 24 hour plume dispersion potentials. The continental wide average wind speed at the altitude of 80 miles falls between 8 and 23.5 miles per hour. Therefore, in a 24 h period radioactive plumes can disperse anywhere in the continental United states between 192 to 564 m, assuming all other factors, such as wind direction or wind speed, remain constant." (p 4170)

Dispersion data would need to be accumulated at the time of the accident. During Fukushima early radiation maps were not available, and some evacuation routes led through the primary contaminated area.

There are currently no real-time radioactive iodine monitoring systems utilized at the nuclear power plants in New York. (p 4174)

The authors' review of recommendations from the American Thyroid Association (ATA) and a body of other evidence shows "that at a minimum, the ATA, recommendation of 50-mile pre-distribution of KI" be implemented" and that the Food & Drug Administration (FDA), the Centers for Disease Control and Prevention (CDC), the NRC, and the National Council on Radiation Protection & Measurements, issue new protective guidelines. KI tablets should be part of all emergency plans and be made readily available within 200 miles of nuclear power plants.

"Pre-distribution of KI is necessary within 50 miles of any potential nuclear accident, and thyroid cancer risk areas extend 300 miles for children. This necessitates KI pre-distribution to all schools, hospitals and other of-interest sites extending 300 miles from any nuclear reactor. Evacuation or sequestering is impossible in congested urban areas. Evacuation protocols such as the plan recommended by the NYS Indian Point nuclear power plant do not extend past emergency planning zones and thus do not effectively address emergency procedures at distances exceeding 20 miles." (p 4185)]

Carpenter DO and Bushkin-Bedient S, Exposure to Chemicals and Radiation During Childhood and Risk for Cancer Later in Life, Journal of Adolescent Health (2013); 52 (5): S21-S29. http://www.jahonline.org/article/S1054-139X(13)00088-8/fulltext.

[Review by researchers from the Institute for Health and the Environment, University of Albany, Rensselaer.

Exposures to radiation and carcinogenic chemicals during gestation, childhood, and adolescence have been demonstrated to lead to cancer later in life. Developmental biology is crucial. Cells are rapidly dividing and organ systems are developing during childhood and adolescence, making these periods a time of sensitivity.

Carcinogens may act via mutagenic, nonmutagenic, or epigenetic mechanisms and may also disrupt endocrine systems. A variety of factors affect cancer risk, including individual genetic susceptibility, the inherent qualities of the chemical or radionuclide, magnitude of exposure, and duration of exposure. But age of exposure is one of the most important factors.

"The most vulnerable ages are the fetal and perinatal periods and the first years of life. Exposure to carcinogenic chemicals during fetal development poses a particularly significant risk because organ systems are developing. During this phase, cells are replicating rapidly and if DNA damage occurs, permanent defects may result and lead to cancer later in life. The fetus is completely dependent on the intrauterine environment, which is influenced by maternal health, diet, and chemical and radiation exposures. Many chemicals cross the placenta. Studies conducted between 2005 and 2011 reported the presence of at least 100 to more than 200 chemicals in the umbilical cord blood of 20 newborns." (p S22) Of these chemicals, 101 were present in every umbilical cord blood sample tested in one study. Known or suspected

carcinogens have been found not only in human cord blood, but also in amniotic fluid and breast milk. (p S22)

Intrauterine and nursing exposures also cause epigenetic changes. Some chemicals persist in the human body of the mother. Reservoirs of elements that deposit in bones and teeth, for example, can be mobilized during pregnancy and lactation.

Puberty and adolescence are also periods of increased vulnerability. "During adolescence, the endocrine, reproductive, neurological, and other systems undergo remarkable development and growth. The developing tissues and functions of these organ systems are particularly sensitive to the effects of carcinogenic and endocrine-disrupting chemicals." (p S22) Endocrine disruption may predispose an individual to eventual onset of cancer in hormone-sensitive organ systems such as the breast, ovaries, prostate, and testes. Evidence from the atomic bomb studies, for example, showed that relative risk for development of breast cancer was much higher for girls who were prepubescent at the time of the blasts than for women who were older than 40. (p S23)

"The combined effects of accumulating carcinogens, exposure to radiation, and epigenetic changes increase the child's risk for cancer as an adult." (p S22) General factors adding to increased lifetime risk for those exposed during early life stages are: (1) Children have a higher respiratory rate, and consume more food and water per pound of body weight, exposing them to relatively greater quantities of pollutants from air, food and water. (2) Children's immune systems are not fully developed. (3) Children have a greater likelihood of oral exposures because of hand-to-mouth behavior. (4) Children are closer to the floor/ground, and are closer to dust and spills. (5) Children have many years of expected life ahead often which time long-latency cancers can develop.

Authors conclude: "We must find ways to reduce human exposure to carcinogenic chemicals and ionizing radiation. Especially important is finding ways to reduce exposure during gestation, childhood, and adolescence because exposure during these critical periods of development leads to cancers later in life." (p S28)]

Grandjean P and Landrigan PJ, Neurobehavioural effects of developmental toxicity, Lancet (2014); 13 (3): 330-338.
http://www.sciencedirect.com/science/article/pii/S1474442213702783.

[Authors Philippe Grandjean and Philip J. Landrigan are with the Department of Environmental Medicine at the University of Southern Denmark (Denmark), Harvard School of Public Health, and the Icahn School of Medicine at Mount Sinai (NY).

Neurodevelopmental disabilities – including autism-spectrum disorder, dyslexia, and ADHD – seem to be increasing in prevalence and subclinical decrements in brain function are even more common. "All of these disabilities can have severe consequences – they diminish quality of life, reduce academic achievement, and disturb behavior, with profound consequences for the welfare and productivity of entire societies." (p 330) Strong evidence exists that industrial chemicals

widely disseminated in the environment are important contributors to "a global, silent pandemic of neurodevelopmental toxicity." (p 330)

"The developing human brain is uniquely vulnerable to toxic chemical exposures, and major windows of developmental vulnerability occur in utero and during infancy and early childhood. During these sensitive life stages, chemicals can cause permanent brain injury at low levels of exposure that would have little or no adverse effect in an adult." (p 330)

Researchers like to focus on acute exposures with a clear and immediate association between causative exposure and adverse effects. A recurring theme is that early warnings of subclinical toxicity are ignored or dismissed until decades pass and mounting evidence finally shows neurotoxicity at much lower exposure levels than had previously been believed safe.

We now know the developing human brain is exceptionally sensitive to injury caused by toxic chemicals and early life epigenetic changes can affect subsequent gene expression in the brain. Exposures to neurotoxicants are particularly likely to lead to functional deficits and disease later in life.

The damage is too often untreatable and permanent.

The authors argue for a precautionary approach that emphasizes prevention and does not mandate absolute proof of toxicity. A rational approach is to start to protect children and take into account the very large individual and societal costs that result from failure to act on available documentation to prevent damage to children. "Additionally, the strength of evidence that is needed to constitute 'proof' should be analysed in a societal perspective, so that the implications of ignoring a developmental neurotoxicant and of failing to act on the basis of available data are also taken into account." (p 336)]

Interagency Breast Cancer and Environmental Research Coordinating Committee (IBCERCC): Prioritizing Prevention: Breast Cancer and the Environment, Report of the Interagency Breast Cancer and Environmental Research Coordinating Committee (IBCERCC), Feb 2013. Link to full report via: http://www.breastcancerfund.org/media/press-releases/ibcercc-breast-cancer-prevention-report.html.

[The Interagency Breast Cancer and Environmental Research Coordinating Committee (IBCERCC) is a large panel comprised of experts from US federal health agencies and numerous institutions. It was established as a result of Congressional action.

Some 227,000 women and 2,200 men a year in the US will be diagnosed with breast cancer, and 40,000 a year will die from it. (Estimates for 2012.) Breast cancer has a large impact on its victims, their families, their communities, and the health care system at large. Survivors require lifelong medical surveillance and, often, additional treatment for additional treatment-related cancers and side effects. The personal and economic toll is enormous.

However, despite decades of research on diagnosis and treatment, “preventing this cancer is the only way to reduce the human toll of this disease that affects 1 in 8 women in their lifetime.” (p 2-1) Most breast cancers occur in people with no genetic history, so environmental factors broadly must play a major role in the etiology of this disease. Yet preventing breast cancer by finding ways to influence environmental causes has not been a priority.

Breast cancer is now known to be not one disease, but many; with differing incidence and mortality patterns by age, gender and race/ethnicity.

Important gene/environment interactions can occur over the course of a lifetime and special attention must be paid to periods of development when the breast may be most susceptible to exposures. Recognized risk factors such as radiation need to be examined in interaction with physical and chemical exposures.

Recent advances in science have helped elucidate previously little understood molecular behaviors. “The molecular and cellular changes that lead to breast cancer can occur early in life and endure across the life span.” (p 2-6) Notably, epigenetics has revealed alterations which do not involve changes in DNA sequences.

Importantly, there appear to be “windows of susceptibility” during the life course when specific exposures might have their greatest influence on lifetime breast cancer risk: “The mammary gland undergoes many stages of development (i.e., *in utero*, neonatal, pubertal, sexual maturity, pregnancy, lactation and lactational involution, post-involution) across the life span. These stages are regulated by endogenous physiology (i.e., hormones, growth factors, inflammatory processes, epithelial-stromal interactions, and metabolism originating from within the body) Epidemiologic and experimental animal studies demonstrate differences in mammary cancer risk and sensitivity to potential cancer-producing or cancer-promoting factors at different developmental stages – referred to as ‘windows of susceptibility.’” (p 5-1)

Two of these windows are *in utero* and puberty. Thus timing of exposure is critical.

Risk is also a factor of race. African American women are more likely than Caucasian or non-black Hispanic women to be diagnosed with tumors that have more aggressive features and arise before age 40.

The Committee urges federal agencies to develop standards that consider the full scope of evidence from *in vitro, in vivo, in silica* and epidemiologic studies regarding health risks and safety to the public to the extent possible. It is also important to develop and apply techniques – including biomonitoring – which measure levels and response to mixtures of exposures relevant to breast cancer with precision. Environmental exposures should be regularly monitored and biospecimens should be collected from diverse populations. Attention should be paid to different exposures which can be a concern in susceptible populations. (p 9-3)

The Committee strongly recommends establishing breast cancer prevention as a priority.]

International Physicians for the Prevention of Nuclear War (IPPNW): Health effects of ionizing radiation, German affiliate of International Physicians for the Prevention of Nuclear War (IPPNW) Summary of expert meeting, Ulm, Germany, Oct 19, 2013. (Provisional translation, Mar 2014). http://www.chernobylcongress.org/fileadmin/user_upload/pdfs/Health_effects_of_ionizing_radiation.pdf.

[Report on October 2013 meeting of physicians and scientists from the fields of radiobiology, epidemiology, statistics and physics in the city of Ulm (the birthplace of Albert Einstein) at the invitation of the German and Swiss affiliates of the International Physicians for the Prevention of Nuclear War (IPPNW). The conclusions of the Ulm experts meeting were:

(1) Even background radiation causes measurable detrimental health effects. "It is therefore misleading to claim that radiation exposure can be considered harmless as long as it falls within the dose range of 'natural' background radiation." (p 2)

(2) The use of radiation for medical diagnostics causes measurable health effects. Certain population groups are particularly radiosensitive, for example, women with a genetic predisposition for breast cancer. "Children and adolescents are at greater risk than adults, while the embryo has the highest vulnerability." (p 2)

(3) Nuclear activities cause measurable health effects. Nuclear accidents and weapons tests have distributed vast quantities of radionuclides around the globe, exposing large populations to increased radiation doses. "Even the event-free routine operation of nuclear power plants leads to discernible health effects in the surrounding population." Workers occupationally exposed to ionizing radiation show significantly higher rates of cancer, even when regulatory dose limits are not exceeded and their children "show a higher incidence of birth defects, leukaemia and lymphoma than other children." (p 3)

"As a result of low-dose exposure to radioactive iodine, thyroid disease, including cancer, can be observed in children, adolescents and adults. Furthermore, low-dose ionizing radiation causes severe non-malignant diseases, such as meningioma and other benign tumour entities, cardiovascular, cerebrovascular, respiratory, gastrointestinal and endocrinological disease, psychiatric conditions, as well as cataracts." (p 3)

Teratogenic effects have been observed in both human and animals following nuclear accidents, even in those only exposed to low levels of radiation. Some genetic effects occur in the first descendant generation, others only begin to appear in following generations. There is also evidence that in-utero and childhood exposure of the brain to ionizing radiation can impair cognitive development. "Numerous other studies also suggest genetic or epigenetic long-term damage caused by ionizing radiation." (p 3)

(4) Health risks of low-dose radiation can be reliably predicted and quantified on the basis of epidemiological studies and the concept of collective dose.

(5) The practice of basing risk factors for low-dose radiation on studies of Hiroshima and Nagasaki atomic bomb survivors is outdated. (Reasons detailed at p 5)

Risk estimates used by the International Commission on Radiological Protection (ICRP) and the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) are out of date. A risk factor of 0.2/Sv should be applied for predicting mortality from cancer and 0.4/Sv for incidence of cancer. But even those risk factors discount the sensitivity to ionizing radiation in fetuses and young children under age ten, which even ICRP has acknowledged is higher than adults by a factor of 3. In addition the "risk factors for predicting incidence and mortality of non-malignant physical disorders (non-cancerous disease), in particular cardiovascular disease, are of the same order as for malignant diseases." (p 4)

(6) An improved risk-based concept of radiation protection is needed, combined with stringent practices to minimize radiation exposure.

"The protection of unborn life and the genetic integrity of future generations should be given highest priority. Radiation protection must therefore supplement adult-based models and take into consideration the increased vulnerability of the embryo and the young child." (p 6)]

Makhijani A, Comments of the Institute for Energy and Environmental Research (IEER) on *Analysis of Cancer Risks in Populations near Nuclear Facilities: Phase I*, Prepublication copy, Jun 5, 2012. http://ieer.org/wp/wp-content/uploads/2012/06/ieer-analysis-cancer-risks-populations-comments-june2012.pdf.

[Comments authored by Arjun Makhijani, PhD, to the National Research Council, Committee on the Analysis of Cancer Risks in Populations near Nuclear Facilities – Phase I. Dr. Makhijani urges, *inter alia*, assessment of the following: (1) Tritium releases to the atmosphere by nuclear power plants. This information is especially important for the understanding of noncancer effects as well as cancer risks, particularly for in utero and early childhood exposures. (2) Exposure to multiple nuclear facilities. (3) Exposure to carcinogens from other facilities. (4) Exposure geography.

Study needs to be done which also looks at incidence of various cancers of *specific* types (eg, different leukemias, brain and nervous system cancers); morbidity; failed pregnancies; malformations as affected by early embryo/fetus exposure; and immune system function as affected by fetal exposure at the time of bone and bone marrow formation. It is thus critical to have reliable determination of strontium-90 exposure.]

Makhijani A, Smith B, and Thorne MC, Science for the Vulnerable: Setting Radiation and Multiple Exposure Environmental Health Standards to Protect Those Most at Risk, Report of the Institute for Energy and Environmental Research (IEER), Oct 19, 2006. http://ieer.org/wp/wp-content/uploads/downloads/reports/Science-for-the-Vulnerable.pdf.

[Detailed, exhaustive review by Arjun Makhijani, PhD, Brice Smith, PhD, and Michael C Thorne, PhD of the health risks of exposure to low level radiation and the extensive gaps and inadequacies in the radiation protection standards, which are long-outdated.

Among the many flaws of the regulatory scheme: Protection is geared almost solely to cancer risk and does not recognize noncancer illnesses, early failed pregnancies, or birth defects. There are no standards for protection of breast-fed children. Combined risks of exposure to radiation and toxic chemicals are ignored, even though some can affect the same systems adversely and may interact synergistically, not just additively. Theoretical models evaluating exposure of the embryo/fetus fail to consider beta-emitting radionuclides (especially low-energy beta emitters like tritium) and alpha emitting radionuclides that may cross the placenta. Considerations of damage to the immune system are ignored (isotopes like strontium-90 notably can affect the red bone marrow). (p 76)

A central principle of environmental protection must be to protect those most at risk.

The current US regulatory scheme utterly fails in the protection of women, children, babies and babies in utero; all groups which are especially vulnerable to radiation's effects.]

Mangano J, Report on Health Status of Residents in San Luis Obispo and Santa Barbara Counties Living Near The Diablo Canyon Nuclear Reactors Located in Avila Beach, California, Report for World Business Academy, Mar 3, 2014. **http://worldbusiness.org/wp-content/uploads/2014/03/Report-on-Health-Status-of-Residents-Near-Diablo-Canyon-Nuclear-Power-Plant.pdf.**

[Trend analysis by the director of the Radiation and Public Health Project (RPHP) of negative health indicators in San Luis Obispo County correlated with the operation the Diablo Canyon nuclear power plant. The two reactors at Diablo Canyon commenced operation in 1984 and 1985, respectively. The findings suggest that federally-permitted radiation releases from nuclear power poses a health risk to the public.

Among the key findings are that after Diablo Canyon commenced operation, (1) cancer rates in the county rose from relatively low to high (with increases translated into an additional 738 people diagnosed with cancer); (2) significant and rapid increases occurred for the incidence of thyroid and female breast cancer, both highly radiosensitive cancers; (3) infant mortality rose significantly; (4) melanoma incidence rose significantly, and became the highest of all California counties; (5) child/adolescent cancer mortality rose rapidly; (6) cancer mortality for all ages rose significantly.

In addition, in the 10 zip codes closest to Diablo Canyon there was a greater rise in the rates of infant mortality, low weight births, and total mortality, than in the 5 zip code areas in the city of Santa Barbara ~90 miles from the reactors.

The major findings of this report show increases in various rates of disease and death in San Luis Obispo County, as compared to California state averages following start of plant operation. This

includes increases in infant mortality, child/adolescent cancer mortality; cancer incidence for all ages – but especially thyroid cancer, breast cancer and melanoma; and cancer mortality for all ages.

The study concludes: "While many factors can affect disease and death rates, the official public health data presented in this report suggest a probable link between the routine, federally-permitted emissions of radioactivity from the Diablo Canyon nuclear power plant and elevated health risks among those infants, children and adults living closest to the reactors."

The data is consistent with earlier studies showing significant declines in local illness and death rates following shutdown of the Rancho Seco nuclear power plant in Sacramento County in 1989.]

Mangano JJ and Sherman JD, Long-term Local Cancer Reductions Following Nuclear Plant Shutdown, Biomedicine International (2013); 4 (1): 1-12.
http://www.bmijournal.org/index.php/bmi/article/viewFile/115/82

[Epidemiologic study authored by Joseph Mangano, Executive Director of the Radiation and Public Health Project (RPHP) and Janette Sherman, MD, a toxicologist. It reports the first long-term study of the full-population health impacts of the closure of a U.S. nuclear reactor. In 20 years following closure, the authors found 4,319 fewer cancers, including notable drops in cancer for women, Hispanics and children.]

Miller, Mark, Catherine Metayer, and Gary V. Dahl, Childhood Leukemia: An Ounce of Prevention, Environmental Health Policy Institute, Jun 24, 2014.
http://www.psr.org/environment-and-health/environmental-health-policy-institute/childhood-cancer-and-the-environment.html.

[Author Mark Miller, MD, is an Assistant Clinical Professor in the Departments of Pediatrics and Occupational and Environmental Medicine at the University of California San Francisco (UCSF) and the Director of the UCSF Pediatric Environmental Health Specialty Unit (PEHSU). Catherine Metayer, MD, PhD, is Professor at the UC Berkeley, School of Public Health and Director of the Center for Integrative Research on Childhood Leukemia and the Environment (CIRCLE). Gary V. Dahl, MD is a Professor of Pediatrics and the Program Director for the Hematology/Oncology Fellowship Training Program at Lucile Salter Packard Children's Hospital at Stanford University.

During the 35 years between 1975 and 2011, there has been a 55% increase in the number of children diagnosed annually (per capita, age adjusted) with leukemia and substantial evidence of risk factors has emerged over the past two decades. "This growing body of literature has implicated the potential role of environmental hazards in the etiology of childhood leukemia. Exposures to ionizing radiation, solvents, pesticides, and tobacco smoke have consistently demonstrated positive associations with the risk of developing childhood leukemia."

The failure to take steps toward prevention of childhood leukemia stems from a reluctance to attribute environmental factors as causative. This is due, in part, to the high bar required to reach consensus on causation and the fact that double blinded randomized studies would be unethical. However, in their paper on smoking, breast cancer and the increasing cost of caution, Glantz and Johnson cite the findings of the Surgeon General's Report on Smoking and Health of 1964 which stopped short of determining smoking caused cardiovascular disease. The report nevertheless concluded action was warranted:

"'Although the causative role of cigarette smoking in deaths from coronary disease is not proven, the Committee considers it more prudent from the public health viewpoint to assume that the established association has causative meaning than to suspend judgment until no uncertainty remains'."

It is time to use the hard-won accumulating knowledge in children's environmental health to begin fostering prevention activities, including those targeted at the prevention of childhood leukemia."]

Mothersill C and Seymour C, Implications for human and environmental health of low doses of ionizing radiation, Journal of Environmental Radioactivity (2014); 133: 5-9. Abstract. http://www.sciencedirect.com/science/article/pii/S0265931X1300091X

[Paper by Carmel Mothersill and Colin Seymour of the Medical Physics and Applied Radiation Sciences Department, McMaster University (Canada).

In the last 20 years there has been a major paradigm shift in radiation biology. Discoveries challenge the DNA centric view which holds that DNA damage is the only critical effect of low-dose radiation.

It was long assumed that the more energy deposition, the more DNA damage and the greater the biological effect. This belief is embodied in radiation protection regulations as the linear-non-threshold (LNT) model.

However the science underlying the LNT model – particularly with respect to environmental exposure – is simplistic and outdated. It is now clear that, at low doses, cells, tissues and organisms respond to radiation by inducing responses which are not readily predictable by dose. These include adaptive (or non-targeted) response phenomena: bystander effects, genomic instability and low dose hypersensitivity. Organism "stress" response behaviors are influenced by genetic, epigenetic and environmental factors.

Recent findings show that low dose radiobiology is very complex and that supra linear or sub-linear (even hormetic) responses are possible and their occurrence is unpredictable at the given system level. Radiation protection models need reevaluation and may further require consideration of the synergistic or antagonistic effects of other pollutants. Radiation protection, at present, only looks at radiation dose but the new radiobiology means that chemical or physical agents, which interfere with tissue responses to low doses of radiation, could critically alter predicted risk.]

National Academy of Sciences, Health Risks From Exposure to Low Levels of Ionizing Radiation, BEIR VII – Phase 2, Report of the Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation to the National Research Council of the National Academies, Washington, D.C. National Academies Press (2005) (known as the "BEIR VII" study). {*Page numbers correlate to prepublication hardcopy}.* http://books.nap.edu/catalog.php?record_id=11340.

[The U.S. National Academy of Sciences Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation (a/k/a, Committee on the Biological Effects of Ionizing Radiation) issued this study as the 7th in a series of reports from the National Research Council prepared to advise the US government. This report is known as the BEIR VII study. Low linear energy transfer (low-LET) radiation (x rays and γ rays) is the subject of the report. (pp 1 & 47) Authors note, however, that high linear energy transfer (high-LET) radiations (α or heavier ion particles) can cause more damage per unit absorbed dose, which is why a weighted quantity, equivalent dose, or its average over all organs – *effective dose* – is used for radiation protection purposes. For low-LET, equivalent dose = absorbed dose. (p 1) "In this report, "only data that are of utility to a quantitative assessment of a dose-response relationship" between radiation exposure and cancer are included. Thus, to avoid dosimetry uncertainties, BEIR VII relies principally on findings from atomic bomb survivor and medical radiation exposure studies, as opposed to ecologic studies of populations living near nuclear facilities, since those do not contain individual estimates of radiation dose. (See, eg, pp 26- 28, 423 & 483)

The BEIR VII Committee concludes there is no level of radiation which can be considered harmless. Ionizing radiation, by definition, contains enough energy to displace electrons and break chemical bonds. Specifically, the Committee judged that the linear no-threshold (LNT) model provides the most reasonable description of the relation between low dose exposure to ionizing radiation and the development of cancer. (pp 13, 154 & 571) (p 12) On average, assuming a sex and age distribution similar to the entire US population, the BEIR VII lifetime risk model predicts that ~1 in 100 persons would be expected to develop a solid cancer or leukemia from a single exposure to 0.1Sv of low-LET radiation, while ~1 in 1000 would develop cancer from an exposure to 0.02Sv. (p 15)

However, sex and age at exposure modify risk at the individual level. Also, a dose-relationship with mortality from non-neoplastic disease mortality has been found in a number of analyses. For example statistically significant associations have been found for the categories of heart disease, stroke, and diseases of the digestive, respiratory and hematopoietic systems. (pp 272 & 282) Studies with large cohorts of both child and adult patients treated with radiation therapy for non-malignant diseases (eg, peptic ulcers, ankylosing spondylitis, benign breast and gynecological diseases) have found statistically significant increased risk for many forms of cancer. (pp 284-299) For example, a study of a cohort of women who received repeated fluoroscopic examinations to monitor lung collapse found that incidence of breast cancer increased the younger the rate of exposure and the excess breast cancer risk – which only became apparent as of 15 years after exposure – remained high for the period of observation of over 50 years. (pp 298-299)]

Treatment for childhood cancers has prolonged the life expectancy of children with cancer, but the risk of developing second cancers later in life is elevated by treatment, notably studies have found significant increased risk of bone cancer and thyroid cancer has been found among patients who received radiation therapy. (pp 283-284)

Pre-natal radiation exposures elevate childhood radiation risk at a statistically significant level, with excess cancers detected at doses of 10mSv. (pp 302-303)

The BEIR VII study concluded that there is no level of radiation which can be considered harmless.]

Olson, Mary, Atomic Radiation is More Harmful to Women, Nuclear Information and Resource Service Briefing Paper, 2014.
https://www.nirs.org/radiation/radhealth/radiationwomen.pdf.

[Briefing based on analysis by Mary Olson, Director of the NIRS Southeast Office, elucidating data contained in the 2006 study of the National Academy of Sciences (NAS) titled "Biological Effects of Ionizing Radiation (BEIR VII) – Health Risks from Exposure to Low Levels of Ionizing Radiation" (known as the BEIR VII study or report).

BEIR VII focuses only on radiation doses delivered from outside the body: gamma (γ) and X-rays – with little attention to doses from radioactivity received internally from alpha (α) and beta (β) particles. BEIR VII also focuses narrowly on cancer risk. "Nonetheless, the NAS report is stunning enough: it finds that harm to women (cancer) is 50% higher than the comparable harm to men from radiation doses that fall within the legal limit to the public over a lifetime. Let's be clear: radiation kills men – but it kills significantly **more** women. *Both cancer incidence and death are 50% higher for women*."

Harm from radiation can depend upon the amount, type (internal/external), and timing of exposure as well as the presence of other carcinogens and stressors. Children are significantly more vulnerable than adults.

While the NAS focus in BEIR VII was on external exposure, internal exposure – *ie*, radioactivity taken inside the body via inhalation, absorption and ingestion – is substantially different. High-LET α and β particles travel with a force which, combined with their greater mass, may inflict greater damage to living tissue than x-rays "Internalized radiation also results in higher doses since every internal emission absorbed, at zero distance to the impacted tissues, will cause radiation impact for as long as it is in the body, and may concentrate in the most vulnerable areas, such as gonads or bone marrow."

Exposure from industrial activity comes on top of natural radiation from terrestrial sources (primarily uranium and its decay progeny in rocks and earth) and non-terrestrial sources (γ rays from space). Notably, "'natural radiation' results in 'natural cancer.'"

Reactors routinely release radioactivity to air, water and as solid waste, with ongoing potential for radiation exposure even without an accident. "The radioactivity generated by a single 1000-megawatt nuclear reactor unit *per year* is on the scale of 1000 detonations of an atomic bomb like the one that destroyed Hiroshima."

The growing number of nuclear accidents (Fukushima; Chernobyl; Three Mile Island; Brookhaven; Santa Suzanna; Fermi 1; Kyshtym; Windscale; et al) also add to levels of so-called "background" radiation; increasing actual received human doses.]

SEER Cancer Statistics Review, 1975-2011, National Cancer Institute. Bethesda, MD, Howlader N, Noone AM, Krapcho M, Garshell J, Miller D, Altekruse SF, Kosary CL, et al., (ed.s), review based on Nov 2013 SEER data submission, published on SEER web site, Apr 2014. http://seer.cancer.gov/csr/1975_2011/.

[The incidence of childhood leukemia (age 0-14 years) in the US has increased an average of 0.7 % per year since 1975.]

Sly L and Drisse MNB, Children's Vulnerability to Their Environment, Journal of Environmental Immunology and Toxicology (2013); 1: 58-65. http://www.stmconnect.com/sites/default/files/20140511082359.pdf.

[Article by scientists from Queensland Children's Medical Research Institute, University of Queensland (Australia) and the World Health Organization, Department of Public Health and Environment documenting the overall vulnerability of children to toxic exposures.

Mothers' exposures both prior to conception and during pregnancy are associated with a variety of outcomes, including spontaneous abortion, stillbirth, neonatal death, and birth defects. After birth, the infant can be exposed via the mother's milk.

Children – both prenatally (via the mother) and after birth – are increasingly exposed to chemicals and environmental toxicants, and exposures are occurring at times when organs are forming or maturing, resulting in structural and functional defects that can adversely affect their health.

Children are actively growing, meaning they are in an anabolic state. They breathe more air than adults relative to body size; they have a higher metabolic rate (thereby ingesting more food and water per unit body mass); they have a larger surface to body mass ratio than; and they have longer expected life spans over which illness may develop.

Children are also more exposed via their behavioral interactions with their environment. Infants can be exposed via the mother's milk. Babies and young children crawl, play on the ground, and put things in their mouth. ("Young children are not adverse to eating sand and dirt." p 63.) Thus, there are pathways in which the young are exposed that are not applicable to the adult.

The developing prenatal immune system is vulnerable to exposures (via the mother) at critical time points in development which can affect later health. After birth, prolonged periods of postnatal maturation of organs and systems such as the lungs, brain and immune systems make them vulnerable to environmental exposures. Major events occur in immune system development for instance: initiation of hematopoiesis; migration of stem cells and expansion of progenitor cells; colonization of bone marrow and thymus; and maturation to immunocompetence and the establishment of immune memory. Similarly, the brain and central nervous system develops well into adolescence, and levels of exposure that produce few or no effects on the adult brain may pose serious threats to the developing nervous system.

Humans in utero, during early post natal life, and up to the end of adolescence are especially vulnerable to environmental insults which may have long-term immune, neurological, and developmental consequences. Moreover, low-dose exposures which may yield small statistical risks applicable to the individual child, does not always reflect the broader implications of exposure for the overall population. A mild but chronic impact could add up to a substantial population level burden. Reducing potentially adverse neurodevelopmental impacts on children age 5 and younger should be prioritized.

When setting exposure standards, authorities need to account for the special vulnerability of children, though few do. Authors strongly urge adoption of the Precautionary Principle.]

White MC, Pelpins LA, Watson M, Trivers KF, Holman DM, and Rodriguez JL, Cancer Prevention for the Next Generation, Journal of Adolescent Health (2013); 56 (5): S1-S7. http://www.sciencedirect.com/science/article/pii/S1054139X13001250.

[Authors from the CDC observe that the number of people with cancer in the US is growing and, during the past decade, rates have increased for all childhood cancers.

Thus primary prevention of cancer remains an urgent public health priority.

Research during the past several decades has vastly changed our understanding of cancer. What was once understood to be a single disease is now understood to comprise distinct types, each of which may have a different etiology. Broadly, cancer development results from a complex interaction of individual, societal, and environmental factors. While the biology is still not completely understood, findings point to multiple cellular pathways by which different cancer risk factors could affect the multistep evolution of normal cells into cancer cells during a person's lifespan. Some researchers, for example, believe cancers like lung cancer and breast cancer may require 5 or 6 steps of development. (Steps can include mutations in oncogenes, mutations in tumor-suppressor genes, and changes that lead to downstream changes in RNA and protein processing.) And recent work into the different intrinsic subtypes of breast cancer has found that one type, basal-like, is characterized by up-regulation of certain proliferation genes and nonexpression of various receptors. Basal-like or triple negative tumors are typically more aggressive, often occur in women younger than age 40, and have a worse prognosis.]

Environmental Justice: African American Women at Heightened Risk of Breast Cancer

DeSantis CE, Fedewa SA, Sauer AG, Dramer JL, Smith RA, and Jemal A, CA (Cancer Journal for clinicians) (2015) http://onlinelibrary.wiley.com/doi/10.3322/caac.21320/full.

[Authors are with the American Cancer Society and Emory University School of Medicine. With the exception of skin cancers, breast cancer is the most common cancer among US women, accounting for nearly 1 in 3 cancer diagnoses. It is the second leading cause of cancer death among women after lung cancer. This article describes trends in breast cancer incidence, mortality, survival, and screening by race/ethnicity in the US as well as state variations in these measures. Data is from the Surveillance, Epidemiology, and End Results (SEER) program of the National Cancer Institute, the North American Association of Central Cancer Registries (NAACR), and the National Center for Health Statistics.

"Although often referred to as a single disease, breast cancer is distinguished by up to 21 distinct histologic subtypes and at least 4 different molecular subtypes, which are associated with distinct risk factors and are biologically variable in presentation, response to treatment, and outcomes." Compared with white women, black women are more likely to have the aggressive HR−/HER2− subtype of the disease known as triple negative breast cancer, which has a poorer prognosis. Triple negative breast cancers account for 22% of the cases among black women (compared with 11% among white women).

African American women are being diagnosed with breast cancer at younger ages and die from the disease at younger ages than white women (whose incidence rates have been stable since 2004). Overall, "the breast cancer death rate is 42% higher in blacks than in whites."]

Field LA, Love B, Deyarmin B, Hooke JA, Shriver CD, and Ellsworth RE, Identification of differentially expressed genes in breast tumors from African American compared with Caucasian women, Cancer (2012); 118 (5): 1334-1344. Abstract. http://onlinelibrary.wiley.com/doi/10.1002/cncr.26405/abstract;jsessionid=4AC3732B16A34D2EC613C6C6D82FA8F2.f04t04.

[Researchers are from the Windber Research Institute; BioReka, LLC; Walter Reed Army Medical Center; Henry M. Jackson Foundation for the Advancement of Military Medicine.

Breast cancer tumors in African American women have less favorable pathological characteristics and African American women have higher mortality rates than Caucasian women. While socioeconomics may influence prognosis, biological factors appear to contribute to tumor behavior.

Here the researchers found that, despite matching of tumors by pathological characteristics, the molecular profiles of African American women and Caucasian women differed in both invasive tumors and benign breast tissues. The differentially expressed genes (including CRYBB2, PSPHL, and SOS1) identified, are involved in cellular growth and differentiation, invasion,

metastasis, and immune response and thus may contribute to the poor outcome in African American women.]

Environmental Justice: Native Americans Unduly Burdened by Radioactivity

Daitz, Ben, MD, A Doctor's Journal: Navajo Miners Battle a Deadly Legacy of Yellow Dust, New York Times Contributing Column, May 13, 2003.
http://www.nytimes.com/2003/05/13/health/a-doctor-s-journal-navajo-miners-battle-a-deadly-legacy-of-yellow-dust.html:http://www.nytimes.com/2003/05/13/health/a-doctor-s-journal-navajo-miners-battle-a-deadly-legacy-of-yellow-dust.html.

[Ben Daitz, a physician and professor at the University of New Mexico School of Medicine describes his visit to a Navajo reservation and a clinic serving Navajo suffering from the ravages of uranium mining:

"The Diné (pronounced dee-NAY) or 'the People,' as the Navajo call themselves, have many stories about their origins. One says that as they emerged from the fourth world into the fifth and present world, they were given the choice of two yellow powders. One yellow powder was corn pollen, and that was the one they chose."

The other was the color of the yellowcake, uranium oxide.

"The Spirits said it had to be left alone. But from the late 1940's through the mid-80's, yellowcake was picked and shoveled and blasted and hauled in open-bed trucks, and then dried in mountainous piles at multiple sites in the American West. The Navajo, whose lands extend over western New Mexico, eastern Arizona and southern Utah, were at the epicenter of the uranium-mining boom, and thousands of Navajos worked in the mines. More than 1,000 abandoned mine shafts remain on Navajo land.

"The consequences are measured today, decades after the mines closed, in continuing health problems and degraded land.

"Under the Radiation Exposure Compensation Act of 1990, people exposed to radiation through uranium mining and milling or through weapons testing are eligible for government compensation."

"More than 500 uranium miners died of lung cancer from 1950 to 1990. Hundreds more will die of lung cancer in the coming years, a study by the Public Health Service predicts. A majority of the deaths stemmed from exposure to radiation from the breakdown of uranium products. These so-called radon daughters attach to dust particles, and when workers inhale the dust, the particles lodge in their lungs, where they release high doses of radiation."

One patient, John James 67, who was on oxygen and sought attention at a clinic for coughing up blood, had started mining in 1956 in Utah. "'We brought dust home on our clothes,' he told the

doctors. ‘We contaminated our families. I saw the yellowcake there. It looked like it was burning.’"

“The doctors saw six patients that morning. Most of the old miners drove at least 100 miles to get there, and they will keep returning for testing, betting that the sad chapter of their past will somehow compensate them for the present, before they die.”

Mitchell Capitan, a former mining technician president of the Crownpoint chapter of the Eastern Navajo Agency, founded Endaum, Eastern Navajo Diné Against Uranium Mining. The group was battling against a plan for uranium mining using a leeching using water from the Westwater Canyon Aquifer under Crownpoint, the sole source of drinking water for the Crownpoint area providing for 15,000 people. Capitan says: "‘People come here from all over these parts, from 50 miles away, to truck this water back to their houses, to drink it, because it's the only pure supply. Their own water is bad -- contaminated….'This uranium impacts on our water, our air and our cultural identity,’” he said. "’We've already had enough uranium.’" At a gathering Mr. Capitan stood under an Endaum banner which said in Navajo and English: "One Mind, One Voice, One Prayer, One People."]

Loomis, Brandon, Abandoned uranium mines continue to haunt Navajos on reservation, The Republic /AZ Central, Aug 4, 2014.
http://www.azcentral.com/story/news/arizona/investigations/2014/08/04/uranium-mining-navajos-devastating-health-effects/13591333/.

[The Colorado Plateau is “scarred, poisoned and frightening a people who still live with the radioactive residue of 521 abandoned mines scattered across their reservation’s 17.2 million acres.” The Environmental Protection Agency (EPA) reached a $1 billion settlement with Anadarko Petroleum Co. for past mining by subsidiary Kerr-McGee Corp., which Anadarko acquired in 2006. The money is part of a record $5 billion nationwide settlement for a number of environmental violations. But the $1 billion for Navajo cleanup is only enough to cleanup a few dozen (49) abandoned uranium mines, and no company has accepted liability for the rest. The federal government has promised cleanup, but at current low funding levels that could take 100 years to complete.

Uranium ore and debris emits alpha particle radiation which does not penetrate skin, but can be ingested or breathed as dust. Much of the toxic material is expelled by the body within days, but with chronic exposure, it can accumulate in the bones and stress the kidneys as they work to expel it. Chronic exposure, the Environmental Protection Agency, says, is known to damage kidneys and increases risk of cancer and liver disease. At certain mine sites, gamma radiation – a high-frequency penetrating radiation – is also well above regulatory doses. EPA scans of 474 abandoned uranium mines on the Navajo reservation found that 403 had gamma radiation at 2 times the background level, and 226 showed radiation at 10 times background.

Church Rock, NM, has two massive uranium waste-rock piles. A 1979 dam break at the site unleashed one of the largest radioactive uranium contamination accident in US history. “Contamination flooded downstream to the Rio Puerco and Gallup, eventually disappearing out

of sight and beyond tracking in the aquifer somewhere above the confluence with Arizona's Little Colorado." While the companies mining at Church Rock ceased digging uranium in the 1980s, site cleanup remains to be done. In addition, a Texas-based corporation, Uranium Resources Inc., wants to use an in-ground leaching system to dissolve and extract uranium for nuclear power plants. Uranium Resources controls a private square of land surrounded by Navajo reservation land, and wants right of access.

A study published in the journal Health Physics in 2000 found Navajo uranium miners had a lung-cancer rate nearly 29 times that of Navajos who didn't work in the mines. From 1969 to 1993, two-thirds of new lung cancers in Navajo men afflicted miners.

Pulmonary fibrosis, multiple-organ failure, and kidney disease also commonly afflicts miners, said Gary Foster, a visiting nurse who treats miners in Colorado. "'If they don't lose their ability to breathe,' he said, 'they're all going to get cancer'. … 'All of the patients I've dealt with, they've had to have it explained to them why they're sick…They don't understand the concept of it sitting in their lungs and staying there forever.'"

Now the children of old miners – who grew up drinking from contaminated wells – are falling ill even as their own children play around abandoned uranium pits and piles.

No funding has been made available to study the full effects of chronic uranium exposure in the Navajo population. Dr. Charles Wiggins, director of the New Mexico Tumor Registry notes: "We don't really have a lot of solid studies that document the effects of exposure."

However a study of health effects upon mothers and babies is being conducted by the University of New Mexico in collaboration with the Southwest Research and Information Center. University of New Mexico researcher Jennifer Ong reports early results show uranium is ubiquitous in blood and urine samples – including in babies. Virtually all of the first 208 samples had uranium levels above the 50th percentile for the US, and 15% spiked past the 95th percentile.]

Moore-Nall, A, The Legacy of Uranium Development on or Near Indian Reservations and Health Implications Rekindling Public Awareness, Geosciences (2015); 5 (1): 15-29. http://www.mdpi.com/2076-3263/5/1/15/htm.

[Author Anita Morre-Nall, of the Department of Earth Sciences at Montana State University, notes energy material from uranium mining and milling activity contains harmful chemical substances that – if mobilized into air, water, or soil – can adversely impact human health and environmental quality. The legacy of uranium procurement in the U.S. has left a legacy of long-lived health effects for many Native Americans. The largest population and some of the most impacted people are the tribes living in the Southwest, especially the Navajo, but also Sioux, Spokane Nation and many others.

"As a result of the mining activity much of the population of the Navajo Nation residing near the areas of mining or milling has had their health compromised. Most of the 1000 unsealed tunnels, unsealed pits and radioactive waste piles still remain on the Navajo reservation today, with Navajo families living within a hundred feet of the mine sites."

Uranium mining has also left a legacy of contaminated groundwater and tailings on the Wind River Reservation, Wyoming, home to Eastern Shoshone and Northern Arapaho Indians. "Increased incidences of cancers among its peoples are attributed to the old Susquehanna-Western uranium mill tailings site. The site is a few miles southwest of Riverton, the ninth most-populated city in Wyoming. In some areas of the Wind River Indian Reservation groundwater contamination is so bad that the Department of Energy (DOE) estimates drinking water from contaminated aquifers could make residents up to 10 times more likely to develop cancer than the general population."

Pacific Northwest tribal groups on nine reservations in Washington, Idaho and Oregon have also been impacted by Hanford Nuclear reservation activities. The peoples of these reservations traditionally used and continue to use the lands and resources from the Columbia River Plateau region including land ceded to the government for which they retained hunting and gathering privileges. "Thus, they may have been exposed to more radiation and contaminants than the general public in practicing traditional lifestyles while fishing, hunting game, food gathering (berries, root plants, *etc.*) harvesting medicinal plants and traditional practices (*i.e.*, sweats), as well as social and spiritual interaction networks."

The other four reservations, the Nez Perce, Confederated Tribes of the Umatilla, Confederated Tribes and Bands of the Warm Springs and the Yakama Nation are known to consume large quantities of fish and likely received higher doses of river borne releases which resulted from radiation releases into the Columbia River water. In addition, liquid waste that had been poured onto the ground or held in ponds or trenches at the Hanford reservation evaporated or soaked into the soil on the site. The contaminated areas are thought to have also created underground "plumes" of contaminants which could also affect native peoples who consume native food sources in the area.

A study in the Eastern Agency of the Navajo Nation, NM found that, despite decades of inactivity in the mines and mills investigated, environmental contamination was widespread, often near homes, livestock grazing areas, and locations frequented by children and families. "The uranium contamination in this area was predominantly in the highly soluble chemical forms that could be spread when disturbed or by the bursts of precipitation that occur in this semiarid region at certain times of the year."]

Health Effects Are Not Just Cancer

Averbeck D, Non-targeted effects as a paradigm breaking evidence, Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis (2010); 687 (1-2): 7-12. Abstract. http://www.sciencedirect.com/science/article/pii/S0027510710000266.

[Researcher from Institut Curie-Section de Recherche, Centre Universitaire (France) questions the scientific validity of the linear no-threshold concept for low-dose radiation exposures.

Modern science has shown that cells and tissues and whole organisms react in complex ways following biologic insult.

The classical paradigm of radiobiology was based on the concept that all radiation effects on living matter are due to the direct action of radiation. But the discovery of non-targeted and delayed radiation effects challenges this concept, and indicates a new paradigm may need to be developed to provide protection against low radiation doses.

Recurrent themes of recent findings are: the low-dose radiation-induced bystander effect; genomic instability; radiation hypersensitivity; hormesis; and radioadaptive and transgenerational responses. "Most of these phenomena include (1) intra- and intercellular signaling, involving reactive oxygen species (ROS). This signaling may be transient or persistent, and may involve the release of cytokines (bystander effect, genomic instability) or epigenetic changes (translesional responses), (2) a large variability of responses depending on the type of radiation, genotype (DNA repair capacity) and physiological state of the cells and tissues. Many more parameters are involved in responses at low doses than at high doses, and different pathways are activated." At low doses, non-linear responses have been shown to occur which cannot be accounted for using the linear no-threshold concept.]

Azzam EI, Jay-Gerin J-P, and Pain D, Ionizing radiation-induced metabolic oxidative stress and prolonged cell injury, Cancer Letters (2012); 327 (1-2): 48-60. Abstract. http://www.sciencedirect.com/science/article/pii/S0304383511007592.

[Authors are faculty members of the UMDNJ – New Jersey Medical School and the Sciences de la Santé, Université de Sherbrooke (Canada).

Cellular exposure to ionizing radiation can result in the immediate alteration of atomic structure and cause DNA damage. However radiation exposure can also be an oxidizing event leading to persisting biologic processes. Oxidative damage may spread from targeted to non-targeted bystander cells through redox-modulated intercellular communication mechanisms. "To cope with the induced stress and the changes in the redox environment, organisms elicit transient responses at the molecular, cellular and tissue levels to counteract toxic effects of radiation."

Specifically, during and shortly following exposure to radiation, metabolic pathways are induced which may or may not adequately cope with the stress. "When the harmful effects exceed those of homeostatic biochemical processes, induced biological changes persist and may be propagated to progeny cells." Physiological levels of reactive oxygen (RO) and nitrogen species (NS) play critical roles in many cellular functions. In cells exposed to radiation, levels of these reactive species may increase due to perturbations in oxidative metabolism and chronic inflammatory responses, thereby contributing to the long-term effects of radiation exposure on genomic stability.

Notably, delayed biologic outcomes can emerge from isotope impact on mitochondrial DNA and mitochondrial protein import. "Defects in mitochondrial functions lead to accelerated aging and numerous pathological conditions."]

Busby C, Aspects of DNA Damage from Internal Radionuclides, Chapter 22 of "New Research Directions in DNA Repair," (INTEC, 2013): 598-637. http://dx.doi.org/10.5772/53942. http://www.nuwinfo.se/files/InTech-Aspects_of_dna_damage_from_internal_radionuclides.pdf.

[Chapter authored by Christopher Busby of Jacobs University (Germany) highlighting evidence which shows that ingestion of particulate – i.e., alpha (α) and beta – radionuclides represent a significant hazard to human health, and one not easily or accurately modeled by analogy with external photon radiation gamma (γ) or x rays. The detrimental health effects of Tritium (H-3) and Carbon-14, and fat soluble gases like Krypton-85, in particular have been effectively ignored by risk models.

A key point is that, for certain exposure regimes, "the ionization density at the DNA and the damage to the DNA can be extremely high even though the absorbed dose, as calculated by the current methodology, may be extremely low." (p 610)

Additionally, risk models ignore the increased risk of noncancer diseases which are being imposed upon populations exposed to increasing buildup of radiation in the environment as a result of continual emissions from nuclear power plants, et al. Cancer yield is simply not a proper representation of the effects of radiation exposure.]

Byun H-M and Baccarelli AA, Environmental exposure and mitochondrial epigenetics: study deign and analytical challenges, Human Genetics (2014); 133 (3): 247-257. http://www.ncbi.nlm.nih.gov/pubmed/24402053.

[Review by authors from the Laboratory of Environmental Epigenetics at Harvard School of Public Health of the literature and research challenges relative to epidemiological studies and the need to increase focus on mitochondrial epigenetics.

"The environment plays a critical role in human health and disease." (p 247) Humans are exposed to multiple environmental pollutants at differing intensities and the hazards can be in the air, water, temperature, and food. Environmental pollutants can cause damage at the molecular level in the cell, which can disrupt cellular function, and the list of diseases linked to pollutants is continuously being updated.

One mechanism through which environmental exposures can induce or impact human disease is by triggering increased oxidative stress in cells. This stress can subsequently lead to alterations in DNA molecules.

Genomic DNA damage in the cell nucleus from environmental exposures has been widely studied. But the nucleus is not the only cellular organelle which contains DNA. Mitochondria also have DNA (mDNA). Mitochondria contain multiple copies of their own genome and mitochondria play an important role in the cellular response to environmental stressors. Notably, mitochondria are particularly sensitive to oxidative stress, with mitochondrial functions often disrupted by increased stress.

There has been insufficient attention paid to the impact upon mitochondrial epigenetics in studying environmental exposures and human disease.]

Chauhan V, Howland M, Kutzner B, McNamee JP, Bellier PV, and Wilkins RC, Biological effects of alpha particle radiation exposure on human monocytic cells, International Journal of Hygiene and Environmental Health (2012); 215 (3): 339-344. Abstract. http://www.sciencedirect.com/science/article/pii/S1438463911002100.

[Researchers from the Consumer and Clinical Radiation Protection Bureau of Health Canada observe radon (Rn-222) gas produces decay progeny that emits high energy alpha (α)-particles. Epidemiological studies have shown a link between exposure to Rn-222 and elevated risk of lung cancer.

While the mechanisms underlying such effects are not clearly understood, cytokines are known to play a critical role in inflammation and their dysregulated production often contributes to disease pathogenesis.

This study reports on the use of Bio-plex multiplex technology to investigate modulations of 27 pro-inflammatory cytokines following exposure of human monocytic cells to 1.5 Gy of α-particle radiation. Cells irradiated with α-particles ranging from 0.27 to 2.14 Gy showed statistically significant, dose-dependent increases in phosphorylated H2A histone family X (γ-H2AX) formation. These data suggest that α-particle radiation causes dysregulation in the production of a number of pro-inflammatory cytokines and results in significant DNA damage.]

Dempf SJ, Azimzadeh O, Atkinson MJ, and Tapio S, Long-term effects of ionizing radiation on the brain: cause for concern? Radiation & Environmental Physics (2013); 52 (1): 5-16. Abstract: http://link.springer.com/article/10.1007/s00411-012-0436-7#page-1.

[Article by researchers for the German Research Center for Environmental Health, Institute of Radiation Biology and the Munich Technology University propose a mechanistic model for radiation induced neurodegeneration with mitochondria as a key element. The authors note that oxidative stress and neuroinflammation are fundamental players in neurodegenerative diseases.

Such questions are important because the cumulative doses of ionizing radiation to Western populations are accruing (especially from medical radiation for imaging purposes) and some research groups have found that low-dose exposure affects cognitive skills. Low doses may lead

to delayed long term cognitive and other defects – albeit at a lower frequency – than those observed from high doses. “Children may be a particular target group with pronounced sensitivity for the correlation of radiation and neurodegenerative diseases: they have a long-life expectancy allowing radiation-induced effects with a prolonged latency to develop, and they have still an immature brain up to adolescence.”]

Forrest D and Wess J, A heartfelt response: new thyroid hormone-sensitve neurons in the hypothalamus, Journal of Clinical Investigation (2013); 123 (1): 117–120. http://www.jci.org/articles/view/67448.

[Scientists at the Nuclear Receptor Biology Section of the Laboratory of Endocrinology and Receptor Biology and the Molecular Signaling Section of the Laboratory of Bioorganic Chemistry, National Institute of Diabetes and Digestive and Kidney Diseases, at the U.S. National Institutes of Health observe that thyroid hormone plays a key role in differentiation of the embryonic neuroblast and – later in life – regulating many developmental, metabolic and cardiovascular functions. Thyroid gland disorders are associated with defects in the maturation and function of many tissues and organ systems. The long challenge to science has been to decipher the mechanisms by which thyroid hormone regulates such a wide range of cellular processes in so many tissues. In recent years, evidence has emerged that signaling through thyroid receptors has differential effects on cells depending on the receptor isoform they express.

In this commentary on recent developments, the authors outline evidence that thyroid hormone receptors are also essential to the formation of parvalbuminergic neurons in the anterior hypothalamus. This function links impairment of thyroid hormone signaling during development to cellular deficits in the hypothalamus. This newly discovered cell group may play a role in regulating cardiovascular function. Impaired thyroid hormone signaling can cause specific effects in the hypothalamus and developmental hypothyroidism may be a cause of cardiovascular disorders later in life.

In sum, recent findings have expanded the perspective on the role of the hypothalamus in the central control of homeostatic functions. While considered an “old” hormone, contemporary techniques are providing unprecedented illumination of how thyroid hormone mediates developmental and physiological functions throughout the body.]

Little MP, Azizova TV, Bazyka D, Bouffler SD, Cardis E, Chekin S, Cumak VV, Cucinotta FA, de Vathaire F, Hall P, Harrison JD, Hildebrandt G, Ivanov V, Kashcheev VV, Klymenko SV, Kreuzer M, Laurent O, Ozasa K, Schneider T, Tapio S, Taylor AM, Tzoulaki I, Vandoolaeghe WL, Wakeford R, Zablotska L, Zhang W, and Lipshultz SE, Systematic Review and Meta-analysis of Circulatory Disease from Exposure to Low-Level Ionizing Radiation and Estimates of Potential Population Mortality Risks, Environmental Health Perspectives (2012); 120 (11): 1503-1511. {*Full study available as pdf on web*}.

[Meta-analysis by scientists from the Radiation Epidemiology Branch of the National Cancer Institute; the Department of Epidemiology and Biostatistics, University of California, San

Francisco; the Department of Pediatrics, Leonard M. Miller School of Medicine, University of Miami; Radiation Health Office, NASA Johnson Space Center; as well as numerous medical and regulatory institutions in France, Germany, Japan, Russia, Spain, Sweden, Ukraine, and the UK.

The current science supports an association between low and moderate doses of ionizing radiation and circulatory disease mortality.

The authors conclude: "The estimates of population-based excess mortality risks for circulatory disease are similar to those for radiation-induced cancer, as also noted previously in relation to noncancer disease… If associations between low-level exposure to radiation and circulatory diseases reflect an underlying causal relationship that is linear at low doses, then the overall excess risk of mortality after exposure to low doses or low dose rates of radiation may be about twice that currently assumed based on estimated risks of mortality due to radiation-induced cancers alone." (p 1510)]

Masuda Y, Molecular nature of radiation injury and DNA repair disorders associated with radiosensitivity, International Journal of Hematology (2012); 95 (3): 239-245. Abstract. http://link.springer.com/article/10.1007/s12185-012-1008-y#page-2.

[Authors are from the Research Institute of Environmental Medicine at Nagoya University and the Research Institute for Radiation Biology and Medicine at Hiroshima University (Japan). They observe that ionizing radiation, like many other chemicals and reactive oxygen species, can cause insults to DNA integrity. However ionizing radiation is distinct from other agents in that it produces clustered DNA damage, especially double-strand DNA breaks (DSBs). Investigation into radiosensitive diseases has revealed molecular mechanisms underlying the impact of cellular responses to radiation and repair of DSBs. Importantly, radiosensitive diseases are also associated with immune system dysfunction and increased risks of leukemia and lymphoma.]

McAllister KA, Lorimore SA, Wright EG, and Coates PJ, *In Vivo* Interactions between Ionizing Radiation, Inflammation and Chemical Carcinogens Identified by Increased DNA Damage Responses. Radiation Research (2012); 177 (5): 584-593. Abstract: http://www.rrjournal.org/doi/abs/10.1667/RR2690.1.

[Researchers are from the Centre for Oncology and Molecular Medicine, Division of Medical Sciences, University of Dundee, Ninewells Hospital and Medical School.

Ionizing radiation and a variety of chemical agents known to increase the risk of developing malignancies and many tumors have been linked to inflammatory processes. Most studies consider the potentially harmful effects of ionizing radiation or other agents in isolation, primarily because of the difficulty and expense of investigating the effects of mixed exposures with different doses and different schedules, as well as the length of time needed to identify disease as a measure of outcome. In this murine study, the research group used short-term DNA damage responses to identify interactive effects of mixed exposures.

The data showed that exposure to ionizing radiation on 2 separate occasions 10 days apart led to an increase in the percentage of cells with a sub-G_0 DNA content compared to cells exposed only once, and this is a greater than additive effect. Short-term measurements of p53 stabilization, induction of p21/Cdkn1a and of apoptosis also identify these interactive effects. The investigation revealed similar interactive effects of radiation with the a mutagenic chemical (methyl-nitrosourea) and with a nonspecific pro-inflammatory agent (lipopolysaccharide). The magnitude of the interactive effects was found to be greater in cells taken from mice first exposed as juveniles compared to adults. Overall, the findings indicate that short-term measurements of DNA damage and response to damage are useful for the identification of interactions between ionizing radiation and other agents.]

Mothersill C, Moriarty MJ, and Seymour CB, Bystander and other delayed effects and multi-organ involvement and failure following high dose exposure to ionising radiation, British Journal of Radiology (2005); S27 (1): 132-138. Abstract. http://www.birpublications.org/doi/abs/10.1259/bjr/69849747?journalCode=bjr.

[Researchers from the Medical Physics and Applied Radiation Sciences Unit, McMaster University (Canada) and Saint Luke's Institute of Cancer Research (Ireland) observe there exists "no doubt that ionising radiation damages DNA and that certain organs in the body are more vulnerable than others to the effects of radiation."

The reasons for the different sensitivities vary and there are now known to be many late expressed effects of exposure that cannot be explained simply on the basis of direct DNA damage. "Examples include transmissible genomic instability, bystander effects and adaptive responses, which seem to be interrelated phenomena occurring even at low doses and affecting very high numbers of cells in the exposed organ or organism."

Both radiation and trauma exposure evidence indicates people exhibit systemic effects that involve general system symptoms that cannot easily be attributed to effects on particular targeted tissues or organs. "There is also evidence for emergent properties of systems that involve communication within and between organs, and concerted responses that are not predictable using reductionist approaches in radiotoxicology."]

Pagano G, Talamanca AA, Castello G, Cordero MD, d'Ischia M, Gadaleta MN, Pallardo FV, Petrovic S, Tiano L and Zatterale, Oxidative Medicine and Cellular Longevity (2014); 2014. Doi: 10.1155/2014/541230. http://www.hindawi.com/journals/omcl/2014/541230/abs/.

[Broad-ranging review of research elucidating oxidative stress mechanisms and mitochondrial dysfunctions across a broad range of immune, neurological, cardiovascular, endocrine, malignancies, and genetic pathologies. (Among the conditions of focus are: Autism Spectrum Disorders; Down Syndrome; aging and aging-related degenerative disorders; cardiovascular diseases; epilepsy; Myalgic Encephalomyelitis; Multiple Sclerosis; lupus erythematosus; breast cancer; lung cancer; myeloid leukemia; melanoma; and cataracts.)

Beyond the focus on individual diseases and disease groups sharing molecular or clinical affinities, the review attempts to elucidate the ways oxidative stress (and resulting reactive oxygen species) and mitochondrial dysfunction (including mDNA damage) act as mechanisms either directly or in concert with other inborn or exogenous causes of disease.]

Majima HJ and Toyokuni S, Mitochondria and free radical studies on health, disease and pollution, Free Radical Research Editorial (2012); 46 (8): 925-926. http://informahealthcare.com/doi/pdf/10.3109/10715762.2012.700784.

[Recent developments in free radical research have shown that numerous human pathological conditions, including aging, cancer, cardiovascular disease, diabetes, neuronal diseases, and prematurity in babies result from or are associated with oxidative stress.]

Szumiel, Irena, Ionising radiation-induced oxidative stress, epigenetic changes and genomic instability: the pivotal role of mitochondria, International Journal of Radiation Biology (2014); Abstract. http://informahealthcare.com/doi/abs/10.3109/09553002.2014.934929.

[Review by Irena Szumiel, of the Centre for Radiobiology and Biological Dosimetry, Institute of Nuclear Chemistry and Technology (Warsaw) of the data on the role played by endogenously generated reactive oxygen species (ROS) in producing non-targeted ionizing radiation effects affecting cell populations, both early after exposure and after multiple cell generations.

Conclusion*:* ROS generation by the electron transport chain of the mitochondria and by the cytoplasmic NADPH oxidases result in both short-term and chronic oxidative stress responses.

Whether induction of oxidative stress and its consequences occur in a cell, depends largely on interaction between the nucleus and the cellular population of hundreds or thousands of genetically heterogeneous mitochondria. High intra-mitochondrial ROS levels can damage mitochondrial (mt) DNA, and mtDNA mutations can affect nuclear DNA epigenetic control mechanisms by decreasing the activity of methyltransferases; thus causing global DNA hypomethylation. Changes can be transmitted to the progeny of the irradiated cells. Chronic oxidative stress can cause cancer and other late post-radiation effects.]

Exposure Zone Populations Mandate Study

European Committee on Radiation Risk (ECRR), 2010 Recommendations of the ECRR: The Health Effects of Exposure to Low Doses of Ionizing Radiation, European Committee on Radiation Risk, Regulators' Edition (2010). http://www.euradcom.org/2011/ecrr2010.pdf.

Fairlie I, A hypothesis to explain childhood cancers near nuclear power plants, Journal of Environmental Radioactivity (2014); 133: 10-17. Abstract. http://www.sciencedirect.com/science/article/pii/S0265931X13001811.

[Dr. Ian Fairlie is former head of the Secretariat of the UK Government's CERRIE Committee on internal radiation risks.

Over 60 epidemiological studies world-wide have examined cancer incidences in children near nuclear power plants. Most show leukemia increases. Especially strong is the evidence from the 2008 KiKK study commissioned by the German Government which found relative risks of 1.6 in total cancers and 2.2 in leukemia's among infants living within 5 km of all German nuclear power plants. One hypothesis proposed is that the increased childhood leukemia cancers result from radiation exposures to pregnant women near nuclear power plants.

However any theory has to account for the >10,000 fold discrepancy between official dose estimates from nuclear power plant emissions and observed increased risks.

An explanation may be that doses from spikes in nuclear power plant radionuclide emissions (such as spikes during refueling) are significantly larger than those estimated by official models which are diluted through the use of annual averages. In addition, risks to embryos/fetuses are greater than those to adults and hematopoietic tissues appear more radiosensitive in embryos/fetuses than in newborn babies. Thus an explanation for the cancers may be a product of possible increased doses and possible increased risks per dose.]

Fairlie, Ian, Childhood Leukemias Near Nuclear Power Stations: new article, Dr. Ian Fairlie blog, Jul 25, 2014. http://www.ianfairlie.org/news/childhood-leukemias-near-nuclear-power-stations-new-article/.

[Dr. Ian Fairlie is former head of the Secretariat of the UK Government's CERRIE Committee on internal radiation risks. In this post, Dr. Fairlie summarizes the findings of his study on increased rates of childhood leukemia near nuclear power plants (NPPs) published in the March 2014 edition of the Journal of Environmental Radioactivity.

Some background is necessary to grasp the new report's significance. In 1990 publication of the Gardner report indicated a very large increase (7 fold) in child leukemia near the Sellafield nuclear facility in Cumbria (UK). Worldwide, over 60 epidemiological studies have examined cancer incidences in children near nuclear power plants. Most (>70%) indicate leukemia increases. Fairlie writes: "I can think of no other area of toxicology (eg asbestos, lead, smoking) with so many studies, and with such clear associations as those between NPPs and child leukemias. Yet many nuclear Governments and the nuclear industry refute these findings and continue to resist their implications. It's similar to the situations with cigarette smoking in the 1960s and with man-made global warming nowadays."

The 2008 KiKK study commissioned by the German Government found a 60% increase in total cancers and 120% increase in leukemias among children under 5 yrs old living within 5 km *{~3*

mi} of all German nuclear power plants. As a result, governments in France, Switzerland and the UK hurriedly set up studies near their own plants. All found leukemia increases. However, because the numbers involved were small, they were claimed to lack "statistical significance". This is misleading. "In such situations, what you need to do is combine datasets in a meta-study to get larger numbers and thus reach higher levels of statistical significance. The four governments refrained from doing this because they knew what the answer would be, viz, statistically significant increases near almost all NPPs in the 4 countries." Farlie and his colleague Korblein helped them out by doing it for them (Korblein and Fairlie, 2012), and "sure enough" there were statistically significant increases.

The combined data {*set forth in a table*} reveals a highly statistically significant 37% increase in childhood leukemias within 5 km of almost all nuclear power plants in the UK, Germany, France and Switzerland. "So the matter is now beyond question, ie there's a very clear association between increased child leukemias and proximity" to nuclear power plants. The only remaining question is the cause.

Any theory involving radiation has a major difficulty to overcome, and that is how to account for the large (~10,000 fold in KiKK) discrepancy between official dose estimates from nuclear power plant emissions and the observed increased risk.

Fairlie postulates that the reasons can be gleaned from KiKK's principal finding, which is that the increased incidences of infant and child leukemias were closely associated with proximity to the effluent chimneys as well as KiKK's observation that the increased solid cancers were mostly "embryonal", ie babies were born either with solid cancers or with pre-cancerous tissues which, after birth, developed into full-blown tumors. This happens with leukemia as well. Cancer increases may thus be due to radiation exposures from emissions to the air. Large annual spikes in emissions may result in increased dose rates to nearby populations. Cancers may arise in utero in pregnant women. Both the doses and their risks to embryos and to fetuses may be greater than current estimates. Finally, pre-natal blood-forming cells in bone marrow may be unusually radiosensitive. Together these factors – discussed in considerable detail in the full 2014 Environmental Radioactivity article – offer a possible explanation for the discrepancy between estimated radiation doses from nuclear power plant releases and the risks observed by the KIKK study.]

Körblein A and Fairlie I, French Geocap study confirms increased leukemia risks in young children near nuclear power plants. Letter to Editor. International Journal of Cancer, (2012); 131: 2970–2971.
http://www.researchgate.net/publication/223973890_French_geocap_study_confirms_increased_leukemia_risks_in_young_children_near_nuclear_power_plants.

Nussbaum RH, Childhood Leukemia and Cancers Near German Nuclear Reactors: Significance, Context, and Ramifications of Recent Studies, International Journal of Occupational and Environmental Health (2009); 15 (3): 318-323.

http://www.ingentaconnect.com/content/maney/oeh/2009/00000015/00000003/art00012 See also **http://www.nirs.org/radiation/radhealth/kikkcommentary0709ijoeh.pdf**

[Review of studies on childhood leukemia and cancers near nuclear plants by Rudi H. Nussbaum, PhD, Physics and Environmental Studies emeritus faculty, Portland State University.]

Sermage-Faure C, Laurier D, Goujon-Bellec S, Chartier M, Guyot-Goubin A, Rudant J, Hémon D, and Clavel J, Childhood leukemia around French nuclear power plants—The geocap study, 2002–2007, International Journal of Cancer (2012); 131 (5): E769–E780. http://onlinelibrary.wiley.com/doi/10.1002/ijc.27425/full

[French research teams from the Institut National de la Santé et de la Recherche Médicale (INSERM), the Institut de Radioprotection et de Sûreté Nucléaire (IRSN), and the National Register of hematological diseases of children in Villejuif, France, demonstrated that childhood leukemia rates are statistically significantly elevated in children living near nuclear power reactors in France. The study established a clear correlation between the frequency of acute childhood leukemia and proximity to nuclear power stations. The researchers could not identify any other environmental factor besides nuclear plant radiation emissions that could produce the excess cancers. Looking at the period from 2002-2007, the scientists found a doubling of childhood leukemia incidence, with an increase up to 2.2 among children younger than five.]

Tsuda T, Tokinobu A, Yamamoto E, and Suzuki E, Thyroid Cancer Detection by Ultrasound Among Residents Ages 18 Years and Younger in Fukushima, Japan: 2011 to 2014, Epidemiology (2015). http://journals.lww.com/epidem/Abstract/publishahead/Thyroid_Cancer_Detection_by_Ultrasound_Among.99115.aspx.

[Authors are from Okayama University's Graduate Schools of Environmental and Life Science and Medicine, Dentistry and Pharmaceutical Sciences. They report here on excess incidence of thyroid cancers in children ≤18 in Fukushima Prefecture.

As the wind shifted direction over time following the inception of the Fukushima nuclear accident on March 11, 2011, I-131, Cs-137 and Cs-134 were released to land areas both northwest and south of the plant.

Exposure information on I-131 has been uncertain because of the destruction of monitoring sites as a result of the accident. There is no precise measurement of external and internal radiation exposure – as delivered by inhalation, ground shine, and ingestion – in Fukushima. However, the World Health Organization estimated the thyroid equivalent doses in 2011 to be 100-200 millisieverts (mSv) in the more affected areas and 10-100 mSv in the rest of Fukushima Prefecture (The radiologic equivalence to I-131, is believed to have been approximately one-sixth that of the 5,200 petabecquerel calculated to have been released by the Chernobyl accident.)

Prior research which measured radioiodine concentrations in breast milk of women residing within 250 km (155.34) of the Fukushima plant found that 7 of the 23 women examined in April 2011 secreted a detectable level of I-131 in their breast milk. The Japan National Institute of Radiological Sciences, using an acute ingestion model, estimated equivalent doses ranged from 119 – 432 mSv among mothers and from 330 – 1,190 mSv in their infants for those living 45 to 220 km (27.9 – 136.7 miles) south or southwest, including Iwaki City in Fukushima Prefecture, Ibaragi Prefecture, and Chiba Prefecture.

Evacuations from heavily contaminated areas within 20 km (12.4 mi), and from additional contaminated areas, mainly northwest of the plant occurred between March 12 and mid-June 2011. Many residents were evacuated to areas within Fukushima Prefecture that resulted in their continued exposure to radioactivity.

"The 'nearest area' to the Fukushima plant, mostly within 50 km {*31 miles*} … was the most contaminated area". The highest childhood thyroid cancer incidence rate ratio (IRR) in the external comparisons was observed in the central middle district of the prefecture, 50 to 60 km {*31 – 37.3 miles*} west from the Fukushima power plant," and area from which the residents were not evacuated.

"Although precise measurements of both external and internal radiation exposure in Fukushima were not obtained, in external comparison, we observed an approximately 30-fold increase in the number of thyroid cancer cases among children and adolescents using the area/district of residence to provide a surrogate for exposure information. … {a finding} consistent with the flow of ^{131}I being primarily in a southern direction from the Fukushima release."

The findings allow the inference that the incidence of thyroid cancer in Fukushima rose more rapidly than expected and by the World Health Organization. The magnitude of the IRRs was too large to be fully explained by the increase in the number of thyroid screenings. Furthermore data on cancer removal surgeries performed at Fukushima Medical University Hospital indicates 74% of their cases had positive lymph node metastases – a finding which suggests that cancers detected by screening were not at a particularly early stage.]

Wing S, Richardson DB, and Hoffmann W, Cancer Risks near Nuclear Facilities: The Importance of Research Design and Explicit Study Hypotheses, Environmental Health Perspectives (2011); 119 (4): 417–421.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3080920/.

[Authors of the study are scientists from the Department of Epidemiology, University of North Carolina – Chappel Hill (US) and the Institute for Community Medicine, Section Epidemiology of Health Care and Community Health, Ernst-Moritz-Arndt University of Greifswald (Germany).]

Wing S, Richardson DB, Armstrong D, and Crawford-Brown D, A reevaluation of cancer incidence near the Three Mile Island nuclear plant: the collision of evidence and assumptions, Environmental Health Perspectives (1997); 105 (1): 52-57. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1469835/.

[Team from the Department of Epidemiology, School of Public Health, at the University of North Carolina, Chapel Hill led by Dr. Steve Wing identifies the flaws of logic and methodology in earlier studies on cancer incidence following the 1979 Three Mile Island nuclear accident in Pennsylvania, U.S. Analysis of the data supports "the hypothesis that radiation doses are related to increased cancer incidence" in the 10 mile area around the plant.]

Yablokov AV, Nesternenko VB, and Nesternenko AV, Chernobyl: Consequences of the Catastrophe for People and the Environment, New York Academy of Sciences (2009).

[This study, published by the New York Academy of Sciences, study is a monograph compiled from tens of thousands of Slavic and other non-English language studies. It constitutes the largest and most complete collection of data on the public health catastrophe resulting from the 1986 Chernobyl nuclear power plant disaster. The list of literature incorporated into the volume includes some 1,000 titles and reflects over 5,000 papers published primarily in the Slavic languages. Janette D. Sherman-Nevinger, MD, of the Environmental Institute at Western Michigan University, served as consulting editor of the English translation.

The lead author of the study, Alexey Vladimirovich Yablokov of the Russian Academy of Sciences in Moscow, served as a consultant to both Gorbachev and Yeltzin. Prof. Alexey Vassil'evich Nesterenko, was a member of the Russian Academy of Sciences in Moscow, Russia. The third author, Vassily B. Nesternenko, served at the Institute of Radiation Safety (BELRAD) in Belarus, Minsk. Trained as a nuclear design engineer, and prior to the Chernobyl disaster, he was Director of the Belarussian Nuclear Center.

In the forward to the volume, Prof. Dimitro M. Grodzinsky, Chairman of the Department of General Biology, Ukrainian National Academy of Sciences, and Chairman of the Ukrainian National Commission on Radiation Protection, writes: "The biological efficiency of cytogenic effects varies depending on whether the radiation is external or internal: internal radiation causes greater damage…With the passage of time, oncological diseases with loner latency periods, in particular breast and lung cancers', become more frequent. From year to year there has been an increase in nonmalignant diseases, which has raised the incidence of overall morbidity in children in areas affected by the catastrophe, and the percent of *practically healthy* children has continued to decrease. For example, in Kiev, Ukraine, where before the meltdown, up to 90% of children were considered healthy, the figure is now 20%. In some Ukrainian Poles' territories, there are no healthy children and morbidity has essentially increased for all aged groups." (p viii)

Dr. Grodzinsky sums up the findings thusly: "The present volume probably provides the largest and most complete collection of data concerning the negative consequences of Chernobyl on the health of people and on the environment. Information…shows that these consequences do not decrease, but, in fact, are increasing and will continue to do so into the future….Over the next

several future generations the health of people and of nature will continue to be adversely impacted." (p ix)

The Chernobyl distribution of gaseous aerosol radionuclides from Chernobyl was extremely nonuniform. "From April 26 through May 5, 1986, the winds around Cehernobyl varied by 360°, so the radioactive emissions from the mix of radionuclides varied from day to day and covered an enormous territory… The daily emissions formed several radioactive clouds, and each such cloud had its own radionuclide composition and geography." (p 6) An area of 2,640 km^2 (1,640.4 square miles) of Belarus cannot be used for agriculture. The 1,300 km^2 (807.8 square miles) Polessk state radioactive reserve near the plant is forever excluded from any economic activity. (p 10). Nearly 350,400 people were forced to leave their homes because of Chernobyl contamination.

Yablokov and colleagues begin the study with a review of the estimates of contamination through both time and geographic space and specify the primary radionuclides involved (e.g., Table 1.5, p 19).

Three most important determinants of affecting the environment and public health to be: (1) spotty/uneven deposits of contamination, (2) "hot" particle impacts, and (3) bioaccumulation of radionuclides. As to the spotty deposits, aerogamma studies, upon which most maps of contamination are based, give only average values of radioactivity for fairly large areas, thereby missing small, local, highly radioactive hot spots. An example given was the findings of the public health services of the French department Vosges on a "glowing" hog hit by a local hunter. Monitors then discovered that the entire mountain where the dead hog had run was radioactive at a level from 12,000 to 24,000 $Bq/m^{2.}$ (p 19)

The epidemic of cancers is exhaustively reviewed.

The Yablokov study also – and uniquely – details the increase in nonmalignant diseases in radiation contaminated regions. Primary morbidities include increased cardiovascular disease; central nervous system disorders; immune deficiencies (including significant changes in cellular immunity, and findings of decreased T lymphocyte, T suppressor, and T helper cells); high incidences of eye problems; and accelerated aging.

"The appreciable increase in newborns with both major and minor developmental anomalies is one of the undeniable consequences of the Chernobyl catastrophe. Everywhere in areas contaminated by Chernobyl radioactivity, increased numbers of children have been born with hereditary anomalies and congenital developmental malformations, including previously rare multiple structural deformities of the limbs, head, and body. … The Occurrence of congenital malformations continues to increase in several of the contaminated territories and correlates with the levels of irradiation." (p 133)

The study stresses recognition of what is known as "Chernobyl AIDS," the overall increased incidence and seriousness of a complex array of multi-systemic morbidities, many of which have the characteristics of accelerated aging.

Among specific health disorders associated with Chernobyl radiation, the authors find an increased morbidity and prevalence of groups of diseases and dysfunctions relating to the: Autonomic nervous system (with clinical signs that present against a background of stress); Central nervous system; Circulatory system (owing primarily to radioactive destruction of the endothelium, the internal lining of the blood vessels); Digestive tract; Endocrine system; Eyes (cataracts, vitreous destruction, refraction anomalies, and conjunctive disorders); Immune system; Musculoskeletal system; Respiratory system; and Urogenital tract and reproductive disorders. (pp 111, 218.)

In contaminated regions, among the general population there is an unprecedented wide incidence of chronic fatigue, impaired memory, diffuse muscular pains, pains in large joints, frequent mood changes, cervical lymph node sensitivity, and decreased body mass. It is postulated that these symptoms are a result of impaired immune system function in combination with disorders of the temporal-limbic parts of the central nervous system." (p 218)]