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Science for the Vulnerable
Setting Radiation and Multiple Exposure Environmental Health Standards to Protect Those Most at Risk

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Preface

The last half century has seen great progress in environmental health protection. The other side of that coin is that most of the standards themselves became necessary as a result of a vast array of hazardous materials and radionuclides that have been introduced into the human environment since the end of World War II. As evidence of the many ways they can harm human health has mounted, maximum exposure limits have been reduced. Children have moved to the center of many of the concerns, as for instance, in the case of exposure to lead. Protections have been put in place to limit radiation dose to the embryo/fetus in the work locations where there are known risks of radiation exposure. That protection is brought to bear only when a woman chooses to declare her pregnancy, thereby protecting her rights in the workplace.

But as knowledge has grown, the gaps in the regulatory framework have become more evident and their importance more transparent. Many radiation protection regulations, notably cleanup standards for contaminated sites, are focused on dose received by "Reference Man" -- defined as a young adult Caucasian male. Children are still viewed as little adults in such contexts since the framework of radiation protection does not cover the variety of ill-health effects that children may experience disproportionately from radiation, but is rather focused on fatal cancer risk. The problems of early failed pregnancies, early miscarriages, and malformations potentially caused by radiation exposure are still not within the regulatory framework. Estimation of health harm as expressed in regulations is generally confined to assessment of one chemical at a time or to radiation. Combined radiation and chemical exposures are rarely considered in research and are practically absent from regulation.

Systematic protection of members of society requires that the horizons of regulation be expanded to cover these yawning gaps. Much scientific work will be needed to fill some of them, while interim strengthening is easily possible and justifiable in other areas (such as bringing U.S. workplace maximum radiation exposure limits in line with best European practice). This report is the first of three in which the Institute for Energy and Environmental Research will analyze the need for

- widening the horizon of health effects to be considered in radiation protection
- systematically integrating key groups--women (who face higher cancer risks per unit of radiation exposure than men), pregnant women, men in their capacity as potential fathers, infants and children, and the embryo/fetus--into the framework of radiation protection
- creating a research framework and scientific paradigm that will allow for assessment of combined radiation and chemical exposures.

In this report, the first of the three, we focus on radiation and, more briefly, on some considerations relevant to combined exposure. The next report will concern itself with the health of the immune system and the carcinogenetic effects of radiation. The last report in this series will propose a theoretical approach, or at least a detailed concept, for determining the individual and combined health effects of toxic chemicals and radiation – at least that is the ambitious goal at present.

This report is written within a rather limited framework that is necessary to understand. We consider here actual or potential harm (health risk) from environmental exposures of anthropogenic origin. Considering health risk does not in any way endorse the imposition of it. This is discussed in some detail in Chapter 1. Radionuclides and hazardous chemicals have been introduced into the environment. Thousands of sites around the country and the world are polluted, many of them severely. Perfect remediation is generally impossible. For example, even if it were possible to remove all residual radioactivity from a nuclear weapons site such as the now decommissioned Fernald plant, near Cincinnati, Ohio, it would have to be taken somewhere else. The community decided to keep some of the waste resulting from clean up in a

waste cell and the rest was sent elsewhere. Very often, health risks can be reduced, but they cannot be eliminated once the problem has been created. Since future generations will bear the brunt of many of these ill-effects, it is critical to know how to assess them.

Risk assessment is a science and art that is often misused – for instance, in justifying imposition of risk without informed consent by appeal to natural risks that already exist. As discussed in this report, we do not consider this a legitimate way to use the science. But risk assessment, used properly, is needed for sensible and effective decision-making in the world in which we live.

Even apart from the question of pre-existing pollution, we need to be able to compare potential environmental health impacts, that is, health risks from proposed activities. Such comparisons are necessary even for implementing the precautionary principle. For instance, if solar energy is needed to greatly reduce carbon dioxide emissions to reduce the impact of global warming, how do we decide between different approaches to solar energy? No approach is without some potential for harm. Silicon cells take a great deal of energy to make, which creates some pollution. Thin film solar cells may require mining of exotic metals. If waiting for the perfect answer is not an option (and it is not in the case of global warming), we must have some tools to assess the impact. Estimating health risk is one tool. Lifecycle assessment is another.

This report is written within the limited framework of assessing a variety of health risks and what needs to be done to strengthen environmental health protection standards to protect those who are most vulnerable to the effects of environmental contamination. This report does not address catastrophic accidents, like the Chernobyl nuclear reactor accident in Ukraine in 1986 or the enormous leak of poisonous gas at the Union Carbide factory in Bhopal in 1984 that killed thousands and sickened many more. For events that may have a relatively small probability of occurrence but catastrophic consequences if they do, risk assessment alone is a poor tool. Low probability of catastrophe does not equate with large probability of a common cold, though a pure risk-based answer (if one could be developed) would equate them. The tool kit must include consideration of such possibilities before deciding whether a technology is socially acceptable.

Another area that we do not address in the report is individual differences in radiation sensitivity. As the genetic constitution of an individual will influence his or her risk of cancer due to exposure to radiation and toxic chemicals, and given that techniques of genetic profiling are increasing rapidly in power and scope, it seems likely that it will in the future be possible to identify specific radiosensitive individuals. The International Commission on Radiological Protection (ICRP) has commented that there are critically important ethical, social, and economic considerations that need to be discussed and resolved prior to the employment of such genetic tests in almost all contexts.[1] However, if such tests are available, the consideration will arise as to whether standards will need to be set to be protective of individuals who are identified as particularly sensitive by such tests, given that such individuals may not be outwardly distinguishable from other members of the population, i.e. not suffering from a particular clinical syndrome. As a precautionary measure, we have incorporated genetic non-discrimination in employment as part of our recommendations.

Arjun Makhijani
Brice Smith
Mike Thorne
October 2006

[1] ICRP 79 paragraph 469

Chapter 1: "Reference Man" and Real People: Protecting the Most Vulnerable

"Reference man is defined as being between 20-30 years of age, weighing 70 kg [154 pounds], is 170 cm [5 feet 7 inches] in height, and lives in a climate with an average temperature of from 10^{o} to 20^{o}C. He is a Caucasian and is a Western European or North American in habitat and custom."
International Commission on Radiological Protection, 1975[2]

"We should try to create a society in which it is never a tragedy to be pregnant."
Annie Makhijani, about 1986

We live in a world bristling with nuclear weapons where signs of severe climate disruption multiply with alarming regularity. The risk of large numbers of people dying due to human-induced catastrophe has never been greater than in the nuclear/fossil-fuel age. Such vast risks tend to overwhelm sense and sensibility, because they can induce feelings of helplessness to change what seems much too large for personal effort. Historically, people have best come to grips with such challenges where they intersect with their own lives, as with air pollution or contaminated food, or as occurred with the central mobilizing force for the 1963 atmospheric test ban treaty, when scientists showed that strontium-90 from the fallout of nuclear weapons' testing was accumulating in babies' teeth and mothers' milk. The nuclear bomb had invaded the home at its most vulnerable and most intimate.

President Kennedy put fallout right next to the threat of reducing the risk of nuclear war when he spoke to the people of the United States at the conclusion of negotiations of the atmospheric test ban treaty in 1963:

> …the number of children and grandchildren with cancer in their bones, with leukemia in their blood, or with poison in their lungs [due to atmospheric testing fallout] might seem statistically small to some, in comparison with natural health hazards. But this is not a natural health hazard -- and it is not a statistical issue. The loss of even one human life, or the malformation of even one baby -- who may be born long after we are gone -- should be of concern to us all. Our children and grandchildren are not merely statistics toward which we can be indifferent.[3]

It was the consideration of the fate of babies, of individual baby teeth which became silent witnesses to the age of the bomb since they had strontium-90 from atmospheric testing fallout in them, that mobilized the people of the United States and the world to demand an end to nuclear testing. The process led to the Partial Test Ban Treaty signed by John F. Kennedy and Nikita Khrushchev in Moscow on August 5, 1963.

We also live in a world in which thousands of chemicals were introduced into commerce without any serious thought to their effects on human health and the environment. As the atmospheric test ban treaty was being negotiated amid reports of strontium-90 in babies' teeth, a little known scientist and naturalist was finishing up a book that was to put the chemical industry and the U.S. Agriculture Department on the defensive. Rachel Carson's *Silent Spring* was the first major event that created broad awareness of the underbelly of "better living …through chemistry."[4]

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[2] ICRP 23 p. 4
[3] Kennedy 1963
[4] Famous advertising slogan of DuPont. (Wikipedia 2006-9-10)

Even before *Silent Spring* was published by Houghton Mifflin in 1962, there was strong opposition to it. As *Time* Magazine recounted in 1999:

> Carson was violently assailed by threats of lawsuits and derision, including suggestions that this meticulous scientist was a "hysterical woman" unqualified to write such a book. A huge counterattack was organized and led by Monsanto, Velsicol, American Cyanamid - indeed, the whole chemical industry - duly supported by the Agriculture Department as well as the more cautious in the media.[5]

Scientist Robert White-Stevens wrote, "If man were to follow the teachings of Miss Carson, we would return to the Dark Ages, and the insects and diseases and vermin would once again inherit the earth."[6]

The thinning of birds' eggshells was however a harsh fact of some chemical pollution, much like the undeniable strontium-90 in babies teeth from nuclear bomb testing fallout. Despite the powerful opposition of the chemical industry and parts of government, President Kennedy's Science Advisory Committee endorsed Rachel Carson's views in a report issued on May 15, 1963 – less than a month before he announced that a meeting on the test ban would be held in Moscow.[7]
Despite their proximity in time and political space, these two aspects of the struggle for a clean environment have stayed on parallel tracks. There has been one struggle for a safer economy where chemicals, including those used in food production and in everyday life, would not turn into deadly loci of cancer and other diseases in the human body. Each chemical is tested separately from the others. Cancer is the main risk that is considered in human health assessments, but risks such as neurotoxicity and teratogenicity are also considered. Data are mainly from animal experimentation. Biologists, physicians, and chemists have been the researchers in the lead.

Policymakers tended to see nuclear-weapons-related issues through the window of "arms control" – that is, what bomb-related treaties of the time were supposed to be about. Even today, the science of health protection from radioactivity arose mainly in the context of the Manhattan Project. It was dubbed "health physics" – it was created by physicists, many of whom were familiar with radiation in the form of cosmic rays. They also understood the properties of the particles emanating from the interior structures of the atom, newly discovered in the decades just prior to World War II. They measured the energy dumped into the body by the particles and assessed how the hazard of radioactivity (that, unlike many chemicals, could not be seen or smelled or touched) would affect the human body. Chemical toxicology has largely been addressed by chemists and biologists.

In radiation, the disease of greatest concern, as time went on, became identified as cancer. That is also one of the principal concerns with chemicals, but there the effects studied have been much more varied. For instance, the neurotoxic effects of lead and mercury have been critical issues. The methods of regulation between radiation and chemicals also were divergent in some areas; those differences arose in part from the different scientific approaches that each has each its strengths and weaknesses. The approach to chemicals recognizes the diversity of toxic agents and their effects, reflecting the complexity and diversity of chemical and biological systems; it tends to operate on a case-by-case basis. This has its positive aspects in that it means that new situations are approached with an open mind. However, it tends to lead to an *ad hoc* approach to regulation, with inconsistencies between the regulatory regimes for different substances in different contexts and a lack of unifying principles to address multiple exposures.

[5] Matthiessen 1999
[6] Truemper 1999
[7] Kennedy 1963b and State Dept. 1963

In contrast to chemicals, radiation protection has very much emphasized the development of a unified approach, where the impacts of a wide variety of types of external and internal exposure are squeezed into a single conceptual framework through the use of absorbed dose and associated derived quantities (equivalent dose, effective dose, committed effective dose). This is tidy and allows different types of radiation exposure to be aggregated in a way that is not possible with chemicals. However, it also makes it difficult to escape from the associated conceptual mindset, and there has been a tendency for radiation protection and radiobiology to become fixed in a particular paradigm from which it is difficult to break free. For instance, given the early observations of microcephaly at Hiroshima and Nagasaki, it is remarkable that effects on mental abilities were not recognized until the late 1970s. This is not to criticize the researchers of the time who worked with the paradigm of the new science they were given as best they could. But what is now the conventional paradigm was so much oriented to genetic effects and cancer as the predominant late effects that it was difficult even to think about investigating other potential consequences.

In brief, much has been learned, and regulations have been improved over the years. For instance, as the cancer risk of radiation has been found to be greater per unit of exposure than originally thought, radiation protection regulations have been tightened. Standards for maximum public exposure today are fifty times lower than they were in the half a century ago (down from 5 rem per year to 0.1 rem per year). Some protections have been put in place for limiting the exposure to the embryo/fetus in the workplace. Concepts such as “maximally exposed individual” with reasonable assumptions have been introduced. But the science underlying both still has large gaps. Neither the regulations nor the research is still fully oriented to protecting the most vulnerable. That is changing so far as research is concerned. But regulations still have a considerable way to go to catch up. And some fundamental issues relating to the paradigms in which research on chemicals and radiation are studied remain to be addressed.

A few features of the regulatory landscape stand out:

1. Knowledge of the different sex and age-related radiation risks has been growing rapidly since the 1986 Chernobyl disaster. Much of this research has been accepted by regulatory authorities and international advisory bodies.
2. Radiation protection regulations as well as cleanup guidelines for contaminated sites are, for the most part, not based on protecting the most vulnerable members of society, but on a concept known as Reference Man, which is defined as a “Caucasian” male in his twenties.
3. Chemicals are, for the most part, regulated individually, and very little is known regarding the combined risks of exposures to different chemicals. However, certain classes of chemicals have been banned because of their common characteristics. PCBs are one example.
4. Exposure to combinations of chemicals and radiation remains largely unaddressed for practical purposes of health protection, with the notable exception of the well-known synergism between smoking and radon (actually the radioactive decay products of radon). Even within the field of radioactivity alone, there are complications. A unified approach to controlling exposure to multiple radioactive materials has been attempted (and implemented in regulations) through the introduction of dosimetric concepts and, in particular, effective dose. However, this approach involves considerable simplifications, e.g., the assumed universal applicability of the radiation and tissue weighting factors adopted, that can be, and have been, challenged.
5. The overall scientific framework that is needed to guide research and to interpret its results for protecting human health when there are multiple exposures is not yet well-defined. This includes exposure to multiple chemicals and combinations of exposure to radiation and chemicals. The development of the science so far has mainly taken the point of view of the pollutant by following its course through the environment to the various organs and tissues of the body. This is useful and essential, since tracing the path of a pollutant helps us understand how it moves through the ecosystem or the sensitivity to the pollutant of different systems and organs within the human

> body. But the human body experiences them as a whole and in combination; hence it is also necessary to trace the modes by which human beings come into contact with multiple pollutants and how the combinations that they encounter affect them.

This report is a description of from where society has come in radiation protection and what is now possible based on what we know and the principles of public health. Also, it aims to begin a process that will allow the assessment of the joint risks of exposures to chemicals and radiation and to multiple chemicals. This is a major scientific challenge which is both conceptually and technically difficult and with which little progress has been made to date. We will develop it sufficiently in this report to establish it as a basic problem and then deal with it substantively in a future report.

As noted, chemicals are, thus far, mainly regulated one at a time and the potential for increased risks from combined exposures is generally not evaluated. As with radiation protection, the study of single cells, of DNA, and of molecular biology, combined with a greater understanding of whole living beings and environmental systems, helps to illuminate the paths where further research efforts might yield the greatest fruit.

Section 1.1--Protecting those most at risk

A part of the inspiration for this study was a simple realization about one radionuclide – tritium (T), which is hydrogen made radioactive by the presence of two neutrons in the nucleus. But chemically, it still behaves like hydrogen.[8] Since water is simply two atoms of hydrogen bound to one atom of oxygen – H_2O – tritium can displace one or both atoms of hydrogen to form radioactive water (i.e. HTO or T_2O).[9] As it combines with oxygen to form tritiated water, tritium can combine with other chemicals in the body, displacing non-radioactive hydrogen with radioactive hydrogen. Water, and nourishment, which consists of hydrogen- and carbon-containing molecules, cross the placenta and are incorporated in the fetus. The tritium present in these molecules can then irradiate the cells within the fetus that are dividing rapidly, which makes them more susceptible to damage than those in adults or even children. Organs may be most vulnerable at certain times when they are being formed (so-called critical periods; see Chapter 6).

One realization in particular seems crucial – a part of each of us is as old as our mother. This is because the specific ovum from which each person is made was formed in his/her mother during fetal development. Radioactive water crossing the placenta, therefore, seems to have the potential both to affect the development of the fetus, possibly resulting in miscarriage, genetic damage, or birth defects. It also seems to pose the risk of multi-generational problems.

Despite the likely greater sensitivity of the embryo/fetus and of children when their systems are growing rapidly, it was a surprise to see that radiation protection research studies and most regulation paid scant attention to a variety of non-cancer problems, including potential non-cancer risks during the most sensitive times of exposure. Indeed, only limited work has been undertaken on investigating whether such critical periods exist. Thus, we can only speak of *likely* greater sensitivity, as the underlying

[8] Chemical properties are defined by the electrons in an atom, which form a cloud around the nucleus. In a neutral atom, the number of electrons in the electron cloud is equal to the number of protons in the nucleus. Radioactivity is a property of an unstable nucleus, which leads to changes in the nucleus by the ejection of particles and, often, photons, or the absorption of electrons from the inner shells of the atom. The most common hydrogen atom contains one proton in the nucleus and one electron. The next most common, also non-radioactive, contains one proton and one neutron in the nucleus. Tritium has one proton and two neutrons in the nucleus. It decays by emitting a beta particle and has a half-life of 12.3 years.

[9] Tritium can also combine with deuterium (D), a non-radioactive isotope of hydrogen to form DTO. This is important in situations where heavy water (D_2O) is used in nuclear reactors, as in Canadian nuclear power reactors or the plutonium production reactors in the United States at the Savannah River Site. The latter are now closed.

research has not been conducted or synthesized into official reviews. In this context it is interesting to note that it was not until the late 1970s that the effects of *in utero* irradiation on intellectual capability of Hiroshima and Nagasaki survivors were recognized, though, as noted, there was some earlier interest in and evidence about this problem. Even then, the first effect that was recognized was severe mental retardation, an obvious clinical symptom in a few individuals. Without this clear signal, it is possible that the more subtle shift in mental capacity of the whole exposed population aged 8-25 weeks post-conception at irradiation may have been neglected.[10]

Another motivation for preparing this report was the realization that clean-up standards for radioactively contaminated sites are being set for "Reference Man," defined explicitly as a young "Caucasian" male with European or North American cultural and dietary habits. A crucial manifestation of this problem is that this Reference Man model is built into the main computer program, called RESRAD, that is used to assess risks from residual radioactivity after remediation of radioactively contaminated sites.[11]

The purpose of this report is to provide an analysis showing that the scientific knowledge and some of the basic concepts for far more effective health protection already exist when it comes to radiation. It also aims to substantiate the need for establishing a paradigm and for new research that would help move us towards a more effective understanding of the risks of combined exposures to radiation and chemicals and to multiple chemicals. (As noted, we will not try to develop that paradigm in this report; we aim to do so in a future one.) A part of the goal is for this work to serve as the main analytical foundation of a letter to President Bush asking him to issue an Executive Order to all agencies in his administration to review the basis of their health and environmental protection standards to ensure that those most at risk are truly being protected regardless of when that greatest risk arises.[12] The time and type of risks will vary and they may be different for different health concerns. In the earliest period, the first two to three weeks, the embryo is most susceptible to damage, with the potential outcome being a spontaneous termination of the pregnancy (technically called a "spontaneous abortion). Later stages of fetal development have different risks such as malformations and even cancer risk associated with them. Young children, adolescents, males just prior to conception, pregnant women may face a variety of health risks in differing degrees. In contrast to "Reference Man" as currently defined, we seek to introduce the concept of protecting the most vulnerable and to discuss theoretical and practical concepts to put that into practice. Some of those concepts already exist (such as "critical group" or limiting dose to the embryo/fetus in the workplace after a voluntary pregnancy declaration); they may need to be developed or put in a new context. Other concepts, for instance, those relating to protection from multiple exposures, will come more slowly because the underlying science and even the paradigm for that science are not well developed. Of course, in doing so, those less at risk would also be protected – more so than at present.

Much of the focus of this report is on the health of the developing fetus, which is dependent on the health of the pregnant woman. We are, of course, aware of the sensitivities of this topic. The status and even the definition of the embryo/fetus in society and who should make decisions on his/her behalf and the point at which the fetus can be considered a human being have all been topics of serious contention and, sometimes, violent action. A woman's right to have an abortion has been the focus of most of the contention. We do not seek here to enter that debate as part of this report.

Our goal is to make whatever contribution we can to help create a society in which it is never a tragedy to be pregnant, at least so far as society might be contributing today to create tragic pregnancies in the form of either avoidable exposures to hazardous chemicals and radiation or of regulatory standards that do not

[10] See ICRP 49 pp. 18-27

[11] The dose conversion factors derived that allow calculations of radiation dose due to a given intake of a radionuclide in RESRAD are based on the Reference Man model.

[12] The letter to President Bush, open for sign-ons, can be accessed via www.ieer.org.

take adequate account of the health damage caused by exposure of the embryo/fetus or of infants and children to anthropogenic environmental risks. This is, in its own way, a central issue of reproductive rights. But, unlike the issues of abortion and contraception, which relate to the rights of women versus the assertion of authority by the State or organized religion when the woman does not want to be pregnant, the issues that concern this report are centered on society's environmental responsibilities when a woman decides that she wants to become pregnant or wants to stay pregnant when the pregnancy was not planned. The time of our concern is both during the pregnancy and after it is carried to term, since children face significantly elevated risks and sometimes different ones than adults for the same level of exposure. Thyroid cancer is an example where the risks are far greater for female children under 5 years than for adult women who, in turn, have a higher risk than adult men. But it is far more than a question of reproductive rights alone. It also concerns men in their capacity as biological fathers and infants, children, and fetuses at various stages of development (see Chapters 4 and 9). Therefore, while a significant element of our work in this report and other parts of the project to examine the process of standard setting is about fetal health and the health of pregnant women, much of our work is on these other aspects of environmental health.

What are the responsibilities of society to pregnant women, to children after they are born, and to women and men who choose to reproduce and thereby give society an essential existential meaning?[13] Do women have the right to have an environment that minimizes the chances of a miscarriage due to the activities of business, industry, government, or other individuals? Do prospective fathers have the right to expect that they will be as fertile as possible and have as healthy sperm as possible when they are ready to have children and want them? In other words, do both men and women have the right to expect that the social, governmental, and economic institutions of society will respect the need for an environment that will be conducive to the potential for giving birth to healthy children and to keeping them that way as they grow? And how do we factor in what is good for infants? For the embryo that is developing into a baby? For the children of the females whose ova, the seeds of their children, develop while they are still *in utero*?

Section 1.2--Inclusion of Children Optional?

There has been much progress made in considering the health of women, including pregnant women in the workplace, so far as radiation protection regulations are concerned. Sometimes, there is even recognition that women, children, and the embryo/fetus should be explicitly considered as the most radio-sensitive. For instance in the *Staff Responses to Frequently Asked Questions Concerning Decommissioning of Nuclear Power Reactors*, the Nuclear Regulatory Commission (NRC) staff provided the following Q&A:

[13] Throughout this report we use the terms "male" and "female" and "men" and "women" in the biological/physiological sense of the words and are not referring to the gender identity of the individual. Our reference to existential meaning refers to the continuity of existence between generations for society as a whole, not to choices particular individuals may make about having children. John Maynard Keynes, one of the most celebrated economists of the twentieth century, argued for economic policies oriented to the short-term by famously declaring: "In the long-run we are all dead." Keynes's declaration contains a crucial philosophical and factual blind spot. It does not recognize the essential difference between a society in which everyone dies at once, and one in which its members can confidently look to succeeding generations populating human society for the indefinite future. If the latter is essential to existential meaning, then it raises the central question that underlies this study: Does society have some obligations to prospective parents, to the developing fetus, to infants, and to multiple generations who cannot make their presence felt except through an underlying value that while we will all die as individuals in the long run, it is still essential to organize society so that those who come after us are not burdened with the effects of the harmful substances that we choose to use?

> Who would be considered an "average member of the critical group?"
>
> The "critical group" means the group of individuals reasonably expected to receive the highest exposure to residual radioactivity within the assumptions of a particular scenario…... If radiation in the soil is the concern, then the scenario used to represent the maximally exposed individual is that of a resident farmer. The assumptions used for this scenario are "prudently conservative" and tend to overestimate the potential doses. *The added sensitivity of certain members of the population, such as pregnant women, infants, and children, are accounted for in the analysis.* However, the most sensitive member may not always be the member of the population that receives the highest dose. This is especially true if the most sensitive member (for example, an infant) does not participate in specific activities that may provide the greatest dose or if they do not eat specific foods that cause the greatest dose. [14]

However, despite this clear and unequivocal statement from the NRC Staff, the practice is far different. For instance, the government-developed computer model, RESRAD, used by the NRC (and the DOE) to verify compliance with regulatory limits explicitly incorporates the 154 pound Reference Man as its one and only built-in, default state.[15] Although it is possible for users of the program to construct their own dose conversion libraries for children using the EPA or ICRP recommendations, it is not possible to directly account for unique exposure pathways such as the consumption of contaminated breast milk by infants, nor is it possible to calculate the doses received by the embryo/fetus.[16]

This disregard of children is sometimes explicitly or implicitly sanctioned, as the following licensing example illustrates. Significantly, the Nuclear Regulatory Commission issued a ruling in 2001 after the *Staff Responses* quoted above were published by the Commission. In summarizing the arguments of Connecticut Yankee Atomic Power Company, a utility seeking approval of its decommissioning strategy, the Commission noted that

> Although the plain language of the regulation does not restrict the terms "critical group," "individual," or "human being" to mean any specific age, race, or gender, CY [Connecticut Yankee Atomic Power Company] argues that the regulation incorporated the Environmental Protection Agency's "Reference Man" concept, which assumes a person is a white male, age 20-30. CY contends that the critical group at Haddam Neck should be composed of resident farmers, as CY described them in its License Termination Plan, and that the "average" member is therefore an average farmer. Doses to children are therefore irrelevant, it argues.[17]

The Commission eventually ruled that the Connecticut Yankee Atomic Power Company should consider the doses to children, but that

> If the evidence shows, as CY claims it will, that doses to children are lower than doses to adults, CY will prevail without the need for an appeal. But even if the evidence shows that doses to children are higher, CY will still have the opportunity after the [NRC's Atomic Safety and Licensing] Board's final decision to argue before the Commission that our regulations prohibit considering doses to children.[18]

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[14] NRC 2000 pp. 40-41 (emphasis added)
[15] The program, known as RESRAD for residual radioactivity, was developed at Argonne National Laboratory and is available free from their website. (Yu et al. 2001 pp. C-22 to C-23)
[16] Some information relevant to such calculations is now available in ICRP Publications 88 and 95. (ICRP 88 and ICRP 95)
[17] NRC 2001 p. 372 (footnotes omitted)
[18] NRC 2001 p. 374

Even when the NRC does choose to consider doses to children, they have sometimes done a very poor job. For example, in the draft environmental impact statement supporting the early site permit application filed by Dominion for the potential placement of a new reactor at their North Anna, Virginia site, the NRC stated that "[n]o infant doses were calculated for the vegetable or meat pathway as infants do not consume these foods."[19] As support, the NRC cited Revision 3 of the corporation's environmental report which contained the very similar language and the same environmental impact analysis.[20] While common sense is sufficient to recognize that this statement is clearly untrue, it is revealing that it is also at odds with the recommendations of studies cited by the Environmental Protection Agency, and the views of the United Nations Scientific Committee on the Effects of Atomic Radiation (see Table 1).

Table 1: Recommendations from the Environmental Protection Agency, and the United Nations Scientific Committee on the Effect of Atomic Radiation regarding the level of fruit, vegetable, and meat consumption by infants compared to adults (all values in kilogram per year).

	Infant (0-1 years)	Adult (> 20 year)
Fruit and Vegetables[(a)] (EPA 1997)	63	213
Fruit and Vegetables (UNSCEAR 1993)	80	230
Meat (EPA 1997) [(a)]	8.4	58
Meat (UNSCEAR 1993)	15	50

Source: UNSCEAR 1993 p. 66, and EPA 1997
(a) These estimates assume an average infant mass of 7.9 kilograms and an average adult mass of 76 kilograms. (NCHS 2005, Tables 4, 5, and 6)

However, there are signs that these approaches may shift and that children and the embryo/fetus are being taken into account in some situations. The U.K. Environment Agency is undertaking research on the radiological implications of authorized discharges of phosphorous-32 (P-32) and phosphorous-33 (P-33) because these radionuclides have substantially higher radiological impacts on the embryo and fetus than on other age groups.[21] Furthermore, although Nirex, the UK waste disposal agency, bases its reference calculations on adults in the context of solid waste disposal, it also undertakes comparative calculations for the 10-year-old child, 1-year-old infant and embryo/fetus, to ensure that it is adequately protective of these groups. There is a trend in the UK to explicitly address protection of the embryo and fetus.[22]

Section 1.3--Informed consent

The democratic process of setting standards is supposed to be one that provides a mechanism for obtaining informed consent. Since there is no way to actually get informed consent from infants, children, the embryo/fetus, not to speak of more distant future generations, we must devise some means of decision-making that respects their right to a life that is not burdened with harm from the activities that adults might choose to engage in for their own benefit. Moreover, even within the context of those who can, at least in theory, participate in decision-making at any given time, there are critical questions raised by the setting of standards of exposure to substances that are known or likely to cause harm. Who benefits? Is the harm imposed on those who get the benefit or on third parties who do not? Is there general agreement about the social usefulness of and need for the activity, as for example in the use of

[19] NRC 2004 p. 5-61
[20] Dominion 2004 p. 3-5-19: "There are no infant doses for the vegetable and meat pathways because infants do not consume these foods."
[21] NDAWG 2006
[22] Thorne 2006 – Issue 1

chemotherapy for cancer treatment or medical radiation for treatment or diagnosis in certain circumstances and with due precautions for minimizing exposure? Or is there a deep division in society, as, for instance, about the use of nuclear power, toxic pesticides, genetically engineered seeds in farming, or the testing of nuclear weapons?

A few principles can be set forth:

- It is improper to base standards setting in relation to risk or harm created by human beings by reference to risks that are present in nature, such as natural radiation or chemical carcinogens. It is part of the natural order that we are born and we will die and in between are exposed to all manner of natural hazards. But clearly one cannot punch one's neighbor in the nose on the ground that natural illnesses can be much more painful. As another example, natural radiation likely causes some ill-effects, including cancer. But, as President Kennedy noted, this "natural health hazard" cannot be a license for some human beings or institutions, even if they are powerful governments, to pollute and expose other human beings without their full and informed consent.
- It is essential to consider whether the imposition of risks is necessary – that is, it is essential to consider the need for the activity and the alternative ways of achieving a goal (such as a reliable energy system).
- Imposed risks, in which the person suffering the risk does not directly benefit from the activity creating it, must be distinguished from voluntary risks, such as those created by medical treatment.
- A democratic decision-making process is essential before society can impose risks on its members.
- If possible, multigenerational risks should be avoided. But if they cannot, there should be an explicit recognition of the problem and social means to deal with it. For instance, a great deal of long-lived radioactive contamination of nuclear weapons sites around the world already exists. This cannot be clean-up in the sense of zero risk to future generations, but risks from it can be reduced both for present and future generations, in particular if the offending activity, such as plutonium production, is immediately halted. The problem of setting standards and the problem of democracy is acute in this case.
- Standards must preferentially protect infants, children, developing fetuses, and their offspring. By extension, the protection of women and men must not only be a goal in itself, but their role as actual and prospective parents must be at the center of the standard setting process. So far, the risks to children, to developing fetuses, to pregnant women, and to prospective fathers have not been at the center of standard setting.

We first provide an overview of radiation protection, and then consider the various issues related to exposure to radiation by sex and age. This is followed by some considerations relating to the study of combined chemical and radiation exposures. We also illustrate the problem of standard-setting with some case studies.

Chapter 2: Overview of Radiation and Radiation Protection

Ionizing radiation consists of particles that are energetic enough to break apart molecules and strip the electrons from atoms, creating electrically charged ions. This process can, in turn, lead to the creation of new chemicals. The processes of breakdown and creation of chemicals in living beings is, of course, part of the process of life, as it is also a part of the process of destruction, disease, and death of living cells.

Ionizing radiation is a concern because exposure to it produces random damage to molecules that constitute the basic structures of living beings, whether they are water, glucose, ATP (adenosine triphosphatase, the energy carrier in cells), amino acids, or genetic material like nuclear or mitochondrial DNA. The vast majority of ionizations result in chemical outcomes easily repaired or otherwise dealt with by the body. Other ionizations may result in cell death, which in most cases is dealt with by the body through the replacement of the cell. But some kinds of ionizations result in damage to genetic material that is misrepaired and thus creates the risk of adverse health outcomes like cancer, miscarriages, genetic damage, or birth defects. Of course, not all such problems are due to misrepair caused by radiation. Indeed, diet, genetic factors, and personal habits (notably in regard to exercise) play large roles in susceptibility to disease, including cancer. Our exploration of anthropogenic radiation should be viewed in this context, but also in the context that radiation of human origin due to the processes associated with commercial nuclear power and with nuclear weapons has not been imposed with full and informed consent.[23]

Radiation has been studied more than most chemicals and much is known about how it acts on the human body. Yet much remains unknown largely because of the complexity of the problem and the fact that the right questions have not been asked or, if they were asked, were not pursued with the vigor needed to create an adequate framework for understanding the ill-health effects, especially their quantitative aspects. In other cases, the answers are known, but their application to the practical world of health protection in society is incomplete or inadequate. As noted, the science is evolving rapidly and in some cases even the regulations are beginning to shift.

The connections between radiation, gene mutation, and cancer were first discussed in the scientific literature as far back as the late 1920s and early 1930s. These works were based on both animal experiments and epidemiological studies of humans that had been exposed to occupational radiation.[24] At that time, only a relatively small number of people had been exposed to significant levels of radiation in the workplace. Examples of some of the most heavily exposed workers included miners working in underground areas with high uranium content, such as in Czechoslovakia, the radium watch dial painters in the United States, and the early x-ray technicians and radiologists.

With the launching of the Manhattan Project in the early 1940s, the U.S. was faced with protecting large numbers of workers who were at risk of considerable exposure. In addition, there were potential exposures to radionuclides that did not exist in significant quantities anywhere in the world. For the project to be successful, it would, at a minimum, be important to ensure that worker doses would remain low enough to avoid acute injuries such as radiation burns or radiation sickness that might impair the employees' ability to complete the atomic bombs. The long-term risks from cancer do not appear to have been a dominant concern during the wartime Manhattan Project.

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[23] Fallout from atmospheric testing is one prime example. In the 1950s, U.S. Atomic Energy Commission scientists knew that iodine-131 from fallout was concentrating in milk, but they did nothing to protect the milk supply. By contrast, they provided advance data on fallout to the photographic film industry so they could protect their products. (Ortmeyer and Makhijani 1997)

[24] See, for example, Muller 1927, Oliver 1930, McCombs and McCombs 1930, Martland 1931, and Pirchan and Sikl 1932.

Section 2.1--Progressive Tightening of Dose Limits

Although the first dose limits in the U.S. had been adopted in the early 1930s to govern exposure to x-rays, it was during the atom bomb program that the fields of health physics and radiation protection in general underwent their most important evolution. As a result, the health physicist's primary focus was on protecting the workers in the nuclear weapons' complex. At the time, this implied a focus on young, otherwise healthy males. During the 1940s, the links between radiation and cancer started becoming clearer. By the end of the 1940s and early 1950s, there were systematic industrial hygiene surveys at many plants where nuclear materials were processed for the nuclear weapons enterprise. The 1950s saw a general tightening of radiation protection standards over those of the wartime years and also the explicit adoption of the male worker as the standard for whom all doses would be calculated.

In 1954, allowable worker doses from exposure to external radiation were reduced to 3 rem per quarter and 5 rem per year over the long term.[25] While the units were not directly comparable, this was a reduction in the worker dose limit of roughly seven times over the standards set during the Manhattan Project. Separate limits were also set for exposure to internal radiation, such as that caused by breathing in radioactive dust particles. The first separate protection standard for the general public, as distinct from workers, in the United States, came in 1959. In that year, maximum allowable exposures for the general public were set at one-tenth of worker doses (i.e., 500 millirem per year).

Who's Who in U.S. Radiation Protection

A number of bodies study radiation or regulate it. Some of the principal ones (as they concern the United States) are:

NCRP: National Council on Radiation Protection and Measurements is a scientific advisory body.

BEIR Committees: The Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation (formerly called the Committee on the Biological Effects of Ionizing Radiation (BEIR)) of the , National Research Council of the National Academies, does periodic reviews of the effects of ionizing radiation that are influential for standard setting bodies. The BEIR VII report is the most recent report in the series.

EPA: The Environmental Protection Agency, issues official guidance documents, called Federal Guidance Reports (FGRs) on radiation that give a regulatory imprimatur to the science, allowing it to be used in regulations (though it may or may not actually be used). FGR 13 is the most current EPA guidance.

Table 2 shows how limits to radiation have been progressively tightened as more has been learned about the risks of radiation exposure. In addition to the tightening of the limits for exposures of the general public from 5,000 millirem per year (the same as those for workers in the mid-1950s) to a maximum 100 millirem per year, stricter standards have also been introduced to control exposures from single nuclear facilities or pathways. For instance, in 1976, the Environmental Protection Agency issued standards for maximum contamination or radiation dose permissible due to the pollution of drinking water.[26] The dose limit for most radionuclides from the drinking water pathway was set at 4 millirem to the maximally

[25] That is, the cumulative dose received by a worker should not exceed five rem multiplied by his/her age over 18 years.

[26] They were first published in the Code of Federal regulations at 40 CFR 141.15 and 141.16, but were later renumbered and consolidated, without change, into 40 CFR 141.66, where they remain as of the date of this report.

exposed organ. Also, effective in 1979, the EPA issued standards for maximum exposure from single nuclear power-related facilities, and set that level at 25 millirem per year to the whole body or to any organ.[27] As a final example, in 1989, the EPA also issued regulations to limit the dose to the maximally exposed individual from emissions of radionuclides to the air from Department of Energy facilities to 10 millirem per year (effective dose equivalent).[28]

These tightened limits reflected three basic realities:

1. A growing scientific and public awareness of the risks of radiation in the context of an overall increase in concern about health damage from environmental causes.
2. Increased understanding that the risks of radiation were higher than believed in the early days of radiation use.
3. Growing recognition, as exemplified by President Kennedy's speech announcing the Limited Test Ban Treaty, that there is a fundamental difference between natural or voluntary risks and human created or involuntary risks, especially since it is often the case that exposures of members of the public cannot be reasonably or easily controlled by actions that they themselves could take.

Table 2: Chronology of External Radiation Exposure Standards[29]

1931-34	U.S. Advisory Committee on X-Ray and Radium Protection (precursor to the National Council on Radiation Protection and Measurements) adopts X-ray "tolerance dose" of 0.1 roentgen per day.
1940-41	U.S. Advisory Committee proposes, but does not implement, lowering the X-ray tolerance dose to 0.02 roentgen per day.
1942	U. of Chicago Metallurgical Laboratory adopts a "maximum permissible exposure" standard of 0.1 roentgen per day. Becomes standard for entire Manhattan Project.
1951	NBS reduces the limit of external whole body radiation to 0.3 roentgen per week.
Mid-1950s	Atomic Energy Commission adopts National Bureau of Standards recommended maximum long-term dose limit of 5 rem per year. Sets additional limits for internal exposures at 15 rem per year for most organs.
1959	Dose limit for workers remains 5 rem per year. AEC also adopts dose limits for the public equal to one-tenth of those allowed for workers: 0.5 rem for external exposure; and 1.5 rem for most organs for internal exposure.
late 1980s - 1990	Department of Energy adopts dose limit for the public of 100 millirem (0.1 rem) per year; dose limit for workers remains 5 rem per year. A new model for calculation of internal doses to workers is adopted, the "committed effective dose equivalent."
1991	International Commission on Radiological Protection recommends worker dose limit be reduced to 2 rem per year. Recommendation is not adopted by DOE.
NOTE: For external radiation sources, roentgen and rem are considered to be equivalent. *Sources*: **1931-34, 1940-41, and 1942**: Hacker 1987, Appendix A, pp. 163-164; **1951**: NBS 47, 1951; **Mid-1950s**: AEC 1954, 0522-01.h and NBS 52, 1953; **1959**: NBS 69, 1959, pp.4-6; **late 1980s - 1990**: DOE 1990, II.1a; **1991**: ICRP 60, p. 72, para. (S25). See also Shapiro 1990, Part VI.	

Section 2.2--Differential Risks of Radiation Exposure

Risks from radiation exposure have been defined largely in terms of cancer mortality and morbidity risks (though the risks of inducing serious hereditary disease in the descendants of the exposed individual were also taken into account), partly because the study of the victims of Hiroshima and Nagasaki allowed the

[27] The standards are for routine operations as specified at 40 CFR 190.10. The limit for the thyroid was set higher, at 75 millirem per year. In addition, the operators of nuclear facilities must meet a more stringent requirement to keep exposures "As Low As Reasonably Achievable" – called the ALARA principle.
[28] EPA 1989, announcing the Final Rule for 40 CFR 61.92
[29] Adapted from SDA vol. 6, No. 2, 1997

risk of developing a cancer to be calculated with rather more precision than would normally be possible in epidemiological studies.[30] Two types of risk came to be defined: risks for adults, defined generally by exposure of an adult male (see below), and average lifetime risks caused by exposure from birth to about 70 years of age. Until about 1990, the overall difference in cancer mortality risks from uniform, whole-body radiation exposure between men and women was thought not to be large for all cancers combined, though differences for specific cancers, notably breast cancer, were known to be significant. In fact, in 1990, the National Research Council of the National Academy of Sciences of the U.S. (hereafter the National Research Council) assessed the risk for women from radiation exposure as being only about 5 percent larger than the risk for men.[31] This estimate was later increased, as describe in Chapter 3.

The greater focus on the specific health risks faced by women in many different areas which has occurred over the last two decades has also led to a greater understanding of the differential risks of radiation. In 1999, the EPA published Federal Guidance Report 13 (FGR 13), which details morbidity (i.e. cancer incidence) and mortality (i.e. cancer fatality) risks from exposure to radiation by sex. By that time, the overall risks to women were recognized to be significantly greater than for men. The most recent detailed analysis, done by the National Research Council, Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation (commonly called the BEIR VII report),[32] provides further details that are discussed in Chapter 3. Suffice it to say here that as knowledge has increased, it has been found that the risks to girls and women are, overall, significantly greater than the risks to boys and men, and the risks to children are generally greater than the risks to adults for a given level of exposure. It should be noted that in some cases, like colon cancer, risks are now thought to be greater for men, in contrast to prior findings.

So far we have been focusing only on cancer risk. This is due, in part, to the fact that there is currently very little comparable data on non-cancer risks, especially at low-levels of exposure.

Although the science has progressed significantly, much regulatory guidance remains stuck in the past. Specifically, in 1975, the International Commission on Radiological Protection (ICRP) published its recommendations for "Reference Man" (sometimes referred to by its older name "Standard Man"). The ICRP described their reference individual quite clearly:

> Reference man is defined as being between 20-30 years of age, weighing 70 kg [154 pounds], is 170 cm [5 feet 7 inches] in height, and lives in a climate with an average temperature of from 10^{o} to 20^{o}C [50^{o} to 68^{o}F]. He is a Caucasian and is a Western European or North American in habitat and custom.[33]

While the model has continued to change and evolve over time, the focus of regulators on a young 154 pound, white male worker remains central to crucial aspects of radiation protection schemes in the United States. Three of the most important are:

[30] These studies have not been without controversy, of course. The point here is simply that compared to many chemicals, for instance, a good deal more is known about radiation risks, not least because of the long-term study of people exposed by the bombings of Hiroshima and Nagasaki and the knowledge about the magnitude of exposure gained from assessments of the irradiation conditions, including a reproduction of certain technical aspects of the bombings in the U.S. nuclear weapons' testing program.
[31] NAS/NRC 1990 pp. 172-173
[32] NAS/NRC 2006. The report was first publicly released in 2005. However, it was published as a book by the National Academy Press in 2006 and is referred to here as NAS/NRC 2006.
[33] ICRP 23 p. 4

- Standards are set according to dose rather than risk of disease. Since adults consume more food and water, they may get a higher dose in some circumstances but may have a lower risk of cancer than children getting the same dose.
- Radiation protection dose limits are set by considerations of cancer risk. Hence, risks of a variety of other problems such as birth defects, infertility and early miscarriages are not part of the regulatory scheme.
- Cleanup levels for residual radioactivity at contaminated sites are set according to results generated by the government-approved program, RESRAD and are estimated for an adult male. For instance, the vast sums of money that have been and will eventually be spent (amounting to hundreds of billions of dollars) to remediate nuclear weapons sites will not take the woman farmer or her children into account, nor the exposure to the embryo/fetus in her, should she become pregnant.

The studies of the effects of chronic exposure to radiation on pregnancy outcomes, on adverse health in the embryo/fetus, on infants and children, and on ova and sperm and the resulting offspring are still plagued with considerable uncertainty, in part due to insufficient knowledge and research. Hence, the potential for harmonizing the protection of prospective fathers and pregnant women has not yet been explored seriously in the regulatory arena.

In the coming chapters, we explore potential health impacts on women, children, and men in terms of the dose per unit exposure and the risks of cancer and other health problems arising from exposure. The aim is not to be exhaustive in the sense of a literature review, but to provide a sound basis for the analysis and to illustrate the nature of the problems confronting radiation protection for various groups. The term "potential health impacts" is used here with particular significance. Some of the impacts discussed are subject to scientific debate as to whether they are induced by ionizing radiation, or to what degree such induction occurs. Where this is the case, the primary requirement is to pursue further research to clarify the issues while adopting a precautionary approach in regulation to ensure that the individuals are adequately protected when there are indications of a connection, whether or not future research confirms the problem as being associated with ionizing radiation. This exploration of potential health impacts, in turn, provides the basis for our findings and recommendations.

Before considering the various groups at risk in detail, however, it is worthwhile considering the costs and benefits of radiation in the context of voluntary or involuntary exposure, since these are an important part of the framework of social decision-making as well as regulatory guidance in considering risk.

Section 2.3—Costs and Benefits of Radiation Exposure

According to the ICRP, when considering activities that may result in exposure to radiation, a system of dose limitation should be implemented according to the following objectives:

> (a) No practice involving exposures to radiation should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes. (The justification of a practice.)
> (b) In relation to any particular source within a practice, the magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received should all be kept as low as reasonably achievable, economic and social factors being taken into account. This procedure should be constrained by restrictions on the doses to individuals (dose constraints), or the risks to individuals in the case of potential exposures (risk constraints), so as to limit the inequity likely to result from the inherent economic and social judgments. (The optimisation of protection.)

> (c) The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits, or to some control of risk in the case of potential exposures….(Individual dose and risk limits.)[34]

These are demanding and complex criteria to implement in practice even for exposures with clearly defined benefits such as medical radiation.[35] This is because the available alternatives are often not explored or may be difficult to compare. We will briefly consider the case of mammograms and MRI imaging for breast cancer detection in Chapter 3. But they are far more complex in the arenas where there is no social agreement regarding the balance of costs and benefits, notably in the case of nuclear weapons and nuclear power. The issues become even more complex when the cost (risk) is borne by the public and the main benefit is profit made by private entities. When the public is in countries other than the ones originating the contamination, as was the case with fallout from atmospheric nuclear weapons testing and some underground testing, the difficulties of considering benefits and costs are greatly magnified. Finally, the most complex issue of all is intergenerational risk. In some cases, future generations may inherit costs and benefits. In other cases, they may suffer the costs, but receive few or none of the benefits.

Section 2.4--Cancer Risk Due to Radiation Exposure

Exposure to radiation is regulated primarily for its potential to cause cancer. Intensive study of its health effects began with the Manhattan Project. The main goal at that time was to prevent serious injury from a hazard that one could not feel or see or smell. In the postwar period, with the development of a vast nuclear weapons infrastructure, the use of nuclear power in the civilian economy, the widespread use of radionuclides in medicine, industry, and research, and concerns relating to radioactive contamination of air, soil, and water that would cause doses considerably below the levels that would produce deterministic health effects (which are readily observable), the health concerns shifted to the potential for radiation to cause cancer.

As the decades have passed, official scientific bodies, notably the Committee on the Biological Effects of Ionizing Radiation (BEIR) of the National Research Council (more recently called the Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation) and the International Commission on Radiological Protection, have increased their estimate of cancer risk per unit of radiation exposure. Much of the data for the risk estimates has come from a longitudinal study of the survivors of the atomic bombings of Hiroshima and Nagasaki in August 1945. The number of cancers in this survivor population has increased beyond the early projections, and so has the estimate of radiation risk.

Much other research, including theoretical work, in-vitro research on the response of cells to radiation, research on animals, and epidemiological studies, has also been conducted. Given the number of cancers that occur due to causes other than human-created radiation exposure, including diet, genetic constitution, exercise, exposure to natural radiation, and exposure to chemical environmental carcinogens, the study of the cancer risk of low levels of radiation is particularly difficult. "Low-level" exposure to radiation is defined as that which does not produce deterministic effects, such as reduction in white blood cells count, skin rash, hair loss, etc. Usually, "low-level" radiation is defined as being less than about 100 rem, though physiological changes, such as reduction in white blood cell count, can be observed at much lower doses.

The lack of deterministic effects can also be due to low dose *rates*, even if the total dose is high. In practice, the main difficulties of radiation research from the point of view of exposure of large numbers of

[34] ICRP 60 p. 71
[35] Medical radiation exposures are discussed in further detail in Chapter 3.

people are related to exposure to low doses and/or low dose rates. One reference point for defining "low dose" is 10,000 millirem (or 10 rem) derived from the analysis of Hiroshima and Nagasaki data.[36] That dose was, of course, delivered in a very short period of time and consisted mainly of gamma rays and neutrons emitted due to the atomic explosions themselves. For comparison, the exposure to natural background radiation at sea level, not including indoor radon, which is an artifact of construction, is about 100 millirem (0.1 rem) per year.[37] Over a lifetime of seventy years (a typical reference lifetime used for purposes of estimating lifetime risk) the total natural dose would be 7,000 millirem. But this dose is received over a long period of time, that is, at a low dose rate of just over 11 microrem per hour, in contrast to the single, acute dose received by the Hiroshima/Nagasaki survivors.

The study of the effects of the Hiroshima and Nagasaki nuclear bombing on survivors has been a huge effort, which is continuing. The estimates of cancer risk used in regulatory practice are largely based on the study of these survivors. However, since the survivors received rather large doses, and since their radiation dose was received over a very short period, extrapolating the risks to low dose levels delivered over long periods of time has proved controversial and difficult. Moreover, some researchers, notably the British physician, Alice Stewart, and her colleagues, have pointed out that the long-term survivors were probably among the healthier people to start with and this complicates extrapolation of cancer risk to the general population from the survivor group.[38] There was also a delay of several years in beginning the study, and the effects of that delay are now difficult to estimate; they have also been the subject of some controversy.

The difficulty is that everyone is also exposed to many other risk factors, including natural and man-made environmental risks, diet, and heritable factors. For instance, besides, natural background radiation, there are also varying levels of exposure to indoor radon, which depends on building construction and on the region in which buildings are located. Since there is a substantial rate of cancer due to all these other factors, it is very difficult to extricate the risks explicitly attributable to exposure to low-levels of man-made radiation, such as nuclear bomb fallout or routine radiation exposure in the workplace. Indeed, given that there are agents that can act in combination (see Chapter 6), it is not clear that a separation of risks can be justified.

Yet, radiation has been much more intensively studied than most chemicals. A considerable amount is known about the effect of radiation at all scales ranging from the cellular level all the way to epidemiological studies, especially in relation to cancer risk. We address gaps in the knowledge in subsequent chapters, but these are in the context of what has become a remarkable consensus in official scientific studies about the best hypothesis of the cancer risk of radiation exposure and the amount of that risk. The consensus, despite the uncertainties that remain about the effects of radiation at low doses and low dose rates, is called the Linear No-Threshold Hypothesis (often abbreviated as LNT hypothesis or LNTH). It states that every increment of radiation exposure, no matter how small, produces a corresponding and proportional increment of cancer risk. It applies to solid tumors, which includes most cancers, including lung cancer, breast cancer, and prostate cancer, but not leukemia. The no-threshold hypothesis is also applied to leukemia, though in that case the model usually preferred is linear-quadratic, which is a combination of those shown in Figures 1 and 2 below.

[36] See, for instance, NAS/NRC 1990 pp. 172-173.
[37] Natural background varies considerably and is higher at higher altitudes and in areas with high uranium or thorium concentrations in the soil and rocks. It is due to radiation from cosmic rays as well as from radionuclides such as uranium isotopes, thorium isotopes, radium isotopes (especially radium-226), and potassium-40 present on Earth.
[38] Stewart 1997

Using the LNT hypothesis, for example, if a person has a certain risk of getting cancer at one rem of exposure, his or her cancer risk would be doubled for an exposure of two rem, and halved at 0.5 rem. Further, if ten people collectively got one rem, their collective risk would be the same as that of one person being exposed to ten rem.

Collective population exposure is expressed as person-rem, which is the sum of all individual exposures in a population. From an estimate of collective dose, one can then apply a constant risk factor to get a statistical estimate of the number of additional cancers that would result from that exposure. Note that these computations apply only at the low doses and low dose rates for which the LNT hypothesis is considered to be justified.

Figure 1 shows the LNT hypothesis. There are other hypotheses about the shape of the dose-response curve. The most common alternative no-threshold hypothesis is the "linear-quadratic" hypothesis. According to this, there is a risk term that is directly proportional to the dose (the linear term) and another proportional to the square of the dose (the quadratic term). Figure 2 illustrates a quadratic dependence of risk on dose (linear term equal to zero).

There are those who believe that there must be a threshold below which there is no increase in cancer risk. They argue that some toxic materials exhibit such thresholds and that radiation has one too. Such thresholds may derive, for instance, from the ability of the body to repair damage caused by lower doses of radiation. Figure 3 shows a threshold hypothesis, with a linear risk response for doses higher than a threshold of T rem but zero below that. However, it has been pointed out that since human beings are already exposed to natural radiation as well as other natural and artificial exposures to other agents that stress the body's repair system, the LNT hypothesis may, in any case, apply to radiation doses imposed by human activities because they are increments to other exposures. Hence, for the purposes of estimating the risks from human activities, the LNT hypothesis could still be valid and is a sound basis for public health protection.

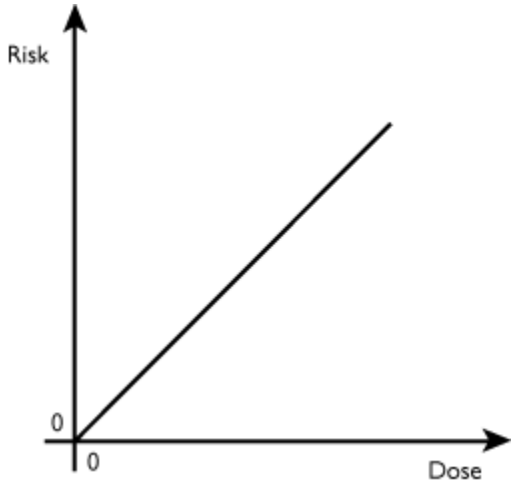

Figure 1: Linear No-Threshold Hypothesis

Figure 2: Quadratic Dose Response

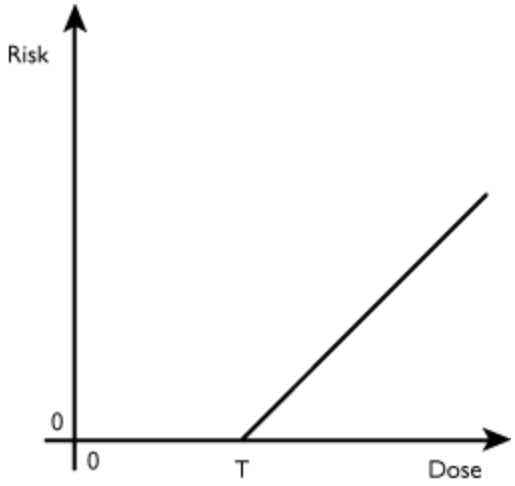

Figure 3: Threshold Hypothesis

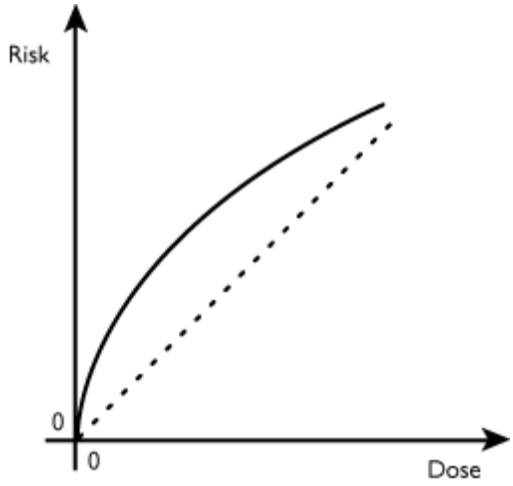

Figure 4: Supralinear Hypothesis

There is also some evidence from recent experiments that low doses may produce a higher level of risk per unit of dose. This is known as the supra-linear hypothesis, and is shown in Figure 4.[39]

Section 2.5—The BEIR Risk Estimates

In 1990, the fifth BEIR panel of the NAS (called BEIR V for short) estimated that the risk of radiation was considerably higher than prior official studies.[40] It affirmed the Linear No-Threshold hypothesis for solid cancers, and provided an estimate of cancer risk. The total risk for all cancers (including leukemia), based largely on the Hiroshima/Nagasaki survivors, was estimated at about 790 *fatal* cancers per million person rem of population exposure. The irradiated population is assumed to have the same distribution as the U.S. population, so that the model assumes that people of all ages are being irradiated.[41]

Under the LNT Hypothesis, the actual distribution of those doses within an age group in a given population would not change the overall outcome in that population, though, of course, individuals who had higher doses would be at proportionally higher risk. The differential risk between men and women was thought to be low at the time, with the risk to women being about 5% greater than that to men. The BEIR V report also discussed the evidence that the risk from low dose rates was lower than that of doses delivered at once, but did not provide a conclusion on how much lower it might be.[42]

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[39] This description is adapted from SDA vol. 8, no. 1, 1999, and the supporting documents are referenced there. There are some who subscribe to the "hormesis" hypothesis, according to which a small amount of radiation could produce some beneficial health effects, by stimulating the immune system for instance. The main evidence put forward for this has been from experiments on mice. According to a summary of the evidence for the hormesis effect, compiled by Charles Waldren, a high dose of radiation produced fewer mutations in some circumstances if preceded by a dose in the 1 to 20 rem range. This supposed protective effect does not appear at lower or higher doses, however, and lasts only for about a day, after which it disappears. (Waldren 1999) Such a hormesis effect, even if it exists in humans, has no public health significance, since the cancer risk of the exposure would be very high and any immune system stimulation would be very temporary. This issue has been extensively addressed by the BEIR VII panel and others. The conclusion of the BEIR VII panel was that "the assumption that any stimulatory hormetic effects from low doses of ionizing radiation will have a significant health benefit to humans that exceeds potential detrimental effects from the radiation exposure is unwarranted at this time." NAS/NRC 2006, p. 335. We do not address the issue of hormesis further in this report.

[40] NAS/NRC 1990

[41] NAS/NRC 1990 pp. 172-173

[42] NAS/NRC 1990 pp. 172-173 and Chapter 7

It has been U.S. regulatory practice to assume that low-level radiation delivered at low dose rates would cause about 400 fatal cancers per million person rem for adult exposure – or one cancer per 2,500 rem of exposure – for adults. This is about a factor of two less than the Hiroshima and Nagasaki risk value to account for the reduced effectiveness of radiation received at low doses and low dose rates (see below). For the general population (exposure at all ages), the risk factor used is higher -- one fatal cancer per 2,000 rem.[43]

Much has changed in recent years. Based on recent data, the risk to women is now estimated to be considerably higher (see Chapter 3). Moreover, the most recent report of the NAS on low-level radiation (the BEIR VII report[44]) also provided estimates of the risk of cancer incidence, in addition to fatal cancer risk. We consider the cancer incidence to be a much more sound basis for risk estimation, since fatality rates are not only dependent on the cancer type, but also on the evolution of medical capabilities in treating cancer and the extent of the use of those capabilities in the health care system. For instance, an early detection of breast cancer or development of more effective therapies may reduce the fatalities from breast cancer, but that does not relate to the risk of breast cancer due to radiation.[45]

The BEIR VII Committee also estimated the differential cancer risk by age. In this report, we mainly use cancer risks as estimated by the BEIR VII Committee when available. We also discuss radiation doses to the embryo/fetus, which are based on the work of the ICRP. Table 3 shows a summary of the BEIR VII Committee's risk estimates.

Table 3: Cancer incidence and fatality estimates per million person-rem, lifetime dose, BEIR VII report – best estimates. Estimates corresponding to 90 percent confidence interval are shown in parentheses.

	Males, solid cancers	Females, solid cancers	Males, leukemia	Females, leukemia	All cancers, males	All cancers, females
Incidence (all cases, fatal and non-fatal)	800 (400, 1,600)	1,300 (690, 2500)	100 (30, 300)	70 (20, 250)	900	1,370
Fatal cases only	410 (200, 830)	610 (300, 1,200)	70 (20, 220)	50 (10, 190)	480	660

Source: NAS/NRC 2006, p. 15.

It is evident that, overall, the risk to females from radiation exposure is considerably higher than it is for males. We take up this issue in the next chapter.

[43] EPA 1999 pp. 1-2
[44] NAS/NRC 2006
[45] In some cases, even cancer incidence does not provide the full picture. For instance, a comprehensive program of colonoscopy examinations of the population over 50 could eliminate the vast majority of colon cancers by allowing pre-cancerous polyps to be removed. Also diagnostic techniques or medical coverage could improve revealing higher rates of cancer than previously believed.

Chapter 3: Women and Radiation Risk

Section 3.1 – The Relative Cancer Risk of Men and Women

Despite the fact that the overall incidence of cancer in the U.S. population is 22 percent higher among men than women, it has consistently been found that women are more sensitive to radiation-induced cancers than men.[46] The higher overall risk of cancer among men is largely due to the prevalence of prostate cancer, which is not a very radiosensitive organ. The difference in risk between men and women when it comes to radiation exposure has been elaborated in detail in recent years by both the Environmental Protection Agency and the National Research Council. The most recent estimates agree that women are at a much higher risk compared with previous estimates. The higher risk to radiation is related to the radiosensitivity of certain organs in females, notably the breast and the thyroid.

The 1990 evaluation of the risks of low-level radiation by the National Research Council (the "BEIR V report") found that cancer mortality risks for women were, overall, only about 5 percent higher than for men. [47] But that view changed dramatically by 1999, when the EPA published Federal Guidance Report 13, *Cancer Risk Coefficients for Environmental Exposure to Radionuclides*. In that report, the EPA concluded that the cancer mortality risk was 48 percent higher for women than for men.[48] The higher risk to women was affirmed in its 2006 review by the National Research Council's Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation. In contrast to the BEIR V report, which examined only cancer mortality risks, the BEIR VII report, released in 2006, considered cancer incidence as well as mortality.[49] Table 4 summarizes the BEIR VII report estimates for all cancers, including leukemia. It shows that the risk of a fatal cancer to females exceeds that of males by 37.5 percent.

Table 4: Sex-Specific BEIR VII Risks, cancer deaths per million person-rem population exposure

	Males	Females	Ratio female to male
BEIR VII, best estimate	480	660	1.375

Source: NAS/NRC 2006, p. 15.

In contrast, the BEIR V report estimated only a five percent excess mortality risk for women compared with men. A comparison of the numerical mortality estimates is more difficult since the BEIR V report did not report its estimates in a comparable way because it did not have any recommended numerical adjustment factor for low dose radiation delivered at low dose rates (a factor known as the DDREF). The EPA has generally used a figure of 500 fatal cancers per million person rem (one additional cancer per 2000 person rem of population exposure), based on the BEIR V report. This can be compared to the BEIR VII average of 570 fatal cancers per million person rem in the BEIR VII report. The main difference is for women, since the old EPA figure is about the same as that for men in the BEIR VII report.

[46] NAS/NRC 2006 pp. 15 and 278. The difference in the baseline cancer risks for men and women is not strongly affected by the sex-specific cancers. Specifically, the number of cases of prostrate cancer per 100,000 is 15,900 whereas the number of cases of breast, ovarian, and uterine cancer is 16,500.
[47] NAS/NRC 1990
[48] EPA 1999 p. 179
[49] NAS/NRC 2006

The more biologically relevant evaluation of risk is to compare estimates of cancer incidence per unit of radiation exposure rather than estimates of cancer mortality. This makes the differences between men and women even more pronounced. In its 1999 Federal Guidance Report 13, the EPA concluded that for all cancers, including leukemia, women would be 58 percent more likely to develop cancer for the same level of exposure to radiation. The BEIR VII Committee reached a similar conclusion and estimated that women would be 52 percent more likely to develop some form of cancer than men following uniform whole-body exposure to the same level of radiation. In addition, the BEIR Committee's 2006 estimates for cancer risk for women and men were 34 and 38 percent higher than the EPA's estimates made in 1999 (see Figure 5). It should be noted here that in evaluating the risk of specific cancers, the exposure to the specific organ or tissue needs to be taken into account. Here the comparison of sex-related risks is more complex (see below).

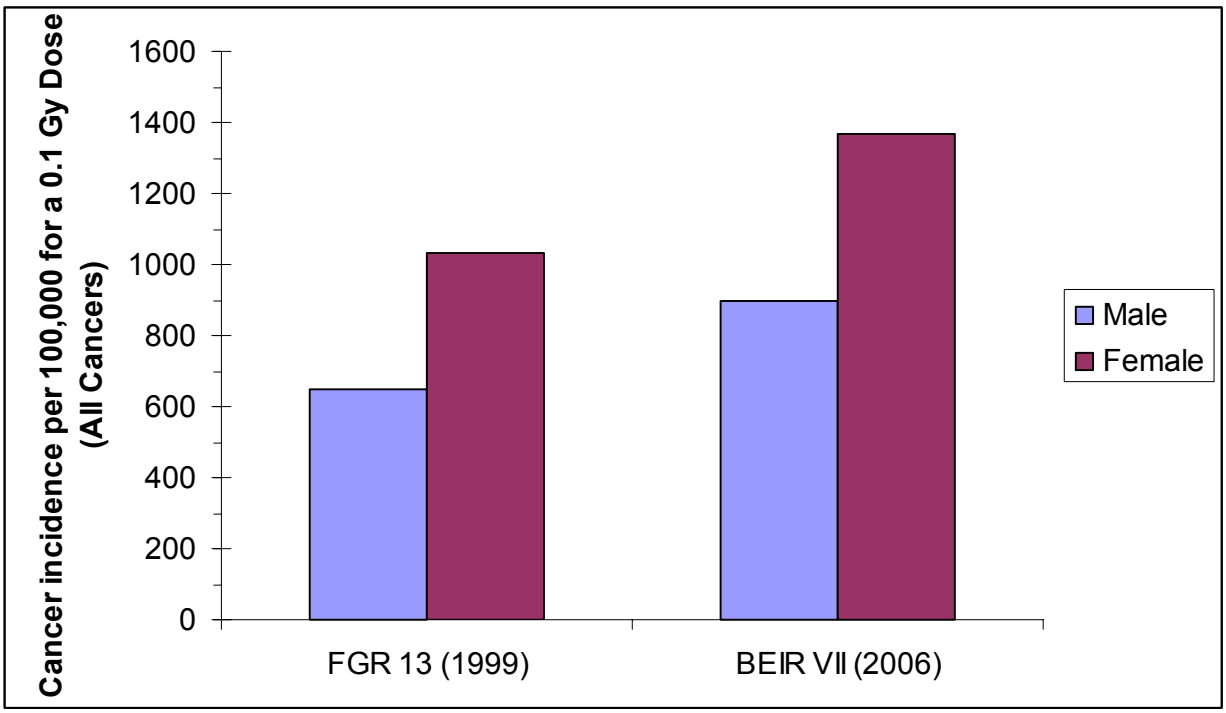

Figure 5: Comparison of the cancer incidence risk per unit of radiation exposure between the EPA's Federal Guidance Report 13 from 1999 and the BEIR VII report from 2006. The BEIR VII report shows an increase in the risk of radiation exposure for both sexes, while both reports agree that women are at a higher risk compared with men. Percentages calculated from NAS/NRC 2006 p. 15 and EPA 1999 p. 182

Although the overall cancer risk for women due to radiation exposure is greater than for men, their relative risks for particular types of cancer varies. For instance, men are now estimated to be at greater risk for colon cancer. This is important to consider for exposures to internally deposited radionuclides that concentrate heavily in one particular organ (such as plutonium which concentrates in the bone or iodine-131 which concentrates in the thyroid). The differences between men and women may be greater or less than the average increase in risk implied by Figure 5 for exposure to these types of radionuclides. Table 5 below compares the estimates from the EPA and the BEIR VII Committee for the relative risk between men and women at the level of specific organs (the risk of leukemia relates to irradiation of the red bone marrow).

Table 5: Sex ratio of cancer incidence risk, women to men, as estimated in two publications - the EPA's Federal Guidance Report 13 from 1999 and the BEIR VII report.[50]

	FGR 13	BEIR VII
Esophagus	2.18	included in residual
Stomach	1.50	1.26
Colon[(a)]	**1.48**	**0.60**
Liver	0.63	0.44
Lung[(a)]	**1.55**	**2.14**
Bone	1.02	included in residual
Skin	1.10	included in residual
Bladder	0.46	0.96
Kidney	0.61	included in residual
Thyroid[(a)]	**2.14**	**4.76**
Residual[(b)]	1.20	0.93
Leukemia	0.73	0.72
Total	**1.58**	**1.52**

Source: EPA 1999 p. 182 and NAS 2006, p. 15, 278-280 (a) These are the organs most responsible for the heightened risk of women compared to men. In FGR 13, the most important single organs were, in descending order, breast, colon, lung, and ovary. In BEIR VII, the most important organs are breast, lung, thyroid, and ovary, while the colon is now estimated to be less radiosensitive for women than for men.
(b) The risk to men of developing breast cancer was assumed to be zero in both reports, but men do have a low rate of breast cancer in the general population and would, as such, be expected to have a small incremental risk of breast cancer from exposure to radiation. Ratios for breast and ovarian cancer are not shown since one is very rare in men and the other does not occur in men.

The most important changes for the estimated risk to women between the EPA's estimates and those of the BEIR VII Committee are the large increases in the risk of developing lung, breast, bladder, and thyroid cancers and the large decrease in the risk of developing colon cancer. For men, the changes between the two estimates of risk were generally smaller than those for women, with significant increases only in the risk of lung and bladder cancer.[51] Among the cancers that commonly affect both men and women, men were estimated by the BEIR VII Committee to be at greater risk for colon cancer, liver cancer, and leukemia, whereas women were at greater risk for stomach and lung cancer and much greater risk for thyroid cancer.

Breast Cancer

Some breast cancer is connected with hormonal system changes. There is also greater risk per unit of exposure if that exposure occurs early in life. As the BEIR VII Committee noted:

> Breast cancer is the most commonly diagnosed cancer and cause of cancer mortality among women in North America and Western Europe... Incidence rates are lower in Asian countries. Ionizing radiation is well documented as a cause of breast cancer in women, *especially when exposures occur in childhood and around puberty.*[52]

There is evidence that radiation may combine multiplicatively with other risk factors for breast cancer. According to the BEIR VII report:

[50] EPA 1999 and NAS/NRC 2006
[51] EPA 1999 p. 182 and NAS/NRC 2006 p. 15, 278-280
[52] NAS/NRC 2006, p. 176 (emphasis added). Worldwide, a million new cases of breast cancer are diagnosed each year. (NAS/NRC 2006 p. 243)

> …the main differences in breast cancer incidence between these two countries [the United States and Japan] are judged to relate to reproductive history and, implicitly, to hormonal factors that would be expected to act as tumor promoters….
> ….
> In a case-control study of breast cancer among A-bomb survivors, Land and colleagues evaluated the interaction of several risk factors for breast cancer with radiation and found that the relationship was better described by a multiplicative model than an additive one.[53]

The above analysis raises the question of whether chemicals that mimic estrogen in the body, like dioxins and PCBs, known as endocrine disruptors or hormonally active compounds, might act in concert with radiation, especially when the exposure to both occurs at sensitive times – that is, in childhood or around puberty. We address these issues in Chapters 4 and 6.

Section 3.2 – Regulatory Aspects

Despite the well-documented differences between men and women with respect to radiation risk, including in its own guidance documents, the EPA continues to use a factor for estimating the cancer risk of radiation that averages the risk to men and women. Therefore, the cancer risk that is estimated from, say, residual radiation, would overestimate the risk to men and underestimate the risk to women for a given level of exposure, according to the EPA's own sex-specific risk factors.[54] Although the use of the average would capture the cumulative risk to a large population, it does not make sense at the level of the individual (who is supposed to be the focus of radiation protection) since, biologically speaking, virtually everyone is either male or female and not half of each. If dose limits were updated to protect women rather than an average of both sexes using the EPA's 1999 estimates of risk, the dose limits would be reduced by about 18 percent relative to today for a given level of risk. If the larger estimates of the risk of cancer incidence from radiation exposure recommended by the BEIR VII Committee were used to update the standards, a further 25 percent reduction in the dose limits would be necessary.[55]

In effect, there is an implicit discrimination against women in the EPA's approach of averaging male and female risks. It is important to remember in this context that while standards are generally expressed in terms of dose limits or maximum contaminant levels, the underlying philosophy from which these limits are derived is one of limiting cancer risk to people. If we accept for the moment that the activity that is causing the risk is socially desirable or necessary and produces benefits to society, then the risk to women (or any other group in society for that matter) should not be greater than what is arrived at through some process of informed consent and democratic decision-making as to the acceptable level of risk.. But if the allowable dose is set to an average risk faced by men and women, the actual risk to women will be greater than the stated maximum acceptable risk, whereas the risk to men will be lower. Hence, the dose limits corresponding to a particular level of cancer risk should be set for females rather than an average of males and females given the significant sex-specific differences in risk.

Of course, this will still mean that men generally have a lower risk than women – this is a biological fact deriving from the nature of radiation exposure that cannot be eliminated except by eliminating human-made radiation exposure. But it will mean that women are not subjected to risk levels that are greater than the maxima specified in the regulatory process.

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[53] NAS/NRC 2006 p. 243
[54] EPA 1999 p. 182
[55] These calculations assume regulation to the same level of risk implied by the current dose limits using the average cancer incidence risk factors.

One consequence of incorporating into the regulatory framework the newer science that shows large sex-specific risk differences would be an increase in both the cost and complexity of the cleanup efforts required following a major radiation disaster such as an accident at a nuclear power plant or successful terrorist attack, since proportionally tighter residual contamination standards would be required. For radiation workers the inclusion of this new science aimed at the protection of women would reduce the annual dose limit from the current 5 rem per year to between 3 and 4 rem per year. This reduction would require changes by the radiation controlled work places but it would not be expected to pose undue obstacles since doses at nuclear installations are already required to be kept as low as reasonably achievable (ALARA). For example, an internal DOE administrative standard from 1999 states that nuclear facilities should be designed to limit worker doses to less than 0.5 rem per year,which is one-tenth of the present annual worker dose limit.[56] Half-a-rem is also the limit that applies to women who voluntarily declare a pregnancy (see below).

It is important to note in this context that the ICRP has recommended that the maximum limit for worker exposure be lowered to 2 rem per year.[57] This recommendation has been ignored in the United States, but adopted in Germany and also, in somewhat modified form, by the European Union.[58] This makes the basic workplace exposure limit of 5 rem in the United States far more lax than in Europe. If the same logic, of doing the male/female averaging, is applied to the 2 rem per year standard, the exposure standard should be tightened to about 1.5 rem per year. Stronger limits are in place in Europe for women who declare their pregnancies (see below).

Section 3.3 – Medical Radiation

Medical radiation procedures are not regulated according to the same criteria as the exposure of the public from environmental contamination due to the operation of nuclear facilities like weapons or power plants or waste repositories. This difference is because medical radiation:

- has the benefit of the individual patient at the center of its purpose,[59]
- the consent of the individual is supposed to be obtained after clear disclosure of the risks by the treating physician,
- the physician is charged with giving the advice and treatment that would best suit the diagnostic or health problem at hand, given available means to address it.

Since the degree of exposure is highly dependent on the health problem at hand, medical exposures cannot be regulated in the same way as involuntary exposures from releases from industrial facilities. Nonetheless, the general criteria regarding the consideration of costs and benefits from radiation exposure and the disclosure of risks still apply. In fact, there is specific ICRP guidance on the topic of medical radiation:

> …unnecessary exposure should be avoided; necessary exposure should be justifiable in terms of the benefits that would not otherwise have been received; and the doses administered should be limited to the minimum amount consistent with the medical benefit to the individual patient.[60]

[56] DOE STD 2004 p. 3-27 and 10 CFR 835.202 2006
[57] ICRP 60 p. 72, para. S25
[58] Verordnung 2001, Section 55 (1) and EURATOM 1996, Article 9 (1)
[59] The issue of human radiation experiments is beyond the scope of this report. IEER has covered this issue elsewhere, including in Makhijani and Kennedy 1994, Makhijani 1994, and Makhijani, Hu, and Yih 2000.
[60] ICRP 26 pp.18-19

Sex-specific questions arise in this context as well. The use of mammography to detect breast cancer affects women only and involves deliberate exposure of the woman to radiation and therefore creates an increased risk of breast cancer. However, at the same time, the use of mammography potentially increases the chances of successful treatment if a cancer already exists since it is capable of detecting the tumor at an early stage of development. For comparison, a typical mammogram involves exposure of 70 millirem; this is about seven times as much radiation as a chest x-ray. [61]

The risks and benefits of the use of mammography constitute a complex topic that is still the subject of considerable study and discussion. There is also a significant issue about the age at which routine mammography is started both because the risk of radiation is age dependent and an earlier commencement involves greater total radiation dose. The medical judgments associated with decisions over when to perform mammography are beyond the scope of this report. Similarly, the use of x-rays in dentistry or for other medical diagnosis must remain decisions for patients and medical practitioners. However, the questions associated with minimizing exposure for a given level of benefit, informed consent, and the implications for research priorities regarding exposures to both chemical toxins and radiation apply to medical radiation exposure as much as to any other activity that has the consent of the participants in the process. Specifically, the requirements of keeping exposure as low as reasonably achievable and of informed consent impose a professional obligation upon health professionals who use x-rays or chemicals in their work to be fully informed about the risks incurred by their patients and the expected benefits. Such discussions are now routine in many parts of medicine. For instance, it is routine for doctors to discuss the risks of surgery or of a colonoscopy with their patients prior to the procedure. That should also become routine for medical radiation, including in the context of chest x-rays, mammography, and dental radiation.

There are Sex implications of this lack of awareness and information because of the exposure of women to regular mammography for early detection of breast cancer. Early detection of the comparably frequent cancer among men, prostrate cancer, is done by analysis of a urine sample and, therefore, does not involve risk to the patient.

Section 3.4--Women, Pregnancy and the Workplace

The maximum allowable limit of exposure for NRC and DOE licensees radiation workers under U.S. regulations is 5 rem per year, with further administrative restrictions and a general working principle to keep exposures as low as reasonably achievable. The routine exposure limit for the general population is 50 times lower -- 100 millirem per year from all human sources of radiation, except medical radiation. Regulations also specify sublimits for the general public of 10 millirem from air emissions of radionuclides, 4 millirem to the most exposed organ from drinking water (for most radionuclides), and 25 millirem per year from nuclear fuel cycle facilities.

Workplace regulations issued by the Nuclear Regulatory Commission do not require a woman to declare that she is pregnant. Measures to limit exposure to the embryo/fetus, by reassigning a pregnant woman to a job that does not involve exposure potential, for instance, are only taken if the woman voluntarily declares her pregnancy, and remain in effect only until she withdraws the declaration. In case of such a disclosure, the maximum exposure to the embryo/fetus is 500 millirem for the duration of the pregnancy. If it has been exceeded at the time of the declaration, the additional exposure allowed for the rest of the pregnancy is 50 millirem.[62] The dose includes external dose as well as internal radiation dose due to

[61] RadiologyInfo 2006 and RadiologyInfo 2006b. Viewed on 6 September 2006
[62] 10 CFR 20.1208 2006

radionuclides inside the body. The main routes of routine workplace internal exposure are by ingestion and inhalation. The external radiation exposure in a field of uniform radiation in the workplace will, for instance, be the same for the mother and the embryo/fetus. In effect, the exposure of the woman is also limited. An evaluation of the dose to the embryo/fetus is done by review of the woman's radiation records (such as dosimeter readings) at the time of pregnancy declaration and at the time of withdrawal of the declaration.

A pregnant woman is normally assigned to non-radiological work areas or job types while her declaration of pregnancy is in effect. However, she may elect to waive her right to be assigned such work and continue to work in radiological areas with the exposure limited to 500 millirem during the pregnancy.

In principle, the most of the framework for workplace protection of pregnant women as well as the embryo/fetus is already in place in the sense that (i) reduced exposure to the embryo/fetus is accepted as an important part of workplace radiation protection and (ii) the disclosure of pregnancy is voluntary, which protects the rights of women in the workplace. Further it is the practice to have zero exposure after the pregnancy declaration as a goal, unless the woman waives her right to be protected from all further exposure or until the woman withdraws the pregnancy declaration. A formal withdrawal of the pregnancy declaration is accompanied by an assessment of the dose to the embryo/fetus. This allows a check on whether the aim of reducing exposure and hence risk to the embryo/fetus at the time of the pregnancy declaration has in fact been achieved.

There is however a gap in this framework in the sense that an embryo/fetus might accumulate considerable exposure before the pregnancy declaration. In practice, this gap may be as long as two months; generally it will not be less than three or four weeks after fertilization. Since certain effects, such as early miscarriages and malformations are important in the first few weeks, this gap needs to be addressed by policy. Further, the limit of exposure in the workplace during pregnancy needs to be tightened, also a standard for nursing mothers who are working is needed and guidance when a pregnant woman continues to work in radiological areas needs further refinement. We discuss this issue in Chapters 4 and 9, but note the German standard here for reference.

Germany's limit for workplace exposure of the embryo/fetus after a declaration of pregnancy is 100 millirem, which is five times more stringent than the United States. Further, breast feeding mothers are also protected. Germany has adopted a Euratom directive in its national standards. That directive states:

> Special protection during pregnancy and breastfeeding
>
> 1. As soon as a pregnant woman informs the undertaking; in accordance with national legislation and/or national practice, of her condition, the protection of the child to be born shall be comparable with that provided for members of the public. The conditions for the pregnant woman in the context of her employment shall therefore be such that the equivalent dose to the child to be born will be as low as reasonably achievable and that it will be unlikely that this dose will exceed 1 mSv [100 millirem] during at least the remainder of the pregnancy.
>
> 2. As soon as a nursing woman informs the undertaking of her condition she shall not be employed in work involving a significant risk of bodily radioactive contamination.[63]

[63] EURATOM 1996, Article 10. Similar provisions are made in the German regulations. (Verordnung 2001 Section 55(4))

Chapter 4: Children and the Embryo/Fetus

Section 4.1 – Children

Children, including infants, are often the most vulnerable population when it comes to a variety of environmental threats, and there is a growing recognition around the world that they should be protected as such. With respect to radiation risks, there are three main factors to consider. The first is the potential for some exposure pathways to be important for children that are not as important for adults. For example, children under 15 years of age often consume a greater volume of milk than many adults, and it often makes up a considerably higher percentage of their daily calorie intake.[64] Since radionuclides such as iodine-131 and strontium-90 are known to concentrate in both cow and human breast milk, this pathway is a potential concern for children's health. Another unique exposure pathway for children is direct soil ingestion. In addition to generally larger routine soil ingestion, some children (and also some pregnant women) will occasionally ingest large amounts of soil on purpose in a behavior known as soil pica or geophagia.[65]

The second reason to consider children's health explicitly in the context of radiation protection is that they have a greater risk of developing cancer compared with adults for the same level of radiation exposure. The increased susceptibility is due, in large part, to smaller organ sizes, higher rates of cell division, and longer remaining lifetime over which a cancer can develop. In addition, a child's gastrointestinal tract can absorb more of some radionuclides than can an adult's, particularly in the first few months of post-natal life,[66] and thus children may receive a higher dose from the same level of environmental contamination.

Finally, the third reason to explicitly consider children is that the difference between the risk to males and females becomes significantly more pronounced in early childhood, adding to the importance of sex-specific analyses. The increased susceptibility of girls to radiation exposure is particularly important for some specific types of cancers such as thyroid cancer.

The heightened vulnerability of children to radiation has been well known for some time. In fact, one of the most important turning points in the entire field of pediatric environmental health was the formation of the Committee on Radiation Hazards and Epidemiology of Malformations by the American Academy of Pediatrics. This committee was set up in 1957 as a result of a growing awareness that the impacts of nuclear weapons testing were disproportionately affecting children due to iodine-131 in fallout. The committee was renamed in 1961 to the Committee on Environmental Hazards as its focus broadened, and it was renamed again in 1991 to the Committee on Environmental Health as its focus shifted more to prevention.[67]

In 2003, the American Academy of Pediatrics, an organization representing 60,000 pediatricians, helped to refocus attention on the issue of radiation and children's health by issuing a policy statement on *Radiation Disasters and Children*. This statement, published in the journal *Pediatrics*, summarized the risks to children from radiation as follows:

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[64] EPA 1999 p. 139
[65] See, for instance, NCRP 1999.
[66] ICRP 72
[67] Goldman et al. 2004 p. 1146-1147. As has long been established, the main way in which iodine-131 in fallout gives a radiation dose to the thyroids of children is via deposition on plants eaten by milch animals, and subsequent concentration in milk.

> Children have a number of vulnerabilities that place them at greater risk of harm after radiation exposure. Because they have a relatively greater minute ventilation compared with adults, children are likely to have greater exposure to radioactive gases (e.g., those emitted from a nuclear power plant disaster). Nuclear fallout quickly settles to the ground, resulting in a higher concentration of radioactive material in the space where children most commonly live and breathe. Studies of airborne pollutants are needed to test the long-held belief that the short stature of children brings them into greater contact than adults with fallout as it settles to earth. Radioactive iodine is transmitted to human breast milk, contaminating this valuable source of nutrition to infants. Cow milk, a staple in the diet of most children, can also be quickly contaminated if radioactive material settles onto grazing areas.
>
> *In utero* exposure to radiation also has important clinical effects, depending on the dose and form of the radiation; transmission of radionuclides across the placenta may occur, depending on the agent….
>
> Radiation-induced cancers occur more often in children than in adults exposed to the same dose. Finally, children also have mental health vulnerabilities after any type of disaster, with a greater risk of long-term behavioral disturbances.[68]

Despite the historical connection between radiation and children's health, it was not until the Chernobyl disaster in 1986 that there was a widespread recognition within the radiation protection community concerning the need to accurately determine doses to children from the inhalation and ingestion of radionuclides. Efforts undertaken in the wake of the Chernobyl accident were integrated with those of the International Commission on Radiological Protection and led to the development of age-specific dose conversion factors for ingestion and inhalation.[69] These age-specific dose models, published between 1989 and 1996,[70] have been referenced in the European Union's European Basic Safety Standards, the International Atomic Energy Agency's International Basic Safety Standards, and the U.S. Environmental Protection Agency's Federal Guidance Report Number 13.[71] In addition, although the Nuclear Regulatory Commission continues to officially use the old "Reference Man" dose models from 1980, a licensee may apply to the Commission for an "exemption" that will allow them to use the more recent ICRP models that take children into account.[72] Further, the compensation law passed for nuclear weapons workers in December 2000 mandates the use of the latest science, which in practice has generally meant the most recent ICRP publications that are relevant to the problem at hand.[73]

To illustrate the importance of considering doses to children, we consider two specific examples. In them, we use the age-specific dose conversion factors and consumption data from the EPA's 1999 FGR 13 and its CD supplement from 2002. With these assumptions we find that an infant drinking milk contaminated with iodine-131 would receive a dose to its thyroid that is 13 times higher than the dose that would be received by an adult drinking from the same contaminated milk supply.[74] This *risk* of thyroid cancer shows an even greater disparity, since infants have a greater risk per unit dose (see below). Because iodine-131 would also pass through to breast milk, the American Academy of Pediatrics has

[68] Pediatrics 2003 p. 1457-1458
[69] NCRP 1998 p. 3 and 9 and, for example, ICRP 72 p. v. The age ranges considered by the ICRP were 0 to 1 years old, 1 to 2 years old, 2 to 7 years old, 7 to 12 years old, 12 to 17 years old, and over 17 years old. (ICRP 72 p. 11)
[70] ICRP 56, ICRP 67, ICRP 69, ICRP 71, and ICRP 72
[71] ICRP 2005 p. A-1 and EPA 1999
[72] NRC 2003 pp. 20-21 and ICRP 30, Part 1 p. 8
[73] The regulations governing dose reconstruction for nuclear weapons workers are at 42 CFR 82.
[74] This calculation assumes that infants (0 to 1 years old) consume 0.34 liters of milk per day compared to 0.22 liters per day for an adult. (EPA 1999 p. 139 and EPA 2002)

recommended that mothers do not breast feed infants following a radiation disaster (unless no alternative is available), even if the mother is taking potassium iodide to limit her own uptake of radioactive iodine.[75]

As a second example, we find that a teenager drinking water contaminated with strontium-90 would receive a bone surface dose that is more than three and a half times the dose received by an adult drinking from the same water.[76] While in some cases the adult dose may be the highest, there are also scenarios in which doses to children may be higher, as illustrated by these examples. Moreover, as we shall see, dose is not always the most relevant measure for assessing health risk differentials per unit of exposure between people of various ages and between men and women. This is the case with both cancer and non-cancer health risks.

Specifically, children have a higher risk of developing cancer from exposure to radiation compared with adults. In fact, the BEIR VII Committee concluded that there is both an increase in the overall risk for children compared with adults as well as a heightened difference between the risks to males and females. For example, the overall risk of developing cancer from radiation exposure as a young child (0 to 5 years) is 2.6 times greater for a boy than the risk for a 25 year old adult male and 3.0 times greater for a girl than the risk for an adult female. For young children, the risk to girls is 86 percent higher than the risk to boys for the same level of exposure. This can be compared to a 52 to 58 percent higher risk for women compared with men when averaged over all ages, as noted in Chapter 3.[77] Figure 6 shows the estimated risk of developing cancer as a function of age at exposure for both males and females. These overall cancer risks are magnified for specific cancers in female children, since they are at greater risk of certain cancers, among the cancers affecting both sexes, notably thyroid cancer (see below).

[75] Pediatrics 2003 p. 1459 and 1463. A peculiar aspect of this problem highlights why it is important to consider both the health of women and infants together. It also reinforces the recommendation of the American Academy of Pediatrics cited here. A woman who is breast feeding and takes potassium iodide (KI) as a thyroid blocking agent following a nuclear accident will not be acting protectively to her infant and may be acting anti-protectively. Her intake of KI means that I-131 that would otherwise have been sequestered in her thyroid can now be potentially excreted in her milk. This effect has been illustrated in cattle, but the issue has apparently not yet been discussed for human beings. (Thorne, Walke, and Beresford 2006)

[76] This calculation assumes that teenagers (12 to 17 years old) consume 0.80 liters of water per day compared to 1.25 liters per day for an adult. (EPA 1999 p. 139 and EPA 2002)

[77] NAS/NRC 2006 pp. 15, 310

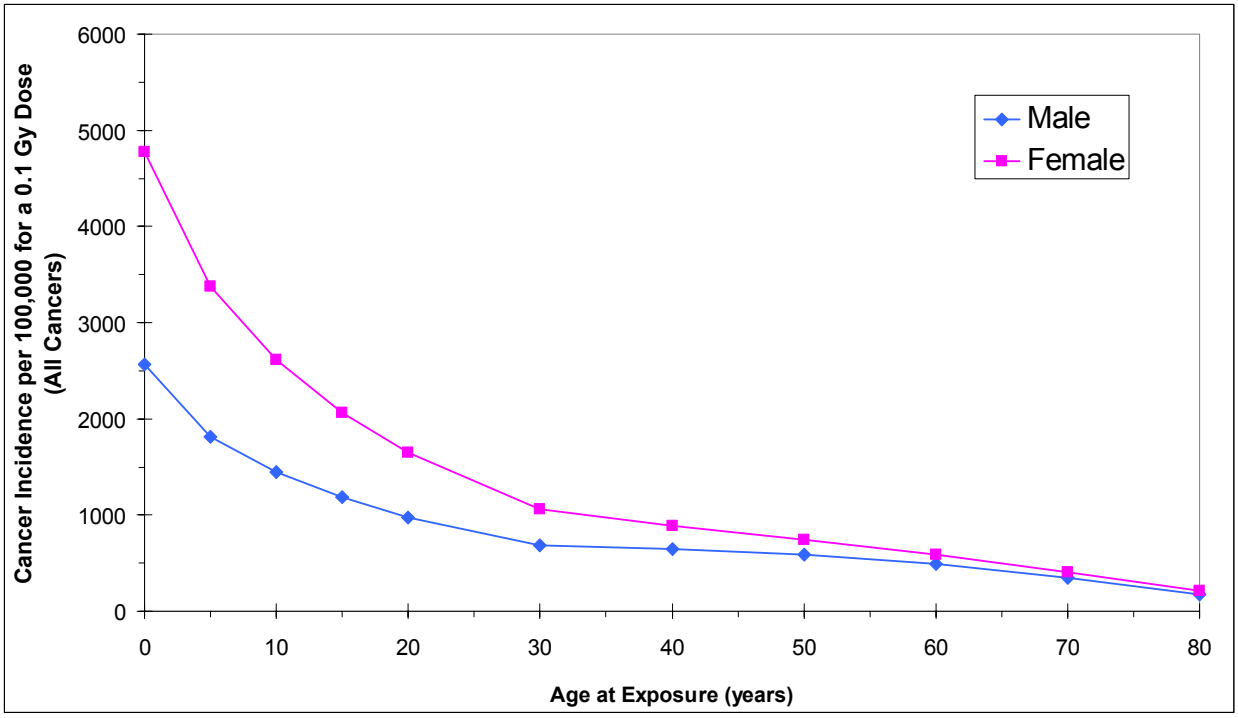

Figure 6: Graph of cancer incidence risk per unit of radiation exposure as a function of age from the BEIR VII Committee. The change in cancer risk for people under the age of 20 is steeper for females than males, resulting in an increase in the difference between their risks. The exposure occurs at the stated age; the risk is over the lifetime remaining after that age.

The BEIR VII Committee assessed the cancer risk expressed as cancer occurrences per million person rem of population exposure, by sex, age, and some cancer types. Table 6 shows some of the results.

Table 6: Cancers per million person-rem of exposure, by age at exposure and sex for some cancer types and all cancers

	Infant		Age 5 Yrs		Age 30 Yrs		Ratio infant:30 yrs	Ratio infant:30 yrs
	Male	Female	Male	Female	Male	Female	Male	Female
Colon	336	220	285	187	125	82	2.69	2.68
Lung	314	733	261	608	105	242	2.99	3.03
Breast	N/A	1171	N/A	914	N/A	253	N/A	4.63
Thyroid	115	634	76	419	9	41	12.78	15.46
leukemia	237	185	149	112	84	63	2.82	2.94
All solid	2326	4592	1667	3265	602	1002	3.86	4.58
All cancers	2563	4777	1816	3377	686	1065	3.74	4.49

Source: NAS/NRC 2006 p. 311.

The pattern of higher cancer risk for most cancers for females is evident at all ages. Further, it is clear that the risks to infants and children per unit of exposure are far greater than they are to adults. In the context of radiation exposure being an initiator of cancer, this needs to be taken into account in setting radiation standards.

We discuss examples of non-cancer risks in the chapters on tritium and depleted uranium as illustrations, since that topic is far less developed.

The BEIR VII risk factors are derived mainly from Hiroshima and Nagasaki survivors, who suffered external doses of radiation (gamma and neutron). Children can face even higher risks when radionuclides are inhaled or ingested, for instance, if it is a radionuclide like tritium that can replace a hydrogen atom in the DNA. Table 7 shows values of radiation dose per unit intake (called a "dose conversion factor") for three organs, based on recent data published by the EPA. We note that in many contexts of environmental exposure intakes of radionuclides by infants and children are less than those of adults, for instance, due to lower intake of food and water. We present the overall risks after discussion of these preliminaries.[78]

Table 7: Dose Conversion Factors for four radionuclides at various ages for three organs

	Dose Conversion Factors for ingestion, Sieverts per Bq, and ratios, Red Marrow				
	Infant	5 Years	Adult	Infant/Adult	5 yrs/adult
Tritium (hydrogen-3)	6.28E-11	3.04E-11	1.82E-11	3.45	1.67
Strontium-90	1.51E-06	2.73E-07	1.79E-07	8.44	1.53
Iodine-131	5.14E-10	2.22E-10	1.01E-10	5.09	2.20
Plutonium-239	1.09E-05	5.87E-07	3.91E-07	27.88	1.50
	Dose Conversion Factors for ingestion, Sieverts per Bq, and ratios, Breast				
	Infant	5 Years	Adult	Infant/Adult	5 yrs/adult
Tritium (hydrogen-3)	6.28E-11	3.04E-11	1.82E-11	3.45	1.67
Strontium-90	1.18E-08	2.85E-09	6.64E-10	17.77	4.29
Iodine-131	5.69E-10	2.32E-10	5.88E-11	9.68	3.95
Plutonium-239	5.50E-07	3.19E-08	1.45E-08	37.93	2.20
	Dose Conversion Factors for ingestion, Sieverts per Bq, and ratios, Thyroid				
	Infant	5 Years	Adult	Infant/Adult	5 yrs/adult
Tritium (hydrogen-3)	6.28E-11	3.04E-11	1.82E-11	3.45	1.67
Strontium-90	1.18E-08	2.85E-09	6.64E-10	17.77	4.29
Iodine-131	3.66E-06	2.06E-06	4.32E-07	8.47	4.77
Plutonium-239	5.50E-07	3.19E-08	1.45E-08	37.93	2.20

Source: EPA 2002. The dose conversion factors represent "committed doses" – that is, doses over the fifty year period following an intake. For most radionuclides, when ingested, the radiation dose is delivered within weeks or months of the intake. However, in some cases, such as inhalation of insoluble plutonium, which stays in the body for decades, the dose is also delivered over decades.

Note that the dose conversion factors for the thyroid are much larger for iodine-131 than any of the other radionuclides in the table. Iodine radioisotopes are, for practical purposes, the main ones of concern for thyroid doses, despite large differences by age in dose conversion factors for other radionuclides. Further, dose conversion factors are only one aspect of the computation overall cancer risk, which may be higher or lower than the ratio indicated by that one factor alone. The fact the infants and children have lower intakes offsets the higher dose conversion factor. This may lead to a higher or lower dose for children compared to adults, for the same level of contamination of food and water. However, infants and children have a higher risk per unit dose to an organ, which offsets the lower intake of food and water or the lower rate of air intake while breathing. When these factors are combined, the risk of cancer due to internal intakes of radioactivity faced by infants and children are generally considerably higher than those faced by adults for the same level of environmental contamination.

[78] See, for example, Thorne 2006 - Issue 1

Table 8 shows an overall comparison of cancer risk for females by age that takes all three factors into account – dose per unit intake, incidence per unit dose, and variation in intake by age for three types of cancer (breast, thyroid, and leukemia, with the bone marrow dose being calculated to estimate the risk for leukemia). The radionuclide considered for leukemia and breast cancer is strontium-90, whereas that considered for thyroid is iodine-131, as iodine preferentially concentrates in the thyroid.

Table 8: Lifetime cancer incidence risk for exposure at stated age, fluid ingestion, females only (fluid intakes adjusted for age)

Cancer	Radionuclide	Infant, Risk/Bq	Age 5, Risk/Bq	Age 30, Risk/Bq	Overall Risk Ratio Infant/Age 30 (Note 1)	Overall risk Ratio 5 Yr/Age 30 (Note 1)
Leukemia	Sr-90	2.79E-08	3.06E-09	1.13E-09	6.2	1.7
Breast	Sr-90	1.38E-09	2.60E-10	1.68E-11	20.6	10.0
Thyroid	I-131	2.32E-07	8.63E-08	1.77E-09	32.8	12.2

Source: Derived from Tables 6 and 7. Bq = becquerel
Note 1: Fluid intakes for females assumed as follows: infant = 350 cc per day, 5-year old = 900 cc/day; 30-year-old = 1,400 cc/day. The fluid intake figures for children were computed at 50 cc/day/kilogram of weight, using mean weights for the United States. These are total fluid intakes, including, but not limited to, water.

Table 8 shows that for *the same level of environmental contamination* the risk for female infants is much higher for all three cancers, but especially so for breast and thyroid cancer.[79] The ratio of children's risk to adult risk decreases as the child grows and may rapidly approach that for adults for some cancers (like leukemia); for others large risk differentials persist for many years. Five year old girls have 10 times the risk of breast cancer relative to thirty year old women, for the same level of strontium-90 in the water.

The risk differentials between female children and male adults are even larger for some cancers that affect people of both sexes. This is particularly so for thyroid cancer. The risk for small female children, infants to 5 year olds, is about *100 times greater than that of a 30-year-old male, drinking the same contaminated milk.*

The ICRP has summed up the relative effect of radiation on children as follows:

> It is well known that the cancer risk is very high after small children are exposed to radiation, and the patterns of cancers are different from those of adults. Questions arise regarding whether this high radiosensitivity also exists for radiation exposures during prenatal development, and whether some embryonic/fetal tissues or organ systems are more radiosensitive than others.[80]

These risk calculations, based on recently published cancer incidence risk data, connect very squarely with the question of standards for radiation protection. For instance, the cumulative breast or thyroid cancer risk accumulated over the first five years of exposure via ingestion by female children exceeds that accumulated by females over their entire adult lifetimes, assuming the same level of environmental contamination.

[79] The ratios for males for various cancers are different but the overall conclusions are broadly similar.
[80] ICRP 90 p. 9

Section 4.2 – The Embryo/Fetus

In addition to the greater sensitivity of children, it is also known that exposures *in utero* can lead to a heightened risk of leukemia and other cancers. These risks accompany external exposures, radionuclides that are in the mother's bloodstream, and exposures to radionuclides that cross the placenta. A variety of ill-health effects can occur, depending on the type and level of radiation and the stage of embryonic or fetal development at which the exposure occurs. The ICRP has published three recent major reports on this topic, one assessing doses to the embryo/fetus due to radionuclide intake (ICRP 88), one discussing health effects of radiation on the embryo/fetus (ICRP 90), and one on medical radiation during pregnancy (ICRP 84).[81]

Consideration of the health effects of radiation during pregnancy, however, is some decades older than recent work. The first scientific work to demonstrate an association between *in utero* x-ray exposures and leukemia that develops later in childhood was published in 1958 by Alice Stewart and others in the United Kingdom. This association was affirmed by a study of the U.S. population published in 1962.[82] Consistent with the findings from exposures during childhood, animal studies have found that irradiation of the fetus results in a higher risk of cancer for females than for males. [83]

It has been demonstrated that prenatal irradiation in animals can interact synergistically with chemical carcinogens, increasing the risk beyond the additive effect of the two exposures in isolation.[84] This last observation may be a particular concern for human health given the large number of chemicals that are known to cross the placental barrier and enter the embryo/fetus.[85] Chapter 6 provides a further discussion of combined exposures to both chemicals and radiation.

The effects of radiation on the embryo/fetus can be considered in the following stages:

1. The pre-implantation stage, which lasts for about the first two weeks after conception (during which the woman is unlikely to know she is pregnant),
2. The period of major organ formation, when the cells of the embryo become differentiated into the various organs and systems of the body and the fetus is formed (2 to 14 weeks),
3. The fetal development period, when development and growth of the organs occurs.

The last period of fetal organ development and growth might be considered in two parts – up to the start of the third trimester and thereafter. In the latter period, the effects of radiation might generally be considered similar to those experienced in the neonatal period by infants.

For irradiation that occurs in the later stages of fetal development, the ICRP concluded:

> It is very well known that ionising radiation interferes to a high degree with cell proliferation. Therefore, biological systems with a high fraction of proliferating cells show high radiation responsiveness. High rates of cell proliferation are found throughout prenatal development. However, although cell proliferation is a key process for the development of radiation effects, the sensitivity of the embryo and fetus is also determined through processes of differentiation and cell migration, and the radiation effects on these biological processes…... Development of the central nervous system starts during the first weeks of embryonic development and continues through the early postnatal period. Thus development of the central nervous system occurs over a very long

[81] ICRP 88 ICRP 90, and ICRP 84
[82] NAS/NRC 2006 p. 172, citing Stewart, Webb, and Hewitt 1958 and MacMahon 1962.
[83] ICRP 90 pp. 8, 128-129, and 138
[84] ICRP 90 pp. 134-135 and 139
[85] See, for example, Houlihan et al. 2005

> period, during which it is especially vulnerable. It has been found that the development of this system is very frequently disturbed by ionising radiation, so special emphasis has to be given to these biological processes.
> …
> … There is a clear constellation of effects of prenatal irradiation on the developing central nervous system – mental retardation, decreased intelligence scores and school performance, and seizure disorders. The first three factors showed strong associations with prenatal radiation exposure, while the association for seizure disorders was weaker, perhaps owing to the sparseness and unreliability of the seizure data….
> …
> … Tissues such as brain, thyroid, bone, and breast appear to be more susceptible if exposed during normal periods of rapid growth (i.e. early childhood or puberty).[86]

Growth occurs by mitosis, or cell division, of the various specialized cells that make up the organs of the human body, and the organs of children are still growing and developing. Damage to a single cell at an early age can, therefore, result in a far larger number of damaged cells in an adult that can later become cancerous. The problem is true in the case of exposure to radioactive isotopes of iodine, which concentrate in the thyroid for a variety of reasons, especially for female children, but it is also true for other radionuclides.

The Early Period: Up to 14 Weeks

The type and magnitude of risks to the embryo/fetus are much less understood for the early period of embryonic development, up to about the time that the major organs are formed, though still immature and very small, that is, up to about 14 weeks after conception.

There are no human data on which to base conclusions about the health damage from radiation in the early period. As the ICRP has noted:

> ...during the pre-implantation period, no observations in humans are available, as conception is not noticed at that time. Therefore, the risk analysis can only be achieved on the basis of animal experiments which have mainly been performed with mice and rats.[87]

The usual problems associated with transferring risk data by extrapolating from mice to human beings are particularly acute in this area, since there are large differences in response even between mouse types.[88] However, experiments with mice do indicate general features of the kinds of problems to be expected in various development periods.

In the first two weeks, the main result of irradiation is an early failed pregnancy. This occurs in two ways. In the early part of this period, the failure usually occurs through resorption of the fertilized ovum in the uterine wall. It may also occur by explusion of the embryo with some bleeding. In this early period, a woman may not recognize a failed pregnancy; the early expulsion would likely be mistaken for a period because the woman generally would not yet know that she is pregnant.[89] It would be very difficult to detect any early failed pregnancies caused by caused by radiation even if women who intend to become

[86] ICRP 90 pp. 9, 118, and 149
[87] ICRP 90 p. 88
[88] See ICRP 90 p. 27, for instance.
[89] ICRP 90 Chapters 2 and 3. Very early miscarriages of the embryo are technically known as “chemical pregnancies.” Since term seems to describe a pregnancy rather than a failed pregnancy; we use the term “early failed pregnancy” here for clarity, to distinguish these very early miscarriages of the embryo when a woman may not even realize she is pregnant from other, later, miscarriages.

pregnant were closely followed, due to the very high rate of natural, or presumably natural, miscarriages in the first two weeks from a variety of causes:

> Miscarriage is the most common type of pregnancy loss, according to the American College of Obstetricians and Gynecologists (ACOG). Studies reveal that anywhere from 10- 25% of all clinically recognized pregnancies will end in miscarriage. Estimations of chemical pregnancies or *unrecognized* pregnancies that are lost can be as high as 50-75%, but many of these are unknown since they often happen before a woman has missed a period or is aware she is pregnant.[90]

As the above quote notes, early miscarriages or spontaneous abortions in recognized pregnancies also occur at a fairly high rate. The causes of the very early miscarriages that occur in the first two weeks are not yet well understood, but they appear to include genetic, dietary, and environmental factors, including exposure to radiation and toxic chemicals. For instance, smoking, active or passive, contributes to miscarriages, including early miscarriages. [91]

The research cited (ICRP 90) definitively indicates that at least above certain levels of radiation, early miscarriage is the result of exposure. Whether there is a threshold or whether some women may be far more susceptible than others, given the large variation in the response of mice as well as the variation in fertility among groups and individuals, it is not possible at present to say. But based on present knowledge it appears that that early failed pregnancy or early miscarriage are more likely in the first few weeks, while malformations may dominate the response to exposure in the weeks after that.

There are some human data on a variety of non-cancer risks that are greater for exposure *in utero* and exposure at an early age. Like cancer, non-cancer effects in humans have been studied largely by the follow-up of the Hiroshima/Nagasaki survivors. However, this follow-up has left significant and critical gaps in the quantitative understanding of non-cancer risks. The following are among the important considerations:

- The study of the survivors began in 1950,[92] leading to a critical gap of five years, in which there may have been excess deaths of children due to a variety of factors, such as infections due to immune systems compromised by radiation exposure of infants or radiation exposure *in utero*.
- The radiation exposure from the atomic bombs was essentially all external exposure due to gamma rays and neutrons. The Hiroshima/Nagasaki data provide no information about the effects of radionuclides that cross the placenta. These radionuclides can affect the development of specific organs in ways that are much more significant than external radiation. For instance, fetal exposure to iodine-131 can adversely affect the thyroid and then harm further development in a number of ways, including the post-partum development of the infant. Exposure of a few cells to tritium at the stage when there are only a few stem cells, e.g., at the blastocyst or early implantation stage, could cause a failed pregnancy in the first two weeks or an early miscarriage. As noted above, such miscarriages would be extremely difficult to detect, much less study epidemiologically.

Section 4.3 – The Regulatory Framework

The argument is often made that there is natural radiation and men, women whether they are pregnant or not, infants, children, and the embryo/fetus are exposed to it. According to this view, levels of radiation

[90] APA 2006
[91] Landrigan 2004 and APA 2006, citing Venners et al. 2004
[92] This was called the Life Span Study. (NAS/NRC 2006 pp. 12-13)

exposure that are smaller that natural background can be considered as having minimal effects or can even be ignored. This premise of often implicit, but nonetheless clear, in the many appeals that are made to levels of natural radiation by the nuclear industry and even regulators.[93]

To date, children and the embryo/fetus have been given specific consideration in the regulatory framework only in relation to medical and occupational exposures.[94] In 1954, the U.S. National Council on Radiation Protection and Measurements (NCRP) recommended that the dose limit for the embryo/fetus should be the same as that for the general public. However, at that time there were no generally accepted models available for determining the transfer of radionuclides across the placenta and thus no accepted way to determine the dose to the embryo/fetus from internally deposited radionuclides.[95]

Moreover, at that time there was no separate exposure limit for the general public. The limit for workers also applied to the public. A separate limit for the general public, ten times less than that applicable to workers, was created in the late 1950s. At the present time, the limit for the general public is 100 millirem per year (except medical radiation); that for workers is fifty times greater, at 5,000 millirem per year.

Consistent with the general trend in radiation protection standards, the allowable dose to the embryo/fetus of a pregnant worker decreased over time as more was learned about the dangers of radiation. For example, in 1990, the ICRP recommended that the external dose to the abdomen of pregnant workers be limited to 200 millirem for the duration of the pregnancy and that the woman's ingestion of radionuclides be limited to 1/20th of the worker limits. In addition they recommended that, during pregnancy, the woman's job "should be of a type that does not carry a significant probability of high accidental doses and intakes."[96]

The ICRP's guidance on ingestion, however, was still based on consideration of the dose to the woman only, given that no dose conversion factors for the embryo/fetus had been adopted. In 1992, the Nuclear Regulatory Commission published Regulatory Guide 8.36, *Radiation Dose to the Embryo/Fetus*, which set forth its recommendations for how to calculate internal doses.[97] These models were used to support the NRC and DOE regulations governing occupational exposures to pregnant women. In contrast to the ICRP, which recommended separate limits for external and internal exposures, the U.S. regulations limit the total dose to the embryo/fetus from both external and internal sources to 500 millirem over the duration of the pregnancy (see Chapter 3). While this limit was consistent with the recommendations of the NCRP set forth in the 1990s, it is five times the annual dose limit for members of the general public. Surprisingly, despite having the same limit for the embryo/fetus as the NRC, the DOE regulation governing occupational exposure of minors has a dose limit of 100 millirem per year, which is five times lower than the NRC limit for children in the workplace.[98]

As noted, in Chapter 3, the U.S. limit for dose to the embryo/fetus is also higher than that in the European directive. This seems consistent, in a rather unfortunate way, with the failure of the United States to

[93] This appeal to "natural" radiation was carried to a rather extreme level by the EPA in the standards it proposed in 2005 for the Yucca Mountain repository for spent fuel and high level nuclear waste. See Makhijani and Smith 2005b.
[94] For a discussion of the ICRP recommendations on medical exposure of pregnant women see ICRP 60 p. 43.
[95] NCRP 1998 p. iii
[96] ICRP 60 pp. 41-43 and 73. The limits of intake are set forth in national regulations covering workers. In the case of the United States, they are at 10 CFR 20.
[97] NCRP 1998 p. 9 and NRC 1992
[98] 10 CFR 20.1207, 20.1208, and 20.1301 2006; 10 CFR 835.206, 835.207, and 835.208 2006; and NCRP 1998 p. iii. Note that NCRP 1998 (p. iii) gives the limit "for the exposure of the embryo/fetus is 0.5 mSv *per month* once the pregnancy is known." This amounts to 50 millirem per month.

adopt the stronger 2 rem per year workplace exposure standard for radiation workers, even though other countries, such as Germany, have adopted it.

Given the heightened susceptibility of children and the embryo/fetus to cancer and other radiation injuries, there is a growing awareness that they should be explicitly considered in such areas as planning for radiation disasters and in determining cleanup standards and not just in regulations governing occupational and medical exposures. For example, the American Academy of Pediatrics has recommended that "[p]ediatricians should be included in all aspects of planning for a radiation disaster."[99] In addition, the International Commission on Radiological Protection has circulated a draft in which it has recommended "the use of all available age-specific dose coefficients in the planning for and response to accidents."[100] Finally, in the context of performing dose assessments, the ICRP draft has also recommended that:

> …if assessed doses to the other age groups include significant contributions from radionuclides known to give rise to relatively high doses to the foetus, and they are approaching the value of the dose criterion, the dose to the foetus or breast-fed infant should be separately assessed to assure that the quantitative recommendations are respected.[101]

As with the differences between men and women, the differences between adults and children should be carefully considered by the regulatory community to ensure that the most vulnerable populations are, in fact, adequately protected. We will discuss specific recommendations in Chapter 9.

[99] Pediatrics 2003 p. 1463
[100] ICRP 2005 pp. 17 and A-1.
[101] ICRP 2005 p. 16. In citing the draft, we recognize, of course, that it may change its recommendations upon finalizing the report. This recommendation is consonant with the analysis in this report.

Chapter 5: Men

The analysis in the foregoing sections regarding the greater sensitivity of women and children shows that the use of "Reference Man" is not an appropriate way to design radiation protection standards for society. If women and children are protected and if adequate attention is given to limiting fetal exposure during pregnancy, men will be at far lower risk than they are today in most respects, including the risk of developing cancer, since the lower relative risk for men is a biological matter.

Section 5.1—Exposures of Prospective Fathers

However, regulators have not yet taken into account the fact that nature has given a role to men in reproduction, even if it is rather modest by comparison with that of the role of women. The issue of whether the exposure of men to radiation might increase risks to offspring is an important one to consider if the protection of children is a central part of the overall goal. Tightening standards for population exposure with the most sensitive population as the reference would also create greater protection for men in their capacity as prospective fathers.

There is some evidence that the progeny of men who were exposed to radiation around the time of conception have an increased risk of cancer. However, this is an area where research is extraordinarily difficult and the results, especially for exposure at low doses, are uncertain and to a large extent unclear from the point of view of causation. The problems of showing statistical significance when there are small numbers of cancers, when there are exposures to multiple risk-inducing agents, and when there are difficulties assigning the correct radiation dose actually experienced by the sperm and to the spermatocytes (which are the stem cells of the sperm) all pose special challenges by themselves and in combination.

Among the best known studies indicating an increased risk of leukemia among offspring of fathers exposed to radiation is the study done by M. J. Gardner *et al*. in the vicinity of the Sellafield nuclear installation in northwestern England. The study found an increased incidence of leukemia that

> was associated with paternal employment and recorded external dose of whole body penetrating radiation during work at the plant before conception. The association can explain statistically the observed geographical excess. This result suggests an effect of ionising radiation on fathers that may be leukaemogenic in their offspring, though other, less likely, explanations are possible.[102]

However, this study included only external radiation, even though the Sellafield installation also has the potential for internal exposure. Another review of the situation attributed the problem to the small numbers of cases involved. In this review, the authors concluded that:

> Cancer in young people is rare, and our results are based on small numbers of events. Overall, the findings suggest that the incidence of cancer and leukaemia among children of nuclear industry employees is similar to that in the general population. The possibility that exposure of fathers to relatively high doses of ionising radiation before their child's conception might be related to an increased risk of leukaemia in their offspring could not be disproved, but this result was based on only three cases, two of which have been previously reported.[103]

Another assessment of the risks of leukemia and paternal exposure to radiation was carried out by Sever et al. under contract to the U.S. Department of Energy for people around three U.S. nuclear weapons plants – Hanford in Washington State, Idaho National Engineering Laboratory, and the K-25, Y-12, and

[102] Gardner et al.1990 p. 423
[103] Roman et al. 1999 p. 1443

X-10 facilities at Oak Ridge, Tennessee.[104] This study found that the data were "consistent with a null hypothesis of no association between paternal preconception exposure and risk of these forms of childhood cancer [leukemia, non-Hodgkins lymphoma and all cancers combined]."[105]

One major limitation of the Sever et al. study was that it included only external radiation exposure in its statistical evaluation because the researchers concluded that radiation "doses are primarily external with limited potential for internal exposure."[106] This conclusion appears too broad and premature. In fact, Sever *et al.* did not attempt to estimate internal doses, other than to gather data for tritium "[w]hen available."[107] .Many conditions at Hanford, for instance, were conducive to significant internal exposure to plutonium, tritium, uranium (including recycled uranium with transuranic trace contaminants) and fission products.[108] Just because the researchers failed to find many records relating to internal exposure cannot lead to the conclusion that such exposure did not occur. In fact, data on internal exposure in the early years tend to be rather sparse at many nuclear weapons facilities, not because the potential did not exist but because the workers were not monitored. This was especially the case for some radionuclides like thorium-232. For instance, at the Y-12 plant, one of the facilities in the Sever *et al.* study, the U.S. government has added workers exposed to or potentially exposed to thorium-232 and several other radionuclides to the Special Exposure Cohort because the data for dose reconstruction do not exist or are too sparse. [109]

In addition, it is known that uranium inside the body can concentrate in the testes making it a potential concern both in terms of radiation and in terms of its chemical toxicity (see Chapter 8). The Sever *et al.* study also did not consider exposure to non-radioactive hazardous chemicals that might affect cancer risk in children. Similarly, Sellafield also has potential for internal exposure, but both the studies cited above included only external exposure in their analyses and neither study considered chemical exposures.

The exclusion of internal exposure is a central problem in such analyses because it prevents the researchers from correctly classifying the study subjects according to their actual radiation exposures. The inability to accurately group the workers by the dose they received can result in a broad failure of the study in arriving at a scientifically sound conclusion and also can provide erroneous results in regard to dose response – that is, the risk per unit of dose and its variation over increasing levels of exposure.

Internal exposure can be very important at some facilities and may, in some cases, be a principal or even the dominant contributor to the dose. For instance, exposure of prospective fathers to tritium in the form of tritiated water could very easily be an important factor in adverse health outcomes. As discussed in Chapter 7, tritium is radioactive hydrogen, and, therefore, tritiated water behaves chemically like ordinary water in the body. Thus, when tritium is ingested, a small portion of it can be expected to become part of the process of spermatogenesis, possibly leading to increases in adverse health outcomes, including cancer in offspring of exposed persons. The relative doses are higher when organically bound tritium is ingested. In sum, the mix of internal and external dose is highly variable due to a number of factors, and external dose cannot be regarded as a surrogate for internal dose as a general matter. Further, since the Sever *et al.* report did not attempt to be comprehensive even about tritium data, its findings remain inconclusive in regard to this aspect of internal dose.

[104] Sever et al. 1997 p. 9
[105] Sever et al. 1997 p. 30
[106] Sever et al. 1997 p. 20
[107] Sever et al. 1997 p. 18
[108] NIOSH 2004 and SCA 2005
[109] NIOSH 2006

A second problem with the existing studies is that the external exposure data are generally readings from film badges or other dosimeters worn at the level of the shirt pocket. The dose to the gonads, which is the most crucial piece of data for assessing risk of damage to sperm and hence adverse health outcomes from external exposures, will depend on the location of the source of the exposure relative to the gonads. In some cases, external exposure fields may be relatively uniform and the badge reading may represent gonadal (and hence sperm) exposure reasonably well. In other cases, the badge data may have very limited utility in accurately estimating exposure; gonadal exposure may be much lower or much higher than that recorded on the badge.

An example of the latter can be clearly observed in a photograph of a worker at the Fernald uranium processing plant in Ohio, taken by Robert del Tredici in 1987 (Figure 7). The gamma radiation dose to the gonads of the Fernald worker was undoubtedly far greater than the dose recorded on the badge dangling from his pocket or even on the wrist dosimeters for more than one reason. First, the main badge worn at the pocket is far from the gonads and the source (the cylindrical ingot or uranium over which the worker is straddled), whereas the latter two are in very close proximity. Since radiation fields vary roughly as the square of the distance (exactly, in the case of a point source), the recorded dose on the badge would be far lower than the gonadal dose, despite the modest shielding provided by the worker's clothes. The same may or may not be true of the wrist dosimeter. Second, badges are calibrated with the radiation incident on the badge at a right angle. Since the worker's badge is dangling from his pocket, almost parallel to the dominant direction of the photons from the uranium ingot, the recorded dose would be a serious underestimate even as an estimate of the dose to the workers chest. Third, the main dosimeter is shielded from much of the source of radiation by the body of the worker, since the ingot is mainly under and behind the worker, but the dosimeter is in front.

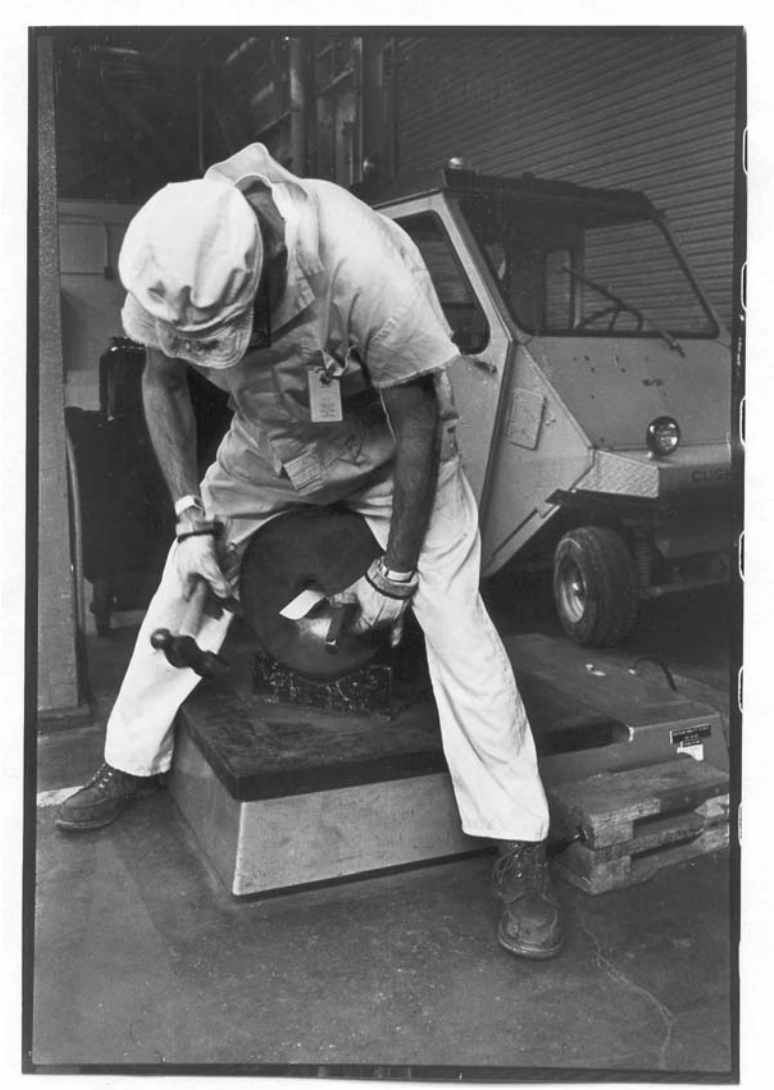

Figure 7: Worker at the Feed Materials Production Center (Fernald) stamping an ID number on a uranium ingot. His film badge is dangling from this pocket vertically. He also has wrist dosimeters. Photo by Robert del Tredici, 1987. Used with permission.

Epidemiologic studies have traditionally not paid attention to such complicating factors that affect the interpretation of recorded external dose. The film badge dose of record is used as the dose of record even when effects on a particular organ are being considered, as was the case in the Sever *et al* study. However, these effects can be critical to the results of the studies, especially when the relevant dose estimate is an organ dose (as it is in the case of exposure of the testes) rather than a whole body dose. Finally, it should be noted that even the external dose data taken into account in the S*ever et al*. study were not comprehensive. Specifically, only deep gamma dose and neutron dose data were collected. However, uranium processing workers at facilities like Y-12 and K-25 (which were part of the study) would be expected to have far higher beta doses than deep gamma doses. While this is unimportant for most organs, the high energy beta radiation (2.19 MeV) could affect the testes.

Section 5.2—Gulf War Veterans

There have also been studies of adverse reproductive outcomes involving U.S. and British Gulf War veterans. These studies have compared war veterans with veterans who were not deployed to the war zone in 1991 and, in some cases, to the general population. An unknown number of the armed forces personnel deployed into the war zone experienced exposure to depleted uranium (DU) dust created by burning munitions and also some external radiation exposure due to the handling of depleted uranium or DU contaminated equipment. However, large numbers of these personnel were also exposed to vaccines, smoke from burning oil fields, and traces of chemical weapons.

In the broad studies that involve examination of health outcomes for large numbers of the children of veterans, the comparison is between those deployed and similar populations. Since there is little or no dosimetric information for those who were deployed to the war zone, this makes any conclusion of a lack of an effect rather problematic, since exposure is likely to have varied a great deal among deployed personnel. Low exposure levels in a significant part of the deployed personnel would dilute the exposed group and render the findings of an association less statistically significant or could wipe out the association altogether.[110]

Statistically significant results are unlikely to be produced when the number of adverse health outcomes is small and there is considerable uncertainty about gonadal dose. However, epidemiological work can be complemented by examining the effects of radiation directly on the testes and on sperm. There are both human and animal data indicating the potential for damage. As regards uranium exposure, an IEER study summarized the reproductive issues associated with exposure of males as follows:

> In regard to the possible effects on men, uranium is found to concentrate in the testes and has been found in the sperm of Gulf War veterans at elevated levels. While no epidemiological data yet demonstrates an impact on reproductive success from the veteran's exposure, the Royal Society noted that the concentration of DU in the testes was a potential concern given the possible synergistic effects between uranium's ability to damage DNA through both chemical oxidative stress and ionizing alpha radiation. In addition, the World Health Organization has noted the observation of "unspecified degenerative changes in the testes" of rats as a result of chronic ingestion of soluble uranium compounds. [footnotes omitted] [111]

More direct evidence of the effects of radiation exposure on sperm were indicated in a 1975 paper by Popescu and Lancranjan. This study examined data for 72 men with occupational exposure to low-level ionizing radiation and 42 controls. This work found an increased incidence of weak sperm, low sperm counts, and malformed sperm in the irradiated group. The exposed population represented a mixture of exposure situations from radium dial painters and uranium miners to workers in medical radiology. Subjects with gonadal diseases or genital injuries were excluded from the study.[112] The main conclusion of the study was as follows:

> The spermatogenesis alterations – especially hypospermia and teratospermia – noticed in protracted irradiation can account for the lowering of fertility of men occupationally exposed to ionizing radiations, previously proved by epidemiologic researches carried out in various countries. As semen of poor quality is known to be associated with spontaneous abortions and stillbirths, and because these were also more frequently found in families of men occupationally

[110] Maconochie, Doyle, and Carson 2004; Doyle et al. 2004; Maconochie et al. 2003; Cowan et al. 1997
[111] Makhijani and Smith 2005 p. 14
[112] Popescu and Lancranjan 1975 pp. 567-568

> exposed to ionizing radiations, our data bring supplementary and direct evidence in favor of a relationship between these dominant lethalities and long-term exposure to ionizing radiations.[113]

Since this study involved direct study of sperm of exposed males compared to the sperm of unexposed males and since all of the subjects were chosen to be otherwise healthy, the results as regards adverse health outcomes can be regarded as more reliable than the epidemiologic studies for cancer outcomes (*Gardner et al.* and *Sever et al.*) discussed above. The Popescu and Lancranjan study indicates that the problem of gonadal exposure may be more broad and problematic than has been recognized in radiation protection, rather similar to the situation for exposure of women and children. We examine this matter more closely by illustrating the problem with a study of tritium exposure in Chapter 7. We discuss the evidence for uranium concentration in testes in Chapter 8.

Section 5.3--Policy Implications

There are no regulations of which we are aware that govern radiation exposure to men who plan to become fathers. The dose limits in the workplace for pregnant women apply to the embryo/fetus, but this is, by definition, a post-conception matter. For prospective fathers, like prospective mothers, there is a regulatory void.

Controls on doses to ova and to cells involved in the process of spermatogenesis are not yet on the regulatory horizon. We consider this question in Chapter 9, where we attempt to indicate an overall direction that regulatory change could take in light of the analysis in this report. We note here that a reduction in workplace exposure to the levels that are now guidelines (rather than requirements) would be considerably more protective than are current regulations, especially in the United States, where workplace limits are lax compared with those in the European Union. Finally, we note that when we take prospective parents into account and include the fact that many pregnancies carried to term are unplanned, the need to protect future generations from harm due to parental exposure points in the general direction of tightening general exposure limits both in the workplace and for the general public (see Chapter 9).

[113] Popescu and Lancranjan 1975 p. 567

Chapter 6: Radiation, Chemicals, and Combined Adverse Health Outcome[114]

Section 6.1—Biological Effects of Radiation and of Chemicals

Ionizing radiation has an adverse effect on health principally through its role as a genotoxic agent. In conventional radiobiological theory, the initial events of significance are considered to be strand breaks in DNA, some of which remain unrepaired and others of which are subject to misrepair. The consequences of such failures of repair include cell death and mutation. More recently, studies of genomic instability and the bystander effect have raised the issue of alternative modes of action of ionizing radiation. Thus, the target may be the whole of the cell nucleus rather than a localized region of DNA and the effect may be mediated by diffusible substances produced by the irradiated cell and affecting others in its vicinity or by other mechanisms involving the transmission of materials or signals across inter-cellular junctions. Nevertheless, the principal locus of action is considered to be the cell nucleus and the primary actions are considered to be cell killing (or sterilization) and the induction of changes in the cell genotype and phenotype that lead to abnormal behavior of the cell.

In the germ line, changes in the genotype (and possibly the phenotype, if epigenetic effects are of significance) can result in serious health effects, e.g. embryonic and fetal death and hereditary disease in impaired individuals that remain viable to term. In somatic cells, abnormal genotypes and phenotypes may result in altered patterns of cellular proliferation and hence cancer.

Chemical toxins may also act as genotoxic agents. However, their modes of action may be both more diverse and more specific than ionizing radiation. Although radioactive substances that are incorporated in the body may be associated with specific organs and tissues, e.g. iodine-131 with the thyroid and plutonium-239 with bone surfaces, the emitted radiation causes non-specific damage. In contrast, chemicals can induce their effects by binding to specific ligands and altering particular biochemical pathways. These ligands and pathways differ between chemicals. Thus, whereas each individual chemical will have a specific mode of operation, the wide range of chemicals to which humans are exposed makes their range of adverse effects more diverse than is observed with ionizing radiations.

Section 6.2—Combined Effects

In the context of potential synergism between the adverse effects of various chemicals and ionizing radiation, these different causative mechanisms have to be kept in mind. If an adverse outcome is a consequence of a multi-stage process (as is thought to be the case in carcinogenesis; see below) and two chemicals adversely affect different stages in that process, then exposures to those two chemicals may operate multiplicatively in inducing the adverse outcome. Conversely, if two chemicals affect unrelated biochemical pathways, then sub-threshold exposure to one of the pair may have no affect on the likelihood or severity of adverse outcomes induced by the other.

It is also important to recognize that the term synergy is not well-defined in the context of chemical toxicology and is used with different intents by various authors. This has been well explained by Hertzberg and MacDonell,[115] who comment as follows.

> A substantial effort has been spent over the past few decades to label toxicologic interaction outcomes as synergistic, antagonistic, or additive. Although useful in influencing the emotions of the public and the press, these labels have contributed fairly little to our understanding of joint toxic action. Part of the difficulty is that their underlying toxicological concepts are only defined

[114] This chapter was authored by Mike Thorne, Ph.D., who is a consultant to IEER.
[115] Hertzberg and MacDonell 2002

for two chemical mixtures, while most environmental and occupational exposures are to mixtures of many more chemicals.

Some of the complexities involved in identifying and quantifying synergy are highlighted by information from the EPA Mintox database on non-cancer toxic effects.[116] As discussed by Hertzberg and MacDonell, whereas approximately one quarter of the evaluations in Mintox showed consistent synergism (greater than additive joint toxicity), the largest group showed mixed interactions. Some studies of a chemical pair showed one type of interaction (e.g. synergy) whereas other studies of that pair showed another type of interaction. Often these differences were explained by, or at least associated with, a different exposure sequence or route, or different target organ or toxic endpoint.

A further complicating factor not mentioned by Hertzberg and MacDonell is that synergy is difficult to define when the two agents exhibit non-linear exposure-response relationships. For some agents, a threshold or pseudo-threshold may exist. With two such agents, two combined sub-threshold exposures may either cross the critical threshold or not, depending on the relative magnitude of the exposures. In such circumstances, the degree of synergy could be interpreted as zero or infinite.

A related effect can occur at high exposures for substances such as vinyl chloride monomer that can be metabolized by the body to only a limited degree. In this case, increasing exposure above a particular level does not lead to any additional production of toxic secondary metabolites[117] and, therefore, other agents that enhance or inhibit production of these secondary metabolites at low concentrations of the toxic agent for which the production mechanism is unsaturated may have a very different effect at high concentrations for which the production mechanism is saturated.

Finally, in some cases, such as intakes of uranium, interactions between radiotoxic and chemically toxic effects may arise in consequence of exposure to one and the same substance. Here, a further consideration arises that the chemical and radiation impacts may be heterogeneously distributed in the body, but may be spatially and temporally correlated. Thus, a uranium particle that is present in the lungs will irradiate cells in its immediate vicinity and, as it dissolves, will subject them to the influence of its chemical toxicity, which includes a mutagenic component. (This is discussed further in Chapter 8).

Existing practice in evaluating the potential significance of exposures to mixtures of toxic agents is described by Pohl *et al.*[118] in relation to the approach adopted by the Agency for Toxic Substances and Disease Registry (ATSDR). In developing Minimum Risk Levels (MRLs)[119] several different approaches were used. In some instances, toxicity equivalency factors were used to estimate the toxicity of the whole mixture (i.e. the individual components were linearly weighted by their relative toxicity and additivity was assumed). In other cases, the most toxic chemical was assumed to drive the health assessment for the whole mixture. In still other cases, the mixture was treated as a single entity and its health effects evaluated directly from laboratory and epidemiological studies (a useful approach if the mixture is very similar in composition for a wide range of exposure situations). Alternatively, sometimes each chemical in the mixture was evaluated separately, but no guidance was provided on how the individual evaluations should be combined to set standards for mixtures of different proportional composition.

[116] EPA 1988b
[117] Thorne, Jackson, and Smith 1986
[118] Pohl , Hansen, and Chou 1997
[119] The ATSDR defines an MRL as follows: "An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure." (ATSDR 2005)

Setting aside hereditary effects, which were once emphasized as a significant concern in radiological protection, but have become increasingly de-emphasized in recent years, the main deleterious effects of exposure to ionizing radiations at low doses and dose rates are generally considered to be the induction of fatal and non-fatal cancers. Although recent observations of the Japanese exposed at Hiroshima and Nagasaki have shown that there is an additional component of health effects (e.g. coronary heart disease), it seems unlikely that these observations will change our numerical perception of radiation risk to any dramatic extent.[120]

Section 6.3—Carcinogenesis

It is generally recognized that carcinogenesis is a multi-stage process. Thus, more than one mutation is necessary for carcinogenesis. In fact, a series of several mutations to certain classes of genes is usually required before a normal cell will transform into a cancer cell.[121]) Only mutations in those certain types of genes which play vital roles in cell division, cell death, and DNA repair will cause a cell to lose control of its proliferation. Evidence for the multi-stage nature of the process is the observation that the incidence of many cancers increases as a high power of age..[122] This suggests that several successive steps of transformation are required for carcinogenesis. Factors involved include initiation or enhancement of the expression of proto-oncogenes, loss of activity of tumor-suppressor genes, escape from control by apoptosis (programmed cell death), alteration in patterns of differentiation, development of capabilities for angiogenesis (blood vessel formation), changes in invasiveness, and, finally, development of the ability to metastasize, which is a key factor in many aggressive and life-threatening cancers.[123]

As discussed by Sarasin, there are debates in the literature as to whether normal mutation rates followed by selective advantage of mutated clones are enough to produce the numerous mutations found in human cancers.[124] Alternatively, the mutator phenotype hypothesis is based on the idea that normal mutation rates are insufficient to cause cancer and that in at least one step of the carcinogenic process a mutation occurs that affects the fidelity of DNA replication or repair, apoptosis pathways, or the cell cycle checkpoint regulations. Effects of chemicals or ionizing radiation on a cell prior and subsequent to transformation to a mutator phenotype may be very different from each other, influencing the degree of synergy that is observed between the two types of insult.

The importance of defects in DNA repair and genomic maintenance to carcinogenesis applies specifically to radiation-induced carcinogenesis. Thus, mutations that predispose people to increases in cancer incidence in general are also associated with increased sensitivity to the induction of cancers by ionizing radiation. For example, the ICRP[125] has stated that " [i]n most, if not all, instances of familial cancer predisposition associated with the dominant inheritance of strongly expressing tumor suppressor gene mutations there will be an absolute increase in the probability of radiation-induced cancer." Also, in the case of cancer predisposition associated with deficiencies of DNA repair, some, but not all, disorders of this type will show elevated cancer risk after radiation.[126]

It is well established that ionizing radiation can act as a carcinogenic agent in its own right. However, it is not clear whether it affects all, or only some, of the stages of carcinogenesis outlined above. The non-specific nature of the damage that it causes, the localization of that damage in DNA, and the generally

[120] Valentin 2006
[121] See Sarasin 2003
[122] Sarasin 2003
[123] Tannock and Hill 2005
[124] Sarasin 2003
[125] ICRP 79
[126] ICRP 79

long latent period between induction of that damage and clinical expression of associated cancers suggests that ionizing radiation primarily has a role in the early stages of transformation of cells from a normal to a malignant phenotype. Chemicals that also cause non-specific damage to DNA might be expected to act similarly. Therefore, mixed exposures to radiation plus such chemicals could reasonably be treated as additive, once a suitable scale factor had been established between the effectiveness of the chemical and radiation in inducing cancer, e.g., by comparing the slope factor recommended by the EPA for the chemical with the slope of the linear dose-response relationship for ionizing radiation.[127]

However, most chemicals will induce a particular category of damage or will have other effects such as suppression of immuno-surveillance. In these circumstances, a synergistic effect between ionizing radiation and chemical toxins is readily envisaged and may be the norm. A mathematical approach model for assessing the synergism between radiation and chemical exposure is rather straightforward, when both types individually produce a linear effect (i.e., effect proportional to the dose). This is shown the box below.

A specific context in which synergistic effects may arise is in the induction of cancers by ionizing radiation and the estrogen agonist diethylstilbestrol (DES). As discussed by Doll and Wakeford,[128] low doses of x-rays to the fetus, especially during the last trimester, cause an increased risk of leukemia and all other types of cancer during childhood. In the case of DES, fetal exposure has been associated with the induction of vaginal adenocarcinoma in young women.[129] As discussed by Birnbaum and Fenton,[130] a wide variety of studies have demonstrated that exposures to endocrine disruptors such as DES can alter the hormonal milieu, reproductive tissue development, and susceptibility to potential carcinogen exposure in the adult. Such endocrine disruptors are not genotoxic, but can have significant adverse health outcomes. There is the potential for a synergistic interaction between *in utero* exposure to ionizing radiation, which can cause early pre-neoplastic changes in cells, as evidenced by the increased risk of childhood cancer after such exposure, and exposure to endocrine disruptors, which alter the environment in which those cells develop and also themselves predispose toward cancer development. Specifically, exposure to ionizing radiation will result in there being present more cells that could develop into cancers and exposure to endocrine disruptors will make it more likely that such cells will survive and express their carcinogenic potential. (See also the box on page 56.) This synergistic interaction could influence both the induction of childhood cancers (which are known to occur in excess after *in utero* x-ray exposure) and of cancers occurring in adults. In this latter context, induction of breast cancer may be of particular concern, since elevated levels of natural estrogens during gestation have been associated with an increase of breast cancer in the children of such women, and there are animal data that show that various chemicals that modify estrogen levels also influence the risk of mammary tumors. These chemicals include dioxins, which are known to be potent tumor promoters.[131] Thus, again, exposure to ionizing radiation could increase the number of cells that have the potential to proliferate to form breast cancers later in life and exposure to chemicals that modify estrogen levels could preferentially enhance the survival of such cells.

[127] EPA IRIS. The IRIS web site contains a glossary that defines slope factor and many chemicals for which it is used.
[128] Doll and Wakeford 1997
[129] Herbst, Ulfelder, and Poskanzer 1971
[130] Birnbaum and Fenton 2003
[131] Birnbaum and Fenton 2003

Synergism Model

Consider an exposure to a dose D of ionizing radiation that increases the number of cells at a particular stage of transformation from N to N+aD at a particular time, where a is the rate of increase per unit dose. Consider also an exposure, E, to a toxic chemical that results in the fraction, b, of those moved to the next stage increasing to b+cE, where c is the fractional rate of increase per unit exposure.

A combined exposure to radiation dose D and a chemical exposure E would result in the number of cells present in the next stage as (N+aD)(b+cE). For radiation alone, the number would be (N+aD)b and for the chemical alone it would be N(b+cE). Without either agent, the number would be bN.

If the two agents were considered to operate additively, the combined exposure would be assessed to give rise to (bN+abD+cNE), the sum of the unexposed value, and the individual radiation and chemical exposure increments, abD and cNE. The excess cells transferred to the next stage as the result of exposure are therefore (abD+cNE).

With the proposed synergistic mechanism, the number is the product of each effect, which amounts to (bN+abD+cNE+acDE). In this case the excess over the unexposed value of bN is (abD+cNE+acDE). By comparing this expression to the value of the excess in the case of the additive model, the degree of synergy can conveniently be expressed as the ratio of the excess in the case of the multiplicative model to that of the additive model:

$(abD+cNE+acDE)/(abD+cNE) = 1 + acDE/(abD+cNE)$

This is more conveniently re-expressed by defining the fractional changes α and β defined by:

$\alpha = aD/N$, the fractional increment in cells transferred to the next stage as a result of radiation exposure
$\beta = cE/b$, the fractional increment in cells transferred to the next stage as a result of chemical exposure

Dividing the numerator and denominator by bN, it is the degree of synergy relative to the additive assumption is given by:

$1 + \alpha\beta/(\alpha + \beta)$

Thus, the degree of synergy depends in a non-linear way on the contributions of the two agents.

Also, as noted above, the carcinogenic effectiveness of ionizing radiation can be increased in cells that lack the ability to repair DNA damage. As shown by Snyder *et al.*,[132] a wide range of metal salts can interfere with the repair of DNA damage induced by x-rays or ultraviolet light. Thus, a synergistic effect between exposure to ionizing radiation, inducing DNA damage, and exposure to metals and semi-metals, such as mercury, nickel and arsenic, that inhibit the repair of such damage, is to be expected.

Furthermore, it is noted that synergy can arise from the interaction of cell transformation and cell sterilization effects. Carcinogenesis is a clonal disease and loss of function in a tissue through cell sterilization is likely to result in signals stimulating cell division in the affected tissue. Such cell division may be stimulated in cells that have already passed through the initial stages of the multi-stage process of carcinogenesis either as a result of exposure to the agent that caused cell sterilization or to another agent.

[132] Snyder, Davis, and Lachmann 1989

Whether a synergistic or an antagonistic effect occurs will be determined by the sensitivity of normal and transformed cells to sterilization by the toxic agents to which they are exposed, the effectiveness of induction of transformation by those agents, and the ease with which normal and transformed cells are stimulated into division by the requirements of tissue repopulation.

Section 6.4 Conclusions

In summary, the multi-stage nature of carcinogenesis makes it highly likely that synergistic and antagonistic effects will exist between different toxic agents, including ionizing radiations. Currently, there is no consensus as to how the effects of different chemically toxic agents should be combined and various approaches have been adopted with little theoretical justification. Although many of the processes involved in carcinogenesis are understood to a greater or lesser degree, there is no overall mechanistic model of the process.

Indeed, the concept of a single mechanistic model is almost certainly inappropriate, as the nature and number of stages likely differ between variously clinically distinguished cancers. Even for a single, well-defined cancer, it is likely that there are multiple ways to get from a normal somatic cell to a cell expressing the full cancer phenotype. For example, it has been argued that the concept of a mechanistic progression is inappropriate and that it is better to think in terms of a model in which phenotypic expression and genomic characteristics are closely coupled dynamically.[133] Evidence for the validity of such an approach is now beginning to emerge.[134,135]

Current understanding of potential synergies between exposures to ionizing radiation and other agents have been discussed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR).[136] They comment as follows.

> Radiobiological research has turned up numerous agents potentially capable of influencing the progression of early radiation effects towards adverse health effects. General conclusions are hindered by the multitude and complexity of the possible interactions and the dependence of the combined effect on the sequence of the exposures. More explicitly, because of the long time period between the initial radiation event and the final effect, a combined exposure to radiation and another agent may occur after simultaneous exposure but also from exposures hours or even years apart…
>
> A very important combined effect is the interaction of smoking and exposure to radon, although even in this case there is still no unambiguous conclusion on the interaction mechanism. Epidemiological data clearly indicate that combined exposure to radon and cigarette smoke leads to more-than-additive effects on lung cancer. These results warrant special consideration in estimating the radiation risks because a large proportion of the world's population is exposed concomitantly to considerable levels of indoor radon and smoking. The combined analysis of 11 miner studies… indicates that the effect of radon may be enhanced by a factor of about 3 by being combined with smoking.

Although UNSCEAR considers that there is no firm evidence for large deviations from additivity at controlled occupational or environmental exposures, they go on to comment that:

> The lack of pertinent data on combined effects does not imply *per se* that interactions between radiation and other agents do not occur. Indeed, substances with tumor promoter and/or inhibitor activities are found in the daily diet, and cancer risk therefore depends on lifestyle, particularly eating habits. Not only can these agents modify the natural or spontaneous cancer incidence, but they may also modify the

[133] Baverstock 2000
[134] Falt et al. 2003
[135] Huang et al. 2005
[136] UNSCEAR 2000

> carcinogenic potential of radiation. Such modifications would influence the outcome particularly when radiation risks are projected relative to the spontaneous cancer incidence.

It is also appropriate to note that current radiological protection standards for humans in respect to carcinogenesis are based almost exclusively on epidemiological data. These standards derive from establishing dose-response relationships in exposed populations and there is limited opportunity for investigating how exposures to other toxic factors influence these relationships, either because of limited information on other exposures or because the small numbers of cases observed limit the degree to which the data can be disaggregated for statistical analyses that will yield meaningful results. An exception to this arises for interactions between smoking and exposure to radon in relation to lung cancer incidence and mortality. However, in general, the first priority has been to disaggregate by factors such as age at exposure, interval between exposure and ascertainment, or age at ascertainment, rather than by exposure to other agents.

In respect to the interaction between smoking and exposure to radon (or more strictly, the decay products of radon), as UNSCEAR has noted, the most comprehensive and complete analysis of radon-induced health risks was published by Lubin *et al.*[137] That report contains a joint analysis of original data from 11 studies of male underground miners. Data on smoking were available for 6 of the 11 cohorts, but the assessments were limited by incomplete data on lifetime tobacco consumption patterns and the sometimes exotic forms of tobacco use.

As pointed out by UNSCEAR,[138] a best estimate from the combined study of the data for miners indicates that the lung cancer risk for smokers expressed in absolute terms is higher by a factor of about 3. To further characterize the association, more detailed data on tobacco use would be needed. Age of starting to smoke, amount and duration of smoking, and type of tobacco were recognized as important determinants of risk.

A best linear estimate of the risk coefficients found in the joint analysis of Lubin *et al.*[139] for the indoor environment indicates that, in the United States, some 10 to 12 percent, or 10,000 cases, of the lung cancer deaths among smokers and 28 to 31 percent, or 5,000 cases, of the lung cancer deaths among never smokers are caused by radon progeny. About half of these 15,000 lung cancer deaths traceable to radon would then be the result of over-additivity, i.e. synergistic interactions between radon and tobacco. Because of the many differences between exposed persons and exposure situations in mines and homes and the additional carcinogens such as arsenic, dust, and diesel exhaust in mine air, these figures should be interpreted with caution. Nevertheless, they indicate that synergy between exposure to ionizing radiation and other toxic agents can be a major public health issue.

Thus, when considering the potential future development of radiological protection standards oriented towards protecting the most sensitive members of an exposed population, it is proper to bear in mind that variations in sensitivity will arise not only due to factors such as age, sex, and intrinsic genetic constitution, but also due to environmental factors such as exposure to toxic chemicals that will interact with radiation-induced damage in complex and poorly understood ways.

[137] Lubin et al. 1994. See also Lubin et al. 1995
[138] UNSCEAR 2000
[139] Lubin et al. 1994

Chapter 7: Case study -- Tritium[140]

Tritium, a radioactive form of hydrogen, in gaseous form generally presents a low health risk because it is exhaled before it can deliver substantial radiation doses to the body. (Tritium is usually denoted by the symbol T, to distinguish it from ordinary hydrogen, H.) However, tritium can displace one or both of the hydrogen atoms in water, thereby creating radioactive water (see box). Since water is essential to life, radioactive water means that radioactivity seeps into all parts of the body and its constituents – approximately 70 percent of the soft tissue in the human body is water.

In addition to tritiated water, tritium can also be integrated into organic molecules and hence into body tissues. The tritium that replaces hydrogen in a carbon-hydrogen bond is difficult to remove and is, therefore, referred to as non-exchangeable organically bound tritium (OBT). Animal studies indicate that 1-5 percent of the tritiated water in mammals is incorporated into such OBT biomolecules. Direct intake of organically bound tritium, for example through food, is more likely to be incorporated as organically bound tritium in biomolecules in the human body than is tritium obtained by drinking tritiated water. However, organically bound tritium is a heterogeneous group of compounds that can behave very differently in metabolic processes, and more research is needed to understand the incorporation of tritium into the body from a variety of compounds.[141]

Both tritiated water and organically bound tritium can cross the placenta and irradiate developing fetuses *in utero*, thereby raising the risk of birth defects, miscarriages, and other problems (see below). The forms of tritium discussed in this chapter are either tritiated water or OBT, unless otherwise specified.

Current radiation protection standards assume that exposure to beta radiation (such as that from tritium) causes the same biological damage as whole-body exposure to gamma and x-rays. However, different kinds of radiation can cause different amounts of health harm – i.e., create different health risks – for the *same amount of energy* deposited in the body. This difference between the effectiveness of different kinds of radiation in causing biological damage is called the "relative biological effectiveness" (RBE) of radiation.

Current radiation protection standards generally assume that gamma rays, x-rays, and all beta particles have an RBE of one.[142] But the biological damage from tritium per unit of radiation energy deposited in the body or a specific organ can be much higher – that is, its RBE can be significantly greater than one. This is due to the fact that tritium emits a relatively low-energy beta particle. This means that it the beta particle is more likely to deposit its energy in a smaller number of cells or even entirely in one cell, compared with a higher energy photon or beta particle that would affect a number of cells, losing a little of its energy all along the way. This is the same reason that alpha particles, which deposit all their energy a short distance, even in a single cells, are assigned an RBE of 20 in regulatory practice. That is, for alpha particles, it is assumed by the radiation protection standards that an alpha particle will do 20 times more biological damage than a gamma ray with the same amount of radiation energy. As we discuss below, the RBE of tritium should not be taken as one, because it is highly dependent on age at exposure and whether the tritium is in the form of water or is organically bound.

A 2002 study by Harrison, Khursheed, and Lambert examined uncertainties in the assumptions of the International Commission on Radiological Protection (ICRP) models for calculating the dose of radiation from the intake of tritiated water and organically bound tritium. It also estimated dose conversion factors for tritiated water and for OBT. The dose conversion factors for various ages estimated in the paper

[140] Parts of this chapter are based on, taken from, or adapted from Makhijani and Boyd 2004.
[141] Harrison, Khursheed, and Lambert 2002, pp. 300, 303, and 304
[142] See for instance, the fact sheet on ionizing radiation published by Argonne National Laboratory. (Argonne 2005)

indicate a relative biological effectiveness of both tritiated water and OBT to be higher than in ICRP models.[143] This means that tritium is often more effective at causing damage per unit of intake than assumed in current models. Also, there are differences in the retention of different compounds of tritium in the body. The integrated effectiveness of tritium relative to the values used by the EPA in current regulations can be estimated from the research of Harrison, Khursheed and Lambert. Our estimates based on their analysis are shown in Table 9 below.

About Tritium[144]

Tritium is a radioactive form of hydrogen with one proton and two neutrons, resulting in a total atomic weight of three... Most tritium in the environment is man-made, however, some tritium occurs naturally due to interactions between the atmosphere and cosmic radiation. With its relatively short half-life (12.3 years), tritium decays at about 5.5 percent annually.

As a gas, tritiated hydrogen is a light and small molecule and hence diffuses readily through all but the most highly engineered containment vessels and mixes freely with the other forms of hydrogen in water and water vapor. It forms tritiated water by replacing one or both atoms of non-radioactive hydrogen in water. Tritiated water is often designated as HTO or T_2O, depending on whether it has one or two atoms of tritium in the water molecule respectively. When tritium is generated by neutron absorption in heavy water (D_2O) the tritium can also displace deuterium to form DTO. All these forms of water containing tritium are rendered radioactive as a result. They behave in a manner that is chemically almost the same as ordinary water, though limited mass-related distinctions in chemical reaction rates can occur. The pervasiveness of tritium is due to the mobility of tritiated water in the environment, since it can move with non-radioactive water (both H_2O and D_2O).

The specific activity of tritium is very high – almost 10,000 curies per gram. Hence a small mass of tritium can contaminate a large amount of water. For instance, one gram (about one-thirtieth of an ounce) of tritium in tritiated water will contaminate almost 500 billion liters of water up to the drinking water limit of 20,000 picocuries per liter. The combination of these two properties -- tritiated water is chemically like ordinary water and tritium is highly radioactive – makes tritium a very pernicious pollutant that is difficult to contain and, once in the water, difficult to remediate, especially when in trace amounts.

Tritium's primary function in a nuclear weapon is to boost the yield of the bombs. It is used both in pure fission weapons and in the primary of thermonuclear weapons. Contained in removable and refillable reservoirs in the warhead, it increases the efficiency with which the nuclear fissile materials are used. Although no official data are publicly available, each warhead is estimated to require an average of approximately four grams of tritium. However, neutron bombs, designed to release more radiation, have been estimated to require more tritium (10-30 grams).

[143] Harrison, Khursheed, and Lambert 2002 p. 308
[144] Adapted from Zerriffi 1996 p. 1

Table 9: Integrated Relative Biological Effectiveness of Tritiated Water and Organically Bound Tritium

Age	Form of tritium	5% Confidence limit	Median	95% Confidence limit
Adult	HTO	1.2	2.3	3.8
Adult	OBT	2.3	5.0	11.6
Fetus (maternal ingestion during pregnancy)	HTO	2.1	4.4	8.1
Fetus (maternal ingestion during pregnancy)	OBT	4.0	9.8	23.1

Source: Estimated from Harrison, Khursheed, and Lambert 2002, Table 8. The Integrated RBEs shown above were calculated by dividing the tritium doses in sieverts per becquerel shown in this paper by $1.73*10^{-11}$, which is the dose conversion factor for tritiated water in sieverts per becquerel in the Federal Guidance Report 11 of the Environmental Protection Agency (EPA 1988). This guide provides the dose conversion factors for "Reference Man." It is the source document for dose conversion factors used in RESRAD.

Note: HTO = tritiated water in which one atom of ordinary hydrogen has been replaced by an atom of tritium. OBT = organically bound tritium. The numbers in the columns for confidence intervals mean that the RBEs would be less than the cited number for the percent of times indicated by the confidence interval were a series of identical experiments to be performed.

The increased risks to pregnant women from tritium do not stop at cancer. The risks of tritium exposure to pregnant women and embryos/fetuses include miscarriages and genetic defects. The risks can also be multi-generational given that all the ova a woman will ever have are produced while in her mother's womb. There are many gaps in the research (discussed below).

Section 7.1—Research Needs

Current estimates of the health risks from exposure to organically bound tritium may underestimate the actual health impacts. Tritiated water is considered to be uniformly distributed throughout the water in the body. According of the EPA's Federal Guidance Report 13, all organs except for portions of the gastrointestinal tract receive the same dose for a given intake of tritium.[145] However, in practice, different organs have different concentrations of tritium -- for example bone and fat have lower concentrations due to their relatively low water content.[146] Further, the distribution of organically bound tritium can be quite localized, which means that relatively small numbers of cells would have relatively high concentrations, while others would have relatively low concentrations. Furthermore, if organically bound tritium becomes incorporated into DNA, it does not uniformly irradiate the whole cell; it preferentially irradiates the nucleus. Hence, the risk of damage to the DNA and of adverse health effects (including cancer but not only cancer) is considerably greater than if the tritium expended its energy in the water in the cytoplasm of the cell.[147] This makes the chemical form in which tritium is ingested important in the outcome. As the ICRP has noted:

> Beta particles which originate from the radioactive decay of tritium have low energies, so radiation energy is absorbed not very far from the place of decay within cells and tissues….. Therefore, if a tritium isotope decays within the cell nucleus, the energy is absorbed almost completely within the nucleus, and if it decays within the cytoplasm, the absorption process takes place in the cytoplasm. Under these circumstances, the chemical form of tritium that reaches the cells is very important.[148]

[145] FGR 13 CD (EPA 2002). See the dose conversion factors for tritium ingestion.
[146] Harrison, Khursheed, and Lambert 2002 p. 305
[147] Hill and Johnson 1993 p. 632
[148] ICRP 90 pp. 13-14.