The 3 R's: Radiation, Risk, and Reason

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Preface

I live within two miles of the Three Mile Island Nuclear Station just south of Harrisburg, Pennsylvania. When we bought our house in 1968 we did not know that the Three Mile Island (TMI) nuclear plant was under construction just across the Susquehanna River. If we had known we may have changed our minds about purchasing the house. Probably not. I was a young nuclear physicist just starting a teaching career at Dickinson College. I dealt with radiation in my job, so what was the problem with living near a nuclear plant?

Then came March 28, 1979. The TMI accident was frightening, even for someone trained in nuclear physics. When I dealt with radioactivity and radiation in the laboratory, I was accustomed to having control over it and regulating the amount of my radiation exposure.

Suddenly I was in a situation where I was being exposed to radiation and had no control over it. The officials at TMI did not have a good understanding of what was being released. Nobody -- not TMI officials, and not the state and federal government agencies -- had a good monitoring system in place. The news media did not understand what was happening and could not get or could not comprehend the important information to communicate to the public. The uncertainty of not knowing what was happening was more fearful than the radiation itself.

Many people in the area solved the problem of uncertainty by leaving. They went to stay with friends and relatives who lived anywhere outside the central Pennsylvania area. We decided to stay. Why? I obviously had an understanding of radiation and what it can do, and armed with this information I should have been comfortable in such a situation. Well, I can say that the uncertainty over what was happening was quite unsettling even for a trained nuclear physicist.

However, we did stay. I always had a radiation survey meter with me so I could measure the radiation dose we were receiving. Without this meter I am not sure if we would have stayed. That is one of those questions we will never be able to answer.

I also had the chance to gather my own information. When a scientist is presented with an opportunity to take data, how can one refuse? One way to avoid fear in a stressful situation is to get personally involved in the situation. I had previously done research on radioactivity in the soil. So, I did what I could do best: collect and analyze soil to measure any radioactivity present in it that may have come from TMI. That kept me busy.

What would you do if you were faced with the prospect of being exposed to radiation? In an emergency such as that of the TMI accident, you might react like many did in this area: leave. But what about those non-crisis occasions? You read about radon being present in your area and public officials urge you to test your house; should you? The news media reports that a shipment of radioactive material is coming by your house. Do you leave or sit in your front yard and watch it go by? Somebody wants to site a low-level radioactive waste facility in your area. Should you consider moving or try to get a job at the new facility?

A person's reaction to hazardous situation depends on that person's knowledge of that hazard. How do you react to the fact the someone you know has AIDS? Would you react the same way if you were a physician who treats AIDS patients regularly? Knowing and understanding the actual risks can make a big difference in how you react.

I heard a story at a professional meeting that exemplifies the concept of fear in a crisis and how we react depending on our knowledge. A radiation worker was taking radiation readings around TMI after the accident. Two police officers were driving the worker to the reading sites. One evening the radiation levels were a bit higher than previous times. The officers were quite uneasy about this situation and insisted on staying in the car while the radiation worker took the measurements. They kept the windows rolled up. Then the officers received a call that an alarm had sounded at some building in their jurisdiction and they needed to respond. The two officers, with the radiation worker in the back seat, raced to the building. When they arrived, the two officers jumped out of the car, drew their guns, and proceeded toward the building. The radiation worker huddled on the floor of the police car.

The purpose of this book is to give readers a better understanding of radiation so you can make a personal decision on how to react when faced with the potential of radiation exposure. Reacting out of fear is one way to respond to such a situation, but this reaction can have disastrous consequences. As I will discuss in one chapter in this book, there is a definite cost to reacting out of fear. On the other hand, not reacting at all can also be disastrous.

When I started writing this book, I had one general goal in mind: to change the reader's reaction to radiation from fear to respect. (Maybe this should be a fourth R.) If I can just change this attitude in a small fraction of the readers, then I will feel all my work has been worth it. On the other hand, I do not want anyone to accept radiation as a totally benign entity. It does demand respect. It does not demand fear.

During the energy crisis in the 1970s, I saw a report on the evening news. It featured a family who were doing everything to conserve energy in their home. In a sense, they were reacting out of a fear of not having enough energy if nobody took conservation measures. The family turned down the heat, did not use many lights, watched very little television, reduced the use of other appliances,

took short showers, etc. However, when the report mentioned that they heated the house with electricity, then I realized the futility of most of their actions.

They did not know one fact about energy: all energy eventually converts to heat. A light bulb gets hot, so does a television (well, maybe just warm). Every appliance is just a heat source; turn them off and the heating system must work harder to supply that heat. The only way that family could have saved energy would be to turn down the heat and to use less hot water. They could have had lights blazing at all hours, watched television until they were bleary eyed and would not have wasted any energy; the lights and TV would just act as part of their heating system.

A more graphic example of how wrong information can lead to wrong conclusions has to do with sharks. We all "know" that the great white shark is a killer. It may attack any human it encounters, and the sighting of a shark near a beach is a reason for great concern, even fear. This characteristics of the great white was the basis for the book Jaws written by Peter Benchley. As he stated: "...it was common knowledge that sharks were not only carnivores, they were omnivores; they would eat anything. They would attack, kill, and devour human beings without much, if any, provocation. They would attack boats" (Brooks 71).

One day Benchley was scuba diving in the Bahamas when he turned around and was face-to-face with a great white shark. "I froze, able only to picture the ironic headlines that would announce my demise. But the shark froze, too. And then abruptly, frantically, implausibly, the great white wheeled around, voided its bowels, and disappeared into a nasty brown cloud" (Brooks 71).

How do we all "know" that the great white is so dangerous? This information came from many stories of shark attacks, most apparently without provocation. These stories slowly grew into "facts." But, as research has shown, the fact of the matter is that sharks suffer from poor eyesight. When they attack humans it is because they think they are seeing food. ". . . a person on a surfboard silhouetted against the is indistinguishable from a sea lion. . . . Some sharks emit warning signals and demonstrate distinctive postures before they attack. Reef fish receive the massages and take cover; humans may not perceive and understand them, and may invite trouble by refusing to retreat" (Brooks, 1995).

This striking example shows how information based on old, inaccurate data becomes "common knowledge" that affects how we react. In fact, Benchley states: " I couldn't write Jaws today. The extensive new knowledge of sharks would make it impossible for me to create, in good conscience, a villain of the magnitude and malignity of the original" (Brooks, 1995).

Similarly, we all "know" the hazards of radiation. This "knowledge" is based on the horrible deaths from the use of nuclear weapons on Japan, the stories we read about fallout, the dreadful predictions of some scientists (and non-scientists) about the effects from the Chernobyl accident, and countless other stories in the news media that depict frightening situations with radiation. But just what are the facts? Does the latest research verify or nullify out "knowledge?" Knowing the correct facts will dictate how we react when faced with a potential radiation exposure.

Incorrect knowledge can produce reactions to a potential radiation exposure that can be futile or even more risky than the radiation exposure itself. During the TMI accident, radioactive krypton gas accumulated in the reactor containment vessel. To reduce radiation exposure to the workers, this gas needed to be vented to the atmosphere.

When the krypton was vented, people were advised to stay indoors. School children were kept inside during recess. About that time I had just finished writing a book on household energy conservation. One fact I learned when writing that book is that the normal house is not air tight. Most houses have about one air change per hour, so any gas in the air outside will find its way into your house quite readily.

The walls of a house do provide some shielding from radiation and would have stopped some of the radiation from krypton outside the house. But it would not protect them from radiation from krypton inside the house. Staying inside may have helped soothe a few nerves, but it did not totally protect a person from the radiation of krypton gas.

During the venting some people left the area just as they did after the accident. Some boarded planes and flew to other parts of the country. In the process they were exposed to cosmic radiation. In fact they received more radiation from that airplane flight than they would have received if they had stayed home.

One reason many left during the accident and during the krypton venting was that "experts" were giving conflicting opinions about the real health risks. Much of the uncertainty in the public mind about radiation arises from conflicting opinions about what radiation can do. However, much of the conflict is not really conflict but science doing its normal thing: trying to arrive at the truth.

People have the idea that science is "exact." We have precise data for everything and therefore we know everything about anything we have studied. To learn physics, all a person needs to do is to memorize all the numbers and equations and that makes you a physicist. Well, that might get you on a television quiz show, but it will not get you far as a physicist. Physics is all about learning concepts and applying those concepts to reality. The numbers just back up your case.

Science is a process that has two major aspects: gathering data and then interpreting what that data means. In the field of radiation science, both aspects have problems. First, the data are not always accurate because the process of gathering the data involves too much natural uncertainty.

Secondly, we need to interpret the data. From our grade-school years we learned that the process of science involves making a hypothesis, taking data, then checking your hypothesis. Unfortunately, if the data have large uncertainties then more than one hypothesis can fit the data so we can have "conflict."

To better understand the reasons for "conflict," I have included some extensive information on how we gather data and why there can be different interpretations. This information may not make you comfortable, but that is reality.

Writing this book has been educational for me. As a scientist I make decisions based on facts. Feelings do not really enter a decision about what to do in the laboratory. However, as I learned from reading what experts say about the perception of risk, people rely on feelings as well as on facts. No matter how many facts someone reads, a person's perception of a risk will not change unless feelings change.

Can a book such as this change someone's feelings? Probably not. But that is not the purpose of this book. The goal of this book is to give you a better understanding of radiation and just what it can and can't do. My goal is not to make you an advocate or an opponent of the nuclear industry, but to be sure you have an understanding of the basics of radiation so you can make up your own mind. I will try to give you an understanding of why there is controversy and let you decide whom to believe.

An incident several years ago emphasized the idea that facts are not the only aspect affecting a person's perception of a risk. I was part of a team of professors teaching a summer school course in environmental science. After one of the other faculty members had given a lecture on air pollution, we were outside with the students taking a break. One student commented that the information he had just received was quite enlightening. He felt that all we needed to do was similarly educate the public on the facts of air pollution and we could solve all our problems. At this time I asked him to read the warning on the package of cigarettes he had in his hands; that was education of facts!

I intend to present the facts and just the facts. However, you need to be mindful that I am human and my biases will creep into the discussions and into what facts I do give. I have tried to gather information from a variety of sources that span the spectrum of beliefs; from radiation does minimal harm, to it is a deadly risk.

When I deviate from the facts, I will clearly identify my opinions based on the information presented. You are welcome to agree or disagree as the case may be. To let you know where I stand, I feel that radiation demands respect, but not fear. I get upset with sensational stories about the effects of radiation. We should not ignore any claims of radiation risk, but all claims must pass the scrutiny of the scientific method.

In the following pages I will use the "weight of the evidence" to come to some of my conclusions. Usually this means that I will favor panels of scientists who have studied radiation effects for many years, have published extensively in peer-reviewed literature, and have become recognized authorities of radiation. However, I will not ignore those who are critical of these scientists.

Introduction

When early settlers immigrated to this country, they knew that there were risks taking the ocean voyage. When they arrived, they cleared sections of the forests to build houses and farm the land. Yet they knew the risks from animal attacks and from floods and insect infestations that could wipe out their crops. They left behind whatever medical assistance they had in their homeland. They took a risk of contacting a fatal disease without whatever treatment that was available then. They perceived that the benefits of a new land and new freedoms were worth the risks.

Today we also face risks, but nothing like those of our ancestors. Most likely we are more concerned with these few risks than our ancestors were with their many risks. They were more concerned with just surviving.

The advances in our quality of life have come about mostly from advances in technology. We have multitudes of objects that make life easier and safer for us. But we also are becoming more concerned that the very same technology that has made our lives better can be a monster in disguise waiting to pounce when we least expect it. The pesticides that make more food available to feed the world population are a potential hazard to those exposed to them.

Two major sources of electrical energy, coal and nuclear, could produce disastrous effects. Carbon dioxide emitted by coal plants could produce global warming. Acid rain from coal plant emissions could have devastating effects on food crops. A nuclear reactor meltdown accident of the magnitude of Chernobyl could affect millions of people. Yet we still demand electricity because it not only makes life easier but it also makes life safer.

The use of technology in a capitalistic society depends on the demand for that technology. However, as a society, we also demand that it be safe. But what is safe? Nothing is totally risk free so we cannot demand that we have zero risk; that would banish all technology. However, we can demand that the risks be small and somehow tied to the benefits.

We seem to accept the risk from driving cars because the benefit of individual freedom of movement is almost like an amendment to the Constitution. Why do we have speed limits on most of our roads? At speeds above these limits we know that more and more severe accidents occur. We, as a society, are not willing to accept those higher death rates. Why do we accept the death rate for 65 mph (or higher in some states)? Should we set the speed limit at 45 mph? Twenty-five mph? We know that at the lower speeds the fatality rates will decrease. Apparently we feel that the loss of benefit (i.e., the ability to get somewhere quickly) is too much to pay for the lower risk of death.

If we are not willing to change the speed limits to decrease vehicle death, then what can we do? We can make a better effort to keep drunken drivers off the road. We can make roads safer by careful redesign and construction. We can get people to wear seat belts and motorcyclists to wear helmets. The required use of seat belts and helmets approaches the line of individual freedom. In our society where everybody shares the cost of accidents through insurance coverage, the "right" of the public to control how their money is spent supersedes the "right" of motorists to not wear seat belts and helmets.

What about the emissions from an industry that uses or manufactures a toxic substance? Do they have a right to emit anything into the air and water, or does the public have a right to breathe clean air and drink clean water? If we deem that the public has the right to clean air and water, can we just tell the manufacturer that they must have zero emission of anything that could be toxic? With very few exceptions, this would be impossible and many industries would simply not exist. So we set emission standards. We say that the risk from small amounts of a toxic material in the environment is worth the benefits derived from the product manufactured by that industry.

Clearly, society is becoming ever more dependant on technology. This technology, although bringing great benefits, can also bring harm, even potential disaster. To be sure the public can make judgments about both the benefits and the risks, the public needs to understand just how a technology acts and what are the actual risks and benefits. Presently the state of scientific knowledge of the public is far from adequate. Even at the crucial institution in our democratic society, the U.S. Congress, this knowledge is lacking.

A former member of the U.S. Congress, Mike McCormack, commented on the lack of understanding by the public:

> During my twenty-four years' service as an elected official, one of the great and repeated frustrations I experienced occurred as I worked with many of my colleagues who were undereducated about -- literally ignorant of -- the fundamental scientific or mathematical realities required for the realistic consideration of policy issues involving such subjects as environmental protection, energy production, and basic research among others.
>
> This inability to handle such issues rationally constitutes a dangerous malaise that is pandemic across our nation. It afflicts all too many public officials, members of the news media, and the entertainment industry. It has, most disturbingly, afflicted much of the educational community, where we would traditionally have expected to find high standards of excellence and intellectual vitality.
>
> For instance, a faculty member of a major university recently related to me an incident in a chemistry class for non-science majors. The students were asked for their suggestion to having a nuclear power plant in their region of the state. The reaction was essentially unanimous and vigorously negative. They were asked who could define or describe radiation. Of a large class of university students, only three hands were raised, and those tentatively.
>
> The message this incident brings to us one that should be disturbing to all thoughtful citizens. These students had been frightened into a state of irrationality without the slightest understanding of the feared subject....
>
> This phenomenon provides a frightening demonstration of the fact that of all the ancient enemies of mankind, ignorance is the most menacing -- the most dangerous. It is the insidious source of fear and superstition -- the vehicles for the enslavement of human minds. (McCormack 2)

Radiation has been, is, and always will be a part of our lives. We can choose to hide our heads in the sand when a radiation issue surfaces. We can "just say no" and hope that it will go away. But it can't go away. The use of radiation does have benefits. We need to decide just when those benefits outweigh the risks.

However, we cannot even start to decide if the public begins any discussion with an abject fear of radiation. No amount of reason will convince someone that a small increase in radiation is worth any benefits if that person has a total fear of radiation. This is like trying to convince someone who has a fear of heights to observe the Grand Canyon from the edge of the canyon.

The purpose of this book is to decrease the public's ignorance of radiation. I am not trying to convince anyone that all nuclear and radiation activities are good. People must make up their own minds about that. Nor am I trying to make the public apathetic to radiation. That is worse than fear.

I just want to give the public the tools to face a radiation issue with a firm knowledge of what is true and what is not. Reacting on the basis of fear, as described later in this book, can lead to disastrous results.

Chapter 1
Radiation: What is it?

The movie The Day After depicts a nuclear weapon dropped over a city in the United States. At the time of the explosion, the people were exposed to radiation. However, this radiation is invisible and there was no way that anybody could see it hitting the people. However, would such a technicality stop Hollywood? No, they must come up with something to make the movie more dramatic.

At the time of the explosion, the movie shows people exposed to a bright light then you see their bones as you would see them in an X-ray picture. The producers obviously knew that the weapon produced radiation and that X-rays are forms of radiation. Well, X-rays (and gamma rays from a nuclear weapon) do penetrate the body. But to see an "X-ray picture," the X-rays must hit a special film. What the movie showed could not happen in reality.

Because of radiation's mysterious nature, the entertainment industry likes to use it to generate unnatural situations for entertainment. Movies, television programs, comic books, etc., are filled with incidents involving radiation. Unfortunately, most situations used by the entertainment industry present an incorrect picture of what radiation does.

Radiation does not cause insects or other small animals to grow into giant creatures that attack people. A giant insect with the same proportions as a normal insect would collapse under its own weight. (Think about the size of the legs of an elephant. They have to be large to support the weight of the body.)

Radiation does not cause someone to turn into a green muscular person when aggravated. It does not cause cute little turtles to mutate into human-sized creatures who eat pizza. The entertainment industry does not give a true picture of the effects of radiation. Therefore, forget all that you have learned from the movies, television, and comic books, and start with a fresh mind.

Radiation: what is it?

If a fire is nearby, we can see it, smell it, feel it, and maybe even hear it, so we know there is danger. Fire is visible energy. We can depend on our senses to avoid harm. But radiation is invisible energy. We cannot depend on our senses to detect its presence and we cannot use our senses to avoid exposure to radiation. This fact makes radiation a bit mysterious and therefore frightening.

Radiation has a variety of sources, some natural and some human-produced. First, let's be clear; radiation is radiation, regardless of the source. Radiation from natural sources (radon, cosmic, radioactive potassium in our bodies, etc.) is no more or less harmful than human-produced radiation (from nuclear power plants, medical treatments, etc.).

Radiation comes in two general forms: particle and electromagnetic. A particle is anything that has a mass; we can put them on a scale and measure their weights just as we can measure the weights of marbles, baseballs, and people. However, the particles about which we are concerned here are microscopic ones. They are the neutrons, protons, and electrons that make up an atom. Most particle radiation comes from radioactive materials.

Electromagnetic radiation has no mass. It is just pure energy. The human body can detect only two forms of electromagnetic radiation: visible light (we can see it) and infrared radiation (we can feel the warmth from it). However, the forms of electromagnetic radiation that are of concern to us are X-radiation (from X-ray machines used in the medical profession) and gamma radiation (from radioactive materials).

Atoms and radioactivity

The atom consists of three major constituents: protons, neutrons, and electrons. We picture protons and neutrons as little spheres, and there is strong evidence that they are spheres. They have a radius of about 10^{-15} meters (0.000000000000001 meters, or one billionth-trillionth of a meter). [See Appendix B for explanation of small and large numbers.] If we could line up a string of protons or neutrons, we could get about 250,000,000,000 across the head of a pin.

The proton and neutron have about the same mass: 1.7×10^{-27} kg. A tablespoon of water (about 15 milliliters) has a weight of 0.6 ounces and contains about 10^{25} (10 billion-trillion-trillion) neutrons and protons.

The electron is more elusive as far as size and shape are concerned. Although we picture an electron as a sphere, it is so small we do not have strong evidence of its shape. However, we do know the mass: 9.1×10^{-31} kg, which is 1800 times less than the mass of the neutron or proton.

The parts of the atom are too small to observe directly; nobody has actually seen a proton, neutron, or an electron. However, from a variety of experiments we have deduced that the atom consists of a relatively heavy nucleus with the electrons orbiting around this nucleus. The nucleus is a sphere and contains protons and neutrons.

Electrons orbit the nucleus much like the planets orbit the sun. Interestingly, the ratio of the sun diameter to the solar system diameter is approximately the same as the ratio of the nucleus

diameter to the atomic diameter. As you can picture in your mind, the atom consists of mostly empty space.

An important property of these particles is their charge: protons have a positive charge, electrons have a negative charge, and neutrons have no charge. As we all learned in elementary school, opposite charges attract and like charges repel. The attraction of the opposite charges (positive protons in the nucleus and negative orbiting electrons) is what keeps the electron in an orbit, just as the attraction of gravity keeps the planets orbiting the sun.

The protons in the nucleus all have the same charge so they repel each other. In addition, there are other repulsive forces that try to push the nucleus apart. However, there is a strong nuclear force that causes the neutrons and protons to attract each other. For most nuclei, the attractive force is the clear winner and the nucleus is stable; it will stay intact forever.

However, certain combinations of neutrons and protons are not stable; the repulsive forces are stronger than the attractive nuclear force. We call these nuclei radioactive. When the repulsive forces overcome the attractive nuclear force, the nucleus emits a particle (either an alpha or beta particle). When many of these radioactive nuclei emit a particle, they also emit gamma radiation.

Particle radiation

Particle radiation includes alpha particles, beta particles, fast-moving neutrons, and fast-moving protons. Fast-moving neutrons exist only in nuclear power reactors, in cosmic radiation above the atmosphere, and in radiation from a nuclear weapon explosion. We find fast-moving protons only in cosmic radiation.

Because neutron and proton exposure is rare (unless you are an astronaut or work in a nuclear power plant), I will ignore them in the discussion of particle radiation. However, neutron radiation was part of the total radiation received by the people in Hiroshima and Nagasaki. This population is the major source of information on radiation risk. Therefore, I will be discussing the effects of neutron radiation in the chapters on calculating risk.

Alpha particles are helium nuclei (two protons and two neutrons bound together by the nuclear force). **Beta** particles are just electrons. Both of these particles exist as radiation only for a fraction of a second after being emitted by a radioactive nucleus.

When a beta particle finally comes to rest, it is the same as any other electron. When the alpha particle comes to rest, it will gather two electrons that may be floating by and become a helium atom. Now it is indistinguishable from the atoms in any helium-filled balloon.

Electromagnetic radiation: x-rays and gamma radiation

Gamma and **x-radiation** are what we call electromagnetic radiation. Electromagnetic radiation takes some explaining. The word "electromagnetic" partially describes the radiation, but a lot is left out. Let's start with the magnetic part of the word.

We have all played with magnets; if not, you might find a supply on your refrigerator door. If you hold two magnets far apart, nothing noticeable happens, but as you bring them together you can feel a force between the magnets. Why is there a force? You cannot see anything connecting them. What you cannot see is a magnetic field generated by the magnet. These magnetic field lines are invisible lines of force.

Magnetic fields arise from moving charges. An electric current is just moving charges. In a wire these charges are electrons. We can make a magnet by wrapping a wire around a steel nail and connecting the wire to both ends of a battery to produce a current in the wire. (The steel nail helps to magnify the magnetic field.)

The interesting aspect about magnetic fields is that they exert forces only on moving charges; they do not affect charges at rest. If you put an electron on a table in the presence of a magnetic field, the electron will not feel any force. However, if the electron were moving on the table, it would feel a force.

Because a current is just many moving charges, a magnetic field will exert a force on a wire carrying a current. This is the principle behind electric motors. One set of coils in the motor produces a magnetic field from a current in the coil. This magnetic field exerts a force on another set of coils that has another current flowing through it.

Now to the "electro" part of electromagnetic radiation. We know that like charges repel and opposite charges attract. What causes the force between them? In this situation, there is an electric field. Like the magnetic field, the electric field is invisible, but we can observe its effects.

Any charge, whether moving or at rest, generates an electric field. When a charge is in the presence of the electric field of another charge, it experiences a force. In this case, the force is exerted on any charge, moving or stationary.

When you run a comb through your hair on a dry day, the comb attracts electrons so it becomes negatively charged. This negative charge generates an electric field that reaches out to something that has an excess of positive charges, such as a piece of paper. The electric field attracts the positive charges (which are in the paper) so the comb attracts the paper.

Electromagnetic radiation is a combination of electric and magnetic fields that moves through space at the speed of light (300,000 kilometers, or 186,000 miles per second). Visible light is

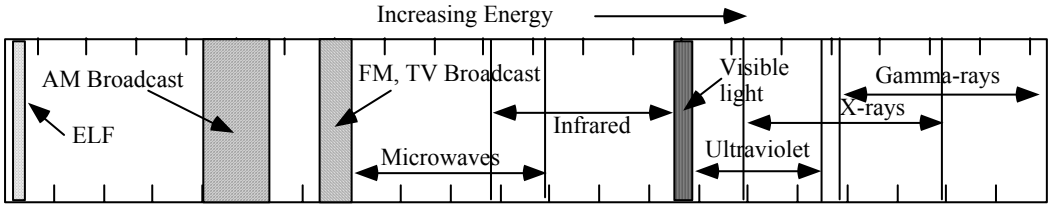


Figure 1-1

one form of electromagnetic radiation. Gamma rays are another. Electromagnetic radiation can have a wide range of energies. The energy of gamma radiation is about 100 billion billion times that of ELF's (Extremely Low Frequency; also called EMF) that come from electrical wires.

Figure 1-1 shows the electromagnetic spectrum. Note that there really are no clear separations from one type of wave to another, and there is always a gray area where one merges into the next. For example, X-rays and gamma rays are defined by their origin. Gamma rays come from the nucleus (from radioactive decay). High-speed electrons (generated in an X-ray tube) hit some special target material to produce X-rays. On the average gamma rays have more energy than X-rays, but some X-rays have more energy than some gamma rays.

The interesting aspect about electromagnetic radiation is that although they differ only in energy, they act in totally different ways. The most notable differences are the ways different materials absorb and reflect the waves. X- and gamma rays can pass though any thin material. Nothing reflects them. However, any material will absorb X- and gamma rays if it is thick enough.

Most solid materials and certain molecules in air (ozone for example) absorb ultraviolet waves. Only non-opaque materials (clear glass, plastic, etc.) are transparent to visible light (even more transparent than to gamma and X-rays). Any shiny surface reflects light. However, even a thin piece of opaque material can stop light. Solid materials, including glass and certain molecules in the air (carbon dioxide for example), absorb infrared waves while other materials can reflect them.

Water and many organic materials absorb microwaves. However, glass and most ceramics do not absorb them, so these materials are good for holding food in a microwave oven. But metals reflect microwaves. To keep the microwaves in the oven, the inside of a microwave oven is lined with a metal. The glass window has a metal covering with holes large enough to see through but small enough to reflect the microwaves. And microwaves, known also as radar, are good for reflecting off metal cars to measure the speed of cars on the highways.

Metals absorb radio waves so the antenna for a radio is made of metal. This property also means that radio waves do not enter a metal building so radio reception is not good inside buildings with lots of metal. Certain layers of the atmosphere that have free charges floating around reflect

radio waves. The ELF (extremely low frequency) waves are absorbed by metal but can pass though other materials easily.

Note that the only group of electromagnetic waves we can sense directly is visible light. We can see the effects of ultraviolet light; that is what tans our skin. If we were exposed to enough microwave radiation, we would feel the heat generated by the absorbed radiation. All the other groups of radiation are detected only by special equipment. Your radio and TV are examples of special equipment.

Penetratability

The most distinguishing factor among the different radiations is their ability to penetrate material. Alpha radiation (two protons and two neutrons together), although commonly the most energetic form of radiation, is the least penetrating. A single sheet of paper is enough to stop alpha radiation. The dead layer of skin stops alpha radiation. Alpha radiation travels only about an inch in air.

Beta radiation (fast-moving electrons) travels many feet in air; the exact distance depends on the energy of the beta particles. In tissue, beta radiation can travel up to a half inch. Beta radiation that originates outside the body cannot affect internal organs. However, if the source of beta radiation is inside the body (i.e., inhaled or ingested so it is spread around the body), then the beta particles can interact with any part of the body near the radioactive nuclei that produced them.

X- and gamma radiation are the most penetrating of all. The medical profession uses this property when taking X-ray pictures. X-rays penetrate muscle more than bone because tissue is less dense than bone. Because bone absorbs more X-rays than tissue, fewer X-rays pass through the bone. Fewer hit the X-ray film behind the arm, leg, or whatever is being X-rayed so when the film is developed, the parts of the film hit by fewer X-rays (i.e. behind the bone) are lighter than the parts behind the muscle.

Gamma radiation, because it generally has more energy that X-radiation, penetrates even more than X-radiation. Gamma rays easily penetrate the body, many passing through without interacting at all. Pipeline companies use gamma radiation to "X-ray" pipe welds.

In later chapters I will discuss the risk from radiation. The risk will be calculated from what we will call a whole-body dose. This means that each part of the entire body absorbs about equal amounts of radiation. The only kinds of radiation that can produce this kind of uniform absorption are gamma or X-radiation.

Chapter 2:
Radiation Dose

In the previous chapter, I described radiation. In this chapter I will discuss how this radiation interacts with matter and define the term "dose." Before we can define dose, however, we need to quickly review the basic structure of the atom.

As described in the previous chapter, the atom consists of neutrons and positively charged protons in the nucleus. Negatively charged electrons orbit the nucleus. The amount of the positive charge on a proton is exactly the same as the negative charge on an electron.

If an atom has six protons in the nucleus, these six protons will attract six electrons. In this situation, the atom has six electrons in orbit around the nucleus. Because the proton and electron have equal but opposite charges, the net charge on the atom is zero. We say that the atom has no net charge, or is neutral.

If, for some reason, the atom has six protons but only five electrons, the atom will have a net positive charge. Such an atom will attract an electron that happens to be nearby, either unattached or maybe attached to another atom. The addition of the sixth electron makes the atom neutral again.

If something hits the atom and removes one electron, the positively charged atom and the negatively charged electron are called **ions.** Together they are an **ion pair**.

Interaction with matter

When radiation interacts with matter, it interacts with the electrons orbiting the atomic nucleus. The charged particles (alphas and betas) attract or repel the electrons as they fly by and either pull or push the electron out of the atom. For example, the positively charged alpha particle attracts electrons and as it moves through matter it pulls on the electrons. However, the alphas travel so fast that electrons do not stick to the alpha particle. They just come loose from the atom thus producing an ion pair.

This is analogous to an adult quickly running past a crowd of small children, grabbing and letting go of hands in the crowd. The adult can pull children out of the crowd but never ends up with anybody from the crowd stuck to the adult's hand. However, with each grab the adult slows slightly, eventually coming to rest.

Beta particles, because they have the same charge and mass as the electrons (remember, betas are electrons), repel the electrons in the atoms. The beta pushes an electron out of the atom producing an ion pair (the ejected electron and the remaining positive atom). Here the action is analogous to a fast cue ball hitting balls on a billiard table. The cue ball can hit one ball, then glance off that ball and hit another ball. In each collision, the cue ball loses a small amount of energy and slows slightly. This process will continue until the cue ball eventually loses all its energy and comes to a rest.

When the cue ball hits the three ball, the three ball suddenly starts moving. This motion is evidence that the three ball now has energy. The collision transferred some energy from the cue ball to the three ball. In the process the cue ball slows a little which is evidence that it has lost energy. The three ball can also hit another ball, say the four ball which causes the four ball to move. Now the four ball has some of the original energy of the cue ball. This process continues until all the energy is spread around some or all of the balls on the table. Eventually friction will stop all motion. Then the original energy of the cue ball has totally disappeared.

This is exactly what happens to beta particles that interact with electrons. The beta hits an electron which moves off with a high speed. The ejected electron leaves the atom, creating one ion pair. The ejected electron hits another electron, creating another high-speed electron and an ion pair. Of course the original beta particle as well as the ejected electron can hit other electrons until they come to a stop. A medium-energy beta particle can create a total of 10,000 to 30,000 ion pairs.

Gamma and X-radiation also interact with the electrons orbiting the atomic nucleus. In this interaction, the X-ray or gamma ray knocks the electron out of the atom, again producing an ion pair. The gamma and X-ray loses most or all of its energy in the interaction. However, the electron with which the radiation interacts will have a large energy and will interact with electrons in other atoms exactly like the beta particle does.

Definition of Dose: rad and Gray

The number of ion pairs produced in some material is directly proportional to the energy absorbed by that material; it takes a certain amount of energy to ionize an atom. If 35 units of energy can ionize one atom, then 350 units of energy can ionize ten atoms. Therefore, a unit that measures the amount of energy absorbed would be a logical way to define a **dose**. The original definition of dose is the **rad** which is one hundredth of a Joule (0.01 Joules) of energy absorbed in one kilogram of material.

How much is a kilogram and what is a Joule of energy? One kilogram has a weight of 2.2 pounds. A liter of water has a mass of one kilogram, so a two-liter bottle of soda has a mass of two kilograms. A person who has a weight of 120 pounds has a mass of 55 kilograms.

Energy can be measured in several different units. One that may be familiar is the calorie. Unfortunately, one calorie can mean one of two energies. A "regular" calorie is the heat needed to raise the temperature of one gram of water by one degree Celsius. The "great" or "large" calorie, or what we physicists call a kilocalorie, is the calorie we use for food. It is 1000 "regular" calories. I am using the large calorie in this discussion.

In terms of Joules, 4186 Joules equal one calorie. It would take one calorie (or 4186 Joules) to raise the temperature of one kilogram (i.e., one liter) of water by one degree Celsius. One Joule can raise the temperature of a kilogram of water only 0.00024 degrees Celsius. It takes 160,000 Joules to heat a cup of coffee from 50°F to boiling.

Although one Joule is a small amount of energy, and 0.01 Joules is even smaller, one rad of dose can produce over 10^{15} ionizations in a kilogram of tissue. This may seem like a lot, but one kilogram of tissue contains over 10^{25} molecules (mostly water) so one rad will ionize only one atom in ten billion.

The earth contains five billion people. Let's represent each atom in one kilogram by each person on the earth. The world would represent one kilogram. With this representation, one-half rad of radiation would interact with only one person on the earth.

This might imply that one rad may not be dangerous. One rad of radiation dose might be considered a medium dose. As we will learn later, a person receiving a few rads at once shows no immediate detectable effects to the body (although there may be some long-term effects). A dose of 100 to 200 rad received at once can produce physical illness, and over 500 rad at once can be fatal.

To standardize units around the world, the rad has been replaced by the new S.I. (Systems International) unit of the **Gray** (**Gy**). A Gray is defined as one Joule of energy absorbed in one kilogram of material. One Gray is equal to 100 rad.

As we will see later, a Gray is a large dose. The public is never exposed to such large doses. The maximum dose to anyone near the Three Mile Island nuclear station during the 1979 accident was only about one-thousandth of a Gray. Only those in the Chernobyl nuclear plant received doses of a Gray or more during that plant's accident in 1986. The maximum dose to people in the areas surrounding Chernobyl was four tenths of a Gray.

To keep units uniform throughout the book, I will use one thousandth of a Gray, or milliGray, as the standard unit in the book. This is abbreviated as **mGy**. The corresponding unit in the "old" system is the millirad, or **mrad**. A mrad is one thousandth of a rad or one hundredth of a mGy. I will keep both systems. Whenever I express a dose, it will be in mGy with mrad in parentheses. A dose of 20 mGy would be expressed as 20 mGy (2,000 mrad).

Measure of Biological Damage: Sievert and rem

But does the rad or Gray tell the entire story about radiation damage? It may be a good measure of the number of ionizations, but not necessarily a good measure of biological damage.

To directly damage living tissue in the human body, radiation must interact with the atoms in the molecules that make up the tissue. Because living tissue consists mostly of water, the radiation interacts mostly with the water rather than with the atoms that make up the tissue.

The ionization of water can produce something called oxidants in the tissue water. Some of these oxidants, especially hydrogen peroxide, depend on two ionizations produced close to each other. (Hydrogen peroxide is a combination of two oxygen and two hydrogen atoms.) If the ion pairs are far apart, the ionized water molecule will just recombine with no oxidants produced. In the next chapter I will discuss the process of producing oxidants in more detail.

These oxidants can interact with and damage living tissue. Because most oxidants remain active for long times, they can move through the water and then interact with the tissue. This type of damage is called indirect because the radiation does not damage the tissue directly. The damage is done indirectly by the oxidants produced by the radiation.

What kind of radiation can produce many ion pairs in a small space? If you remember that the different kinds of radiation have different abilities to penetrate material, then we can deduce which kind produces the most damage. Alpha radiation loses its energy in a short distance (less than a millimeter in tissue). This short stopping distance means that the alpha interacts with many electrons in a short distance thus producing many ion pairs near each other.

Alpha radiation produces more oxidants per dose (rad or Gray) than beta, gamma and X-radiation. Thus, it produces more biological damage per dose than beta, gamma and X-radiation.

To account for this disparity in biological damage, scientists use the unit of **rem** ("old unit") or **Sievert** (S.I. unit, abbreviated **Sv**). This is called the **dose equivalent**. The units are related by a quality factor that depends on the type of radiation. The rem is a rad multiplied by the quality factor; the Sievert is the Gray multiplied by the quality factor. The quality factor for beta, gamma and X-radiation is one, so for these radiations one rad produces one rem and one Gray produces one Sievert.

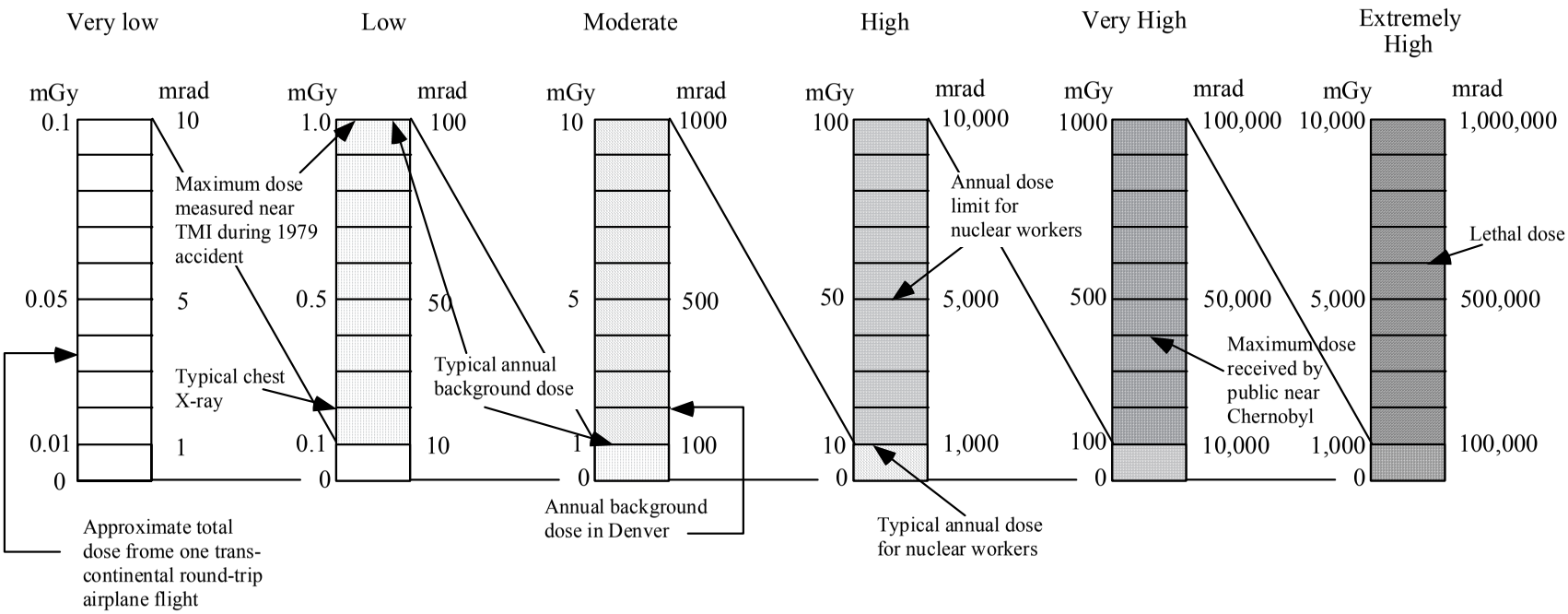
For alpha radiation, the quality factor is 20. One rad of alpha radiation produces 20 rem, or, one Gray produces 20 Sievert. Because of its high ion pair density, alpha radiation does 20 times the biological damage to living tissue than beta or gamma radiation for the same dose received. However, this value does depend on several factors such as the energy of the alpha particles, which changes as the alpha comes to rest. Therefore, the accuracy of the dose equivalent (rem and Sievert) is questionable in many situations.

For this book I will deal with mostly beta, gamma and X-radiation. Except a few isolated cases, we will not have to deal with the quality factor and the rem and Sievert units. When I discuss radon, which involves alpha radiation, I will use data that does not use dose to calculate the risk values.

The information on risks from radiation comes mostly from gamma and X-radiation doses. The most common forms of radiation the public receives from natural sources, medical procedures, and potential nuclear accidents is beta, gamma and X-radiation. Therefore, I will use only the units of Gray and rad throughout this book. These are measurable units and consequently are more reliable to use than the Sievert or rem which depend on a quality factor which is of questionable accuracy.

To put these units in some sort of perspective, let's consider some numbers we will see in later chapters. The average background dose from the earth, cosmic rays, and radioactivity in our own bodies is about one mGy (100 mrad) per year. A typical diagnostic X-ray gives about 0.2 mGy (20 mrad) to the part of the body examined.

Figure 2-1
Radiation Exposures



The maximum radiation dose a radiation worker can receive is 50 mSv (5,000 mrem) per year. However, the average annual dose actually received by nuclear power plant workers is about 10 mSv (1,000 mrem). Here I have used dose equivalent units of Sievert and rem. Because a nuclear plant worker can also receive radiation doses from alpha and neutron radiation, the dose limits are given in dose equivalent units. However, most of the dose received is from gamma and beta radiation, so the average dose is about 10 mGy (1,000 mrad).
Figure 2-1 shows the range of doses we will consider in this book. I have included some values for reference. Note that each succeeding block of doses, from left to right, is ten times the prior block.

Effective Dose and Population Dose

Dose is the energy absorbed per mass. However, a person who receives an X-ray dose over the trunk of the body will have more biological damage than a person who only has a hand X-rayed. Each may receive the same dose (i.e., the same energy per mass), but one has more of the body receiving that dose. How can we make the two somewhat equivalent?

In this situation, we use appropriate weighting factors to account for differences in the extent of the dose to give an effective dose (effective to the whole body). These weighting factors depend not just on the ratio of the mass, but also on the sensitivity of the organ receiving the dose. For example, the weighing factor for the lungs is 0.12. A one-Gray dose to the lungs has the same effect as 0.12 Gy to the entire body. So, an annual dose equivalent of 10 mGy to the lungs is effectively the same as 1.2 mGy to the entire body.

When we discuss radiation dose to a large population, we can either use the average dose or a total dose to that population. We will see in later chapters that radiation increases the probability of getting cancer. Therefore, to determine the risk to a population the total dose is a useful quantity. The total dose is given in **person-mGy** (**person-mrad**). To determine the total dose, we just add up the doses received from each person in the population. For example, if each of 100 people receive 2 mGy, and each of 70 people receive 0.5 mGy, then the population dose would be 100 X 2 mGy + 70 X 0.5 mGy = 235 person-mGy.

However, this unit is only useful only if the effects from a radiation increase linearly with the dose. Linearity means that if the dose doubles the effects double; if the dose increases by a factor of three, the effects similarly increase by a factor of three. As I will discuss in detail later, radiation effects may not increase linearly with dose, so the concept of person-mGy may not be an appropriate unit to use for large population doses.

Roentgen

One of the first attempts to quantify the amount of radiation was the definition of exposure. The exposure is a measure of the radiation energy coming from a radiation source. Historically exposure is defined for gamma or X-rays only, not beta and alpha radiation. The actual definition is based on the number of ion pairs produced in air.

The unit of exposure is called the **Roentgen** (abbreviated as **R**), named after W. K. Roentgen who discovered X-rays. One Roentgen of exposure of gamma or X-radiation produces almost one rad in tissue. So, we can assume that any person in an area that has an exposure of 1 R, will receive a dose 1 rad (10 mGy). I will not use this unit in this book, but you may see it in other references.

Chapter 3
Biological Effects of Radiation

All matter consists of atoms. A piece of aluminum contains only aluminum atoms. Charcoal contains only carbon atoms. Living tissue consists of many different types of atoms, but mostly carbon, hydrogen, oxygen, and nitrogen. Living tissue also contains many water molecules (hydrogen and oxygen).

When radiation interacts with a piece of aluminum, the radiation ionizes a few of the aluminum atoms. However, the electrons that leave the aluminum atoms eventually return to the aluminum atom from which they came, or maybe to another atom that lost an electron. After all is said and done, the piece of aluminum is just as it was before the radiation hit it.

When radiation hits living tissue, however, the story is different. Because living tissue consists of many different kinds of atoms that form long and complicated molecules, the recovery from the ionization may not be complete.

Atoms join to form molecules. For example, a water molecule consists of two hydrogen atoms and one oxygen atom. A methane gas molecules consists of one carbon atom and four hydrogen atoms. The DNA molecule is a long chain of millions of carbon, hydrogen, nitrogen, oxygen, and phosphorous atoms. For the DNA molecule, the location of each atom in the molecule is critical to the function of the molecule.

All molecules are bound together by the electric forces between the atoms. This binding is stable until some external force disturbs it. Some examples of external forces include heat, an electric current, and radiation. Heat applied to methane, in the presence of oxygen, will cause methane to burn. In the burning process, the carbon and hydrogen atoms join with the oxygen atoms, producing heat, water (oxygen and hydrogen) and carbon dioxide (carbon and oxygen). An electric current passing through water can separate water into its constituent gases hydrogen and oxygen. Radiation can cause water to break apart or change the order of the molecules in the DNA molecule.

DNA, Cells, Organs, and Body

Before we get into the process of biological damage, we need to discuss the function of different parts of the body. The human body consists of organs (muscle, heart, lungs, etc.) that each have a specific function to keep the body working. Each organ is made of cells and each cell has a specific function in the organ. For example, the lung has some cells that have the sole purpose of transferring oxygen from the air to the red blood cells. Other cells in the lung move dust from the lungs up the esophagus. While others just supply a support structure for the lung (i.e., the tracheal and bronchial tubes).

Each cell contains, among other things, DNA. The DNA is the "control room" of the cell. It dictates how the cell functions. In addition, DNA is in charge of reproducing new cells when needed. We might liken the DNA-cell-organ-body interconnections to society. Society as a whole (a country or maybe just a state) would be the body. It "lives" because various parts (i.e., organs) are doing specific functions. One "organ" might be the (only) factory that makes cars. Another might be the farmer-food processor-grocer system that provides food for the body. Under this analogy, I guess the brain would be the governing system.

The auto plant consists of "cells" that carry out specific functions in the car manufacturing process. Some cells are the assembly line, other cells are the parts manufacturing groups, etc. But each cell has someone in control, or the DNA. This could be the supervisor. The supervisor decides how the cell (i.e., the workers) work to produce parts or the finished car.

Suppose something happened to one of the parts supervisor and the workers in that section. That segment of the manufacturing process would stop. But there are more than one group (or cell) making parts. Another supervisor would then direct the process of replacing the lost parts section. Cars would continue to come off the assembly line without interruption.

But what happens if something happened to many supervisors, so many that other sections could not carry out their functions and attempt to replace the lost sections. Then the manufacturing process would grind to a halt. Because society depends totally on cars, society (i.e., the body) would not survive.

In a society, there are always losses of supervisors. Some change jobs, some retire, and some die. But the process allows for replacements with no harm to the manufacturing process. Likewise, cells in the body die, some from "old age" and some because of certain chemicals or other agents such as heat, cold, or radiation. But life goes on. The living cells reproduce making new cells to replace the

dying ones. Only when some agent overwhelms the system (poison, too much heat, too much cold, etc.) will the organ and, consequently, the body succumb.

Chemical Changes and Radiation Damage; Oxidants

A typical cell consists of about 70 percent water, so water absorbs most of the radiation that interacts with living material. A water molecule consists of two atoms of hydrogen and one atom of oxygen. When ionizing radiation hits a water molecule, it removes an electron from it. If the missing electron is one that forms the bond between one hydrogen atoms and the oxygen atom, the hydrogen atom (just a proton now) becomes detached from the oxygen atom.

Through a series of interactions, the hydrogen atom, the remaining oxygen-hydrogen molecule, and the free electron create one or more molecules called oxidants. The most notable oxidant is hydrogen peroxide (two hydrogen atoms with two oxygen atoms).

Oxidants are compounds that are in search of an electron. When an oxidant contacts a molecule it will grab an electron and consequently can change the makeup of that molecule. Oxidants chew up organic molecules (those found in living matter). We use hydrogen peroxide as a bleach because it attacks the organic materials in the color dye thus causing fabrics to lose their color. It is also used to kill many viruses and bacteria.

Radiation is not the only substance that produces oxidants. Many chemicals that we ingest, both natural and man-made, produce oxidants in the body. The human body also produces oxidants.

What is the harm in having oxidants in the body? In the cells of living tissue, the oxidants can attack the DNA molecules with one of three consequences. First, the DNA molecule can just repair itself. This is the most common result. When it does, there is no effect from the oxidant reaction.

Second, the oxidant can destroy the DNA molecule. Now the DNA molecule can no longer perform its prescribed functions and the cell becomes useless. However, if other cells are not affected, they can replace the lost cell and there is no effect from the oxidant reaction.

The third consequence is that the oxidant alters the arrangement of the DNA molecule so that it can still function but not function properly. The DNA molecule may produce the wrong protein or enzyme. If cells of the small intestine do not produce the proper enzyme, some food is not digested and the body receives less nutrition.

When a cell divides, the DNA sets the pattern for the new cell. If the DNA of a cell is damaged so that the cell functions improperly, any new cells it produces will also function improperly. If enough cells in an organ are damaged over time, then the organ will not function properly.

Biological Effects from Radiation

We know that direct radiation, and the oxidants produced by radiation, affect the DNA molecule. However, we do not know what happens next to cause the observed effects. The process from DNA damage to observable biological effects is a mystery. We know that it happens, we just don't know how.

How do we know what radiation does? For the answer to this question we need to turn to statistics. Suppose a hospital emergency room were to receive a sudden increase in the number of stomach problems and all the people who had this problem had just eaten at the same restaurant. Any amateur Sherlock Holmes could deduce that some food in that restaurant contained something to cause the illnesses.

Similarly, if some or all of a group of people exposed to radiation exhibit specific biological problems, then we can deduce that radiation caused the problems. The effects of low doses of radiation, though, are hard to detect because other agents can cause the same effects. In these cases, we need detailed information about the magnitude of the radiation dose and careful analysis of the effects. Sherlock Holmes will need many Watsons to get the necessary data to determine if the effects are really from radiation or from another agent.

Radiation effects fall into three broad categories: **acute somatic**, **delayed somatic**, and **genetic**. Somatic means that the effects are to the person who received the dose. Genetic means that

the effects appear in the offspring of the irradiated person. Genetic effects could show up in the next, or even later, generations.

An acute radiation dose is a dose received in a short time, within a few hours to a day, and the effects appear within hours to weeks. Such doses can be received by workers if they accidentally walk into a high radiation area, by a medical patient undergoing radiation therapy, or by individuals near a nuclear weapon's explosion.

Delayed refers to the effects that are observed years after the radiation exposure. Radiation causing delayed effects can be received within a short time or over a period of years.

Acute somatic

As long as large radiation sources exist somewhere, there is a possibility of an accident or a willful misuse of radiation. I do not expect any member of the public to suffer what I am about to describe. Even during the Chernobyl accident, the only persons who received such large doses were the nuclear plant workers and the pilots who dumped sand and water on the burning reactor. People living near the plant received large doses to the thyroid gland from radioactive iodine, but not a large dose to the whole body.

Knowing the effects of radiation on the cells, we can make some predictions about what physiological effects we might observe. If radiation affects the DNA, then we would expect an impairment of the DNA's function. DNA's two major functions are to produce enzymes and proteins used in certain biological processes, and to produce new cells.

Blood cells do not have DNA, so radiation does not affect them. The cells in the blood remain even after a large radiation dose. However, blood cells do not live forever. They are constantly being replaced by the bone marrow which does have DNA, so radiation can affect the production of the red and white blood cells. A large radiation dose -- above 3,000 mGy (300,000 mrad) -- reduces or completely stops the production of new cells.

The typical lifetime of a white blood cell is about a week. A red blood cell has a lifetime of about 17 weeks. Therefore, in a week or two after a large radiation dose, the number of white blood cells in the blood is greatly reduced and the number of red blood cells is decreasing. In addition, platelets, the part of the blood that controls the clotting of blood, have a lifetime of about two weeks, so they are also greatly reduced.

Another organ that has constant cell activity is the small intestines. This organ digests our food. If radiation affects the small intestines, then the ability to digest food can be impaired.

Nerve cells are permanent fixtures in the body; they do not produce new cells and do not produce any specific enzymes or proteins. We might expect that nerves are less sensitive to radiation than the bone marrow and the small intestines.

In fact, the effects from acute exposures do follow what we expect. Table 3-1 gives the effects from an acute exposure to whole body doses of gamma radiation. The effects to any one person are not predictable. However, if a large population receives the radiation, then we can predict what will most likely happen to groups of these people.

Table 3-1: Acute Radiation Effects

Dose		Effects
mGy	(mrad)	
0-250	(0-25,000)	No immediate detectable effects
250-1,000	(25,000-100,000)	Decrease in red and white blood cell count. At the upper range some may have nausea.
1,000-3,000	(100,000-300,000)	Mild to severe nausea, vomiting, possible infections. At the higher levels some may not survive.
3,000-6,000	(300,000-600,000)	More severe nausea, vomiting plus hemorrhaging, diarrhea, infection, loss of hair, and temporary sterility. Between 4,500 and 5,000 mGy (450,000-500,000 mrad) about half of those exposed will die within 30 days; others will survive.

>6000	(>600,000)	Same effects as above plus central nervous system impairment. Death within 30 days. Above 10,000 mGy (1000 rem), incapacitation.

Acute radiation effects from a 3,000 to 6,000 mGy (300,000 to 600,000 mrad) dose follows a four-stage pattern. The first period, lasting about 48 hours, is characterized by nausea, a general tired feeling, and loss of appetite. These symptoms go into remission during the second stage and the person feels well for 48 hours to about two weeks.

During the third stage, infections begin to appear because the loss of white blood cells makes it difficult to ward them off. Hemorrhaging may take place with few platelets to initiate clotting, and intestinal problems may develop. At the higher doses hair may fall out and a fever may develop. If a person survives for 60 days, then that person will probably recover. We call a dose of 4,500 to 5,000 mGy (450,000-500,000 mrad) the LD50/60 dose: lethal dose (LD) to 50 percent of those receiving this dose. The deaths occur within 60 days.

The recovery depends greatly on the amount of bone marrow affected. If radiation kills all the marrow, then the person has no chance of survival. However, even a small amount of bone marrow can regenerate more marrow in a few weeks. If the person lives for 60 days after the radiation dose, the new marrow will produce enough red and white blood cells so the person will survive.

The treatments of leukemia and some cancers produce these effects on the patient. One method to treat leukemia is to irradiate the entire body with a lethal dose of radiation to kill the leukemia cells in the bone marrow. This dose also kills the bone marrow, which would be fatal to the patient. However, before treatment, physicians find a matching bone marrow donor. After the radiation kills the bone marrow (and leukemia cells), the physicians replace the bone marrow with some from the donor. These patients then suffer from nausea, vomiting, hair loss, and possible infection.

Acute somatic effects appeared in people exposed to high levels of radiation in Gioânia, Brazil. In September 1987, junk dealers found an abandoned medical teletherapy unit that contained a large source of radioactive cesium. They took the source home and tried to break it open but were unsuccessful. After about three hours the men became nauseous and were vomiting. One man had diarrhea. Another junkman, who received the source a few days later, noticed that the source was luminescent. After keeping it in the house for a few days, the junkman broke it up and shared it with relatives and friends.

Fifteen days after the source had been found, the junkman's wife noticed that many people had health problems so she took the source capsule, on a public bus, to the local sanitary office (Oliveira 17-24).

During this entire process, many people received significant radiation doses. Scientists used blood changes to estimate the radiation dose to 20 people who showed symptoms of radiation exposure. Four of these people died. The estimated doses to these four people ranged from 4,500 to 6,000 mGy (450,000 - 600,000 mrad). Two people who received estimated doses of 6,200 and 7,000 Gy (620,000 and 700,000 mrad) survived. The rest of the people had doses of 4,400 mGy (440,000 mrad) or below. The only symptoms of most of these people were skin burns (Brandão-Mello 31-39).

Let me reiterate that it is highly unlikely that anybody in the public would receive any such large dose. Except for cancer and leukemia treatments, radiation received for medical diagnostic and therapeutic procedures will not produce these effects.

Delayed Somatic Effects

Scientists know of only six documented delayed effects of radiation exposure: solid tumor cancer, leukemia, degenerative changes and life shortening, cataracts, birth defects, and sterility.

Leukemia and solid tumors are both forms of cancer. Because leukemia is a cancer of the bone marrow or lymph glands, it has some different characteristics than other cancers so we generally treat the two types of cancer separately.

Cancer: solid tumors. From the survivors of the atomic bombing in World War II, we know that large radiation doses produce a measurable increase in the incidence of solid tumors. This increase in the number of solid tumors does not appear until about 10 years after the exposure. This time from exposure to the onset of cancer is called the latency period.

Not everybody who is irradiated develops solid tumors. Irradiation does not mean that a person will develop a solid tumor, it only means that the probability of a solid tumor increases.

The development of cancer is a two-stage process. The first stage, initiation, requires some mechanism that can change the nature of a cell. To make this change, some agent must break a bond in a molecule. Possible agents include a whole host of carcinogenic chemical compounds as well as radiation. The initiator needs to act only once; once the damage is done, then that ends the initiation process.

The second stage, promotion, requires the presence of another (or the same) agent that helps to further the process of cell change. This agent does not need to break bonds, but it must have a continued presence. Scientists think that the promotion process is the reason for the latency period. Radiation seems to act as a promoter as well as an initiator. (Alpen 263)

The major controversy about radiation causing cancer is not whether it causes cancer, but how much radiation increases the probability by a measurable amount. Because cancer is responsible for about 20 percent of the deaths in the United States, trying to detect a small increase from radiation exposure can be difficult.

Although radiation is a known carcinogen, it is far from the greatest cause of cancer. Of all the cancers observed in the United States, about 35 percent are a result of diet, 30 percent from tobacco, ten percent from infections, seven percent from sexual behavior, four percent from occupations, and three percent from alcohol. Pollution contributes about two percent. Radiation -- from natural, medical and occupational sources -- contributes about another 2 percent (Doll and Peto 1256). These numbers have large uncertainties, but they do show that radiation is a minor cause of cancer.

Radiation is not equally effective in causing all types of cancers. Table 3-2 gives the relative sensitivities of the different kinds of solid tumors and leukemia to radiation.

Cancer: Leukemia. When radiation is the cause of leukemia, the latency period is between two and four years. The exact time depends on the age of the irradiated person. After this latency period, a person is at risk of getting leukemia for 25 to 30 years. After that time the risk of leukemia from that radiation becomes zero.

Leukemia follows the same initiation/promotion steps as solid tumor cancers. Like cancer, exposure to radiation increases the probability of getting leukemia, but does not make it a certainty.

Table 3-2
Sensitivities of various solid tumors and leukemias to radiation

Type	Spontaneous incidence	Radiation sensitivity
Female breast	Very high	High
Thyroid	Low	Very High, especially females
Kidney and bladder	Moderate	High
Ovary	Moderate	High
Lung	Very high	Moderate
Liver	Low	Moderate
Brain; nervous system	Low	Low
Bone	Very low	Low
Skin	High	Low
Prostate	Very high	Low
Uterus and cervix	Very high	Very low to not observed
Pancreas	Moderate	Very low to not observed
Testis	Low	Not observed
Leukemia:		

Acute myelogenous	High	Very high
Chronic lymphocytic	Low	Not observed

Adapted from National Academy of Sciences (1980) 266-267, but modified based on United Nations Scientific Committee on the Effects of Atomic Radiation 147

Degenerative effects and life shortening. Radiation affects the ability of cells to reproduce themselves accurately (or at all). After years of exposure to radiation, the cells may not reproduce in sufficient number. Or, the cells may not reproduce with the needed accuracy to keep the organ functioning properly. As the organ degenerates, it is not able to fulfill its function in the body and can contribute to an early death for the individual.

Most of the information on these effects is from animal studies and large occupational and medical exposures. However, no accurate data is available to quantify the effect. In fact, further analyses of the animal data suggest that the life shortening observed may have been the result of an accelerated onset of tumor growth (National Academy of Sciences (1990) 363).

Cataracts. A cataract is the clouding of the eye lens caused by abnormal lens fibers. The minimum dose needed to cause cataracts is about 2,000 mGy (200,000 mrad) in a single exposure or about 4,000 mGy (400,000 mrad) over a long period. A person may or may not get cataracts from a single dose to the eye between 2,000 and 8,000 mGy. Everyone who receives above 8,000 mGy in a single dose to the eye will get cataracts. Cataracts from radiation appear between six months and 35 years after the radiation exposure. (Jorgenson 179-180)

This type of response is different from that for cancer or leukemia. The probability of cancer incidence increases with dose, but there is apparently no minimum dose nor is there a dose that produces cancer in everybody. The response of cataracts to radiation is called a **threshold** effect; there is a threshold below which there is no effect and above which everyone is affected. However, the opacity of the cataracts does depend on the dose. In addition, cataracts induced by radiation develop in a way that is distinctly different from those resulting from other causes.

Birth defects. Because radiation affects the ability of cells to reproduce properly, cells that are rapidly dividing are the most sensitive. During the gestation period, the time from fertilization to birth, the body is going through its greatest cellular reproduction. Therefore, the embryo and fetus are sensitive to radiation damage. The degree of sensitivity of each organ depends on the exact time during gestation that the dose is received.

The effects from radiation depend greatly on the stage of fetal development at the time of the radiation exposure. During the first two weeks the embryo consists of a few cells and is going through the process of implantation. No organs are developing during this time. If the embryo receives a radiation dose during the first two weeks, the embryo either dies or is not affected. Based on data from rats, a dose of about 1,000 mGy (100,000 mrad) is needed to produce an appreciable possibility of embryo death.

The next month (weeks three through six) is the time of greatest cell activity, and the time when the organs are being formed. At this time the organs consist of only a few cells and if radiation destroys some or all of these cells then that organ will not develop, or will develop abnormally. The fetus is at the greatest risk for suffering congenital abnormalities during this time. However, the doses needed to cause death or abnormalities during the first few weeks of pregnancy is about 100 to 1,000 mGy (10,000 to 100,000 mrad) with nearly a 100 percent change of death or abnormality at the 1,000 to 2,000 mGy (100,000-200,000 mrad) range (Alpen 222).

After the organs reach a stage where they have many cells, they become less sensitive to radiation. Therefore, after the first six to seven weeks of gestation, the fetus is less likely to suffer from congenital abnormalities. From this time to birth the effects of radiation exposure diminish and are not as conspicuous.

Effects such as lower birth weight, decreased physiological activity, and lower mental abilities may fall within the normal range of these conditions. We may not observe them as radiation induced. Again, to produce these effects, the radiation dose must be more than a few hundred mGy (tens of thousands mrad) -- a dose mothers in the public do not receive.

Radiation is not the only cause of birth defects. About seven percent of all births involve some kind of abnormality arising from natural risks during pregnancy. These defects appear as developmental abnormalities, growth retardation, and chromosomal abnormalities. Viral infections, such as German measles (rubella), can also cause birth abnormalities. As with radiation, the embryo is the most sensitive to the virus during the first few weeks of pregnancy.

In addition, certain chemicals (usually in medication) can cause birth defects. Thalidomide, used as a sedative in the 1950s, caused many birth defects in Europe.

Compared to natural risks, the risk of radiation is only significant if the dose to the fetus is more than 1,000 mGy (100,000 mrad). This is a dose that nobody in the public can expect to receive.

Sterility. High doses of radiation can cause sterility in humans. Temporary sterility in males can occur at doses above 2,000 mGy (200,000 mrad) and permanent sterility in males or females at doses above 8000 mGy (800,000 mrad) (Turner 231).

Genetic effects

Genetic effects arise from damage to the DNA in the reproductive cells. The arrangement of these DNA molecules determines the physical makeup of the offspring and, if it is changed, the offspring can be born with one or more mutations. If the parent passes the damaged DNA onto the offspring, then successive generations may also have these mutations. Most mutations are detrimental to the offspring, though they can be beneficial if it gives the offspring an advantage in its competition for the basics of life.

Mutations can appear in many forms including physical abnormalities, different mental abilities, and different physiological characteristics (changes in metabolism, ability to utilize different foods, etc.). Physical abnormalities include color blindness, hemophilia, Down syndrome, club foot, cleft palate, spina bifida, congenital heart disorders (such as heart murmur), extra fingers or toes, and skin blemishes. Anything that can damage DNA can cause genetic damage; radiation is just one of many potential causes.

The actual amount of damage per radiation dose is difficult to determine because large groups of people need to be exposed and then successive generations studied. Intentional exposure to humans is not acceptable so we must depend on animal data and on the Japanese survivors of the atomic bombs.

The animal data are quite extensive and the genetic effects on mice are well documented. From these data we can extrapolate to humans to quantify the human genetic risk. However, the data from the Japanese bomb survivors suggest that the extrapolation from animal data overestimates the damage to humans (National Academy of Sciences (1990) 69). I will discuss the actual values derived for genetic risk in a later chapter.

Hormesis

Can radiation be beneficial? We use radiation for beneficial effects such as cancer treatment, but can radiation produce some beneficial effects?

To determine the deleterious effects of radiation I discussed in the previous sections, we observe the effects from humans or animals that are intentionally or accidentally exposed to radiation. Because the effects are hidden among a sea of the same effects from other causes, good data come only from those experiments that use large radiation doses (order of magnitude of thousands of mGy, or hundreds of thousands mrad). However, virtually all of the radiation doses received by the U.S. population are low doses; background radiation is a few mGy (few hundred mrad) per year and even radiation workers generally receive less than 10 mGy (1,000 mrad) per year.

We can also compare two large populations, one exposed to “normal” levels of radiation and one exposed to slightly higher levels. Scientists have done several studies on large human populations. These studies compare a population living in an area that has high background radiation with a similar population living in an area that has low background radiation.

Several studies have been done on large population of animals (mice, dogs, fish) exposed to low levels of radiation and comparing them to populations of animals not exposed to any additional radiation. The conclusions from some of these studies say that annual radiation doses between 1 mGy (100 mrad) and 10,000 mGy (1,000,000 mrad) are beneficial, with about 100 mGy (1,000 mrad) per year being optimal. Some of the observed benefits are increased growth rates, greater fertility, increased life spans, and greater immunity to disease (Luckey 239).

At levels below 1 mGy (100 mrad) per year (which is about the average background), the effects are deleterious; i.e., we need some radiation to maintain the status of health we do have (Luckey 239).

At this time I am not saying that hormesis is a valid theory. I have just reported what some researchers have found. When I discuss all the studies on radiation effects in later chapters, I will put the hormesis studies in context with the other studies.

Chapter 4
Evidence for Radiation Effects

In the previous chapters we discussed the basics of radiation: what it is, how it interacts with matter, and what it does to living tissue. There is little controversy about these topics. All scientists who are knowledgeable about the effects of radiation agree, maybe with some small differences, on the effects of large acute doses of radiation. However, very few people are exposed to high levels of radiation. Even from the Chernobyl accident in Ukraine, where many people received high doses of radiation, the vast majority of people received doses that we consider as low.

Low Dose

What do we mean by "low" dose? Although there is no definite definition, low doses have a magnitude about that of background radiation, a few mGy (few hundred mrad). Background radiation itself would be considered a low radiation dose.

The most concern is for radiation exposures from sources other than normal background. These exposures include the radiation received by those who lived near the Three Mile Island Nuclear Station during its accident in 1979, those in Europe and Asia far from the site of the Chernobyl reactor in 1986, those exposed to fallout radiation from nuclear testing in the 1950s and 1960s, and those who live in areas that have background radiation levels (including radon) two to three times higher than average.

How do we determine the risk from low doses of radiation? The typical method would be to expose subjects to known amounts of radiation and observe the effects. However, this method suffers from two major problems: first, ethics dictate that nobody should intentionally expose humans to radiation and, second, other effects mask those that we observe at low doses.

One alternative is to use animal studies: for example, expose mice to radiation and observe the effects. This is done and the results have been valuable for understanding some mechanisms of radiation effects. But humans are not mice and the effects we observe in mice are not necessarily the same that we would observe in humans.

What data do we have on humans at low doses? Not much. We do have data on the effects from high doses, but using it for low-dose effects is not easy. The use of high-dose data to predict effects at low doses is the controversial part of the whole process of risk determination. In this chapter, I will discuss the studies that provide information on the risk at high doses. In later chapters, I will use the data from these studies to show how scientists determine the risk at low doses. The controversial part of the low-dose risk determination is not the data at high doses, but the extrapolation from high dose to low dose.

Starting the Long Walk Toward Radiation Risk at Low Doses

At this point I could just say that the risk is such-and-such and end the book right here. The purpose of this book, however, is to provide an understanding of how scientists arrive at risk values, not just to provide numbers. When someone must make a decision about some action that involves risk, the person generally bases any decision on feelings as much as numerical data. Part of the feeling is based on confidence, or lack of it, in the data. Over the next few chapters I will not only provide numerical data, but some feeling for that data.

The process of arriving at some risk value for low doses of radiation is not straightforward. To get a final answer, we need to consider such things as how science gathers and handles data, how uncertainties affect the data, and how scientists use data at high doses to determine risks at low doses. I will discuss these topics in the next few chapters before we get into the actual risk determinations in Chapter 9.

We may think of this process as an injury trial in which the jury must make a monetary award to the litigant. In such a trial, the litigant must present evidence that the injury actually happened. Then the litigant must provide some background information as to help the jury to understand why the defendant was negligent. This evidence could be technical in nature depending on the cause of the injury.

In this "trial," I will present evidence in this chapter that radiation does affect the health of living organisms. Injury is possible. In the next four chapters I will present some (technical) background information on the scientific method, on statistics and uncertainty, on making extrapolations, and how we express the risk. Chapter 9, which presents the analysis of the data, is the heart of the trial. I present witnesses, in this case groups of scientists, who will explain how they arrived at a risk value at low doses.

As in any trial, witnesses are open to cross examination. So it is here. In Chapters 10 through 12, I have witnesses who back up the data or argue that it is not correct. Some say that the calculated risks are too low, some say too high. You can act as a juror. You will have an opportunity to hear all these witnesses, then you can render a verdict.

The only difference between an injury trial and our trial on radiation risk is that an injury trial has an end. Ours does not. As you will see in later chapters, the data are not complete and, in many cases, uncertain. Nobody can say they are 100 percent confident in their information. Maybe we should ask for a continuance until we have all the data and we know the risks precisely.

But we cannot wait. We need an interim decision to go on with our lives. We have had various interim decisions in the past and as expected they have changed as more evidence became available. In this and later chapters I will present the evidence we have at this time and you can make a decision. However, you must be aware that new evidence could change your decision in the future.

If we don't have the evidence that will conclusively give the risk from radiation, what do we do in the meantime? This situation is much like a defendant charged with a serious crime; should that defendant be released on bail until the time of the trial or should the judge refuse bail? Should we continue using radiation and radioactivity and use what we know now to set standards for protection? Or, do we refuse bail, lock up all nuclear facilities, and not let them open until we have the evidence to prove that the risk is definitely small enough to be acceptable?

Quality of the Evidence

We know from Chapter 3 that the long-term effects of radiation are leukemia, solid tumor cancers, and genetic effects. We know these are the effects from human and animal populations exposed to high levels of radiation. Our task now is to determine whether low levels of radiation also cause these same effects and, if so, determine the actual risks. Our determination of these risks depends on the quality of the data in the various studies.

We have three major categories of human data on radiation risk: unintentional radiation exposure (accidents and the atomic bombs dropped on Hiroshima and Nagasaki), medical exposures to radiation, and miners who worked in mines with high concentrations of radon. However, there are distinctive problems with each of these studies that affect the quality of the data.

In an unintentional exposure the radiation dose is unknown. In some cases, accident reconstruction can give a good estimate of the dose, but in others the dose can be quite uncertain.

Medicine uses radiation for both diagnostic and therapeutic purposes. Diagnostic doses are quite low unless a patient requires many tests involving radiation. Therapeutic doses are generally quite high. Although the doses are generally known, if the patient needs a diagnostic procedure then that patient must have some medical condition that needs treatment. One condition could be cancer. These patients, by virtue of the fact that they have a medical condition, could be more prone to cancer or leukemia than the public as a whole.

Most miners in the radon studies were smokers. Because both smoking and radon cause lung cancer, the risk from radon calculated from these studies is really only applicable to smokers. Applying these data to nonsmokers produces a large uncertainty in the calculated risk.

However, the major problem with any study is that the natural incidence of these conditions mask the effects of radiation. About 25 percent of the U.S. population succumb to cancer and leukemia (American Cancer Society 1) and between 3.7 and 4.7 percent suffer from genetic disorders (National Academy of Sciences (1990) 70). In a population of one million people, we would expect about 250,000 to die of cancer or leukemia and about 40,000 to suffer some kind of genetic disorder. With such large natural incidence rates it is difficult to measure small increases from radiation.

Another major problem with studies involving cancer is the length of time between the radiation exposure and when the cancer appears. Solid tumors and leukemia have a latency period during which no effects appear. Then after the latency period, there is a period of many years when the exposed individual has a possibility of getting cancer.

The latency period for leukemia is two or more years. The exact period depends on the age of the person at the time of exposure. The latency period for cancer is 10 years. The at-risk period for these diseases apparently is 30 to 40 years, so to get good data on the cause and effect of carcinogens we need to study a large group of people for more than 50 years. And, genetic disorders require at least one generation to get even some partial data. You can see why the data are not complete.

Four committees or organizations have published their interpretations of the data on the risk of low doses of radiation. These four include: the Committee on the Biological Effects of Ionizing Radiation (BEIR) of the National Research Council of the National Academy of Science, the International Commission on Radiological Protection (ICRP), the United Nations Scientific Committee on the Effects of Ionizing Radiation (UNSCEAR), and the U.S. Environmental Protection Agency (EPA). These groups of scientists have published interpretations periodically over the past several years, and they have changed their results several times. BEIR I, in 1972, was the first attempt to ascribe a risk factor to low levels of radiation. BEIR III in 1980 and BEIR V in 1990 are later testimonies. BEIR IV, published in 1988, is a study of the health risks from radon.

Because of differences in the four groups, trying to explain and compare each would be confusing. To give some order to this attempt to cover lots of unfamiliar territory, we will first look at the data that all these studies used. Then in chapter 9 I will concentrate on the BEIR IV and BEIR V studies as "test cases" because these studies are the most widely known and used. In Chapter 10, I will compare each of the other three studies to BEIR V. In Chapters 11 and 12, I will discuss opinions of those who feel that the BEIR IV, BEIR V or other findings are too high or too low.

What is the Evidence?

The four groups studying radiation used six major studies: the Japanese atomic bomb survivors, Ankylosing Spondylitus patients, women treated for cervical cancer, Canadian Fluoroscopy patients, New York State postpartum mastitis study, and the Massachusetts fluoroscopy patients (National Academy of Sciences (1990) 182-188). The BEIR IV study on radon considered four miner groups (National Academy of Sciences (1988) 100-130).

Most of the studies use solid tumor and leukemia mortality and not incidence in the risk calculations. There can be a large difference between the two. Medical treatment cures about half the solid tumor cancers. Cure rates range from 4 per cent for pancreatic cancer to 100 percent for prostate and thyroid cancers if diagnosed early. (American Cancer Society 14) However, data on

incidence are not as accurate as that on mortality because mortality is recorded on death certificates. Not all cases of incidence are reported. Of course, there can be some uncertainty due to an incorrect determination of death.

Japanese Bomb Survivors

Data on the survivors of the atomic bombs is the major source of information on radiation risk. This study began in 1950 and includes 120,321 individual residences of Hiroshima and Nagasaki, of which 91,228 were exposed to radiation from the atomic bombs. This group has shown a statistically significant number of excess leukemia and cancers of the esophagus, stomach, colon, multiple myeloma, female breast, ovary, bladder, and lung.

One major problem with the data on this group is the calculation of the dose each person received. Obviously nobody took measurements at the time of the bombing. To determine the dose, scientists calculated the expected dose from the two weapons. Then they determined the dose at the location of each exposed person at the time of the bombing. They also corrected for shielding by buildings between the bomb and the person.

Before 1986, the radiation dose included a component from neutrons from the bombs. However, further calculations completed in 1986 showed that the dose from neutrons was insignificant compared to the dose from gamma rays. In addition, a new calculation of the gamma dose produced an increase in the Hiroshima dose but a decrease in the Nagasaki dose. The net result of all these changes is that the total calculated dose is smaller than previously calculated.

To determine the risk from radiation, we divide the excess deaths by the dose to get a ratio of death to dose; if we have a lower dose then the calculated risk increases. Therefore, calculated risks made with the new dose data are higher than those made from previous data.

However, a study published in 1992 shows that the 1986 neutron correction may have been too severe. Data show that the low-energy neutrons may have been more significant than the 1986 revision had suggested. Thus, the actual dose could be higher. (Straume 421-426) If this is true, then the most recent calculated risks by the various agencies may be too high.

The new dose calculations apply to about 76,000 of the survivors, so the data on those individuals are used for this study. However because there is considerable uncertainty in the doses of the most highly exposed, only survivors with doses below 4,000 mGy (400,000 mrad) are used in the analysis.

Another problem with the bomb survivor data is that the population is not representative of a typical population. The group is missing most of the young adult males who were in military service. This missing group would be more healthy, so as a group the overall health of the survivors may not represent a normal population.

However, even with the problems with the dose and population, this group offers the best data we have on radiation risk. Scientists have followed this group for more than 40 years so the data are extensive.

Ankylosing Spondylitis

Ankylosing spondylitis is a disease of the backbone that leaves the vertebrae joints hardened and effectively fused together. From 1935 to 1954 physicians treated ankylosing spondylitis patients in the United Kingdom with large doses of X-rays. Unlike current technology, the X-ray machines of that generation could not narrowly focus the X-ray beam. X-rays not only hit the spine but also hit many other organs in the body. Because of variability in machines and beam direction, not all patients received the same dose to the different organs. A study of about seven percent of the patients' radiotherapy records determined an average dose to all the patients.

The group consists of about 6700 patients and follow-up data are available through 1982. This study provides information on several organs with the bone marrow being the most significant.

Women treated for cervical cancer

One treatment for cervical cancer involves radiation, either by external radiotherapy or by implanting a radium source in the cervix so that the gamma radiation irradiates the cancer. Physicians in several countries treated about 100,000 women with radiation. Of these, 4188

developed a second primary cancer. Organ doses for these women were calculated and compared with the doses for 6880 women in the group who did not develop a second cancer.

The doses are well known for this study. The group, though, already had cancer before the study began. The largest risk factor for cancer is having a close relative who had cancer. Susceptibility to cancer may be genetic. Therefore, a person who already has one cancer may be prone to getting another so "extrapolation of the results to the general population must be made with some caution" (National Academy of Sciences (1990) 186).

Canadian fluoroscopy study

Between 1930 and 1952, Canadian physicians treated tuberculosis patients in Canada with fluoroscopy (X-rays). About 26 percent received a dose of more than 100 mGy (10,000 mrad) with a maximum dose of 20,000 mGy (2,000,000 mrad). Because the X-ray beam was directed at the lung, this study is useful for data on female breast cancer. The group consists of 37,710 women and the mortality data is from 1950 to 1980.

Doses to the individuals varied because the number of treatments and the orientation of the X-ray beam varied. To get an average dose to the group, scientists interviewed physicians who administered the radiation and used dummies to measure typical doses to the lung and breast. The major weakness of this study is the effect of tuberculosis. Does it make the patient more or less likely to get cancer?

New York postpartum mastitis study

In the 1940s and 1950s physicians treated some women for inflammation of the breast with X-rays. The group is small, only 610, so the information is limited. Most women were between the ages of 20 and 40. The individual doses received were significant, from 600 to 1,400 mGy (60,000 to 1,400,000 mrad).

Massachusetts fluoroscopy patients

Between 1930 and 1956, two Massachusetts sanatoria treated 1742 women for tuberculosis by collapsing the lung. The treatment involved the use of fluoroscopy, so this study is useful for data on breast cancer. The dose calculations are good but the small size of the group limits the usefulness.

Miner studies for radon

Scientists studied groups from three uranium mining areas and from one iron mine. Two of the uranium mining areas are in Canada: Ontario and Eldorado, Saskatchewan. The one area in the United States is the Colorado plateau. The iron mine is in Malmberget, Sweden. The Ontario group had 11,076 miners plus 570 surface workers for controls, the Eldorado group had 6,847 miners and 1,580 in the control group, and the Malmberget group had 1,292 miners. The Colorado had 3,347 in the group but only 2,975 were used in this study. The major problem with these data is that many miners smoked and the effects of smoking and the effects of radon are difficult to separate. (National Academy of Sciences (1988) 33)

Chapter 5
Doing Science

Before we can get to risk calculations, we need to know the rules of the game. On a recent trip I asked a fellow traveler if he would review the rules of cricket. He refused to even start, saying that he would need several days to explain the game to me. Well, the rules of science may not be as complicated as the rules of cricket, but they are important for understanding radiation risk.

Scientific Method

Many people believe that scientists simply derive equations and gather lots of numbers and then, heaven forbid, memorize them. This may be appropriate for participating in a question-and-answer quiz show, but it doesn't make anybody a scientist. It is true that scientists must gather facts, but these facts are just the tools used to understand some physical phenomenon.

I enjoy working with wood. I have hammers, saws, a wood lathe, tape measures, etc. If I have the tools of a carpenter, does that make me a carpenter? No. Being a carpenter means that I must know how to use these tools. Only when I can make a table or cabinet or house can I call myself a carpenter. And so it is with science. Knowing the facts does not make anyone a scientist. Being a scientist means that you can use these facts correctly to come to some logical conclusion.

In later chapters we will look at the data scientists have gathered about the effects of radiation. As we will see, some data contradict other data. Doing science is studying these data and then deciding what is the most likely truth. The gathering of facts is only part of the game of science. The real science will be the interpretation of these facts to determine what they tell us about the effects of radiation.

The process of doing science is much like the process of reaching a verdict in a court trial. To get a fair trial you would expect to have the prosecution call several witnesses, the defense attorney cross-examine these witnesses and call other witnesses, and then a jury decide the accused guilt or innocence. This is exactly the process we follow in science to reach a verdict about the effects of radiation. Is it guilty or innocent of causing the damage that some claim?

Examining the Witnesses

A scientist does a study that shows some effect of radiation. The scientist then writes a paper describing the experiment and either submits this paper to a professional journal for publication or presents the paper at a professional meeting. This paper acts as a witness in our "trial."

At a professional meeting, other scientists in the audience have the opportunity to question (cross-examine) the presenter. If the scientist submits the paper to a professional journal, the editor sends the paper to two or three referees who read the paper and look for errors, omissions, and unsubstantiated conclusions. If acceptable, the journal publishes the paper and readers can respond through letters or through another article. In this situation, the referees and readers are the ones doing the cross-examination of the witness. In the sciences we call this peer review.

Some scientists making claims about radiation do so without publishing in peer-reviewed journals. They tend to publish in popular magazines or in books. Does this make them less credible than the "establishment" scientist? Not necessarily. One reason they cannot get their papers published is that the scientists who referee the articles do not agree with the conclusions and therefore reject the papers. Of course, this leaves these scientists without a place to publish except in the popular literature. But, what if they are right and the "establishment" is wrong?

Many years ago, one scientist believed in a theory and presented evidence to verify it. Although he published his ideas in a scientific journal, others severely criticized his work, mainly because they didn't understand it. However, slowly Albert Einstein convinced the rest of the world that his Theory of Relativity indeed was correct.

Therefore, we should not reject these ideas out-of-hand. They need to be considered carefully in light of all the evidence. However, they must satisfy all the basic rules of science. If they do not satisfy the rules, then the conclusions should be considered highly questionable.

Reproducibility

In a trial, one witness usually is not sufficient; each side calls many witnesses. Similarly, science requires several studies to verify some study that shows a new or unexpected result. Other researchers may duplicate the experiment or use the results to do another similar study. This process of verifying the original work is called reproducibility. Scientific data is acceptable only if others, following the same procedures, arrive at the same conclusions. If other research verifies the

original work, the original paper becomes accepted fact. If not verified, it is rejected by the scientific community.

Reproducibility comes in different forms. The common definition of reproducibility is one scientist repeating an experiment of another scientist and getting the same results as the first.

However, reproducibility can have other meanings. If the exact same conditions apply to two different situations then the results should be the same. For example, suppose that a new industry starts near a farm and the industry produces a chemical that falls uniformly over four fields that each contain the same number of cattle. When the farmer brings in the cattle, the cattle in all fields yield 100 percent weight gains, except one. The cattle in one field yields only 75 percent gain. Can the farmer conclude that the chemical caused the decrease in the one field?

Here is a case of non-reproducibility. All fields were subjected to the same potentially harmful effects of the chemical. If the chemical causes a lower weight gain, then it should act equally in all the fields. There could have been another reason for the one field to have a smaller weight gain, such as illness or insufficient grass growth.

Epidemiological Studies

Much of the information we have about the effects of radiation comes from studies of populations of animals and humans. (Although scientifically humans are in the animal kingdom, when discussing radiation studies I will refer to human and nonhuman animal studies as just human and animal studies.) These studies are called epidemiological studies. For animal studies, scientists may expose one group of animals to a known dose of radiation and record any effects that might be attributable to radiation. For human studies, scientists study groups of people exposed to radiation. However, epidemiological studies are difficult to control and can give erroneous answers if not done properly.

One key aspect about epidemiology is a control group. If we want to determine the effects of radiation on one group of people exposed to radiation, then we must also have a control group. This control group is very similar (same age distribution, sex distribution, diets, etc.) to the group under study, except they are not exposed to radiation. Then to prove that radiation has some effect, the group under study must have a higher incidence of some effect attributable to radiation than the control group.

As an example of the necessity of a control, let's do a simple experiment. We want to see how temperature varies in the north-south direction in the northern hemisphere. To do this experiment, we have a thermometer and a car. We get in our car and start driving north at 8:00 in the morning. As we drive, we take temperature readings. We start at 60°F, then we measure 65°F, then 70°F, 75°F and finally 80°F. We find that as we drive north, the temperature increases.

About 3:00 in the afternoon we turn around and take temperatures as we drive south. Sure enough, the temperature decreases: 75°F, 70°F, etc. back to 60°F when we get back about 10:00 at night. Well, that surely is conclusive evidence that temperatures are warmer up north.

What is wrong with this little experiment? We didn't have a control. We needed to have someone stay at the starting location to measure the temperature there. Had someone done this, they might have found that the temperature rose from 60°F to 85°F during the day. The control is a way to measure other factors that can affect the temperature such as the effect of daytime heating from the sun. Using the control data, we would arrive at a totally different conclusion: north is colder.

If the farmer on farm A wanted to decide if industrial chemicals were harming cattle exposed to the chemical, the farmer should find a nearby farm B that is very similar to farm A but not exposed to industrial chemicals. Because cattle weight gain depends greatly on the cattle's food, each must have the same kind of grass in the fields, the same rainfall, sunshine, etc. The only difference between the two farms should be the industrial chemicals on farm A. If the cattle on farm A gain less than those on farm B then the chemicals would be the likely cause.

The greatest problem with epidemiological studies is getting a proper control group. Many chemicals in the foods we eat are carcinogens. This makes it difficult for scientists to precisely match

a control group with the study group. One of the two groups could be exposed to an additional carcinogen so radiation exposure may not be the only difference between the two groups. In later chapters I will discuss the problems of some control groups and also discuss the studies that do not use control groups.

Chapter 6
Statistics and Uncertainty

As indicated previously, we do not have good data on the effects of radiation at low doses. When scientists calculate the risk values for low doses of radiation, they use information about the risk at high doses. From these data, they extrapolate from the risk at high dose to make predications about the risk at low dose. In the next chapter, I will discuss the process of making these predictions.

Although the process of making this extrapolation has considerable uncertainty, the actual data also has uncertainty. In this chapter, I will discuss the concept of statistics and how that affects the accuracy of calculated risk factors.

In the following sections of this chapter, the numbers get a bit intense. I suggest that you read this chapter slowly. Also, I would suggest that you have a calculator handy and do the additions and subtractions yourself. This will have two benefits. First, it will slow you down. Second, you will get a better feel for the numbers if you do the calculations yourself.

Statistics: Unavoidable Uncertainty

We base all of our quantitative risk values on experience. If we want to predict a risk in the future we need to investigate the history of this risk. For example, suppose we want to predict the number of vehicle accidents for the next year. To make this prediction, we would just study the number of accidents each year for the past five to ten years. Then we could predict the same number for next year. Or, we might modify that number if we want to account for changes in the number of vehicles on the road, changes in road quality, changes in speed limits, or any other factor that could affect the number of vehicle accidents.

But suppose the conditions remained exactly the same from last few years to next year. It rained in the same places on the same days, the same people were on the road on the same days, etc. Would we expect to have the same number of accidents every year? No. although we have the same conditions, we will not have the same number of accidents. Even under the same conditions, the fact that accidents are random in nature means that we will not observe the same number of accidents.

Similarly, if two groups of scientists were to expose two identical groups of mice to radiation, would they observe the same number of effects (solid tumors, leukemia, etc.)? No. Even if researchers do their work with precision and accuracy, their results will still have some uncertainty because of the random nature of events.

For example, the toss of a coin is a random event. The probability of getting a head is equal to that of getting a tail: 50 percent. Based on the probability of a 50 percent chance of getting tails, can you predict the number of tails if you toss a coin 100 times? Fifty? Would you bet lots of money that you will get exactly 50? If you would, give me a call. Toss a coin 100 times and see how many tails you get. If you should happen to get 50, don't call me to tell me I am wrong. Do it again. If you do it enough you will eventually get all numbers from about 30 to 70, including 50.

The high temperature on a particular day is a random value depending on local weather conditions on that particular day. Over the years, local weather bureaus record the high temperatures every day for many years to determine the average high temperature for that location for each day of the year. If the average high for a particular date, say May 25, is 70°F, then we would expect to have highs on every May 25 that are near 70°F, but not necessarily exactly 70°F. Most years on May 25, the high temperature is not 70°F.

However, this may not be the message transmitted by some local weather reporters. Some call the average high the "normal" high, thus implying that we should have a high of 70°F every year on

that date. If the high on May 25 is, for example, 75°F, the reporter may say that the high was 5°F higher than what it should be. In fact, 75°F would be a perfectly normal high temperature for May 25.

The message I want you to understand is that variation from the average is expected; the average is not normal. You will not get the same number of vehicle accidents in two consecutive years even under the same conditions. You will most likely not get 50 tails if you flip a coin 100 times. The high temperature for any particular day has only a small probability of being the same as the average high for that date. Two or more scientists preforming the same experiment will not get the same result.

The World of Probability

To get some idea of the meaning of probability, let's do some "supposing." Suppose you are on a seat above a tank of water. People are paying to throw baseballs at a circular target that when hit will send you into the tank of water. The target has an area of one square foot. Suppose further that the people don't have good aim and randomly scatter their throws over an area of 25 square feet. Therefore, the probability of being dunked is the ratio of the area of the target to the area over which the balls hit, or one square foot divided by 25 square feet. This gives a probability of $1 \div 25 = 0.04 = 4$ percent. If 100 people each throw five baseballs, for a total of 500 throws, then you can expect to be dunked $0.04 \times 500 = 20$ times.

One person pays to throw five baseballs. Will you be dunked? Can you say with certainty that you will or will not be dunked? You know that there is a probability of 0.04 (4 percent) per throw that you will be dunked, or a 0.20 (20 percent) probability that you will be dunked with five throws. But you cannot say for certain that you will or will not be dunked.

If five people pay, the probability becomes $5 \times 5 \times 0.04 = 1$ (or 100 percent). Does this mean that you will be dunked? No. It means that the probability of being dunked is large, but not absolutely certain. You could not be dunked, or you could be dunked two or three times. Even if 100 people were to take five throws each, you can expect to be dunked 20 times, but even that number is not certain; it could be 19, or 21, or 22, or 15, or . . . However, if millions of people paid, the average number of dunks per 100 people would be very close to 20. The world of probability allows for predictions, but does not give certainties.

This is the world governed by the bell-shaped, Normal, or Gaussian curve. The Gaussian curve can tell us the probability, not certainty, of an event happening. In our example of baseball throwing, for 500 throws of the baseball the average number of dunks is 20. However, in actual practice, you may be dunked more or less than 20 times for every 500 throws.

Let's do an experiment: you are now the official dunkee for a carnival and every day 100 people each throw five baseballs to watch you get wet. The summer carnival season is 100 days long, so we will have 100 bits of information to gather: the number of dunks each day. On the average, as noted above, you can expect to be dunked 20 times. If we plot a graph of the number of days you were dunked a certain number of times (vertical axis) as a function of the number of dunks (horizontal axis), we should get the traditional Gaussian curve. Figure 6-1 shows what we might expect for this example. Even this plot is uncertain; if you did this experiment for five summers, no two plots would be the same.

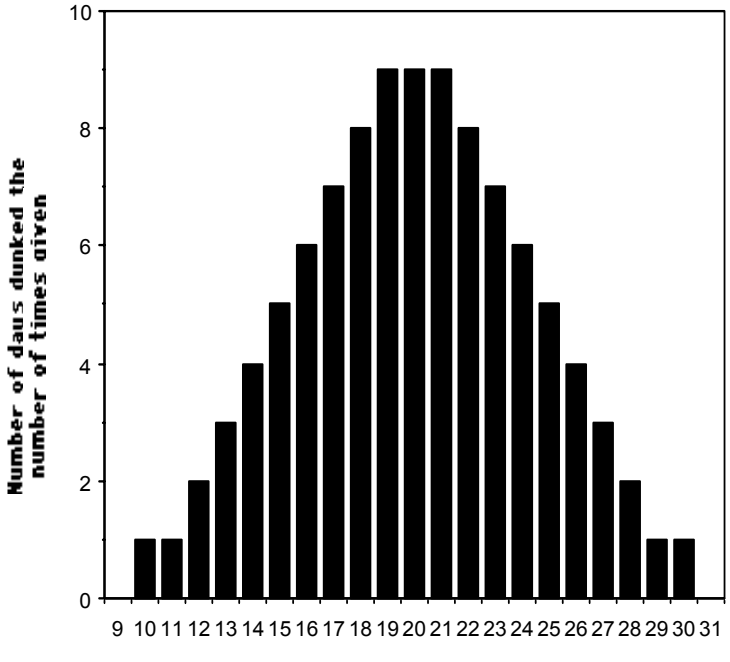


Figure 6-1

Although the average is 20 dunks per day, for only nine of the days will

you expect to be dunked exactly 20 times. In fact, you will expect that on one day you will get dunked 30 times and on another day only 10 times. Note the use of the words "expect" and "should" rather than "will." All numbers are just probabilities and we cannot make any definite statement about the results, we only say what "could" happen. Even with 100 days of data you should not expect to get exactly the graph shown; you may have 12 days with 19 dunks and maybe even a day with only five dunks; they are all possible, although less probable than the values shown.

Looking at Figure 6-1, we can determine the probabilities of each number of dunks. If we expect to get dunked 20 times on only nine days out of 100, then the probability of being dunked exactly 20 times is 9 ÷ 100 = 0.09 which is 9 percent. The probability of 16 dunks is 6 ÷ 100 = 0.06 or 6 percent. Note that if you carefully add up the number of days you will get 101, not 100; this comes from rounding the probabilities to whole numbers; the actual probability of 20 dunks is 0.089 (8.9 percent, or 8.9 days out of 100) and of 19 or 21 dunks is 8.7 percent (or 8.7 days out of 100). But we must deal with entire days, not fractions of a day, so we must round to the nearest whole day.

For a Gaussian curve, 68 percent of the values fall within what is called one standard deviation. The standard deviation is a measure of the uncertainty in the measurement. It tells us how much variation we might expect in our measurements. This variation is not due to errors in taking data, just on the inherent uncertainty from random events.

The standard deviation is just the square root of the average number of events. In the dunking example, the average number of events is 20 so the standard deviation is the square root of 20, or 4.47. Because we can't have 0.47 of a dunk, we need to round off 4.47 to 4. Therefore, we would expect that 68 percent of the days (i.e., 68 days) you would be dunked between 20 - 4 = 16 and 20 + 4 = 24 times. Counting the total number of days with between 16 and 24 dunks, we find that the total is 69; again the slight difference is from rounding.

We can also use the 95 percent confidence limits: that is 1.96 times the standard deviation. Here, the 95 percent confidence limit is 1.96 X 4.47 = 8.76, or, rounding to 9, the range would be 20 ± 9. We should find 95 percent of the number of dunks between 11 and 29. Looking at Figure 6-1, we find that 99 out of 101 are in this range, or about 98 percent. Again, because of rounding we get a number that is slightly different that expected.

What if we don't have 100 trials to determine the average number of dunks? Rather have only one. How certain are we that the number we get is the average? We apply our probabilities to the one trial in the following way.

Suppose that on one day you are dunked 22 times. The square root of 22 is 4.7. Rounding off to 5, this means that the standard deviation interval is between 17 (i.e., 22 - 5) and 27 (i.e., 22 + 5). What I can say now is that I am 68 percent confident that the real average (that determined from hundreds of trials, i.e., 20 in this example) falls in this range. The 95 percent level of confidence is 1.96 X 4.7 = 9.2, or 9. I am 95 percent confident that the real average is between 13 (i.e., 22 - 9) and 31 (i.e., 22 + 9). To be more confident, you need to make the range of possible values larger.

What if the 100 people throwing the baseballs were professional baseball players? Now we would expect that the probability of hitting the target will be higher than 4 percent and you would expect to be dunked more than 20 times. So, although we can determine probabilities based on random numbers, other biasing factors can change those probabilities.

Effect of Uncertainty on Risk Calculations

How will the uncertainty from statistics affect our calculations of risk from radiation? To get some idea of how the uncertainty plays a role, let's play a game of pin the tail on the donkey. In this game, a blindfolded person tries to attach a paper tail on a picture of a donkey attached to a wall. The process of pinning the tail is somewhat random. The probability of getting it on the correct location is small; most times the tail is too high, low, to the left or to the right. The unblindfolded people find enjoyment watching the blindfolded person try to get the tail on the appropriate place on the donkey.

To the unblindfolded people, the process may seem easy; they can see the picture and it may look simple to get the tail in the correct place. However, to the blindfolded person, the process is

more difficult. So, let's look at an example of radiation risk calculation, first from an unblindfolded perspective then from the blindfolded person's perspective.

As outlined in Chapter 5, an epidemiological study requires a study group and a control group. The control group is identical to the study group except that the study group receives a larger (and measurable) radiation dose. The effect we will use in this example is cancer; we do know that high doses of radiation cause an increased risk of cancer. Therefore, we would expect the study group to have a larger cancer fatality rate than the control group.

Suppose that the risk of cancer from a certain radiation dose is 0.001 (0.1 percent). I have two groups of 100,000 people; one exposed to this radiation and one not. The first is my study group and the second the control group. Also suppose that the natural fatality rate of cancer is 20 percent.

As an unblindfolded researcher I now do my study. Remember that because I have no blindfold, there is no uncertainty; all numbers are accurate. I have a clear view of the entire donkey. First, I would observe 20,000 cancer fatalities (20 percent of 100,000) in both the study and control group from natural causes. However, the study group would have an additional 100 cancer fatalities (0.1 percent of 100,000; or 0.001 X 100,000) for a total of 20,100. Now I do my calculation. The difference between the two groups is 100. Dividing this by 100,000, we get a risk of 0.001, or 0.1 percent. That was easy.

Now you can play the part of a blindfolded researcher making an attempt to get the tail in the correct place. Your numbers will have uncertainty so you will not get the precise numbers I did. You do not know the exact location of the donkey. Suppose you have many groups available to study so let's see what numbers you might observe.

First, let's consider the control groups. When you get the data of cancer fatalities, will you find exactly 20,000 fatalities in every control groups? No. Because of statistical uncertainties, there is a standard deviation of the square root of 20,000, or 140. Therefore, you would expect that 68 percent of the groups to have fatalities between 19,860 and 20,140 (i.e., 20,000 ± 140 at the 68 percent confidence level). Of the remaining 34 percent, half will have numbers less than 19,860 and half will have over 20,140 fatalities. You will find that 95 percent of the control groups have between about 19,720 and 20,280 fatalities (i.e., 20,000 ± 280 at the 95 percent confidence level).

Would you find exactly 20,100 in each of the study groups? No. Because of statistical uncertainty we would find 68 percent of the groups with 20,100 ± 140 fatalities, or between 19,960 and 20,240. At the 95 percent confidence level, that range is 20,100 ± 280, or between 19,820 and 20,380.

Now you are ready to do your study. Remember, from my unblindfolded perspective I needed only one study because all the numbers were exact. Even if I repeated the study I would always get the same numbers. But you will not get the same answer for every study. Your results will be random. And, you have only one chance to get the tail in the right place on the donkey. What is the probability of doing this, i.e., what is the probability that you will happen to get an exact difference of 100 between the study and control groups?

I might note that the largest radiation study group is that of the Japanese bomb survivors which totals less than 100,000. Also, the risk from cancer at the doses received by the bomb survivors is higher, but not that much higher than the 0.1 percent assumed here. Therefore, what follows is similar to the actual numbers we face when trying to determine the risk from radiation.

The probability that you will get a risk value of 0.1 percent is small. Most likely you will get something different. For example, you might get 20,050 in both the study and in the control groups. This would tell you that the radiation does not cause cancer. At one extreme, you could get 20,240 in the study group and 19,860 in the control group (one standard deviation high and low respectively). The difference is 380. Dividing this difference by 100,000, you get a risk of 0.0038, or 0.38 percent. It is even possible to get 19,960 in the study group and 20,140 in the control group (one standard deviation low and high respectively) and you would conclude that the radiation has a negative risk of 0.0018. The radiation has a 0.18 percent chance of preventing cancer.

What if you had 50 groups of each? You have 50 attempts to get the tail in the right part of the donkey. If you did these 50 studies and took the average value of all 50, you would you have a better chance of getting 0.1 percent risk.

The important message that I want to get across here is that statistical uncertainty can greatly affect the risk value. Because we will be dealing with small differences in large populations, the calculated risk values will have large uncertainty.

These examples also show three important conditions that must exist for any quality radiation risk study. First, the groups must be large. If the study and control groups in our above example were 1,000,000 rather than 100,000, the uncertainties would be less. The standard deviation from 200,000 cancer fatalities (20 percent of 1,000,000) is 450. This is a larger number that the 140 from a group size of 100,000 (and 20,000 cancer fatalities), but the percent uncertainty is only 0.2 percent rather than 0.7 percent from the smaller group.

Second, we need a good control group. In my example, I have assumed that the only difference between the study and control group is the radiation exposure. In reality, it is difficult to get an exact match for a control group. We need to have similar age distributions, socioeconomic conditions, races, etc., to have a perfect control group. Any deviation from the ideal can cause more uncertainty in the calculated risk value.

Third, the radiation dose must be large enough to produce a statistical difference in the cancer fatality rates. In my above example, if the risk were only 0.01 percent rather than 0.1 percent, the probability of finding a statistical difference between the study and control groups would be infinitesimal. As we will see in later chapters, we need some large doses to get statistical differences in the study and control groups.

From a blindfolded person's perspective, the pin-the-tail game is not easy to play. Any valid study must satisfy the above criteria. Anybody who does a study that does not meet the above criteria cannot honestly claim that the resulting risk value is valid.

Chapter 7
Making Predictions

I learned Newton's laws, including the classic equation, F = ma, in a college physics course. The professor commented that Newton's laws are simple. It is just the applications and implications that are difficult. It didn't take long to see what this meant.

Similarly, the concept of the Gaussian distribution is simple, it is just the applications and implications that are difficult. Not fully understanding the implications, especially in the field of risk analysis, can produce misleading conclusions.

Clusters

Let's return to the dunking example. Remember that the average number of dunks per day is 20. However, according to figure 6-1, you have one chance out of 100 of being dunked 30 times. If the people hit the target 30 times in one day, you might look for some reason for this exceptionally high number of dunkings. You might suspect that many of the people were professional baseball players. If you couldn't see the balls the people throw, you might suspect that they were throwing softballs or even basketballs. When something appears out of the ordinary, it is human nature to suspect some cause, possibly devious in nature.

Sometimes a small population (section of a town, maybe the entire town) suffers a significantly higher incidence of some affliction. Such an occurrence is called a cluster. I remember an incident during my childhood that involved a cluster. One summer, before polio vaccines were available, several polio cases appeared in the region where we lived. In our town, my father noted that nine of the 10 polio cases were children who lived within a block of a drainage ditch that ran through our town. Rats lived in that ditch and it was known that rats carried the polio virus. It might appear that the rats in that ditch were spreading the polio virus to children in the area.

Or was this a situation where we were seeing the high end of the Gaussian curve? If we had 100 summers of similar conditions, we might have seen a normal distribution of polio cases throughout the town with one summer having many cases near that ditch. Maybe another summer

there wouldn't be any near the ditch. Nobody did study this situation and nobody tested the rats. So we never knew if the rats were the cause, or if the higher polio incidence was just part of a normal distribution.

When a cluster appears, we need to be careful about our conclusions. To blame it on a certain cause requires more than just numbers. First, we need to have a reason to suspect that something is causing the cluster. Is there some agent present that we know causes this affliction? Second, we need to measure the amount of the suspected agent to verify that it is present in quantities that could cause the affliction.

In a later chapter, I will discuss clusters as they apply to radiation. In that section I will discuss the unique properties of radiation that make it difficult, if not impossible, to be the cause of clusters of cancers.

Risk Determination: The ins and outs of science

What I will cover in this section may seem a bit esoteric, but it all has utility later in the book. When I discuss the possible fits to radiation risk data, the method of fit and the kinds of fit are very important. Before I go on, I need to define what I mean by a fit.

When we try to find a relationship between a cause and effect, we make a graph. On this graph we plot the effect as a function of the amount of the agent suspected of causing the effect. For example, we know that salt in our diet increases our thirst so we drink more water. We can put salt into the food of a large group of subjects and we can measure how much each person consumes over a period of several days or weeks.

Then we measure the amount of water each subject drinks each day. Finally, we plot the amount of water consumed as a function of the salt in each person's diet. The results could look like one of the graphs in figure 7-1. Each dot represents the amount of water consumed for the given amount of salt in the diet.

Graph (a) in figure 7-1 shows that the amount of water consumed does not depend on the amount of salt in the person's diet. We would write this relationship as W = C where W is the water consumed and C is a constant. This equation is called the fit to the curve.

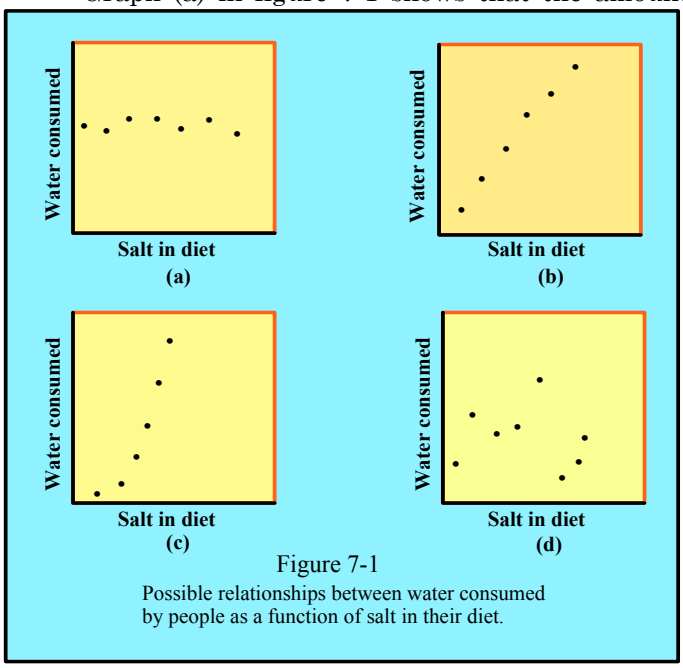


Figure 7-1
Possible relationships between water consumed by people as a function of salt in their diet.

In graph (b), there is a linear relationship. If the amount of salt doubles, the amount of water consumed also doubles. We would write the fit as W = aS where S is the amount of salt in the diet and a is a constant (the slope of the line which determines how rapidly W changes with S).

Graph (c) shows that the amount of water increased faster than linearly. If the salt is doubled, the water consumed is more than doubled. Graph (d) shows that there is no relationship between the two. The amount of water is random, independent of the salt in the diet.

The two kinds of fits for radiation risk data are linear (graph (b) in figure 7-1) and linear-quadratic. These are not easy ideas to understand. You may want to brush up on your algebra: specifically the $y = mx + b$ and $y = ax + bx^2 + c$ equations. Appendix B has some information on these equations, so you might want to take a side trip there before you proceed.

Using data to predict

Science is not just the process of making measurements and then putting these numbers into a computer to generate a new accurate number. Science is a living, breathing entity that eats facts, digests them, then pronounces some conclusion. This conclusion is based not only on the facts, but also on ideas stored in the minds of the individuals studying the facts. Once the scientific community gathers the data for a particular situation, then humans do the interpreting of these data. Different people will arrive at different conclusions.

If we feed twin runners the same food before a race, they will not necessarily end the race in a tie. The running times depend on many factors: the conditioning program each has followed, the state of each runner's health, and their diet many weeks before the race.

Similarly, when we feed the same facts to two scientists (they could even be twins), they will not necessarily come to the same conclusion. Part of the reason is that the data always has some uncertainty so each person places a different weight on the values. Just as the actions of the runners before the race can affect the results of the race, each scientist has a history about the subject. That knowledge can affect an individual's conclusion.

For example, suppose we want to know the relation between the number of oak trees per acre and the number of squirrels per acre in a forest. We assume that the number of squirrels affects the number of oak trees because squirrels collect and bury acorns, and there is a probability that some of these acorns will be left to grow into mighty oak trees. We assume that as the number of squirrels increases in a forest, the number of oak trees also increases, but just what is the relationship between these two numbers? To find out, we study the number of squirrels and oak trees. To make the study more accurate, we study many different one-acre sections of forests.

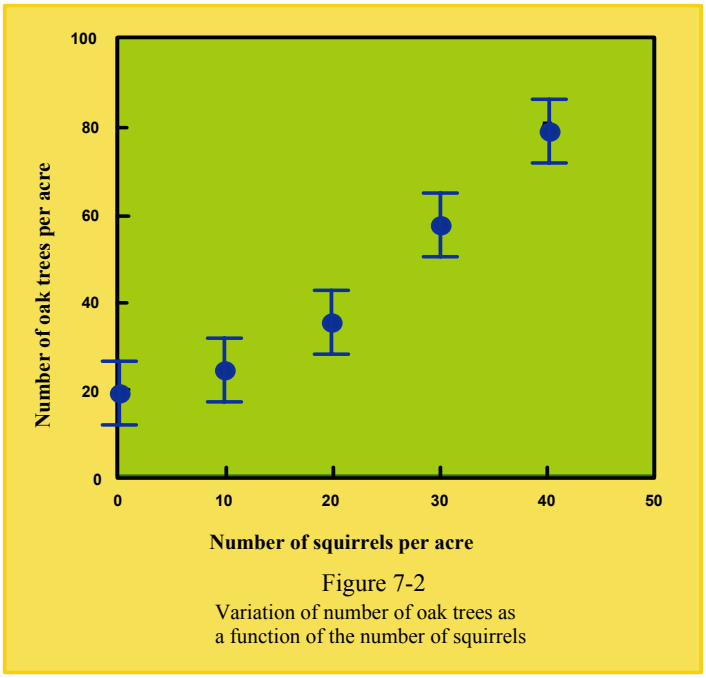


Figure 7-2
Variation of number of oak trees as a function of the number of squirrels

The first set of one-acre plots we study are ones with no squirrels; the average number of oak trees is 20 per acre. Of course, each one-acre plot did not have exactly 20 trees, the numbers ranged from 11 and 29 with 68 percent of the numbers falling between 13 and 27. This means that the average value for the number of oak trees has an uncertainty of ±7.

We plot this point (20 trees for zero squirrels) on a graph with vertical bars to represent the ±7 uncertainty. Then we find several one-acre forest plots with 10 squirrels and count the number of oak trees and get an average of 25. Just as with the first data point, this average number has an uncertainty, let's say ±7 again to keep things simple. Repeating the process we get an average number of 39 trees per acre of for 20 squirrels per acre, 57 for 30 squirrels, and 80 for 40 squirrels; each with a ±7 uncertainty. Figure 7-2 shows the plot of the data.

The graph seems to show that our assumption is correct: the number of oak trees does increase with the number of squirrels. A forest with no squirrels will have 20 oak trees, but the presence of squirrels increases the number of trees above 20 per acre. Now we need to determine the mathematical relationship between the number of trees and the number of squirrels.

With no other information to go on, one may just assume that the relation is linear, i.e., if the number of squirrels doubles, the number of additional oak trees will double. If the number of squirrels goes up by a factor of five, so will the number of trees. Ten squirrels cause an additional five trees (25 for 10 squirrels minus the 20 for no squirrels). From this ratio we would expect that 20

squirrels should cause an additional 10 trees (i.e., double each), and 40 squirrels an additional 20 trees. But our data show that 40 squirrels cause an additional 60 trees; something is not right!

Linear fit to the data

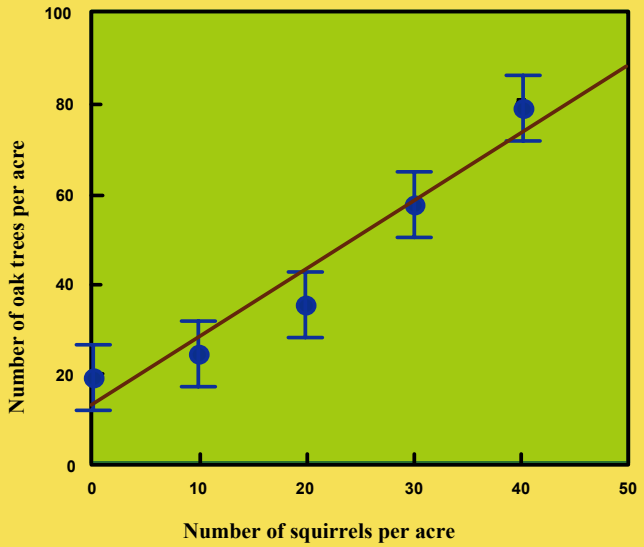


Figure 7-3
Variation of number of oak trees as a function of the number of squirrels with a linear fit to the data

In the above example, we have used only two data points (those for no squirrels and for 10 squirrels) and each data point has uncertainty. To find the relation between squirrels and trees, we need to use all the data points. Can we draw a straight line close to each data point such that the line stays within each of the error bars? Figure 7-3 shows the a straight line fit.

If the linear fit is good then the line should pass through each error bar; the best fit is when the line passes through or close to each data point. The line passes close to the data points for 10 and 30 squirrels, but not the others. The line just catches the error bars for the first data point error (zero squirrels) and the last data point (40 squirrels) and just misses the middle point (20 squirrels) error bar (the line is just above the top of the error bar). As data goes, this actually is not a bad fit; not perfect, but could be acceptable.

This linear fit gives an equation $y = 13 + 1.52x$ where y = number of trees and x = number of squirrels. This means that a forest with zero squirrels will have 13 trees and each squirrel in the forest will cause an additional 1.52 oak trees to grow. We can then use this equation to find the expected number of trees for any number of squirrels: a forest with 25 squirrels per acre would have $13 + (1.52 \text{ X } 25) = 51$ trees per acre.

However, the fit could be better. Note how the line passes through the bottom of the zero and 40-squirrels points and above the other three. This implies that we need a curved line that slopes upward. But we need some basis for this possible fit.

Suppose someone reads an article in a scientific journal or maybe in a nature magazine that describes a study about squirrels and hickory trees. The study shows that as the number of squirrels increases each hides more hickory nuts.

Maybe a person who studies squirrels just guesses that as the squirrel population increases, each needs to hide more nuts because other squirrels steal some during the winter. So, if the squirrel population doubles, there are more than double the number of hidden hickory nuts in that acre. Consequently, there is a greater chance that more than twice the number of hickory trees will grow. If this is true for hickory trees, maybe it should also be true for oak trees. This will give us a reason to try a different curve to fit the data.

Sometimes this process works in the opposite direction. A scientist will find a curve that fits the data then look for a reason for this fit. In either case, the best fit to the data must have some reason for it. We can't just find the best fit and say that is the truth without some justification for that fit, even if that reason is just guess.

Linear-quadratic fit

The next kind of mathematical relationship to try would be a quadratic. This is represented by the equation $y = bx^2$. For a quadratic relationship, if the number of squirrels doubles, the number of trees should increase by the square of two, or four times. If the number of squirrels increases by a factor of three, the number of trees should go up by a factor of nine (the square of three). Ten squirrels cause an additional five trees, so 20 squirrels should cause four times that number, or an additional 20 trees. This agrees quite well with the 19 additional trees we measured. For 30 squirrels we would expect 45 additional trees, slightly more than the 37 additional we measured, and 40 should cause 80 additional trees, considerably more than the 60 we measured. This doesn't seem to be a good choice either.

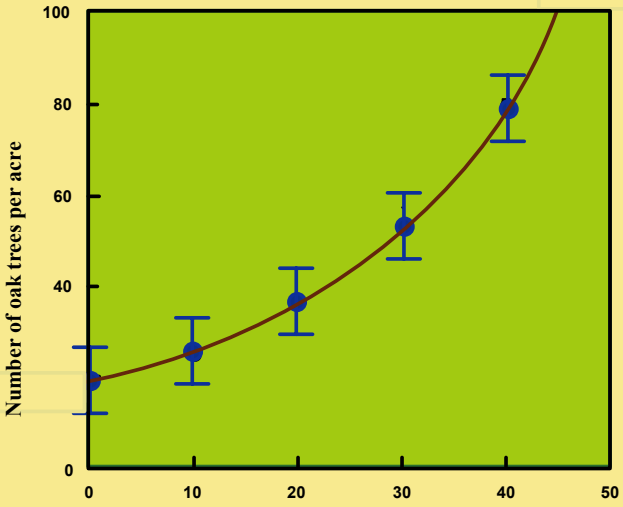


Figure 7-4
Variation of number of oak trees as a function of the number of squirrels with a linear-quadratic fit to the data

Another choice is a combination of a linear fit and the quadratic fit -- a linear-quadratic fit. This is represented by the equation $y = ax + bx^2 + c$. Figure 7-4 shows this fit. You can see that the fit is much better than the linear fit.

The equation for this fit is $y = 20 + 0.15x + 0.034x^2$ where y is the number of oak trees and x is the number of squirrels per acre. This equation predicts (correctly) that for zero squirrels (when x = 0), we would have 20 oak trees. As the number of squirrels (x) increases then the number of oak trees increases both linearly (0.15x) and quadratically ($0.034\ x^2$). For 25 squirrels, the equation predicts 45 oak trees per acre.

In this example we have determined the mathematical relationship between the number of trees and the number of squirrels. We based this relationship on the idea that the number of trees depends on the number of squirrels, i.e., the squirrels cause the increase in the number of oak trees.

However, someone could also argue that the reverse could be true: the number of squirrels depends on the number of trees. If the forest has more oak trees, then more squirrels will find food and therefore call the forest home. Or, there could be no connecting relation between the two; maybe a third factor (possibly the amount of water available for both oak trees and squirrels) may cause the increase of both squirrels and trees. Just because there is a correlation between two factors does not mean that there is a connection. To establish a connection, there must be some substantiating evidence to verify that connection.

Using data to predict risk: Being attentive

What does all this mean for those trying to understand risk? It means that you need to understand the process of how the risk values are derived to understand them and how accurate they are. Fischhoff (National Research Council 217) identifies three essential steps for understanding the science associated with risk analysis.

First you need to identify the factors that determine the amount of the risk. In our tree-squirrel example, we need to identify that the number of trees depends on the number of squirrels. We also need to realize that squirrels are not the only factor that determines the number of trees. The amount of rain and sunshine and the type of soil can also affect the number of trees.

The second step is to identify the accepted facts that affect the risk. The facts are the data given; can we all agree that these are valid data, or is there a flaw in the data?

The third step is:

> "knowing how [the science of risk] depends on the educated intuitions of scientists, rather than on accepted hard facts; although these may be the judgments of trained experts, they still need to be recognized as matters of conjecture that are both more likely to be overturned than published (and replicated) results and more vulnerable to the vagaries of psychological processes" (National Research Council 217).

In our tree-squirrel example, this corresponds to the way we justified the linear-quadratic fit. If this fit is based on conjecture, then that can throw some uncertainty into the use of these numbers to predict the number of oak trees in other forests. This is where further studies are needed to verify or reject the conclusion.

When do we need to be attentive to assumptions made by risk assessors? The answer depends on how much data is verifiable and how much "assuming" goes into the assessment.

There are two ways to make risk assessments. One way is to use experience, adjusting to account for different circumstances. The other way, especially for cases where there is no experience, is to make assumptions about what could happen based on data from related situations. The first requires little work except to verify the original data; the second requires considerable research.

The risk assessments for fatalities from disease or accidents best exemplifies how we use experience in making assessments. Knowing the past rates of cancer, we can make risk assessments for the future. We can make adjustments, but usually they make only small differences.

The calculated risk of a nuclear power plant accident is derived from assumptions because we do not have a sufficient number of accidents to study. We have had one serious accident in the United States that could provide any data for predicting future accidents. But that is only one accident, hardly enough upon which to base predictions of future accidents. Also, the modifications made to existing plants because of the lessons learned from that accident will change the risk factors for other plants.

For example, we have data on failure rates of large steel vessels of the kind that make up the reactor vessel. However, the data are for similar vessels, not the exact same kind, so we need to make some assumptions about how we can use the data for a reactor vessel. We also have data on pipe failures, valves leaking, etc. Then there is the problem of what is called a "fault tree": in which one failure causes another failure. Putting all these risks together, including the fault trees, we come up with the probability that a nuclear power plant will have an accident.

How accurate is this risk calculation? If we include every possible failure, it can be a reasonable estimate although the data may have some uncertainty.

> "[R]isk estimates are based on a chain of conservative decisions about each choice point in the analysis (e.g., studying the most sensitive species, using the extrapolation model that produces the highest risk estimate, giving benign tumors the same weight as malignant ones, etc.). Despite the uncertainties, one may have great confidence that the 'true risk' is unlikely to exceed the estimate resulting from such a conservative process. In other words, uncertainty and subjectivity do not imply chaos" (Slovic 49)

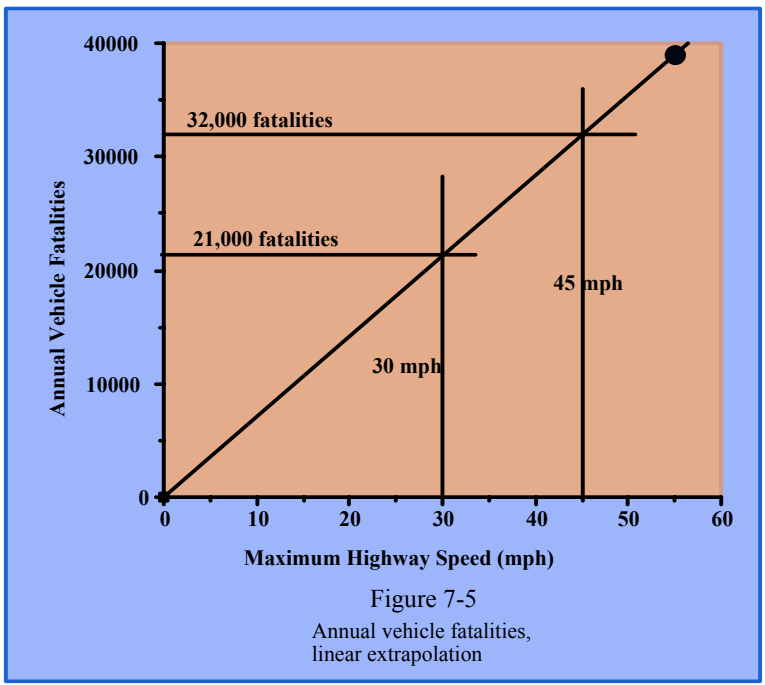


Figure 7-5
Annual vehicle fatalities, linear extrapolation

Extrapolation

The major problem with any risk determination is to get accurate numbers on the harm. If the death rate or the number of people studied is too small, the statistics are not adequate to give reliable results. A disease that causes an average of 1000 deaths each year has an uncertainty of 32, i.e., normally we would expect between 968 and 1032 fatalities per year. This means that

if you believe a new agent is causing an increase in this disease, you could not determine the risk factor unless the increased annual fatalities are more than 32. Even this number would mean that you are only 68 percent confident that the suspected agent is really the cause of the disease; you would need more than 64 additional cases to be 95 percent certain. In addition, if there is a long delay between the initiation of the disease and the time that the disease is evident (as with cancer), trying to relate the cause with the effect can be difficult.

One way to get around the problems of statistics is to do studies on animals with doses of the suspected disease-causing agent much higher that the levels normally found in the typical diet. If the higher doses cause cancer then we can determine the risk factor in deaths per dose and extrapolate to the lower dose.

However, this method can have serious problems.

> "Nowhere are these problems [of risk assessment] more evident than in the assessment of chronic health effects caused by low-level exposures to toxic chemicals and radiation. The typical assessment uses studies of animals exposed (relatively briefly) to extremely high doses of the substance, in order to draw inferences about the risk to humans exposed to very low doses (sometimes over long periods of time). The models designed to extrapolate the results from animals to humans and from high doses to low doses are controversial" (Slovic 49).

But how do we extrapolate? Let's use vehicle fatality rates for an example. Suppose we wanted to know the annual number of fatalities on the highways if the maximum speed limit on non-interstate roads were reduced to 45 mph or even to 30 mph. We could get data if we actually reduced the speed limit. However, that would involve so much effort (and lots of politics) that this kind of study would be impossible.

We are left with the extrapolation method. First we determine the annual death rate when the maximum speed limit is 55 mph. All the following data are from studies when the maximum speed limit was 55 mph except for some interstate highways. To account for the fact that some highways had a 65 mph maximum speed limit, let's assume that the annual number of fatalities would be 39,000 if 55 mph had been the maximum speed limit everywhere.

Now, let's assume that the risk of fatalities is a linear function of the maximum speed. We would then put that one data point on a graph and draw a straight line through the origin of the graph as shown in Figure 7-5. This extrapolation shows that the expected number of fatalities would be 32,000 at 45 mph and 21,000 at 30 mph.

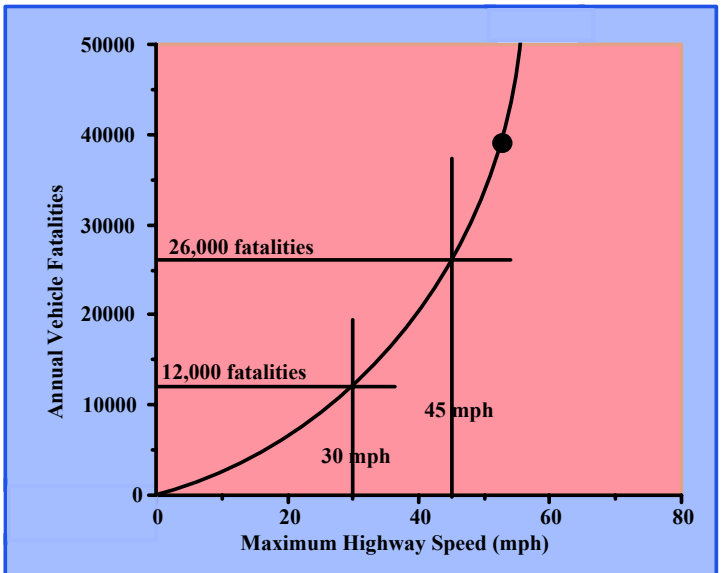


Figure 7-6
Annual vehicle fatalities, quadratic extrapolation

Is the linear extrapolation valid? From our knowledge of physics we know that the kinetic energy of an object (a car does fall into this category) depends on the square of the speed of the object. This means that a car traveling at 60 mph has four times the energy as it would at 30 mph. And, it can do four times the damage.

This reasoning would suggest that the fatality rate should depend on the square of the speed. This would be a quadratic curve. For this case, we put the fatality rate of 39,000 at 55 mph on the graph and draw a line that changes with the square of the speed and passes through the one point on the graph as shown in Figure 7-6. The results show that the annual fatality rate should be about 26,000 at 45 mph and 12,000 at 30 mph; considerably less then the values given by the linear extrapolation.

We have assumed that the fatality rate depends on the maximum speed. In fact the rate should depend on the average speed of the cars (which of course is related to the maximum speed). The average speed of cars, averaging over all roads, interstates, local highways, city streets, alleys, etc., is impossible to determine, but in this exercise of supposing, we can make some reasonable estimate and work from there.

Making an estimate that for a maximum speed of 55 mph, the average speed is 40 mph, then we will put 39,000 annual fatalities at 40 mph on the graph. Using the quadratic extrapolation, we get the results shown in Figure 7-7. This shows that at an average speed of 30 mph (corresponding to a maximum speed of something between 40 and 45 mph; your guess is as good as mine), the death rate would be 21,000; somewhat lower that the previous value of 26,000 at 45 mph maximum speed. Increasing the average speed to 45 mph (maximum speed of about 65 mph) will result in 61,000 fatalities.

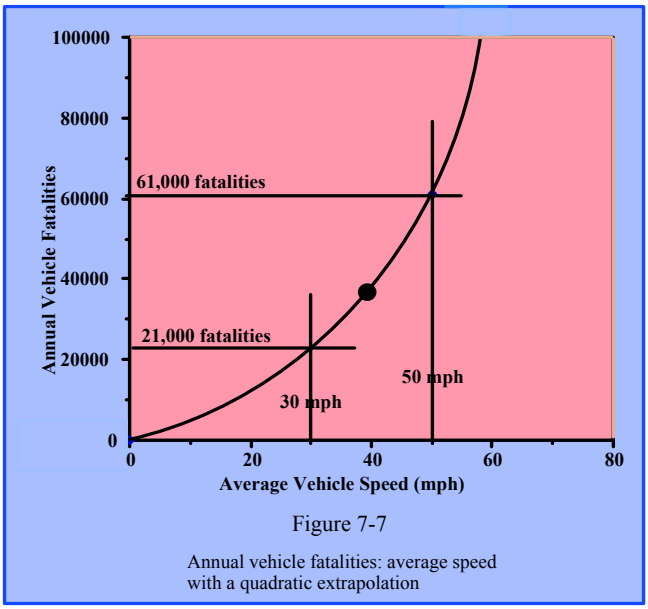


Figure 7-7

Annual vehicle fatalities: average speed with a quadratic extrapolation

In all of the previous examples of vehicle fatalities as a function of speed, we have assumed that fatalities can occur at any speed. Yes there can be pedestrian fatalities at any speed. However, if we consider two cars colliding at 5 mph, the probability of a fatality is most likely zero if we do not include secondary causes such as heart attacks or heavy object shifting that injure the people in the vehicle. If there is some minimum speed at which no fatalities occur, then we have what is called a threshold.

In actuality, we need to consider many more factors beside speed as the cause of vehicle fatalities, but this example does show how our assumptions can greatly affect the extrapolated value. Using the linear extrapolation, the expected number of vehicle fatalities at a maximum 30 mph speed limit would be 21,000. For the quadratic extrapolation, the number would be 12,000. Using the average speed, and the assumption that an average speed of 20 mph corresponds to a maximum speed of 30 mph, then the expected number of fatalities would be slightly less than 10,000. Obviously the choice of the extrapolation makes a big difference.

As a real-life example, consider the study done on saccharin when the tests were done to see if it is a carcinogen. Scientists fed rats large doses of saccharin until the rats showed a measurable increase of bladder cancer. From the increase in cancer, the scientists determined the risk of cancer per dose. Then they determined the risk factor for a lower dose by linearly extrapolating to the amount of saccharin typically consumed by a human. The dose that caused cancer in rats was about 1 to 2 percent of the rat's diet. This is equivalent to a quarter pound of saccharin per day for humans. This extrapolation gave a risk of one chance per 100,000 of bladder cancer (Lewis 149-155).

Some serious questions arise from this process. Are rats and humans equally susceptible to cancer from saccharin? Is the linear extrapolation valid, or is a quadratic, or other function, a more appropriate extrapolation? Is there a threshold below which saccharin does not cause cancer and is that threshold above the typical amount consumed by humans? For example, using a quadratic extrapolation, the risk becomes something in the order of one chance per 1,000,000,000 (one billion) rather than one per 100,000.

Summary

The determination of risk values is a complicated process. First we need to be sure that what we suspect is causing a harmful effect is really the cause. Just because there may be a correlation between a suspected cause and effect does not mean that there is a connection. To determine a connection we need to either determine a path of events that show the connection or have sufficient epidemiological data to verify the connection.

To determine the radiation risk per dose we need to gather sufficient data to overcome uncertainty in the data. We can extrapolate the risk at low doses of radiation from data for the risk at high doses. However, the extrapolation that we use is critical, especially if the difference between the high and low dose values is large.

Chapter 8
Expressions of Risk

Several years ago while discussing the public's lack of understanding of radiation risk, someone mentioned the risk of eating peanut butter. My first reaction was: "Why is eating peanut butter risky?" Now should I worry about my peanut butter and jelly sandwich?

Something called an aflatoxin can develop in peanut butter. Aflatoxin can cause cancer. People who calculate such things tell me if I eat one tablespoon of peanut butter a day, on the average, I will lose one day of life.

Radiation from a nuclear power plant gives anyone who lives near the plant a small dose each year. Using risk factors for radiation, we can calculate the average loss of life. According to one study, I might expect to lose one day of life from living near a nuclear power plant all my life. (Cohen 333)

Does knowing that eating peanut butter is just as risky as living near a nuclear plant make me feel better about living near TMI? Not really. Some people may read this comparison and become frightened of eating peanut butter.

Communication experts say that the method of comparing a new unfamiliar risk to an old familiar risk does not work. I cannot simply compare the risk of radiation to other risks and let you decide if it is safe or not. Therefore, to understand and get a feel for the risk from radiation, we need to build an understanding of radiation risk from the ground up.

Quantitative measures of risk: there are lies, damn lies, and then there are statistics

Determination of risk has one major problem: what do we use to measure risk? The way someone presents a risk greatly affects the perception of that risk. So, what will be our measure of harm?

When people are involved in vehicle accidents, they can suffer a range of injuries: a few bruises, cuts, broken bones, serious internal injuries, and even death. So, how do we quantify that risk? Do we count every injured person? Does a bruise count or only a broken bone? Cuts? Is there a measure for pain or the loss of life's pleasures? How can we quantify the time lost during recovery, the pain of the treatment, and the loss of vitality?

As defined by our legal system, a person who gets sick or is injured endures "pain and suffering." Courts can quantify pain and suffering with a monetary award from the person or organization that causes the injury. However, the awards given to the injured people are inconsistent and therefore virtually impossible to quantify, so we need a better measure. As morbid as it may sound, that measure is death.

Physicians always record the cause of death although the cause may not always be accurate. Several government and private organizations keep records of deaths so these statistics are readily available and assumed to be accurate. For example, the National Safety Council records fatalities by the cause of the accident, the American Cancer Society records cancer fatalities by type of cancer,

the American Heart Association records fatalities from various heart diseases. There are data on the number of people dying from drowning, explosions, bicycle accidents, suicide, respiratory cancers, leukemia, cerebrovascular disease, asthma, etc.

Table 8-1 lists a few of the causes of death and the numbers of fatalities from those causes.

Table 8-1(a)
Annual U.S. Fatalities from Various Accidents

Accidents	Deaths (USDC)[1]	Deaths (NSC)[2]
Motor vehicles	43,500	43,500
Falls	12,600	12,200
Accidental poisoning		
Drugs and medicine	5,215	---
Solids and liquids[3]	483	5,600
Gasses and vapors	736	800
Drowning	3,967	4,600
Fires	4,120	4,200
Firearms	1,441	1,400
Objects inhaled or ingested	3,240	2,900
Medical complications	3,240	---
Electric current	626	---

[1]U.S. Department of Commerce, Bureau of Census; 1991 data
[2]National Safety Council (reported in World Almanac 962); 1991 data
[3]The NCS data include drugs and medicine

Table 8-1(b)
Annual U.S. Fatalities from Disease

Disease	Deaths[1]	Deaths per 100,000[2]
Heart	720,000	283
Cancer	521,090	204
Stroke	143,640	56
Chronic obstructive lung disease and allied conditions	91,440	36
Pneumonia and influenza	76,120	30
Diabetes mellitus	50,180	20
HIV infection	33,590	13
Suicide	29,760	12

[1]National Council for Health Statistics (as reported in the World Almanac 956); 1992 data
[2]Deaths per 100,000 population

Table 8-1(c)
Annual U.S. Occupational Fatalities and Injuries

Occupation	Deaths[1]	Deaths per 100,000[2]	Disabling injuries
Construction	1,300	22	300,000
Service	1,300	3	740,000
Agriculture	1,200	37	140,000
Transportation and utility	1,200	20	250,000
Trade	1,000	4	720,000
Manufacturing	600	3	600,000

Mining and Quarrying	200	29	30,000
[1]USDC, 1994 (U.S. Department of Commerce, Bureau of Census); 1991 data			
[2]Deaths per 100,000 workers in that occupation			

Looking at the numbers in Table 8-1(c) for occupations, we can see that fewer people die from mining and quarrying (200) than from service jobs (1300). Does this mean that mining and quarrying are safer than service jobs? If we compare the death rate (deaths per 100,000 workers in each occupation) we see that the death rate for mining and quarrying (29) is ten times larger than that for service jobs (3). Therefore, the death rate, rather than total deaths, is a better measure of the risk of an activity. The difference arises because there are many more workers in the service industry than in mining and quarrying.

Risk Factors

Here is where we get serious about mathematics. I have done all the following calculations on a simple hand calculator. I suggest that you find your calculator and do all the calculations mentioned below. I think that you will get a better understanding of the mathematics if you do the calculations along with the reading.

What we eventually want is a risk factor; something quantitative that we can use to tell us the risk of an activity. This is expressed simply by the death rate. We calculate the death rate by dividing the number of deaths by the number of people engaging in a certain activity. Then, we can express this probability of death as a fraction or as a percent.

For example, suppose 100,000 people take part in a certain activity and 10 people die each year. The number of deaths per participant is 10 divided by 100,000, which gives 0.0001. This value is the risk factor, expressed as a fraction. If 250,000 people take part in this activity, then the expected number of fatalities is the probability of death per participant (0.0001) multiplied by the number of participants (250,000). This multiplication gives 25 expected fatalities per year.

To convert the probability to a percent, multiply by 100. When you multiply 0.0001 by 100, you should get 0.01 percent. If you are in this group, your risk of death is 0.01 percent per year. This is the risk factor expressed as a percent.

Another way of expressing this risk is by a chance per some number: one out of a thousand, one out of a million, one out of 54,000, etc. If we divide the number of people engaged in this activity by the number of deaths, we get a number that tells us how many people need to take part in this activity to result in one death. In the above example, that would be 100,000 divided by 10. This gives one chance out of 10,000. This means that for every 10,000 people engaged in this activity each year, we would expect one death per year.

For vehicle accidents, approximately 40,000 people die per year (1992 data). If we assume that everyone either drives a vehicle or is a passenger in a vehicle, then there are 260,000,000 people (the U.S. population) who are either drivers or passengers in vehicles. The risk per year per person is just 40,000 deaths divided by 260,000,000 people which equals 0.00015 deaths per participant per year. (Numbers from the World Almanac 962; source: National Center for Health Statistics and Bureau of Census)

Or, we can look at the number of participants needed to produce one death per year. For vehicle accidents, dividing 260,000,000 participants by 40,000 deaths gives 6500 participants per death. This means that your chance of dying in a vehicle accident is one out of 6500 per year. If you live in a town that has a population of 6500 people, you would expect, on the average, that one person in that town will be killed in a vehicle accident each year. Note that 6500 is just one divided by the risk factor, 0.00015, or the inverse of the risk factor.

Figure 8-1 shows a variety of risks expressed as a fraction and as a chance per million, etc. Note in the figure that moving from left to right, each bar represents ten times the risk of the previous bar.

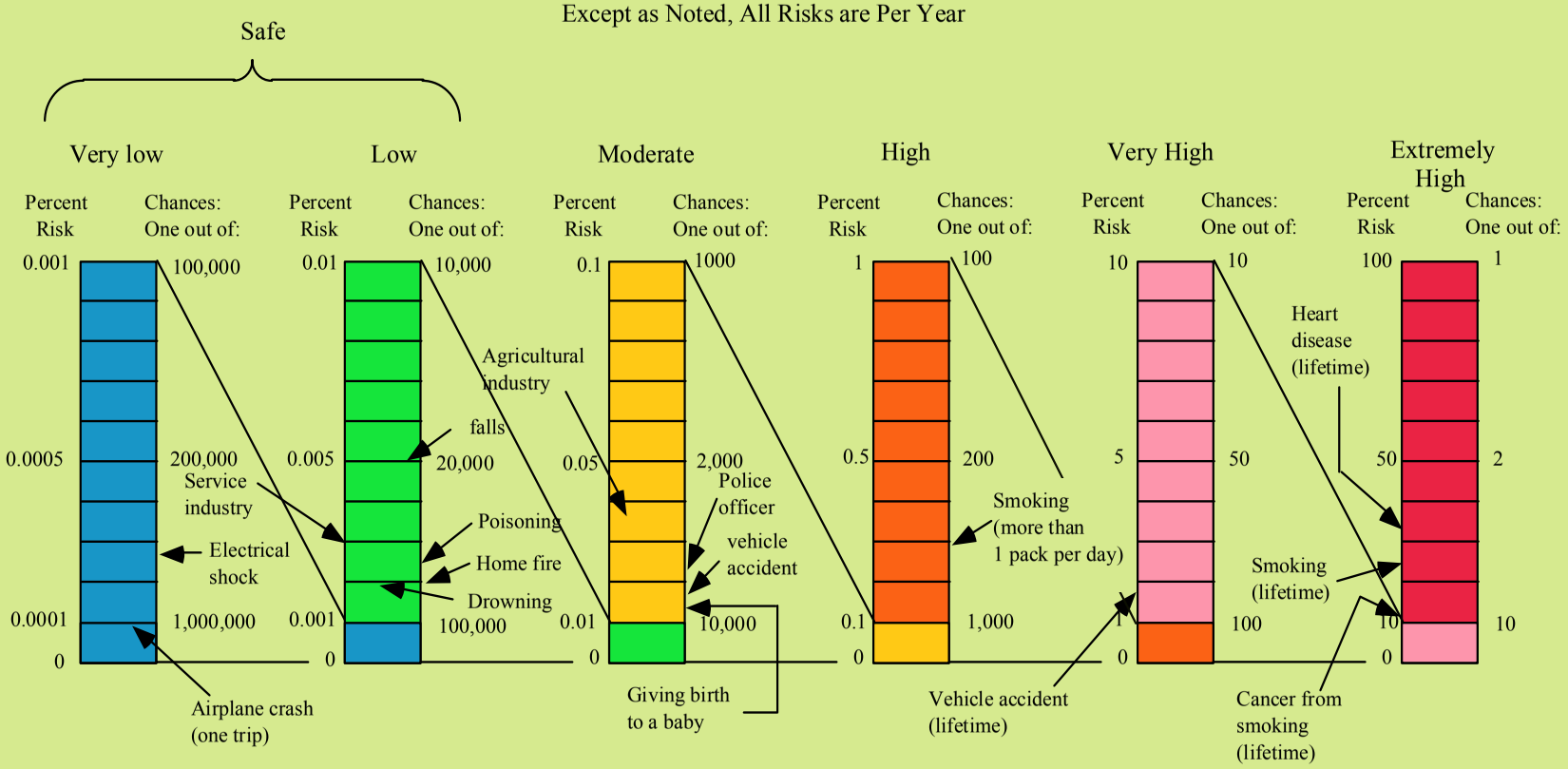
Relative Risk, Excess Risk and Excess Relative Risk

In the above example we have assumed that if nobody took part in the given activity, then nobody would be at risk. If nobody were to drive or ride in a vehicle, then there would be no vehicle fatalities. However, the situation for radiation is different. Even if nobody were exposed to radiation, there would still be fatalities from cancer from other causes.

Figure 8-1

Various Risks

Except as Noted, All Risks are Per Year



Sources: National Safety Council, 1991 data
The World Almanac (1994)
Breaking the Vicious Circle, Breyer, 1993

To determine the risk from radiation we need to determine the tumor and leukemia deaths that are attributable to radiation and use those numbers to determine the risk. We can represent the total cancer fatalities in a population as O (the Observed number) and the number of fatalities that we would expect from natural causes as E (the Expected number).

One way to use the observed and expected risk is to define a relative risk. This is just the ratio of O to E, or **relative risk** = O/E. If the observed number and the expected number are the same, then the relative risk is 1. This means that there is no risk. A relative risk of 1.5 means that we would see a 50 percent increase in natural mortality rate.

Another way to use O and E is to define excess risk. The number of fatalities that we can attribute to radiation, or the **excess risk**, would be just the difference between the observed and expected, or O - E. If O and E are equal, then the excess risk is zero.

For example, suppose a population of 10,000 people were exposed to a large dose of radiation and that the number of cancer fatalities in that group were 2200. The number of expected cancer fatalities in that group is 2000 (20 percent of 10,000). The relative risk would be 2200 ÷ 2000, or 1.1. The excess risk would be 2200 - 2000 = 200.We can also express the excess risk as a fraction (or percent). By dividing the excess risk by the expected number of fatalities, then we get a number called the **excess relative risk**. In our example, the excess relative risk would be 200 ÷ 2000 which is 0.1, or 10 percent. Note that a relative risk of one is equal to an excess relative risk of zero; a relative risk of 1.1 is an excess relative risk of 0.1.

What does this term mean? It means that if a person would be exposed to the same radiation dose as those in the example, the risk of developing cancer would be 10 percent <u>higher than the natural risk</u>. Note that it is not a 10 percent chance of developing cancer, it is <u>relative</u> to the natural cancer rate.

We can use these data to determine the risk to a general population. Suppose the excess relative risk is 10 percent and the natural cancer rate is about 20 percent. In a population of 100,000 we would expect to have 20,000 naturally occurring cancer fatalities and 200 additional fatalities from the radiation. The 200 cases represent 0.02 (or 2 percent) of the 100,000 population (200 ÷ 100,000). Therefore, the risk is 2 percent for the entire population.

In the previous chapter I discussed the linear, quadratic, and linear-quadratic curves. As we will see in the next chapter, a linear relationship gives the best fit to the risk of solid tumors from radiation. A linear-quadratic curve gives the best fit to the risk of leukemia.

For linear curves, we can define an **excess relative risk per dose**. This is just the excess relative risk divided by the dose that produces this risk. If you remember your algebra, this would be the slope of a graph of risk vs. dose.

If the dose in our example were 250 mGy, the excess relative risk per dose would be 0.1 ÷ 250 mGy, or, 0.0004 per mGy. This means that a dose of 1 mGy will increase the risk of a cancer fatality by 0.04 percent. Again, this is relative to the natural rate of 20 percent. The absolute risk would be 20 percent of 0.04 percent (0.2 X 0.0004 = 0.00008), or, 0.008 percent. As we will see in the next chapter, this is approximately the risk of cancer from radiation.

Loss of Life Expectancy (LLE)

Another way to quantify risk is to calculate the average loss of life expectancy (LLE). How many times do we say when an 80 year old dies in an accident "Well, that person had a long and full life." But if a 20-year-old dies, we say: "That person had so much to live for; it is a shame to die at such a young age." With these statements what we are really saying is that the life of the 20 year old has more value than that of the 80 year old. An 80 year old (average male and female) has 8 years of life expectancy while a 20 year old the life expectancy is 57 years.

Let's use an example to understand LLE. Suppose the risk of each of two activities is one death per 1,000 male participants per year. As a fraction this is 0.001 deaths per participant. But suppose that the average age of the participants on the first activity is 20 years and the average age of the participants in the second activity is 50 years. Now the two activities have different loss of life expectancies.

A 20-year-old male has a life expectancy of 57 years (i.e., is expected to live 57 years after the age of 20) while a 50-year-old male has life expectancy of 29 years (U.S. Department of Health and Human Services, 1989). The LLE of the 20-year-old group is 0.057 years (57 years multiplied by the probability 0.001), or 21 days (derived by multiplying the time in years by the number of days in a year: 0.057 X 365 = 21 days). The LLE of the 50-year-old group is 0.029 years, or 11 days.

This type of measure puts more value on the life of a younger person (National Research Council 259). Using LLE means that if we have limited resources to reduce risks, then we would spend it to save the young rather than the old.

One drawback with using LLE as a measure of risk is that it does not include the time from the initiation of an injury to the actual time of death. The best example is cancer. If a person is exposed to a carcinogen that will eventually cause that person's death, the death will occur many years after the exposure. For several years after exposure, the person has no visible effects from the carcinogen. When the cancer does appear, the person endures months to years of suffering before eventually succumbing to the disease. LLE only considers the time lost after death, not any of the time before the symptoms first appear nor the time of suffering before death.

In calculations of risk, many who compare risk from radiation with other risks like to use LLE. The major effect of radiation is to increase the risk of cancer. Like chemical carcinogens, there is a long time (years) between when a person receives the radiation dose and when the cancer first appears. Using the LLE risk calculation, the risk of radiation is reduced when compared to the risk of things like vehicle accidents, fires, and electrocution.

Let's consider an example of how an LLE calculation reduces the risk from radiation when compared to risks that have immediate effects. Consider two groups of one million people each. Group F is exposed to fires and 15 die in those fires. This is a risk of 15 out of one million. Suppose that Group R is exposed to a high enough dose of radiation that the risk of cancer mortality is also 15 out of one million. Both groups have an average age of 20 years so each has a life expectancy of another of 57 years.

Group F suffers the 15 deaths immediately, so the LLE of group F is 57 years X 15 deaths ÷ 1,000,000, which equals 0.00086 years or 0.31 days. However, the 15 cancer deaths in Group R appear an average of about 30 years later, when the average age of the group is 50 years. The life expectancy of this group is now 29 years, so the LLE is 15 X 29 ÷ 1,000,000 = 0.00044 years , or 0.16 days. This is about half that for the fire victims, yet both groups suffered the same number of deaths.

Those who argue for LLE say that, yes, the same number of people die but they had an average of 30 years to live after the radiation exposure. Those who argue against LLE say a death is a death, early or late in life, the result is the same.

As an absolute measure of risk, LLE has no meaning. A delayed death is just the same as an immediate death. Therefore, we will use mortality as the basis for our risk factors.

How much is a Human Life Worth?

If someone were to ask me to put a value on the life of a friend, a family member, or even a total stranger, I would refuse to answer. I would be upset even at the thought of assigning some monetary value for a life. There are too many aspects to a life that have value other than monetary. How can I put a price on caring, cheerfulness, compassion, happiness, and health?

In case of a fire in our house, we would not rescue the television, computer, workshop tools, or other "things." We would grab family pictures and family heirlooms first. Why? We could not replace them even if our insurance would pay for them.

In spite of our reluctance to put a value on life, we do just that every day. And not just for money. I get upset at drivers who weave in and out of traffic just to get somewhere in a hurry. Those drivers increase the risk of an accident for everybody else on the road. In effect, they are saying that their need to get somewhere in a hurry is worth risking the lives of others on the highway. If these drivers save a total of ten hours of time but cause one death, then their value for human life is ten hours of time saved. In terms of dollars, that is not much.

Courts decide the value of a human life every day. Families of victims of medical malpractice, industry and personal negligence, product liability, etc., sue for monetary settlements. Juries then decide how much to compensate the families for the loss of a life. These values can range from a few hundred thousand dollars to several millions.

Are there any guidelines for how much to award? Generally not, although sometimes attorneys use the potential lifetime earnings as a tool to convince a jury that the victim was worth millions (at $40,000 per year, a 30-year career means a total earnings of $1,200,000). However, "it [future earnings] undervalues those in society who are underpaid and places no value at all on people who are not in income-producing positions. In addition, it ignores the interpersonal effects of a death which may make the loss suffered much greater than any measurable financial loss." (Fischhoff et. al. 267)

When we decide how much money to spend to avert some risk of death, we are assigning a monetary value to a human life. In these cases the range of monetary worth of a human life can vary even more than what we see in courtrooms. A study of how much various activities cost to save one life show that the value of a life can vary from nearly zero to several billion dollars (Tengs, et.al. 369). I will present more details of this information in Chapter 18.

Every industry wants to have a safe work place so worker safety is something that receives considerable attention and money. Of course, a company wants to keep costs down to stay competitive, so the budget for worker safety cannot be unlimited. Someone must make choices about how to spend limited funds to get the most benefit.

Suppose that a chemical company must spend money to reduce the risk from a potential leak of some toxic material. As best determined by a risk analysis, the present risk of a toxic material leak is one chance in 10,000 for causing one fatality per year. By spending $1,000 a year that risk can be reduced to zero. To save one life, the company would have to spend $1,000 each year for 10,000 years, or, $10,000,000 to save one life.

Because they dread radiation, the public demands safeguards to limit their exposure to radiation. This kind of request is reasonable as long as the costs are not outrageous. However, when the costs of these safeguards per life saved is far above the cost of other activities, we need to consider the real value of these measures. In Chapter 18, I will discuss how radiation reduction measures compare with other lifesaving measures.

Can we define "safe?"

The purpose of all this work is to answer the question: "What is safe?" How do probabilities relate to something being safe? As we know, nothing is risk-free, so nothing is absolutely safe. Zero risk is impossible. To many people, the word "safe" means that the risks are acceptable. However, each person has an individual level of acceptable risk for different activities. A skydiver feels that jumping out of an airplane is safe, but most of us would put that in the unsafe category. A few people even feel that any airplane is unsafe.

To define "safe" we need to define an acceptable risk and call that "safe." If we define our homes safe from fires, falls, and electric shock, then we accept a risk of 0.00006 (one chance out of 17,000) per year. If we accept automobiles as safe, then we accept a risk of 0.00015 (one chance out of 6500) per year, almost three times less safe. If we accept manufacturing as a safe occupation, then we accept a risk of 0.00003 (one chance out of 33,000) per year. Note that manufacturing jobs are twice as safe as our homes.

Because nuclear power falls into the unknown and dreaded categories, people tend to associate a level of safety with a lower risk than our homes or automobiles or manufacturing jobs. To say that nuclear power should be as safe as our homes would mean that 15,000 fatalities per year would be acceptable. Obviously, this would be unacceptable!

Chapter 9
Analysis of the Evidence

The previous chapters have been preparation for this and the next three chapters. In these chapters I present the evidence that we have for the risks from radiation. To really understand what is in these chapters, you need a good understanding of the basics of radiation, radiation dose, how we do science, uncertainty, presentation of risk, and how we make extrapolations.

What I will do in this and the next chapter is to present the data from various reports on radiation risk. These reports use many individual studies. I could present the data from all these studies, but that would leave your head spinning! I feel that the reports I will cite are from groups of well-qualified scientists who have done a credible job summarizing all the studies.

The basic game plan is to present the findings of one group: the Biological Effects of Ionizing Radiation (BEIR) Committee of the National Research Council. These findings are in this chapter and cover the two latest reports, BEIR IV and BEIR V. Then, in chapter 10, I will discuss the findings of other reports that corroborate the findings of the BEIR reports. In chapters 11 and 12, I will discuss the studies of other scientists who disagree with the BEIR results. Some think the BEIR risks are too high (Chapter 11) while other think they are too low (chapter 12).

In these four chapters, I will present the cases with no, or at least very little, editorial comment. Finally, in chapter 13, I will present my own summary of the arguments presented in these chapters. This will be my editorial. You will be welcome to agree or disagree as you see fit.

The methods of analyses for BEIR IV and V are significantly different so we will treat them separately. BEIR V raises some basic questions about how to treat the data so I will discuss this report first.

The analysis of radiation risk presented in BEIR V is applicable to gamma and X-ray radiation only. Most of our radiation is from gamma radiation and X-rays. The only major contributor is radon, which we treat separately, Therefore, the risk values reported in these chapters are applicable to most radiation the public receives.

High Dose/High Dose Rate to Low Dose/Low Dose Rate

We are concerned with the effects of low doses only. However, most data we have on risk are from high doses. Let's first look at the data we have and consider how we can use it to predict a risk for low doses.

All of the studies mentioned in chapter 4 use subjects who received the radiation dose in one or more short periods. The atomic bomb survivors received a large dose at the time the bombs exploded then received high to low doses from the fallout from the bombs. The subjects who received medical exposures received high doses in one or more short periods. Because the individual received the dose in a short time, the dose rate (thousands of mGy per hour, for example) is also high. Therefore, the data are for high dose and high dose rates. But we want to predict the risk of radiation received at low doses and low dose rates; how do we get from high dose/high dose rate to low dose/low dose rate?

To make the jump, we must extrapolate from the data for high doses to that for low doses. <u>This method of extrapolating to low doses is the controversial part of risk calculations.</u> To make the extrapolation, the BEIR reports depended on studies of animals and on studies of cells exposed to radiation. Although we cannot apply animal data directly to humans, it does show some characteristics of the effects of radiation on living systems. Similarly, studies of cells can show how radiation causes changes in the DNA molecule although it cannot give any risk numbers directly.

To understand the reasoning behind the extrapolations used in the BEIR reports, we need to understand how radiation affects the DNA molecule. The DNA molecule is a double strand of spiraling molecules joined by a series of molecules between the spirals. Think of it as a twisted rope ladder.

Radiation, either directly or indirectly by oxidants produced by the radiation, can break a chemical bond in the spiral. Think of breaking one of the side ropes in a rope ladder. If the broken bond is repaired exactly as it was, then no damage is done. But if the bond is repaired incorrectly, then the DNA does not provide the correct code for producing enzymes, proteins, etc., or when

reproducing a new cell. Among the possible malfunctions of the incorrectly repaired DNA molecule is the production of cancerous cells.

When a DNA molecule suffers one broken bond, it has a good chance to repair itself. However, if it suffers two or more breaks, especially if the breaks are close to each other, the possibility of repair decreases. Therefore, a double break in a DNA molecule at one time is more damaging than two single breaks at different times.

The probability of breaking a single bond is proportional to the radiation dose. If the dose is doubled, twice as many X- or gamma-rays pass through the tissue and interact with twice as many molecules. Therefore, the probability of breaking a bond is doubled. The probability of starting a cancerous growth is also doubled.

This would argue for the risk being a linear function of the dose. From the discussion in Chapter 3, a linear function is a straight line represented by the equation $y = mx + b$; y is the cancer risk and x is the dose. The slope of the curve, m, is the ratio of the increase of y (risk) over the increase of x (dose), or the risk per dose.

The term b gives the value of y when x is zero. For radiation, we would expect that when the dose is zero ($x = 0$), the risk of cancer is also zero ($y = 0$). This means that b equals zero (i.e., there is no risk when there is no radiation). When we set b to zero, the equation becomes $y = mx$. In more descriptive terms, let R represent the response or risk and D represent the dose so the equation is $R = mD$. This equation is shown in figure 9-1(a).

When one spiral suffers a break, the other strand holds the DNA molecule together. This would be like breaking one side of a rope ladder. The other side will hold the ladder together until the first side can be repaired.

What if the DNA molecule suffers two breaks in the spiral, one break in one strand and the other break in the other strand? When this happens, especially if the breaks are near each other, the DNA molecule may become completely separated. The probability of a correct repair is much less than for a single break. This would be like breaking both sides of a ladder at the same rung. Making a repair is now more difficult especially if the pieces mix with other pieces of broken ladders.

To get two nearby breaks, we need two oxidants produced near each other. We have this situation for the types of radiation that produce many interactions close together, like alpha and neutron radiation. This is the reason that these kinds of radiation produce more biological damage per dose than gamma and X-radiation.

There are two ways that gamma and X-ray radiation produce oxidants close to each other. First, as indicated above, doubling the dose produces twice the number of oxidants and the probability of an oxidant breaking a bond doubles. But we are specifically concerned with breaking two bonds close to each other. How does the probability change with dose?

This is a case of simple probability, just like tossing coins. The probability of getting a head is 50 percent, or 0.5. If we toss two coins, what is the probability of getting two heads? It is just the probability of getting one head (50 percent) for the first toss multiplied by the probability for getting a head (50 percent) for the second toss. The total probability is proportional to the square of the probability of tossing one head (Probability = $(0.5) \times (0.5) = (0.5)^2$).

For a double break, the probability is the square of the probability for a single break. Because the probability of a single break depends on the dose, the probability of the two breaks will depend on the square of the dose. The equation for the probability of a double break is $R = aD^2$ where R is the risk, D is the dose. Figure 9-1(b) shows the shape of this curve.

What are the implications of the $R = aD^2$ curve? As shown in Figure 9-1, the risk from the dose-squared curve (curve (b)) is lower than that from the linear curve (curve (a)) at low doses. This is because the probability of a double break is smaller than a single break at low doses. However, at higher doses, the risk from double breaks becomes greater than from single breaks. This is because at the higher doses there is a larger probability of getting two interactions close enough to create two oxidants close to each other.

Because the risk will depend on both single and on double breaks, we need to combine these two into what we call a linear-quadratic equation: $R = mD + aD^2$. This function is shown in Figure 9-1 (c). Note that the values of m and a in the linear-quadratic equation are less than the corresponding values in the linear and quadratic equations. Adding curves (a) and (b) without

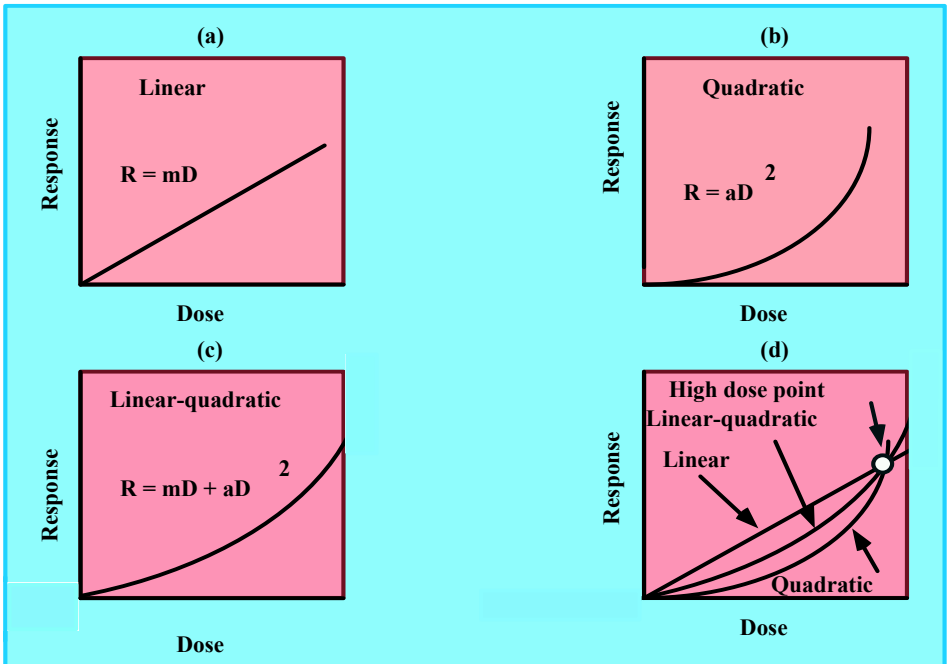


Figure 9-1

Comparison of linear, quadratic, and linear-quadratic curves

changing the values of m and a would produce a curve that passes above the high dose point shown in Figure 9-1(d). However, all curves must pass through this point.

All three are compared in Figure 9-1(d). Because we want to extrapolate from high- to low-dose data, all the curves have the same value at a particular high dose (for which we have data). We draw the shape of the curve starting at the high dose point (where the three curves intersect in Figure 9-1(d)). In addition, all must pass through the origin (zero risk at zero dose), so we get the curves shown. This is exactly how we extrapolated the highway fatalities in Chapter 7.We took the known fatality rate at the present maximum speed (which is equivalent to the high-dose point) and extrapolated to zero speed (or to zero dose). The number of predicted fatalities at lower speeds (or lower doses) depended on the kind of extrapolation.

What do these curves tell us? The linear curve has a constant slope so the risk increases steadily as the dose increases. However, the linear-quadratic curve starts out with a lower slope than the linear curve, so the risk does not increase as fast as the linear curve. This says that a small increase in dose produces a smaller increase in risk than the linear curve. However, as the dose increases, the slope increases. Eventually the slope becomes greater than that of the linear curve, so the risk increases faster with dose than the linear curve. In the lower dose region, the linear-quadratic curve says that radiation is safer than what the linear curve predicts.

The quadratic curve, which is not really used in radiation risk calculation, predicts an even smaller increase in risk at low dose. However, as the quadratic curve approaches the high-dose region, the slope is greater than either the linear or linear-quadratic curves, so it predicts a greater risk per dose in this region.

The second way to produce two oxidants close to each other is to have a high-dose rate. Oxidants have a finite lifetime. If radiation produces two oxidants at the same location but at different times, the oxidants cannot break adjoining DNA strands simultaneously. The DNA molecule may repair the damage from one oxidant by the time the second oxidant breaks the other strand. However, any dose, even a low dose, that occurs in a short time produces many oxidants at once and several could be close enough to produce a double break in the DNA molecule.

Figure 9-2 shows the results from a study of chromosome aberrations from different types of radiation. These curves clearly show that the type of radiation, the dose and the dose rate all affect the damage to the cell. The figure shows that at a given dose, the number of aberrations is greater for higher dose rates. For example, the number of aberrations from gamma rays at high-dose rate (500 mGy per minute) is about twice that from gamma rays at low-dose rate (3 mGy per minute). There is a similar relationship between high- and low-dose rate X-rays.

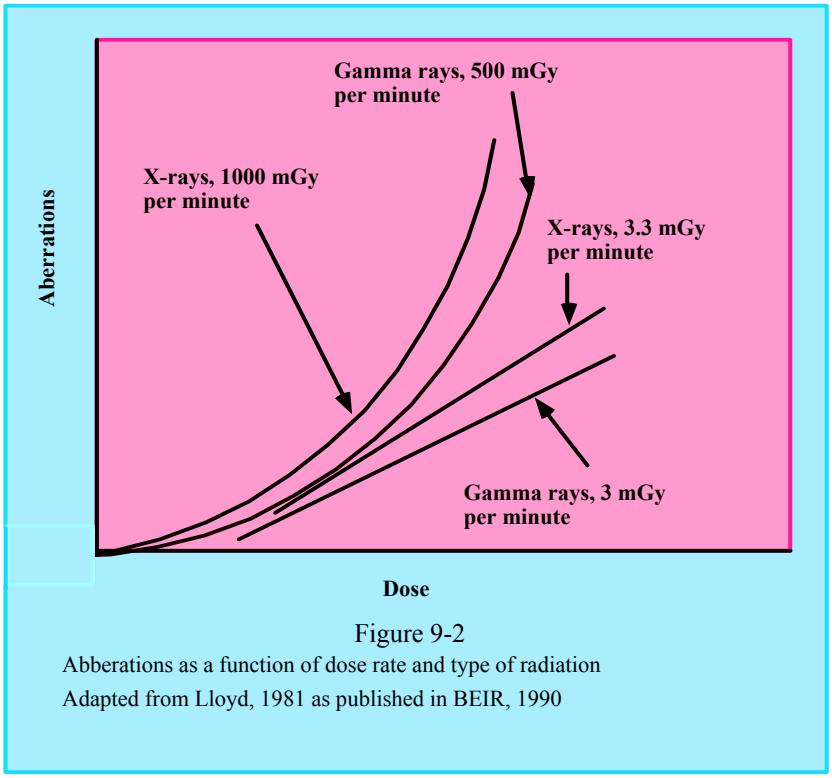


Figure 9-2

Abberations as a function of dose rate and type of radiation

Adapted from Lloyd, 1981 as published in BEIR, 1990

Studies show that higher-dose rates can produce risks between two and ten times larger than the same dose received at a low-dose rate (National Academy of Sciences (1990) 23).To account for dose rate, we will need to adjust the risk by a factor that relates the risk at high dose rates to low dose rates.

Similarly, the UNSCEAR-1988 report (United Nations Scientific Committee on the Effects of Atomic Radiation (1988)) considered the low dose and low-dose rate effects and concluded that "the carcinogenic effects of low-LET radiation are generally smaller at low doses and at low dose rates compared with those at high doses and high dose rates. The reduction factors will vary with dose and dose rate and with organ system but generally fall within the range 2 to 10" (United Nations Scientific Committee on the Effects of Atomic Radiation (1988) 492).

Extrapolations

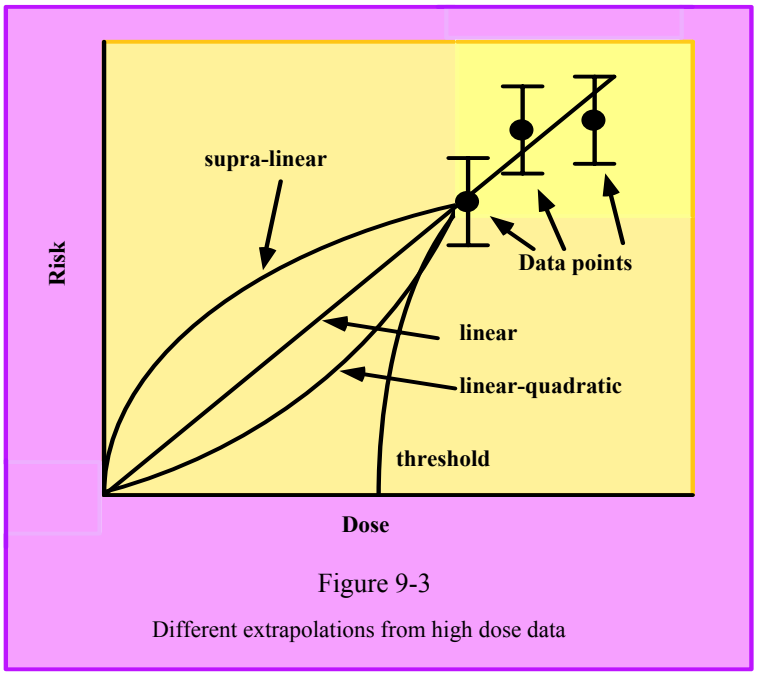


Figure 9-3

Different extrapolations from high dose data

Now we can apply possible extrapolations to the data we have. Figure 9-3 shows possible extrapolations from the high dose region to the low dose region. These extrapolations include not only the linear and linear-quadratic curves, but two other possible curves.

The extrapolation labeled threshold says that below a certain dose there is no effect from radiation. This curve is applicable to effects like cataracts and sterility discussed in Chapter 3. For these effects, a certain amount of cell damage is needed before the effect is apparent. Although the number of cells damaged may increase linearly (or linear-quadratically) with dose, the observable effect is not observed until enough cells are damaged.

The curve labeled "supra-linear" predicts that the risk increases rapidly at small doses but does not change much at higher doses. The slope of the curve gives the risk per dose, so this curve says that the risk per dose is larger for small doses than for high doses. I will discuss the

implications of this curve in Chapter 13 when I summarize the arguments about predicted risks being too high or too low.

Note that the linear curve is more "conservative" than the linear-quadratic curve. The real risk curve could be something between the linear and threshold curves (for example, it could be the liner-quadratic). If this is so, then the risk at low doses is actually less than the linear curve predicts. By conservative, we mean that if we use the linear model to predict cancer risk, then we should observe fewer cancers than the model predicts. This is like the safety factor that engineers use when designing a structure.

For most solid tumors, the linear extrapolation seems to give the best fit to the data. For leukemia, the best fit is linear-quadratic. For bone cancer, however, there apparently is a threshold. The minimum threshold is about 10,000 mGy (1,000,000 mrad) (Mills 147). This agrees with the BEIR V findings: "The data currently available from the study of Japanese A-bomb survivors provide no evidence of bone cancer resulting from low-LET irradiation at levels in the 0 to [4,000 mGy] range" (National Academy of Sciences (1990) 310). (Low dose rate gamma and X-radiation is low LET radiation.)

Absolute and relative risk

Now we need to consider how to apply the risk factors that we determine. Are the risks absolute or relative? To understand the difference between these two possibilities, let's do some supposing. Suppose a large population is exposed to some chemical or biological agent that causes severe itching of the skin. Suppose further that the incidence of severe itching of the skin is purely random, and not dependent on factors such as age, sex, health, nor the condition of any person's skin. Also suppose the probability of severe itching is 10 percent at a dose of one unit of exposure, 20 percent at two units, etc. If 100,000 people are exposed to 1.5 units of the agent, then we would expect 15,000 people to get severe itching (1.5 units X 0.1 chance per unit X 100,000 people). This would be considered an absolute risk of getting severe itching.

What if the risk of severe itching depends on a skin condition, say the number of wrinkles in the skin? As people age their skin has more wrinkles, so the older population has a greater probability of itching from the agent. For example, a 30 year old with one wrinkle per square inch could have a probability of 10 percent of severe itching from one unit of the agent. A 60 year old with two wrinkles per square inch would have twice that probability, or, 20 percent per unit. In a population of 100,000 where 2,000 are 30 years old and 2,000 are 60 year olds, the 30 year olds would have 300 with itchy skin but the 60 year olds would have 600 with itchy skin. This would be considered a relative risk; the risk is relative to some other factor.

The incidence of cancer increases with age. Slowly the cells lose their ability to repair damage from oxidants so they become more sensitive to any chemical, biological, or radiological agent that can damage the cell. They become cancerous more readily.

The question then is: does the risk of cancer from radiation depend on the natural risk of cancer (relative risk) or is it independent of the natural cancer rate (absolute risk)? Before the BEIR V report, scientists either used the absolute model, or they used both with no preference given for either. However, the findings published in BEIR V show that the relative risk model is more appropriate. Therefore, the risk determined in BEIR V is a risk that depends on the naturally increasing risk of cancer.

The risk factors that we will develop later represent a combination of the relative risk and the natural cancer rate for that cancer. For example, the relative risk from radiation over the natural risk factor could be 0.1 (10 percent). If the natural risk at one age is 0.01 (1 percent), then the expected number of cancer fatalities due to radiation at this age would be 0.001 (0.1 X 0.001), or 0.1 percent of the population. If the relative risk is the same for some later age where the natural risk is 0.05 (5 percent), then we would expect to have 0.005 (0.1 X 0.05), or 0.5 percent, of this population as cancer fatalities. Note that although the relative risk does not increase with age it still produces more cancer fatalities with age because the people at a later age are more likely to get cancer. This is how the relative risk model works.

BEIR V Risk Values

Now that we have our models, we can look at the risk that they predict at low doses. Ideally we would like to have risk values for each type of cancer for any dose at any age of the person and for either sex. To get such numbers with some reasonable accuracy we would need thousands of people in each group. We just do not have sufficient data to do this. As a compromise between accuracy and variety, BEIR V limited their analysis to leukemia and cancers of the respiratory tract, breast, digestive tract, and thyroid.

Here I would like to insert an important word of caution. Most of the risk values discussed in this chapter are based on the atomic bomb survivors. Although this group is large and received a large dose, the excess number of cancer deaths for this group is less than 350 -- about 80 are from leukemia and the others from solid tumor deaths. The number in each category of cancer type, sex, and age is small. As a result, the uncertainty in any risk calculation for any subgroup is large. Taking the group as a whole will give a risk value that is more certain, but not age, sex, or type specific. Though we would like to glean as much information out of these data, we must remember that the small number of cases make any risk value highly uncertain. But it is the best we can do.

Another problem with the atomic bomb survivor data is that the natural incidence rate of different types of cancers is not the same in different countries. The Japanese have a much higher incidence of stomach cancer but a lower incidence of colon/rectal cancer and a significantly lower incidence of breast cancer than U.S. citizens. The risk values in this chapter had to be adjusted to fit the cancer incidence rates for the United States.

Latency and at-risk period

The risk from radiation does not appear immediately after the radiation exposure and the risk apparently does not extend forever. Figure 9-4 shows a graph depicting the latency period and the period at risk for leukemia. This figure is a composite of different age groups exposed to radiation from the atomic bombs. BEIR V uses a two-year latency period for leukemia and a ten-year latency period for solid tumors.

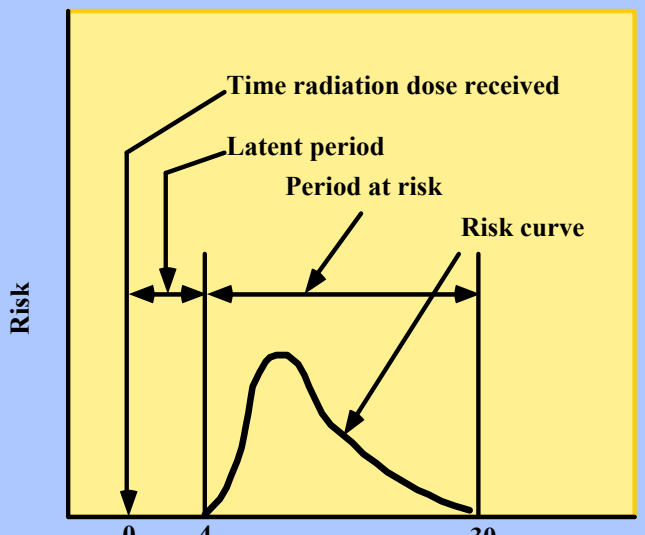


Figure 9-4
Composite of leukemia risks
(adapted from p. 339, BEIR, 1980)

The actual latency period for leukemia ranges from two to about eight years depending on the age at exposure. At ages less than 15 years, the latency period is two years. At ages above 45 years, the latency period is about eight years.

After a person receives a radiation dose that produces cancer, the cancer appears in the time labeled "period at-risk." For leukemia, that period at-risk lasts between 15 and 30 years after exposure. The younger ages have the shorter periods at risk. For solid tumors the data do not show an end to the at-risk period. However, this information is coming from the bomb survivors and if there were a similar end to the at-risk period it would be in the 1980s to 1990s. Those data are still under analysis.

Risk values

To give numbers for the risk from a radiation exposure we need to specify the age of the person, the age at the time of the exposure, the dose received, and for some cancers, the sex of the person. These five factors make it difficult to present the risks in a simple, straightforward manner.

However, we can add up all the annual risks to get a lifetime risk that has a reasonable accuracy. Such a number is more understandable than a set of risk numbers for each year. Tables 9-1, 9-2, and 9-3 give the lifetime risks from BEIR V for leukemia and solid tumors. Because the lifetime risks depend on three factors (age at exposure, dose and sex) we would need many tables to cover all possible situations. BEIR V chose exposures that are similar to real situations.

Table 9-1 gives the risk from a single exposure of 100 mGy (10 rad) received by an entire population (with all age groups represented). This would represent a situation where an entire population received a radiation dose from something like a nuclear weapon detonation or a nuclear power plant accident (for example, Chernobyl). For the solid tumors the risk is linear with dose so the risk factor should be applicable for any dose, i.e., a dose of 50 mGy (5,000 mrad) would be half that given in Table 9-1.

The risk factor for leukemia follows a linear-quadratic model so that is not directly transferable to other doses. The leukemia risk from half this dose would be slightly less than half the risk shown. The risk from twice this dose would be slightly more than twice the risk shown.

Table 9-1
Excess cancer mortality from a single exposure to 100 mGy (10,000 mrad)
Risk per 100,000 exposed persons
Adapted from National Academy of Sciences(1990) 172

	Male			Female		
	Leukemia	Tumors	Total	Leukemia	Tumors	Total
Expected Mortality	110	660	770	80	730	810
Range	50-280	420-1040	540-1240	30-190	550-1020	630-1160
Normal expected	760	19,750	20,510	610	15,540	16,150
Excess relative risk[1] (percent per mGy)	0.14	0.033	0.038	0.13	0.047	0.050
Actual risk[2] (percent per mGy)	0.0011	0.0066	0.0077	0.00080	0.0073	0.0081
Average years of life lost per excess death			16			18

[1]The excess relative risk is derived by dividing the expected mortality by the normal expected mortalities. The excess relative risk in percent per mGy is the excess relative risk divided by 100 mGy and multiplied by 100 to convert the fraction to a percent. The two 100's cancel so the number is the same.

[2]Derived by dividing the expected mortality by 100,000, dividing by 100 mGy and multiplying by 100 to convert the number to a percent.

The total risk of this population is calculated from the annual risks for each age group. Although this calculation gives a number, this number has considerable uncertainty. The range gives the number of expected cases that BEIR V is 90 percent confident will come from radiation; i.e., we are 90 percent confident that the actual number of fatalities will be somewhere in this range.

In Chapter 8, I defined the excess relative risk as the excess risk relative to the natural risk. It is the excess fatalities divided by the number expected naturally. From the data in Table 9-1, the excess relative risk for male tumors is (660 ÷ 19,750) which is 0.033, or, 3.3 percent. For females the excess relative risk is (730 ÷ 15,540) which is 0.047 per mGy, or, 4.7 percent per mGy. These values

are listed in Table 9-1. To put these values into perspective, the excess relative risk for smoking is close to 20.

The excess relative risk per dose for males is 0.033 ÷ 100 mGy, which is 0.00033 per mGy, or, 0.033 percent per mGy. For females, the excess relative risk per dose is 0.047 percent per mGy.

Table 9-2 shows the excess cancer mortality from continuous exposure of 1 mGy per year (100 mrad per year). This is about what we receive from background radiation, excluding the lung dose from radon. This information would be useful for calculating the expected risk in areas that have background radiation higher than the average (Denver, for example). The data in Table 9-2 suggests that about 3 percent (average of 2.5 and 3.4 percent risks for male and females) of all tumors and leukemia are from background radiation, excluding radon. I will have some pertinent comments about this at the end of Chapter 10.

Table 9-2
Excess cancer mortality from a lifetime exposure of 1 mGy (100 mrad) per year
Risk per 100,000 exposed persons
Adapted from National Academy of Sciences (1990) 172

	Male			Female		
	Leukemia	Tumors	Total	Leukemia	Tumors	Total
Expected mortality	70	450	520	60	540	600
Range	20-260	320-830	410-980	20-200	430-800	500-930
Normal expected	790	19,760	20,560	660	16,850	17,520
Excess relative risk	0.089	0.023	0.025	0.090	0.032	0.034
Percent risk[1]	0.07	0.45	0.52	0.06	0.54	0.60
Average years of life lost per death			16			18

[1]Derived by dividing the expected mortality by 100,000 and multiplying by 100 to convert to a percent.

Table 9-3
Excess cancer mortality from a continuous exposure to 10 mGy (1,000 mrad) per year from age 18 until age 65
Risk per 100,000 exposed persons
Adapted from National Academy of Sciences (1990) 173

	Male			Female		
	Leukemia	Tumors	Total	Leukemia	Tumors	Total
Expected mortality	400	2480	2880	310	2760	3070
Range	130-1160	170-4560	2150-5460	110-910	2120-4190	2510-4580
Normal expected	780	20,140	20,910	650	17,050	17,710
Excess relative risk	0.51	0.12	0.14	0.48	0.16	0.17
Percent risk	0.40	2.48	2.88	0.31	2.76	3.07
Average years of life lost per death			15			17

Table 9-3 gives the risk from a continuous exposure of 10 mGy per year (1 rad per year) from age 18 to age 65. This is about what nuclear power plant workers receive. These risk values would be useful for calculating the risk to nuclear workers.

Often we would like to have a single-risk figure (i.e., risk per person-mGy or per person-mrad) for "quick and dirty" risk calculations. From Table 9-1 the average number of solid tumors

and leukemia fatalities in males and females from a single 100 mGy (10,000 mrad) dose is 790. The total dose to the 100,000 persons is 10,000,000 mGy (100,000 people X 100 mGy). This gives a population dose of 10,000,000 person-mGy. The risk per person-mGy is 0.000079 (790 fatalities ÷ 10,000,000 person-mGy). Or, the risk is 7.9 X 10^{-7} per person-mrad. We will round this off to 0.00008 per person-mGy or 8 X 10^{-7} per person-mrad.

A similar calculation for a 1 mGy (100 mrad) continuous exposure in Table 9-2 also gives a risk of 0.00008 per person-mGy. The risk to nuclear power plant workers is a little lower. The total dose to the 100,000 workers during their 47 years between the ages of 18 and 65 is 47,000,000 mGy (100 mGy X 47 years X 100,000 people). The average number of fatalities is 2980 so the risk is 0.000063 per person-mGy (2980 ÷ 47,000,000 mGy), or 6.3 X 10^{-7} per person-mrad.

Table 9-4 gives a breakdown in the risk depending on the age at exposure. The high risk from cancer at the early ages is because the at-risk period extends over most of the individual's lifetime. The risk of leukemia is high both at the early and late ages because the natural rate of leukemia is high at these ages.

Table 9-4
Excess mortality as a function of age at exposure
from an exposure of 100 mGy (10 rad)
Risk per 100,000 person exposed
Adapted from National Academy of Sciences (1990) 175

	Male			Female		
Age at exposure	Leukemia	Tumors	Total	Leukemia	Tumors	Total
5	111	1165	1276	75	1457	1532
15	109	1035	1144	72	1494	1566
25	36	885	921	29	1149	1178
35	62	504	566	46	511	557
45	108	492	600	73	468	541
55	166	450	616	117	388	505
65	191	290	481	146	240	386
75	165	93	258	127	100	227
85	96	14	110	73	17	90

Before we leave the BEIR V calculations, let's look at the numbers in Tables 9-1, 9-2, and 9-3 to see what they mean. If we had a population of 100,000 men exposed to the radiation doses given, could we actually observe the increase in radiation-induced cancers above the natural incidence rate? When considering solid tumors from a single 100 mGy (10,000 mrad) dose to males in Table 9-1, the normal number of fatalities would be 19,750. If we use the square root of this number to determine the uncertainty (68 percent confidence level), then we get a number of 140. The 660 expected cases are significantly above this number so they would be observable. If we want to use the 90 percent confidence level, then we would need 230 cases, still within the 660 expected.

However, if the population had only 10,000, and 66 expected solid tumors from radiation, the results would be different. For this population we would expect 1,975 cases naturally. The uncertainty for this number is 45 at the 68-percent confidence level and 73 at the 90-percent confidence level. Here the expected increase is above the 68-percent confidence level but less than the 90-percent level. If we did observe an increase of 66 cases, we would be only 84-percent confident that those cases were from radiation-induced cancers and not just from a statistical fluctuation in the natural cancer rate.

Another question to ask is how many people would we have to study to see the expected number of excess cancers from radiation. Let's use Table 9-2 as an example. From Table 9-2 the expected excess for cancer is 540 for 100,000 women exposed to 1 mGy (100 mrad) per year, which is a 3.2-percent increase over the natural incidence rate of 16,850. How many cancer fatalities would we have to study so that the uncertainty in the natural rate is less than 3.2 percent? We would need

1000 cancer fatalities, or we need to study a population of about 6,000 females. To be at the 90-percent confidence level we would need to have over 2,500 cancer fatalities which would be found in a population of over 15,000.

Genetic Risks from BEIR V

Radiation causes genetic mutations. However, as with cancer, we do not have good data to determine how many mutations radiation causes per dose. Radiation is only one of many causes of mutations, so the natural incidence completely masks those caused by radiation. One major difficulty with getting human data on mutations is that the minimum time lag between cause and effect is one generation. This makes the minimum follow-up time 30 to 40 years. Because no good human data exists on the genetic damage done by radiation, all predictions of risk come from studies with animals, mostly mice.

The genetic disorders studied included autosomal dominant disorders, recessive gene disorders, chromosomal disorders, and congenital abnormalities. Autosomal dominant disorders include diseases such as Huntington's disease, dwarfism, and mortality (i.e., the fetus or infant does not survive). Recessive gene disorders include diseases such as cystic fibrosis, hemophilia, Tay-Sachs disease, and sickle-cell anemia. Chromosomal disorders include Down syndrome, Klinefelter syndrome (sterile male), and Turner syndrome (non maturing of female sex organs). Congenital abnormalities include cleft palate, spina bifida, and congenital inguinal hernia.

Using animal data, the BEIR V Committee made risk estimates for the number of cases in the first generation and the number of cases per generation after many generations. Table 9-5 gives the risks for these disorders.

Table 9-5
Estimated Genetic Effects from 10 mGy (1 rad) exposure
per Generation per one million liveborn offspring
Adapted from National Academy of Sciences (1990) 70

Disorder	Natural incidence	First generation	Equilibrium
Autosomal dominant	10,000*	5 - 30	100
Recessive	2,500	<1	very slow increase
Chromosomal	4,400	<6	very little increase
Congenital	20,000 - 30,000	10	10-100
Total	37,000 - 47,000	20 - 45	

*2,500 clinically severe, 7,500 clinically mild

The BEIR V Committee studied the bomb survivors for genetic disorders and reported: "A pregnancy termination study analyzed some 75,000 births, of which 38,000 had at least one parent who was exposed to radiation. No significant effects on still births, birth weight, congenital abnormalities, infant morality, childhood mortality, leukemia, or sex ratio were found. A significant distortion of the sex ratio had been reported, but the effect subsequently disappeared" (National academy of Sciences (1990) 94).

Besides expressing congenital disorders per dose, another way to express the genetic effect of radiation is to determine the dose needed to double the natural incidence rate. This is called the doubling dose. For genetic effects the doubling dose is estimated to be 1,000 mGy (100,000 mrad).

Radon and Lung Cancer from BEIR IV

The BEIR IV Committee (National academy of Sciences (1988)) had a choice of two methods for calculating the risk of lung cancer from radon: either a dose model, or using epidemiological data. A dose model would require the calculation of a dose to the lungs from radon and the radioactive elements generated by the decay of radon. Then the model would use this dose with the BEIR V risk models to calculate the risk of lung cancer.

Such a dose model requires many notable assumptions about the deposition of the dust particles in the lung, the dose to lung cells, breathing rates, and clearance rates of radon progeny. Because these assumptions would cause large uncertainties in the calculated risks, the Committee chose to use epidemiological data to generate the risk values.

The Committee used the Working Level Month (WLM)[1] as the measure of dose because the radiation dose received by the respiratory system is mostly from radon progeny. (The Working Level Month is a measure of the total alpha radiation energy deposited in the lungs by radon's radioactive progeny, polonium.) All but the Colorado study had risk data up to about 250 WLM. The Colorado study had data up to 2,500 WLM. The Committee plotted the risk of lung cancer from radon progeny exposure as a function of WLM for each of the four groups.

Three of the four showed a linear relationship between risk and dose. The fourth, the Colorado, had a linear relationship for exposures below 2,000 WLM, so the committee did not use any data above 2,000 WLM in the final analysis. Then they combined the four studies to get better statistics.

As we learned in the last section, the effects of radiation depend not only on the dose but also on the age of the person and the age at exposure. Therefore, besides the cumulative dose, the Committee considered factors such as the duration of the exposure, the age of the individual, the age at exposure, and the time since the first and last exposure for those miners who did not have continuous exposures.

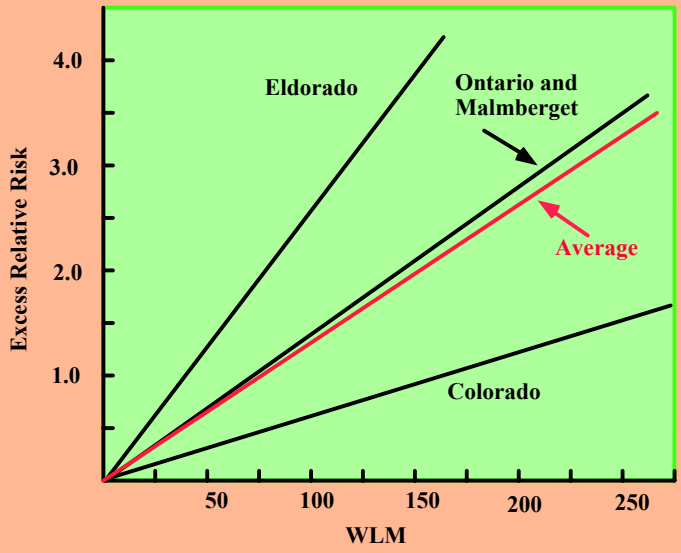


Figure 9-5
Lung cancer risk as a function of Working Level Month for three miner studies.

Although the data provided a linear relationship between risk and dose, the slope of the line was not the same for each group. Figure 9-5 shows the best fits to each set of data and the average fit for all four sets. The average slope is 0.0134 per WLM, just a little below the Ontario and Malmberget curve. The average, of course, has some uncertainty; the 95-percent confidence range is 0.008 to 0.023 per WLM. A risk of 0.0134 per WLM means that a miner has a 1.34 percent increased risk of lung cancer for each WLM of exposure.

The model used by the Committee for the relative risk accounts for the age of the miner and the time since past exposures. The risk to miners decreases after the age of 55, by 27 percent between the ages of 55 and 65, and by 67 percent after the age of 65. Any dose received more than 15 years before the selected age of the miner is considered half as effective as a dose received within 15 years. For example, the dose received by a 60-year-old miner before the age of 45 is considered only half as effective as the dose received from age 45 to 60.

Uncertainties

Three major questions arise about the risk projections based on these four groups of miners. Can we project data from miners to people in houses? How can we get risk data for females if all the miners were males? How does smoking affect the risk?

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[1] The Working Level Month is a measure of the total alpha radiation energy deposited in the lungs by radon's radioactive progeny, polonium.

The conditions in a mine are considerably different from those in a dwelling. The main differences are the size of the dust particles in the air and the breathing rates of a miner compared with a person sitting in a house. Calculations of comparative risk per dose show that the risk per dose to people in a house is about 70 to 75 percent of that of miners (National research Council 45).

To answer the question about projecting a risk to females, the committee had little data to guide them. Based on what data they had, they assumed that the relative risk for females should be slightly higher than that for males. This is consistent with the relative risk model for radiation exposure to the lungs from the BEIR V study. However, to use the relative risk we must apply it separately to the natural lung cancer risk for men and women which produces a lower total risk for females.

Multiplicative effects of smoking

Three of the four groups did not have any data on the number of miners who smoked, only the Colorado group had such data. However, the Committee also used data from a group of New Mexico uranium mines and data from the atomic bomb survivors to draw conclusion about the effect of smoking. In each of these studies there is information on the smoking habits of the subjects. The results of these studies showed that the risk of radon progeny exposure and smoking is multiplicative, i.e., the risk from the combination of the two is much higher than the addition of the separate risks.

For example, a male nonsmoker exposed to 8 pCi/L[2] for all his life has a risk of 0.0173 of lung cancer mortality. A male smoker who has no exposure to radon progeny has a lifetime risk of 0.123 of lung cancer mortality. For a smoker exposed to 8 pCi/L for a lifetime, the two risks added together would be: 0.0173 + 0.123 = 0.140. However, the risk calculated by the BEIR IV Committee is 0.182, which is a factor of 1.3 above the added risks. For females, the ratio is 1.33. At higher pCi/L values, the ratio increases, rising to 2.7 for males and 3.3 for females at 80 pCi/L.

Table 9-6 is derived from the BEIR IV report and gives the relative risk and lifetime risk for lung cancer from radon progeny as a function of pCi/L. Figure 9-6 is a plot of this data. You will note that relative risk (R_e/R_o) for smokers is less than that for nonsmokers. However, this is a relative risk. To get the total risk, multiply the relative risk by the natural risk, which is much higher for smokers. As a result, the actual risk (R_e) for nonsmokers is much lower than for smokers. The loss of life column (L) clearly shows the difference between the risk for smokers and nonsmokers.

The natural risk (R_o) for each is the top value in the R_e columns. Values of R_e are derived by multiplying the R_e/R_o column by the top value in the R_e column. Table 9-6 also gives the values for years of life lost for the conditions given. These values clearly show the multiplicative effects of smoking and exposure to radon progeny.

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[2] pCi/L is picoCuries (pCi) of radioactivity per liter (L) of air. A picoCurie is a measure of the activity of a radioactive material. A one pCi sample of radon will emit 133 alpha particles per hour. In the home, a concentration of 8 pCi/L will produce about 1 WLM per year.

Table 9-6

Lifetime Relative risks (Re/Ro), total risk (Re), and years of life lost (L) from the radon exposures listed. These risk values are based on 70 years of exposure in a house for 12 hours per day and 50 percent equilibrium between radon and its progeny

Adapted from National Academy of Sciences (1988)

	Males						Females					
Radon	Nonsmokers			Smokers			Nonsmokers			Smokers		
pCi/L	R_e/R_o	R_e	L	R_e/R_o	Re	L	R_e/R_o	Re	L	R_e/R_o	Re	L
0	1.00	0.011	0.000	1.00	0.123	1.50	1.00	0.0060	0.00	1.00	0.058	0.81
2	1.08	0.013	0.022	1.13	0.138	1.74	1.14	0.0068	0.016	1.14	0.066	0.95
4	1.27	0.014	0.045	1.24	0.153	1.98	1.28	0.0078	0.030	1.27	0.074	1.10
6	1.41	0.016	0.068	1.37	0.167	2.21	1.43	0.0086	0.045	1.41	0.082	1.24
8	1.54	0.017	0.091	1.48	0.182	2.44	1.57	0.0095	0.061	1.54	0.090	1.38
10	1.68	0.019	0.113	1.59	0.196	2.67	1.71	0.0103	0.076	1.67	0.097	1.53
15	2.02	0.023	0.169	1.86	0.229	3.22	2.07	0.0125	0.114	2.00	0.116	1.88
20	2.35	0.026	0.225	2.12	0.250	3.75	2.42	0.0146	0.151	2.32	0.135	2.22
30	3.02	0.039	0.337	2.58	0.317	4.76	3.12	0.0188	0.228	2.92	0.170	2.90
40	3.68	0.041	0.447	2.99	0.368	5.68	3.83	0.0231	0.301	3.51	0.204	3.55
80	6.24	0.070	0.883	4.24	0.521	8.77	6.59	0.0398	0.598	5.56	0.324	5.98

Derived from corrected Table 2-4, National Academy of Sciences (1988)

Let's look at some examples from Table 9-6 and Figure 9-6 to see what they mean. The risk, R_e, given is the absolute risk of a person dying of lung cancer from radon. For example, the risk of dying of lung cancer for a male nonsmoker who has zero radon exposure is 0.0112, or 1.12 per cent. For a male smoker exposed to a radon level of 10 pCi/L, the risk is 0.196, or 19.6 per cent. Or, for 100,000 male smokers who have radon exposures of 10 pCi/L, about 19,600 will die of lung cancer.

Let's get some idea of how these risks compare with that from those in BEIR V. Data from Table 9-1 show that a single 100 mGy dose produces an actual cancer risk of 0.0077 percent per mGy for males. How many mGy would we need to produce a lifetime risk of 19.6 percent for a male smoker living in a house with 10 pCi/L radon concentration? To determine this, we just divide 19.6 percent by 0.0077 percent per mGy. This gives 2550 mGy, or about 2.5 Gy (255,000 mrad, or 255 rad). Over a 70-year lifetime, the average annual dose would be 36 mGy.

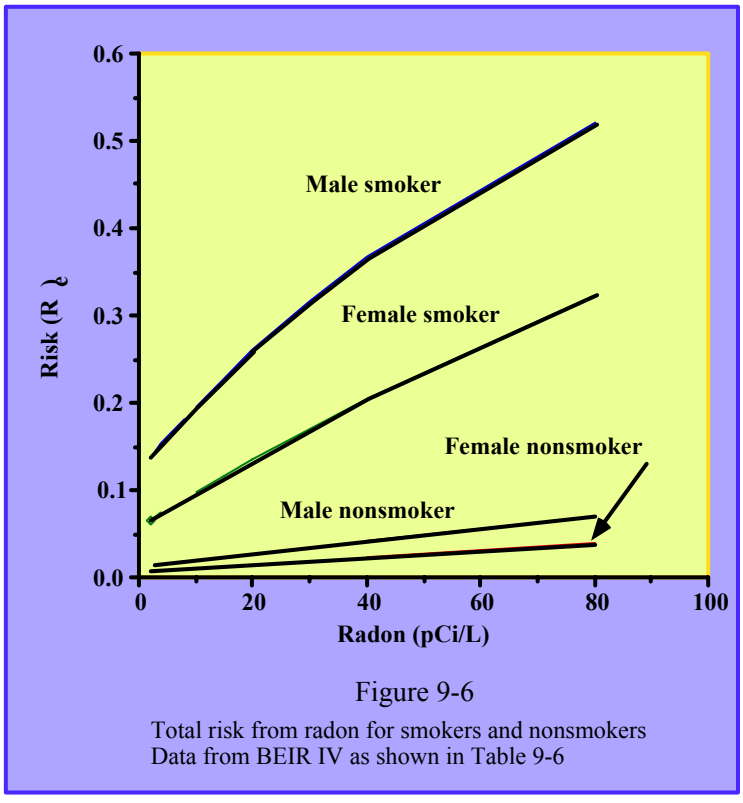

Figure 9-6

Total risk from radon for smokers and nonsmokers
Data from BEIR IV as shown in Table 9-6

Or, consider a nuclear worker. Over a lifetime of work receiving 10 mGy (1,000 mrad) a year, a male nuclear worker has a cancer risk of 2.88 percent. Dividing 19.6 percent by 2.88 percent per lifetime, we get 6.8 lifetimes. This means that a male smoker living in 10 pCi/L for a lifetime has the same risk as a nuclear worker getting 6.8 X 10 mGy per year, or 68 mGy (6,800 mrad) per year. This is 18 mGy (1,800 mrad) above the 50 mGy

annual limit for a nuclear worker.

The literature published by the EPA on the risk of radon sets the annual number of radon deaths at between 7,000 and 30,000, with 14,000 as the best estimate. (EPA, 1992) If every house miraculously had zero radon, would 14,000 lives be saved every year? Not according to the data in Table 9-6. Those smokers who would have died from radon and smoking induced lung cancer would still have a large risk from smoking. The number of lung cancer fatalities among smokers would decrease more from everybody quitting smoking than from reducing the radon levels to zero in all the houses.

A closer analysis of the Colorado miner data showed some interesting results about the period of radon exposure relative to the period of smoking. Those with the radon exposure prior to smoking had a slightly higher risk of lung cancer than those who smoked prior to the radon exposure (with the end of the smoking period coming before the beginning of the radon period). This might suggest that radon may be more of an initiator of cancer than smoking, and smoking more of a promoter (Thomas et al.).

Chapter 10
Corroborating Evidence

One report is not enough to justify a risk value. Although the BEIR V (BEIR, 1990) report is based on many individual studies, it represents just one group looking at the data. To be more certain of the risk values derived in BEIR V, we need more studies, and for other groups to look at the same and new data to verify the values in BEIR V. This chapter will summarize several other large studies. At the end of this chapter I will summarize the studies and suggest a risk value that I think is appropriate based on the various studies considered in this chapter.

Risk values

The other major studies on the effects of radiation are: the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) reports published in 1988 (United Nations Scientific Committee on the Effects of Atomic Radiation (1988)) and 1994 (United Nations Scientific Committee on the Effects of Atomic Radiation (1994)), the United States Environmental Protection Agency (EPA) report published in 1989 (Environmental Protection Agency), the International Committee on Radiological Protection (ICRP) report 60 published in 1991 (International Committee on Radiological Protection), and the Radiation Effects Research Foundation (RERF) report in 1994 (Thompson et al., Preston, et al.). All these studies used the atomic bomb survivors as the major source of data. However, each report included data from a variety of other studies.

Although these may seem like independent studies, they are related. The BEIR V, UNSCEAR and RERF reports are independent but the other two are not. The ICRP uses the results of BEIR V and UNSCEAR for its risk calculations.

The EPA report uses the results of BEIR III (National Academy of Sciences (1980)) and, according to one assessment, “the EPA has based its risk assessment on outdated information” (Wahl). Therefore, I will not include the EPA risk assessment values in this summary. The major problem with the BEIR III report, and therefore the EPA report, is that the BEIR Committee based its risks on the atomic bomb survivor radiation doses calculated before the dose corrections in 1986. As a result, those risk predictions are lower than those that use the corrected dose data.

I must also note that the studies had several other differences. As in BEIR V, the other studies used the relative risk model but UNSCEAR-1988 and EPA also used the absolute risk model for some calculations. BEIR V, ICRP, and EPA used U.S. population statistics for their sample population; UNSCEAR used a combination of Japanese and United Kingdom populations. All but the ICRP study used a two-year latency period for leukemia; ICRP used 10 years. All used 10 years for solid tumors.

All four studies used the linear extrapolation for the solid tumor data, but the ICRP reduced the low dose risk by a factor of two to account for the difference in high and low dose rates. Two of the studies used the linear-quadratic extrapolation for leukemia and two used the linear.

Table 10-1 gives the calculated risks for leukemia and four types of cancers for the reports that came out in 1988 through 1991. Except the higher value for digestive cancer by ICRP, the other values are quite close.

To help understand these numbers, let's convert the fractional risks in Table 10-1 to one chance out of some number. To convert from a percent risk, add two decimal places (to make the number a fraction) and then invert the number. For example, the UNSCEAR leukemia risk is 0.00100. Adding two decimal places gives 0.0000100. Inverting this (1 ÷ 0.0000100) gives 100,000. The risk is one chance out of 100,000. The average leukemia risk of 0.00098 percent is one chance out of 102,000; the average breast cancer risk of 0.00035 percent is one chance out of 286,000; and the total average risk of 0.00833 percent is one chance out of 12,000. Refer to Appendix B for a table of conversions.

Table 10-1
Lifetime Risk Estimates for Leukemia and Four Types of Solid Tumors
All values are percent risk per mGy

Type	BEIR V	UNSCEAR[1]	ICRP	Average
Leukemia	0.00095	0.00100	0.00100	0.00098
Breast	0.00035	0.00030	0.00040	0.00035
Respiratory	0.00170	0.00151	0.00170	0.00164
Digestive	0.00230	0.00239	0.00480	0.00316
Other	0.00260	0.00190	0.00210	0.00220
Total	0.00790	0.00710	0.01000	0.00833

Source: Gilbert
[1]UNSCEAR-1988; the UNSCEAR-1994 study was published after the analysis by Gilbert. The UNSCEAR-1994 report gives slightly higher risk values than those in the 1988 report.

Solid tumors and leukemia initiated by radiation have a probability of appearing anytime after the latency period. The period at risk is 30 years or longer, so to get the best data we need data for many years after the population receives a radiation dose. The UNSCEAR-1994, and RERF reports use data on the bomb survivors through 1987. The BEIR V report uses data through 1985.

Table 10-2 summarizes the solid tumor and leukemia actual risk values for the four independent studies. As shown from the numbers in the table, the later reports have higher risk values for solid tumor but lower values for leukemia.

Table 10-2
Actual Risks from Four Reports
Values in percent per mGy

	BEIR V	UNSCEAR 1988	UNSCEAR 1994	RERF	Average
Solid tumors[1]	0.00695	0.00610	0.0083	0.0110	0.0082
Leukemia	0.00095	0.00100	0.0007	0.0007[2]	0.0008
Total	0.00790	0.00710	0.0090	0.0117	0.0090

[1]Risk values from Table 10-1 (BEIR V and UNSCEAR-1988), from later text (UNSCEAR-1994) and calculated from value in later Table 10-3 (RERF).
[2]RERF (Preston, et. al., 1994) does not list a specific risk value for leukemia; the UNSCEAR value is used here.

Maybe I should try to give you some feel for what these numbers mean. First, how much of a dose is a mGy? One year of normal background radiation (from the earth, cosmic sources, and from

radioactivity within our own bodies) is about one mGy. A person flying at 35,000 feet for a total of about 300 hours (about 30 cross-country round-trips) receives a dose of one mGy. The average person in the United States receives a dose of 0.7 mGy from medical procedures each year, or about one mGy in about a year and a half. One mGy is also the limit of radiation dose that any person in the public can receive from an industry that might release radiation to the public.

What risk is 0.0090 percent? First, it is one chance out of 11,000. Referring to Figure 8-1 on risks, we see that a risk of 0.009 percent falls in the region of activities that we might consider "safe." It is safer than being on the highways for a year and safer than being a police officer or a farmer. Later I will discuss why even the risks in Table 10-2 are really too high.

Solid Tumors

The later reports also include other information that verifies findings of the BEIR report. They also give more detailed information than that found in BEIR V.

Linearity

The key assumption about the solid tumor risk from radiation is that the effects depend linearly on the dose; double the dose and the effects are doubled. The data used in BEIR V indicate that the risk is linear. Data from the UNSCEAR-1994 report verifies the linearity of the solid tumor risk. Figure 10-1 shows the plot of the excess relative risk as a function of dose and the linear fit to the data is nearly perfect.

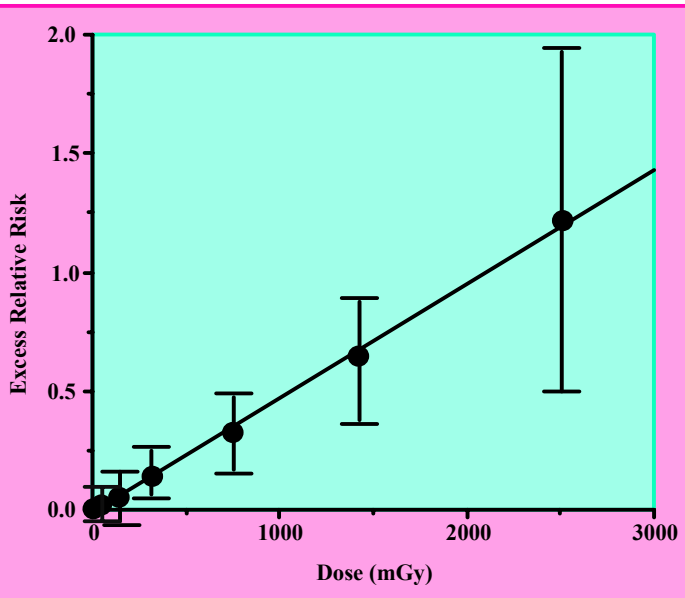

Figure 10-1

Excess Relative risk for atomic bomb survivors
Data from Table 7, Annex A, UNSCEAR, 1994

The slope of the line is 0.00048 per mGy, or 0.048 percent per mGy. This is a relative risk; relative to the natural solid tumor risk. Note that this is not the same thing as the values found in Tables 10-1 and 10-2. The values in these tables are lifetime actual risks.

How does this compare with the BEIR V excess relative risk? To compare the two studies, we need to compare similar situations. Here, the best comparison is with the single 100 mGy dose presented in Table 9-1. That table gives an excess relative risks of 0.033 and 0.047 percent per mGy for males and females respectively. The values from the UNSCEAR report used in Figure 10-1 are for both males and females together, so we need to use the average BEIR V risk value of 0.040 percent per mGy. The UNSCEAR value of 0.048 percent per mGy value is 20 percent higher than the BEIR V value.

This 20-percent difference agrees with the difference between BEIR V and UNSCEAR risk values shown in Table 10-2. The 0.0083 percent per mGy risk from UNSCEAR is 20 percent higher than the 0.00695 value from BEIR V.

Linear to zero?

If you look closely at the data points in Figure 10-1, you will see that the range of uncertainties for the low dose values includes zero risk. Because of these uncertainties, we are not

really sure that the risk is linear to zero dose. The UNSCEAR-1994 report clearly states that below 200 mGy the statistics do not show a definite risk. "Statistically significant risks for solid tumors in the life span study are presently seen only above 0.2 Sv [200 mGy], i.e., the relative risks for solid tumors in the lower dose categories, namely [10-50, 60-90 and 100-190 mGy], are not significant from unity" (United Nations Scientific Committee on the Effects of Atomic Radiation (1994) 41).

Risk by type of tumor

The UNSCEAR-1994 and RERF reports give detailed analyses of cancer risk by type of cancer. Both reports use the atomic bomb survivor data on cancers diagnosed through 1987. Table 10-3 gives the excess relative risk per dose (in percent per mGy) for 15 different cancer types based on the atomic bomb survivor data (called the Life Span Study, or LSS). Appendix A gives even more details of these two reports, including risk values for age at exposure and time since exposure.

Note that the values listed in Table 10-3 are relative risks. This means that the values are the expected risks relative to the natural risk. You cannot compare these values to those in Table 10-1 because the values in Table 10-1 are actual risks.

Table 10-3
Excess Relative Risk per Dose from Various Solid Tumor Cancers
Mortality: values in percent per mGy

Cancer Type	UNSCEAR-1994			RERF		
	Male	Female	Both	Male	Female	Both
Bladder	0.145	0.088	0.122	0.048	0.223	0.102
Brain & Nervous System	0.003	0.305	0.052	0.026	0.026	0.026
Female Breast		0.179	--		0.159	--
Colon	0.047	0.047	0.047	0.076	0.068	0.072
Esophagus	0.037	0.161	0.059	0.004	0.183	0.028
Gall Bladder			0.028[1]	0.027	0.023	0.012
Liver	0.054	0.027	0.044	0.067	0.017	0.049
Lung	0.021	0.159	0.076	0.048	0.193	0.095
Oral Cavity			0.019[1]	0.016	0.046	0.029
Ovary		0.026	--		0.099	--
Pancreas			0.020[1]	0.022	0.011	0.018
Prostate	0.031		--	0.029		--
Rectum			0.009[1]	0.00	0.051	0.021
Stomach	0.017	0.026	0.022	0.018	0.051	0.032
Uterus		0.009	--		-0.015	--
All solid tumors			0.047[1]			0.063

[1]Male and female average; not separated by sex.

Note that the two reports do not always agree on the risk values for a given type of cancer. Because the data for these risk factors were derived from small numbers, each value has a large uncertainty. For example, the UNSCEAR report lists the risk of stomach cancer as 0.022 percent per mGy. At the 90-percent confidence level, the actual value, however, could be between 0.010 and 0.040. The 0.032 value from the RERF report has a 90-percent confidence range of 0.016 to 0.050. Although the RERF value for stomach cancer is 45 percent higher than the UNSCEAR value, the two ranges of uncertainty have considerable overlap (0.016 to 0.040).

The uncertainty in the stomach cancer risk is the smallest of all the cancers because the Japanese population is particularly susceptible to stomach cancer so the number of stomach cancers is the largest of all the different types. All other cancers, though, have larger uncertainties. For example, the 0.122 percent per mGy value for the risk of bladder cancer has an uncertainty range

between 0.03 and 0.33 at the 90 percent confidence level. Therefore, do not consider the values in Table 10-3 as accurate. However, they do show the relative risks of the different kinds of cancers and of the differences between male and female risks.

Age dependence

The risk values reported in Chapter 9 and here in Chapter 10 are for an average population. The population has a mixture of people at all ages. However, because of the long latency period of solid tumors and the long period at risk, a person receiving a dose at a young age will have a higher lifetime risk than someone receiving the same dose at an older age. Table 10-4 shows this dependence.

If we take the average of the values in Table 10-4, we should get a value that is close to the corresponding values in the tables for average populations. The average of the values in Table 10-4 is 0.0079 percent per mGy. This agrees amazingly well with the average actual male and female risk of 0.0079 from Table 9-1.

Table 10-4
Actual Lifetime Risk

Age at Exposure (years)	Excess Lifetime Risk (percent per mGy)
10	0.0145
30	0.0081
50	0.0064
70	0.0026

Adapted from Table 28, Annex A, UNSCEAR-1994

Leukemia

The UNSCEAR-1994 report does verify the linear-quadratic response of leukemia to dose.

Low Dose -- Low Dose Rate Correction

The data used to derive all the risk factors in the previous tables come almost exclusively from the atomic bomb survivor study. The doses received by this population ranged from zero to 4,000 mGy. The population received this dose in a short time (most in a matter of seconds, some over a period of several days). However, most doses received by anybody, public or radiation worker, are at a low dose rate. As indicated in Chapter 9, radiation absorbed at low dose rates does less damage to tissue than doses absorbed at high dose rates.

The Committee on Interagency Radiation Research and Policy Coordination (CIRRPC) considered this problem and recommended that the solid tumor risks be reduced by a factor of two to correct for low dose-low dose rate situations. Because the leukemia risks are based on linear-quadratic curves, they already account for the lower risk at lower doses, so those values do not change. (Wahl 1) Table 10-5 shows the corrected risk values.

Table 10-5
Actual Risks from Four Reports
Values in percent per mGy

	BEIR V	UNSCEAR 1988	UNSCEAR 1994	RERF	Average
Solid tumors	0.0035	0.0031	0.0042	0.0055	0.0041
Leukemia	0.00095	0.0007	0.0007	0.0007	0.0008
Total	0.0045	0.0038	0.00049	0.0062	0.0049

Summary

The purpose of this chapter is to compare the risk values from other reports to those in the BEIR V report. Three independent reports give risk values that are comparable to those in BEIR V. I can easily round off the Table 10-5 average value of 0.0049 percent risk per mGy to 0.005 because the number is not very certain. This means that a person receiving a dose of one mGy has a 0.005 percent risk of a fatal leukemia or a solid tumor. This is a fractional risk of 0.00005, or one chance out of 20,000.

In addition, these other reports verify that the solid tumor risk is linear with dose at the higher doses and the leukemia risk shows a linear-quadratic relationship with dose. However, none of the reports can say with any certainty that the linear extrapolation is valid below about 200 mGy.

What does this uncertainty of the risk for doses below 200 mGy mean? It has some serious implications for risk calculations for low doses. The fact that we are not sure that the solid tumor risk is linear at low doses means that statements like "about 3 percent of all cancers and leukemia are from background radiation," which I made in Chapter 9, may not necessarily be true.

The "3-percent" value suffers from two problems. First, the data used in Table 9-2 to derive the 3-percent value were based on high dose data. To correct for low doses and low dose rates (one mGy over one year is definitely a low dose and low dose rate), we need to divide the solid tumor risk by two. That brings the value down to 1.7 percent.

Second, the linearity in this region is in serious question. If, as some data seem to show, the risk is less than linear at these low doses, then the percent of cancer from background radiation would be even less than 1.7 percent. It could be close to zero.

When large populations are exposed to radiation, we can multiply the average dose by the number of people to get the person-mGy (or person-rad) for that population. This is the same as adding up the individual doses of everybody in the population if we know the individual doses. For example, if 1,000 persons were exposed to 50 mGy the total dose for the entire population would be 50,000 person-mGy (5,000 person-rad).

To use this number to calculate the expected number of cancer deaths, we need to multiply the person-mGy by the fractional risk. The values in Table 10-5 are percent risks, so we need to divide them by 100 to get the fractional risk. The average value of 0.005 percent per mGy risk becomes 0.00005 per mGy fractional risk.

Three independent calculations of the population dose from the TMI accident give an average value of 32,000 person-mGy (3,200 person-rad) for the population around TMI during the 1979 accident (Daniels 88). Applying the above risk factor to the TMI accident, the expected excess cancer and leukemia deaths would be 32,000 person mGy X 0.0005 percent per mGy which gives 1.6 excess deaths.

However, the maximum dose received by anybody during the TMI accident was about one mGy. This is definitely a low dose. The uncertainty of the linear extrapolation in this low dose region means that the expected number of cancer deaths could be much less than 1.7. It could easily be zero.

Quality of the Reports

I have cited three groups -- BEIR, UNSCEAR, and RERF -- in the above discussion. These three groups come with excellent credentials. BEIR, which stands for the Biological Effects of Ionizing Radiation, is a committee formed by the National Academy of Sciences. According to the introductory statement about BEIR V, "The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competencies and with regard for appropriate balance" (National Academy of Sciences (1990)). The

members of the BEIR V committee included scientists from several cancer institutes, medical schools, hospitals, and environmental science departments.

The scientists on the committees that produced the two UNSCEAR reports came from many different countries. Some counties have extensive nuclear facilities. These countries include the United States, United Kingdom, Japan, France, the Russian Federation, Canada, and Germany. Other countries on the committee included Australia, Egypt, Indonesia, Mexico, and Sudan.

The RERF (Radiation Effects Research Foundation) is funded jointly by the United States and Japan. It is the group that gathers the data for the life-span study. The foundation is composed of scientists from both the United States and Japan. This group gathers information on the death of the bomb survivors and determines the doses receive by the survivors. All the other reports that calculate radiation risks use the basic data from the RERF. The reports by Thompson, et. al. and Preston, et. al. give the foundation's analysis of their own data.

Finally, I need to emphasize the number of cases that these reports have to work with. The atomic bomb survivor solid tumor study group consists of about 40,000 survivors. The control group consists of about 46,000 unexposed people. Since the beginning of data collection in 1950, the total number of solid tumor cases in the bomb survivors is about 8,600 with 6,900 deaths. Of these, about 8,100 cases would be expected, with about 6,600 deaths. This means that the risk factors for solid tumors mortality are based on about 300 excess deaths (United Nations Scientific Committee on the Effects of Atomic Radiation (1994) 104).

The leukemia study group consists of about 41,000 survivors and the control group consists of about 45,000 unexposed people. In the study group, about 230 cases of leukemia have been diagnosed, For this group, about 155 would be expected (United Nations Scientific Committee on the Effects of Atomic Radiation (1994) 104). This leaves only 75 cases that can be attributed to the radiation.

You can see that, although this group received a high dose, the number of deaths, although significant, is not high enough to give accurate risk values. When the group is divided into smaller groups (male/female, age, or type of cancer) the uncertainties get even larger.

Chapter 11
Objections: Risks are Too Low

In the last chapter I presented the risk values from what I will call formal studies. Several scientists feel that the risks calculated by the formal studies are too low. These scientists arrive at their higher risk values either by using the same data but making different assumptions, or by gathering their own data. This chapter summarizes some of these studies.

Gofman

In 1981 John Gofman, Professor Emeritus of Medical Physics at the University of California at Berkeley, published an extensive text, Radiation and Human Health (Gofman, 1981) which is his own analysis of the risk from radiation. In 1990 he published a follow-up to this study, Radiation-Induced Cancer from Low Dose Exposure: an Independent Analysis (Gofman, 1990). In both publications Gofman provides his conclusions of radiation risks along with the data and the calculations used for his conclusions.

Gofman bases his work almost exclusively on human data from the atomic bomb survivors. In these two publications, he discusses many of the assumptions and conclusions used in the formal studies, agreeing with many but significantly disagreeing with others.

Gofman recognizes the existence of a latency period for cancer and leukemia and a finite period for risk of leukemia. He uses a three-to-five-year latency period for leukemia and a 10-year period for solid tumors. He assumes that 90 per cent of the leukemia will occur within 29 years. His analysis shows that the at-risk time for solid tumors is longer than 25 years. The exact time, if there is one, is unknown because the data do not extend far enough in time (Gofman, 1981, Gofman, 1990). These conclusions agree with the formal studies.

Also in agreement with the formal studies, Gofman feels that radiation from any source does the same harm. This is contradictory to some people's feeling that radiation from natural sources is less harmful than that from human activities, especially nuclear power production. Gofman points out that all radiations are the same: "A curious notion is commonly expressed, that such natural sources of radiation are less damaging than man-made radiation. A variant of this myth is that natural radiation can be held up as some sort of evidence for the lack of harm from ionizing radiation" (Gofman, 1981 45).

Gofman does agree with the formal reports about the risk at high doses. "Substantial agreement exists between this book and the new UNSCEAR and BEIR reports with respect to cancer-risk per rad from moderate and high doses acutely delivered . . . . This finding indicates that our independent methods and theirs can lead to the same results" (Gofman, 1990 1-3).

However, Gofman disagrees with the formal studies on the extrapolation from high doses to low doses. The formal studies use cellular and animal data to determine this extrapolation. From this data the formal studies find that the linear and linear-quadratic curves should be the best predictors of cancer risks at low doses. However, Gofman believes that animal and cellular studies are not as reliable for predicting the risk from low doses as actual human data. He prefers to use human data from the atomic bomb survivors to extrapolate to low dose. Based on this data, he decides that a supra-linear curve is a better predictor than the linear or linear-quadratic extrapolation for the risk from low doses.

Figure 9-3 shows the supra-linear curve with the linear and linear-quadratic curves for comparison. The implication of the supra-linear extrapolation is that at low doses the risk per dose is much greater than that predicted by the other curves. If you look at the slope of the curve you will see that the slope is steeper at low doses than the slopes of the other curves.

The implication of supra-linearity is that the risk of radiation exposure is much more harmful at low doses than at high doses. This argues that low doses from sources such as nuclear power plants (normal operation) and medical procedures that produce such low doses (diagnostic X-rays and nuclear medicine studies) are more harmful per dose than high doses from such sources as nuclear explosions (atomic bombs dropped on Japan), major nuclear accidents (Chernobyl), or radiation therapy.

Because of the supra-linear extrapolation, Gofman's prediction of the risk to a population from low doses is greater than predictions based on the linear or linear-quadratic extrapolations. Table 11-1 gives Gofman's value along with the BEIR V values for comparison. As seen in the table, Gofman predicts a risk almost four times greater than the BEIR V uncorrected value. Gofman's value is nearly eight times the corrected value.

Table 11-1
Excess solid tumor mortality from 10,000 persons exposed to 100 mGy (10,000 mrad).

	BEIR V		Gofman
	Male	Female	
Excess tumor deaths	660	730	2600[1]
Percent of normal	3.3	4.7	14.7[2]
Risk factor (uncorrected)[3]	0.0066	0.0073	0.026
Risk factor (corrected)[4]	0.0033	0.0037	

[1]Gofman does not separate male and female; this is the total for the U.S. population. Value from Gofman, 1990.
[2]Derived from an average of male and female normal incidence values.
[3]In percent per mGy; from Table 9-1.
[4]In percent per mGy; derived by dividing the uncorrected values by two.

Gofman also strongly disagrees with any suggestion of a threshold or "safe dose" for radiation. He feels that any radiation is harmful, even very small doses.

In addition to his cancer studies, Gofman studied genetic effects. He criticized the BEIR III Report for not including some genetic diseases that do not become evident until adulthood. (Genetic effects are covered in Gofman, 1981 but not in Gofman, 1990, so the comparisons are to the earlier version of the BEIR report.) He derived a doubling dose of between 300 and 500 mGy (30,000 - 50,000 mrem), between two and three times smaller than that from BEIR V.

Gould and Goldman

Another book that predicts higher risks from radiation is Deadly Deceit by Gould and Goldman (Gould and Goldman). They base their predictions on epidemiological data, much from data on infant mortality supplied by Ernest Sternglass. Although the authors do not derive any values of risk per dose, they do attempt to show that low doses from human activities have significantly affected cancer and mortality rates for infants. Most of the material concerns fallout from the nuclear weapons testing in the 1950s and 1960s, the Savannah River plant releases in 1970, the TMI accident in 1979, the operation of the Peach Bottom reactors in Pennsylvania (just south of TMI) and the Millstone reactors in Connecticut, and the Chernobyl accident in 1986. In addition, they have a section that discusses the BEIR V Report.

Most of the graphs showing increases in infant mortality are based on the fact that infant mortality is decreasing in the United States but at times of accidents (Savannah River and TMI for example) the rate of decrease slows or mortality increases slightly. They claim that the lack of continued decrease is evidence that radiation has affected the infant mortality (Gould and Goldman 62).

In their study of radioactive iodine from the Chernobyl accident, they correlate the percent increase in infant mortality across the United States with radioactive iodine in milk and find a supra-linear fit to the data. They also correlate the decrease of bird reproduction in the California Point Reyes Bird Sanctuary with rainfall in the summer of 1986 following the Chernobyl accident (Gould and Goldman 18, 29-38).

In their section discussing BEIR V, Gould and Goldman point out that the major difference between the BEIR results and their belief is that the extrapolation for low dose should be supra-linear rather than linear or linear-quadratic.

Other Studies

Studies published in the late 1970s and early 1980s on the effects of radiation on workers at the Hanford, Washington National Laboratory also showed excess cancers from radiation (Mancuso et al. and Kneale et al.). In these studies, the doses are known because the workers wore badges that measured radiation and the follow-up on the workers' health was thorough. The plant began operation in 1944, so there were adequate solid tumor data available after the 10-year latency period. Unfortunately the papers do not give a risk factor that we can compare with BEIR V or Gofman values.

A study of radiation workers at the Oak Ridge National Laboratory also found that the risk of solid tumors and leukemia per dose is higher than predicted by the BEIR V report. This study included 8318 men who worked at the laboratory since the early 1940s. Most of the doses occurred before 1965. The study looked at deaths through 1984. Therefore, the time from when the workers received most of their dose to the time of study was greater than the 10-year latency period of solid tumors (Wing, et. al. 1397).

Some scientists feel that the formal studies ignore an effect that they feel radiation may cause. That effect is Down syndrome. Down syndrome is a disease resulting from a specific pair of chromosomes having an additional chromosome. Because radiation can affect the DNA, and therefore the chromosomes, there is concern that radiation to one or both of the parents can cause Down syndrome in a child. Gofman summarizes several studies that give statistically significant increases in Down syndrome when one or both of the parents received high (several mGy; 1,000 to 2,000 mrad) of radiation (Gofman, 1981 817-839). In addition, higher incidences of Down syndrome

have been reported in the Kerala Coast area of India and Gaungdong Province in China where the background doses are high (National Academy of Sciences, 1990 384) and in Germany (Sperling, et al.) and Scotland (Ramsay, et al.) after the Chernobyl accident.

The studies mentioned here do not include the entirety of reports that claim that the risk from radiation is significantly higher than predicted by the formal studies. However, the purpose of this chapter is not to give information on every possible report, but to give a representative sample.

Radon Studies

Although I am not aware of any studies that show the BEIR IV risk values for lung cancer from radon are too low, there are some papers that support the BEIR IV findings. In the face of some severe criticism discussed in the next section, you should be aware that there is some support for the BEIR IV values. One study included 586 women and 774 men in Sweden who had lung cancer. The group included 178 nonsmokers and 108 ex-smokers. The results of this study showed that both smokers and nonsmokers had an increased risk of lung cancer with increasing levels of radon in their homes. The risk values are about equal to those found in the BEIR IV report. Also in agreement with BEIR IV, they found that the risk of smoking combined with the risk from radon is multiplicative rather than additive (Pershagen, et al.).

Another study of 433 female lung cancer cases, along with 402 controls, showed that the risk of lung cancer increased directly with the radon in the house. (Schoenberg, et al.) Radon levels ranged from less than one to 11 pCi/L. The relationship between cancer and radon is strongest for smokers. However, the number of lifetime nonsmokers in the study is small so any conclusion about the actual risk is tentative at best.

A third study looked at the same basic data used by the BEIR IV Committee but separated the group into heavy smokers, light smokers, former smokers and nonsmokers. BEIR IV had the former smokers in the nonsmoking category. This study found the lung cancer risks for female smokers and nonsmokers about the same as those determined by BEIR IV. The male nonsmoker risk was the same in both studies but the new study found a lower risk for the light and heavy categories of smokers as compared to the BEIR IV smoker values. For both male and female groups, the former smokers had a risk about midway between the smokers and nonsmokers (Maillie, et al.).

A recent study by 14 scientists from almost as many different institutions around the world pooled the radon data from 11 miner studies. They found that the relative risk for lung cancer from radon exposure increases as the rate of radon exposure decreases (Lubin, et al. (a) and Lubin, et al. (b)). This says that the risk per dose of a miner is less if the miner receives a high dose rather than a low dose. But, the total risk for the higher dose is still higher.

However, the study only showed this inverse relationship for total radon exposures above about 50 WLM. To have this exposure in a house, the air in the house must have a radon concentration of about 6 pCi/L for 70 years.

As discussed later, there is some question about the validity of the BEIR IV risk factors for nonsmokers. One group of researchers did a detailed study of the nonsmokers in the Colorado Plateau group. The results of this study showed a correlation between radon and lung cancer. (Roscoe, et al)

Several case-control studies of residential radon exposure are now underway and these results should be published in a few years. These studies will involve up to 3,200 lung cancer cases (Environmental Protection Agency).

Unfortunately, these studies may not provide any more reliable information on the risk. In a recent paper, Lubin, Boice, and Samet generated hundreds of simulated radon epidemiological case-control studies with 700 cases and 700 controls. For some of the simulated studies they assumed a radon risk equal to zero. For the others they assumed a risk equal to the BEIR IV risk. They found that the probability of one study accurately predicting the risk is small. A set of simulations with 2,000 cases and 2,000 controls showed a better probability of getting reliable results but only under ideal condition. Under more realistic conditions of exposure and mobility uncertainty, the reliability decreases dramatically. "[T]hese simulations imply that it is unlikely that case-control studies alone

will be able to determine precise estimates of risk from indoor radon, and that even further efforts at pooling epidemiological studies may not adequately address issues of risk from residential radon exposure" (Lubin, Boice, and Samet 329).

The implication of this paper is that the results of any new epidemiological study on the risk from radon are not reliable by themselves. Therefore, do not believe any results from one study. Even the results of many studies may not be valid.

Chapter 12
Objections: Risks are Too High

Many studies on the effects of low doses of radiation show data which suggest that the risk from radiation is less than that proposed by the BEIR V report. I will discuss some of these studies in this chapter. These studies involve large numbers of people. However, in all but two the doses and dose rates are small so there is a chance of some uncertainty.

China Study

One investigation is studying a population that lives in an area of high background radiation in China. The major advantage of this study is that the population is very stable; most of the people live in the same town or village in which their ancestors lived. The areas under study have background radiation levels about three times the average. Based on the predictions of BEIR V, we should observe some measurable increase in cancer mortality if the population is large enough.

The population in the three regions under study is about 80,000, and the average background dose is 5.5 mGy (550 mrad) per year. A control region, similar in geography, size, culture, education, medical care, etc., has an average background of 2.1 mGy (210 mrad). These doses include terrestrial, cosmic, internal, and radon doses. The difference in the dose between the two regions is primarily from a difference in the terrestrial and radon doses (Kondo 51-55).

To get some idea of what differences we should observe between the high dose and control populations, refer to the Table 9-2 excess relative risk values. A population with a lifetime exposure of 1 mGy (100 mrad) per year should experience a 2.8 percent (average of male and female values) increase in solid tumor mortality and an 8.8 percent increase in leukemia mortality. However, the difference between the two regions is 3.4 mGy (340 mrad). Therefore, we should see differences of 9.5 and 30 percent respectively for solid tumor and leukemia between the high dose and control regions.

Although this study has data for only about 20 years, we can still compare the high dose and control groups to look for significant differences with the present data. The population and dose are large enough to show any possible difference. Among the 77,000 people studied in the control area, the solid tumor mortality is 47.7 per 100,000 person-years. (A person-year is one person studied for one year; 74,000 people studied for 20 years is 1,480,000 person-years. This unit is a way to normalize the numbers between the control and high-background region.) The mortality rate for the 74,000 people studied in the high-background region is 45.8 per 100,000 person-years.

The leukemia values are 3.39 and 3.02 for the control and high-dose regions respectively (Kondo 54). The control and high dose values for both cancer and leukemia are not significantly different. This shows that the increased background does not cause any increase in cancer.

Although the population is stable, we do not have proof that everybody always lived in the same area. To be accurate the study should extend for 70 to 80 years.

The above values are for the entire population. We know that the risk of cancer increases with age so a more sensitive group of people to study would be the older segment of the population. The solid tumor mortality of the 40 to 70 year age group is 168 and 144 per 100,000 person-years for the control area and high-background regions respectively. The high-radiation area mortality is 16.7 percent lower than that of the control area (Kondo 54).

But how good are the statistics for the older group? The solid tumor rates of 168 and 144 per 100,000 person-years are based on 377 and 299 actual cases respectively. The standard deviation of

each of these two numbers is about 5 percent. This means that the "real" value of 144 per 100,000 person-years could actually be between 137 and 151 at the 68-percent confidence level. At the 90-percent confidence level, the range would be 130 to 158.

Likewise, the "real" value of 168 per 100,000 person-years could really be between 155 and 181 at the 68-percent confidence level and between 143 and 193 at the 90-percent level. Therefore, it is possible that the two values are the same, which would show that there is no real difference in the solid tumor rates. However, that probability is only about 15 percent. Apparently this study indicates that high background radiation does not cause any excess solid tumor or leukemia mortality.

Worker Studies

A population that has good data on doses is that of radiation workers. Over the past 40 to 50 years, nuclear power plants, hospitals, shipyards that install nuclear reactors in ships, and Department of Defense establishments that manufacture nuclear weapons have employed many people. All these workers have dose records, so we know the total does to each of these populations with some accuracy.

I discussed one study on the Hanford workers in the previous chapter (Mancuso et al.). However, several subsequent papers have criticized the methods used by Mancuso, Stewart, and Kneale (Anderson; Hutchinson, et. al.; Gilbert and Marks). One study concurrent with the Mancuso study found that the exposed Hanford workers had higher longevities than the non-exposed workers, out-of-plant controls and even siblings of the exposed workers (Sanders).

Later studies show that the solid tumor and leukemia mortality of the Hanford workers is not only less than predicted by the BEIR V projections, but the mortality is equal to or less than the population as a whole.

> "We also analyzed the Hanford data for consistency with predictions of the models recommended by the BEIR V Committee . . . . Hanford data were analyzed by expressing estimates and confidence limits as multiples of risks predicted under the BEIR V models, and this involved weighting each annual dose according to age, time from the effect, and gender . . . . Using this approach, the leukemia risk estimate was - 0.6 times the BEIR V predictions and the upper limit (two-sided 90% interval) was 0.8 times the BEIR V predictions. The BEIR V leukemia model included reduction of risks by a factor of 2 for low doses . . . . For all cancers except leukemia, the risk estimate was almost exactly zero and the upper confidence limit was 1.5 times BEIR V predictions based on a linear model with no reduction for low dose rates." (Gilbert, et. al, 1993)

One study of several nuclear facilities in the United States contained a group of about 36,000 workers (Gilbert et al., 1989). Another study of facilities in the U.S., Canada, and the United Kingdom, included almost 100,000 workers, the largest worker group ever studied (Gilbert). For the study of the U.S. facilities, there was "no evidence of any correlation between radiation exposure and mortality from all cancer or from leukemia" (Gilbert et al., 1989 19). For the larger group as a whole, the standardized mortality rates (SMR) were less than one (i.e., no excess cancer fatalities were detected). "Occasional elevated SMRs for specific cancers were found, but, in most cases, these were not statistically significant, and no consistent pattern across studies emerged" (Gilbert 676)

In this study there was a correlation with leukemia in the workers at Sellafield, England. However, because some of these workers received doses up to 500 mGy (50,000 mrad), the results were "not inconsistent with predictions based on atomic bomb survivor data. None of the other studies provided evidence of a positive correlation with leukemia" (Gilbert). Also, there was a correlation of multiple myeloma (blood disease) in the Sellafield and Hanford workers, the two studies with the greatest total dose.

A review of the 1991 Department of Energy study on Nuclear Shipyard workers says that the cancer mortality among the exposed workers was about the same as non-exposed workers.

> "The NSWS [Nuclear Shipyard Workers Study] is the world's largest epidemiological study of the health effects of radiation to nuclear workers . . . . Deaths from various causes of the 29,000 nuclear workers were compared with deaths from a control group of about 33,000 shipyard workers of similar age with similar jobs but who were not exposed to radiation on the job. The study considered deaths from about 1960 to 1982. There was less cancer among the radiation workers but the difference wasn't statistically significant" (Lenihan 23).

Other Studies

In an address to the 1991 Northeast Regional Symposium on The BEIR V Report and Its Implications, Rosalyn Yalow discussed several studies that suggest that the BEIR V solid tumor and leukemia risks are too high. One study was on military X-ray technicians whom each may have received up to 1,000 mGy (100,000 rad) over a period of three years in the early 1940s. This group of technicians had the same solid tumor mortality as a control group of technicians not exposed to radiation (Yalow).

For breast cancer, BEIR V uses the atomic bomb data to predict the risk as a function of age. The risk curve in BEIR V for breast cancer shows a significant risk in the 45- to 60-year age group. However, the original data show that for women in the over-40 age group who received 500 mGy (50,000 mrad) or more, the incidence of breast cancer is equal to that of the control group (Yalow).

Yalow also discussed two studies on patients who received radioactive iodine as either diagnostic uptake tests for thyroid disease or for treatment of a thyroid tumor. The group of 35,000 patients who received radioactive iodine for diagnostic purposes received a thyroid dose of about 500 mGy (50,000 mrad). They had a thyroid cancer incident rate 38 percent lower than the control group (Yalow).

Another investigation studied 30,000 patients treated for hyperthyroidism with either large doses of radioactive iodine or with surgery. Those treated with radioactive iodine received a whole body dose of about 100 mGy (10,000 mrad). This group showed a 50-percent increase in leukemia, which would suggest that the radiation from the radioactive iodine was the cause for the increase. However, the group treated surgically also showed a 50-percent increase in leukemia, so the increase was more likely due to the hyperthyroidism rather than the radiation (Yalow).

As in the previous chapter, I have not included all the studies which indicate that the risk values are too high. What I have given here is only a representative sample of what is in the literature.

Radon Studies

Some studies on populations in high-radon areas have shown no correlation between lung cancer and indoor-radon concentration. One study investigated the correlation between radon and lung cancer in 411 U.S. counties. The results not only show no correlation but found a negative correlation. Counties with the higher radon levels had the lower lung cancer rates. The radon levels ranged from zero to 7 pCi/L (zero to 260 Bq/m^3). Even correcting for smoking (based on cigarette sales in each county), family income, education, employment, number of physicians, and age, the negative correlation did not change significantly (Cohen).

Similar studies in China, The United Kingdom, and Japan also show zero or negative correlations between lung cancer and radon levels. The Japanese study looked at a city that contains radon spas where people come for treatment for a variety of ills. Lung cancer rates in the city were compared with those in the suburbs. The lung cancer mortality of the city residences was only 55 percent of mortality of the control group from the suburbs. In fact, the mortality of all types of cancer in the city was significantly less than those in the suburbs (Kondo 63).

The study in China involved a 14-year investigation that compared a population exposed to an average of 3 pCi/L to a control group exposed to 1.3 pCi/L. According to the BEIR IV predictions,

the population exposed to higher levels of radon should have a mortality from lung cancer about 10 percent above the control group. In fact the rates were essentially the same: 2.7 per 100,000 people in the high-radon area as compared with 2.9 per 100,000 in the control area (Hofman, Katz, and Chunxiang). However, the actual numbers of cancer deaths were only 23 and 27 in the two groups respectively. The uncertainty of these numbers is about 20 percent so we cannot be very confident that the rates are really the same; one could easily be higher than the other.

The most serious problem with the BEIR IV study is that most of the subjects were smokers, so the data on nonsmokers is small and consequently uncertain. Therefore, the predictions for the lung cancer risk to nonsmokers may not be very accurate. We need other methods to determine this risk. One method is to study the location of lung cancers in smokers and nonsmokers. According to several cancer experts, most cancers in nonsmokers are located in the deeper portions of the lungs. Cancers in smokers and those exposed to radon are found in the tracheobronchial region. "Among the 148,000 lung cancer deaths expected in 1991 it is likely that no more than 7,500 will occur in nonsmokers. Most of these are in the region of the lung where radon daughters do not penetrate" (Yalow 71).

Hormesis

All of the previous discussion has assumed that radiation is harmful. Is it? Some scientists believe that low doses of radiation can be beneficial. The beneficial effect is called hormesis. The possibility of the hormesis effect has come to light in the past several years when researchers, trying to gather data on the effects of low doses of radiation, got data that would indicate the effects were beneficial rather than harmful.

In his book Radiation Hormesis, T. D. Luckey summarized the study of hormesis.

> "Although virtually no systematic research was devoted to radiation hormesis, it is supported by the preponderance of evidence. The consistency of the data gleaned from human experiences and animal experiments is particularly convincing when one considers that most of the work reported was performed with no preconceived concept of radiation hormesis. Indeed, most of the research was conceived, performed, and interpreted within the constraints of the zero thesis [radiation is harmful]. The appearance of results which conflicted with the zero thesis usually came as a surprise" (Luckey 33).

If radiation hormesis is a valid description of the effects of radiation, then the dose response curve would look like that shown in Figure 12-1. The figure also shows the linear, linear-quadratic, and supra-linear curves for comparison. The curves are plotted such that harmful effects increase from the zero point upward. Where the hormesis curve drops below zero, the harmful effects are negative, or beneficial.

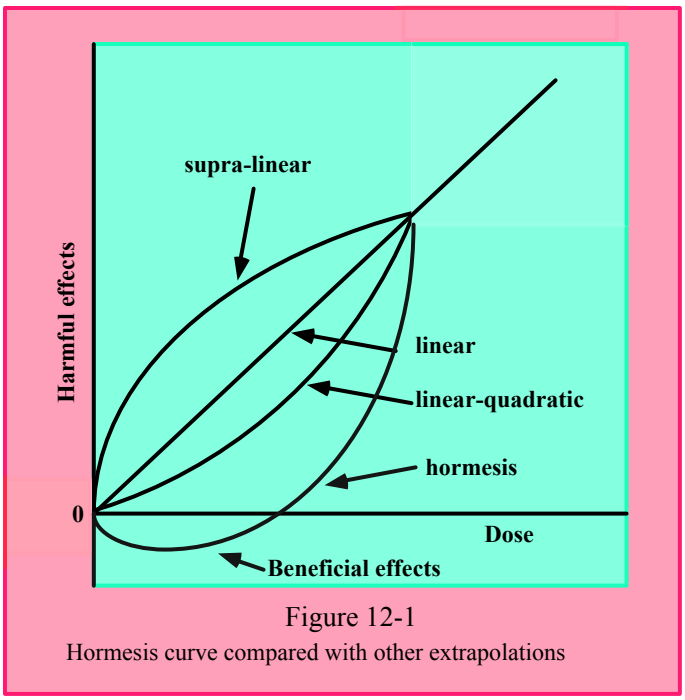

Figure 12-1
Hormesis curve compared with other extrapolations

The curve shows that at zero dose, radiation has no effect; it is neither good nor bad. But for doses slightly greater than zero, the radiation has beneficial effects, reaching a maximum benefit where the curve bottoms out. The benefits decrease at yet higher doses until the radiation again gives neither benefit or harm where the curve passes through the zero line again. Although the hormesis curve shows beneficial effects at low doses, it still does predict harmful effects at high doses in agreement with all other models.

Some data for hormesis are from human studies, but most come from

studies on animals. For example, compared with mice not exposed to radiation, the growth rate of mice was higher for those exposed to X-radiation for doses up to about 60 mGy (6,000 mrad) per day. Mice exposed to doses up to 1,000 mGy (100,000 mrad) had fewer cancers than the non-exposed control group. Fingerling salmon exposed to less than 50 mGy (5,000 mrad) per day had higher growth rates than the controls not exposed to radiation. (Luckey 59-60)

Among the human studies cited are the studies in China and on the Hanford workers mentioned in the previous section. Also cited are studies on the bomb survivors. Some data show that for doses less than 50 mGy (5,000 mrad) the cancer incidence is significantly lower than the control population (Lenihan 25). Other data from the bomb survivors show that the death rate from all causes except cancer of those who received less than 2,000 mGy (200,000 mrad) was less than those who receive no radiation (Kondo 30). However, a different analysis concluded that the data do not show any hormetic effect (Shimizu, et. al.).

One argument for hormesis is based on the fact that many substances that are toxic at high doses are beneficial, and even necessary for survival, at low doses. For example, we need trace elements such as copper and zinc to live, but at high doses they are toxic. Why should this not also apply to radiation? Humans have evolved in an environment that has always contained radiation, so some argue that humans have adapted to it. Even if we conclude that background radiation causes mutations, these mutations, both good and bad, have played an important role in giving humans an advantage for survival (Laterjet).

Chapter 13
Summation of Risks

In the previous chapters I have presented the evidence, mostly without editorial comment or rebuttal. However, in a few cases I have inserted clarifications that tend to show the validity of the evidence. I have tried to present each side as stated by those cited without bias on either side. If there is any bias on my part it would be from what I feel is important and valid information. Now I would like to play lawyer and summarize the data as I see it to give you some sense of what all these numbers and assumptions mean. You can play jury and decide if you agree with my analysis and come forward with a verdict.

Extrapolation, Threshold and Hormesis

The greatest divergence of opinion is the extrapolation from high doses to low doses. Which is correct? Linear, linear-quadratic, threshold, hormesis, or supra-linear?

In many ways we face a situation similar to that which judges and juries face in courts across the country. When trying to decide the guilt or innocence of a person charged with a crime, the prosecutor and defense attorneys present the evidence that supposedly shows the defendant to be guilty or innocent. The best evidence that a prosecutor or defender can have is a videotape of the crime that clearly shows the defendant or someone else committing the crime. The next best evidence is to have several believable, irrefutable witnesses. In our trial we have neither: there is no videotape and nobody can present irrefutable data.

Lacking a videotape and believable witnesses, the prosecutor tries to show that the circumstantial evidence is so overwhelming that the only logical conclusion is that the person actually commited the crime. The defense attorney then tries to present evidence that the defendant could not have committed the crime. The judge or jury then has to use the evidence of both sides to decide the guilt or innocence of the defendant.

In our trial, no evidence clearly shows that one extrapolation is correct and is the only one that fits the data. So now we are left with circumstantial evidence. We must weigh the evidence and, based on “the preponderance of the evidence,” decide what is the truth, or what most resembles the truth.

If we don’t have any really conclusive evidence, then maybe we should ask for a continuance and wait until more data are available so we can make a more informed decision. But the accused

has the right to a speedy trial. We need to make a decision on the extrapolation question so we can use this decision to guide us in many public policy decisions facing us. Do we build more nuclear power plants? How dangerous is a radioactive waste facility? Is a medical procedure worth the risk?

The one difference between this kind of decision and the verdict in a criminal trial is that as more evidence becomes available, we can change our decision either way; increase or decrease the risk values. In a criminal case only a guilty verdict can be reversed, not an innocent verdict.

Hormesis

Let's consider the question of hormesis first. Is radiation good for you? Most of the evidence for hormesis is based on animal data; very little good data are available on human effects. The study by Cohen (Cohen) that shows a negative correlation between lung cancer and indoor radon concentration is not a controlled study; there are too many factors that can affect the lung cancer rates. The fact that smoking is such a large confounding factor makes this type of analysis highly uncertain. One of many articles critical of Cohen's conclusions states: "smoking-related bias and other problems make ecologic analyses uninformative regarding the magnitude of risks from low-level exposures to radon" (Gilbert 197).

The other major human data used as evidence of hormesis is the negative correlation between cancer rates and background radiation. Again, this kind of study is problematic because there are too many other factors that can affect the cancer rate in any one area. In addition, the mobility of people, especially in the United States, means that most of the population in one area has not lived an entire lifetime in that area; they bring a history of different background radiation doses with them. Because of these reasons, I do not see that we can conclude that low levels of radiation can prevent anybody from getting cancer.

What about the radiation preventing other illnesses and therefore being beneficial? The theory that radiation "activates" the immune system so it is ready to fight off disease (Luckey 4) could be valid. There is some logic to this theory, but I do not feel that the circumstantial evidence is strong enough to conclude that this is true. We need more information on how radiation affects the immune system before we can say that low levels of radiation are truly beneficial.

Threshold

Is there a threshold? The amount of circumstantial evidence is not insignificant. For bone cancer, the evidence is strong that a threshold exists. For other kinds of cancer, the evidence is marginal, mainly because of statistics. Although some studies have enough data to suggest the risk values in BEIR IV and V studies may be too high, I am not convinced that they have shown "beyond a reasonable doubt" that a threshold exists. However, if someone pressed me to make a decision either way, I would tend toward saying that a threshold does exist.

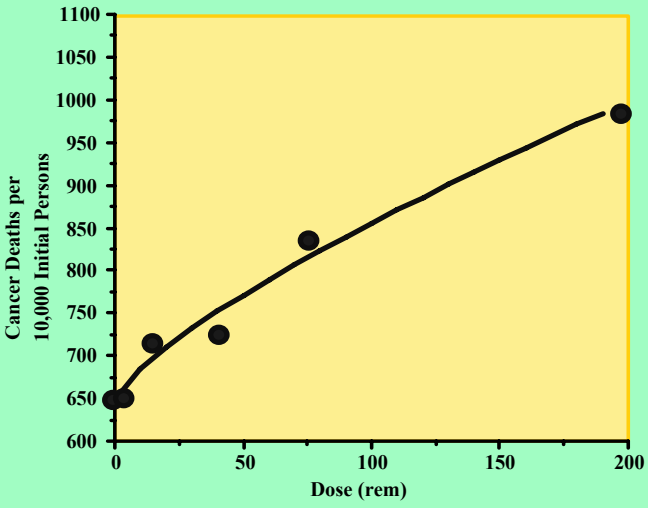

Figure 13-1

Gofman atomic bomb survivor data
with supra-linear fit to the data

Supra-linearity

Now, let's skip to the other side of the question; is the supra-linear extrapolation valid? Gofman uses six data points to derive his supra-linear theory. (Gofman, 1990 13-10 &11). Figure 13-1 shows Gofman's data and the supra-linear curve that he fit to the data. (Note that the dose is in rems; I have used the data as Gofman presented it. Essentially one rem is equal to one Gray, or 1000 mGy.)

These data have two major problems. First, it does not contain any

error bars. All data have uncertainty, and the uncertainty limits the accuracy of any fit to the data. By not including any uncertainty, the reader cannot make any judgment about the accuracy of the fit.

To get some indication of the accuracy of Gofman's data, we can use the raw data that he used for the curve. For the number of deaths reported for each group, the uncertainties range from 2 percent at the lowest dose to 6 percent for the three highest doses. These are at the 68-percent confidence level. If we use the 90-percent confidence level then the uncertainties range from 4 to 12 percent. Figure 13-2 shows the same data with the 90-percent confidence uncertainty on each point. A linear fit passes through every point's uncertainty range, so a linear fit would be just as good as the supra-linear fit.

Second, the supra-linear fit really depends on one data point, the one at the highest dose. If the risk were only about 15 percent higher at 200 rem (equivalent to 200,000 mGy), then a supra-linear fit would not be appropriate. A linear fit is the only choice.

The data used by Gofman was that available before 1990. Recent data show more conclusively that the fit should be linear above 200 mGy. Figure 10-1 shows the data from the 1994 UNSCEAR report. That plot shows a linear relationship even without the error bars.

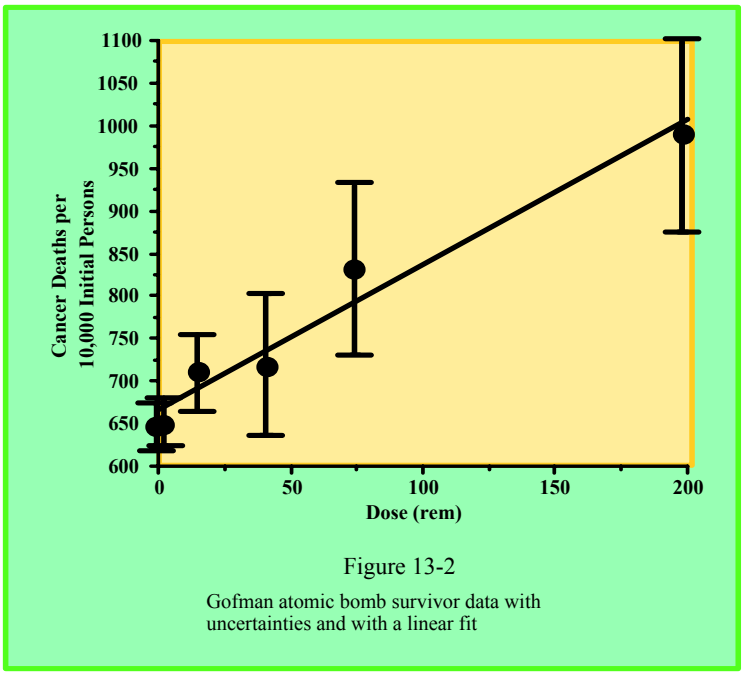

Figure 13-2

Gofman atomic bomb survivor data with uncertainties and with a linear fit

In addition, a philosophical argument would argue against the validity of a supra-linear risk curve. Everybody agrees that the risk from radiation received at different times is cumulative; the risk from one milliGray at age 10 plus one milliGray at age 30 is additive. The average radiation dose rate to the U.S. population is about 3.6 mGy (360 mrad or 0.36 rem) per year (natural, including radon, plus human produced, including medical) (National Academy of Sciences, 1990 18). By the age of 20, a person will have accumulated a total dose of 72 mGy (7,200 mrad, or 7.2 rem). This puts the person on the supra-linear curve where the slope, and therefore the risk per dose, is considerably less than that at lower doses. This would imply that a person becomes more immune to radiation with age.

Therefore, based on uncertainty problems and on philosophical arguments, I do not feel that the supra-linear extrapolation is valid.

Linear and linear-quadratic

The validity of two remaining extrapolations, the linear and linear quadratic, depend on the evidence of low dose epidemiological studies and on the evidence for the interaction of radiation with the cells. The linear extrapolation is the more conservative of the two. It predicts a higher risk than the linear-quadratic extrapolation. Cellular studies imply that the cell response to radiation should be linear-quadratic at low doses but for solid tumors there is no real epidemiological evidence that this is true. For leukemia, the epidemiological evidence points to a linear-quadratic response. The combination of this and cell studies would indicate that either the linear or the linear-quadratic extrapolation would be valid for low doses.

Infant Mortality

The studies on increased infant mortality argue for the supra-linear extrapolation. However, just like the studies on the hormetic effects of radiation, too many other factors can affect the data in these studies. Infant mortality depends on pre-natal and post-natal medical care, poverty, nutrition, etc., factors which are hard to quantify and separate from the effects of radiation. Ernest Sternglass, as referenced in Gould and Goldman, bases his conclusions on changes in infant mortality of the entire country that occur when there is a radiation release at one location in the country. He then correlates the two and concludes that the change is from the radiation. This is just not logical.

However, the major problem with this study, and others that follow, is that there are no control groups. Had a control group from states far from Pennsylvania or from South Carolina shown a steady decrease in infant mortality while there was no change or an increase near TMI or the Savannah River plant, then there might be a case for radiation being the cause. This type of analysis is just like traveling north to see how the temperature changes without having a control temperature reading at the starting location.

Others have examined the Sternglass infant mortality studies and have disagreed with the idea that the cause of infant mortality is radiation. "Sternglass's conclusions were rejected by many critics, largely on the basis of internal consistencies, unexplained selectivity in choice of observations, arithmetic errors and inattention to questions of statistical significance." (Fuchs 847) In addition, some studies on the effects of the radiation from the Chernobyl accident found no increase in birth defects (Czeizel), congenital malformations, preterm births, stillbirths, or malformed children (Harjulehto, et al.). The average dose to these populations was much higher than the subjects Sternglass used in his study.

Gofman also questions the studies of infant mortality.

> "In particular Sternglass has published a summary of his findings linking radiation releases to ostensible increases in infant mortality. Unfortunately, for almost none of the situations in the Sternglass reports can the dose received by the population be determined, with the exception of the claim that weapons-testing fallout (where dose estimates are possible) produced a great increase in infant mortality . . . by their very nature, his studies can compare infant mortality rates before and after some radiation releases, but they cannot provide a control group" (Gofman, 1981 756).

Gofman also uses his atomic bomb survivor data to show that the increase of infant mortality of the survivors was 0.054 percent per mGy (0.00054 percent per mrad) (Gofman, 1981 757).

From the TMI accident, the average dose was less than a milliGray to people near the plant. Therefore, the maximum expected increase in infant mortality would have been a small fraction of one percent. This increase would be totally unnoticeable in the immediate area. Yet Sternglass states that the national infant mortality rate increased after the accident, implying that the radiation from TMI was the cause of this increase.

Sternglass said that radiation from the Chernobyl accident was the cause of a decrease of bird reproduction in California. The area received increased rainfall when the Chernobyl accident fallout was over the Western United States (Gould and Goldman, 1990 24-26). However, there are no data to show that the rainfall actually contained more radioactivity than what fell on other sites in California where there were increases in bird reproduction. Without data on the amount of radioactivity in the rain and without a control group, any conclusion from this study cannot be considered valid.

Down Syndrome

Although several studies make a connection between radiation and the incidence of Down syndrome, there are other studies that show no correlation and even a negative correlation. The group with the highest dose is the atomic bomb survivors. Studies of this group do not show any correlation, in fact they show a negative correlation (National Academy of Sciences, 1990 84;

Gofman, 1981 835). There is a possibility of under reporting of this disease because of the stigma attached to being an atomic bomb survivor and to having an abnormal child, so the negative correlation for the atomic bomb survivors may not be totally correct (Gofman, 1981 837-838).

When considering all the evidence, both the BEIR V report (National Academy of Sciences, 1990 84) and Gofman conclude that we cannot definitively say that radiation is a cause of Down syndrome.

> "Overall, the existence of and the magnitude of any causal relationship between radiation of the mother and Down syndrome in later offspring must remain moot. The various studies have been presented. There are problems with just about every one of them. None can be considered definitive" (Gofman, 1981 838).

Based on these comments, I do not feel that the evidence supports the claim that radiation can cause Down syndrome. Similarly, the evidence does not support the claim that radiation does not cause Down syndrome. "We must leave open the possibility that numerical chromosome aberrations, such as trisomy-21 [Down syndrome], may yet be conclusively shown to increase appreciably with each rad of maternal radiation" (Gofman, 1981 838).

Low Dose Rates

Several studies, mostly cellular, show that radiation at low doses and low dose rates is less hazardous that radiation received at a low dose but at a high dose rate. If this is the case, then the BEIR V risk values would be too high for low dose rates. Gofman contends that low dose rates are actually more harmful than high dose rate exposures. Some studies on radon agree with this argument, but only for the combination of radon and smoking (Thomas et al.) or at high radon exposures (Lubin, et al. (a) and Lubin, et al. (b)). I would agree with the cellular studies and say that the risk of radiation exposure at low dose rates is less than that predicted by BEIR V, UNSCEAR and ICRP without correction for low dose and low dose rate. I would believe that the corrected risk values in Table 10-5 are the best estimate of radiation risk we can get with the available data.

Person-Dose

Scientists use the concept of person-dose (person-mGy or person-mrad) as a way of calculating the total dose to an exposed population. If we assume that the effects of radiation depend linearly on dose and that even the tiniest amount of radiation (one gamma ray!) can produce some effect, then this way of calculating a population dose is valid. However, the evidence shows that the risk of leukemia is not linear and there is significant evidence that a threshold may exist for other cancers. If a threshold does exist, then a person-dose can lead to erroneous numbers of expected cancer cases.

Let's use an analogy to see how this type of population dose calculation can overestimate expected cancer rates. I will use another carcinogen: cigarettes. I will define the unit of person-cig; this is one person smoking one cigarette. A person who smokes two packs of cigarettes a day has a daily "dose" of 40 person-cigs. This person will have a 30-year "dose" of 440,000 person-cigs.

Health studies tell us the chance of dying from smoking is about one in four (0.25, or 25 percent), mostly from lung cancer or heart disease. (I have also seen numbers like one in three and one in two, but I will use the lower value for this argument.) I will assume that one of these health effects will happen after 30 years of smoking. We can now define the risk of smoking as 0.25 ÷ 440,000, or, 0.00000057 per person-cig. For heavy smokers, this could be a good measure of the risk. But is it a good measure of the risk to those who smoke one or two cigarettes a day, or in the extreme case, those who smoked one cigarette in their life?

Like many people I have tried smoking. When I was at about 11 or 12, someone got a cigarette and asked me to try it. I did, and that was the end of my smoking. Suppose that the 60 to 65 percent of the population who never smoked tried one cigarette in their lifetime. This would be about 160,000,000 people in the United States. The "dose" for this population would be 160,000,000

person-cigs. Based on our risk of 0.00000057 per person-cig, we would expect to find about 90 deaths from lung cancer or heart disease in this group due to smoking just one cigarette each. This does not seem logical to me.

This shows how we can apply a tiny risk to a very large population and produce a large number of expected deaths. Similarly, when we apply the risk per person-dose to a population in which each individual receives a tiny radiation dose, we can predict many deaths that just will not be there.

Radon

Finally, we have the risk from radon. We have seen that most of the risk data is from miners and most of these miners also smoked. The accuracy of the data used by BEIR IV is probably adequate to make the prediction for the risks of radon to smokers. The studies by Lubin, et. al. (Lubin, et al. (a); Lubin, et al. (b)) tends to verify the BEIR IV risk numbers. However, the accuracy of the risk to nonsmokers depends on the ability to determine the exact effects of smoking on the miner data. As indicated by some studies cited previously, the question of the effects of smoking is far from being completely answered. Therefore, the risk to nonsmokers is highly uncertain.

> The evidence for radon-associated lung cancer among nonsmokers in the home is very weak. Even among nonsmoking miners, lung cancer is not found among those exposed to less than 1000 times the 70-year indoor levels that the EPA estimates would result in 1 [to] 5 lung cancer deaths among 100 so exposed. Smoking is such an overwhelming cause of lung cancer, that variation in smoking patterns would tend to obscure any possible effect of radon exposure. (Yalow, 1991 72)

In 1991 the Health Physics Society's Ad Hoc Working Group on Radon published a draft statement that, based on the information available, I would agree gives a good description of the situation.

> "Inadequate information on radon health risks and the meaning of screening measurements is leading many homeowners to spend money on reducing indoor radon that may not significantly reduce their risk of lung cancer. The widely publicized estimate of 20,000 lung cancer deaths a year due to indoor radon implies that reductions in radon could save 20,000 lives a year. This will not happen because more than 70 percent of those estimated deaths are due to the combined effects of radon and cigarette smoking. . . . Homeowners should be advised that if they have a cigarette smoker in their house, reductions in radon levels may not significantly reduce lung cancer risk to family members because cigarette smoking remains the primary cause of lung cancer" (Mills, 1991).

Just who is the culprit? Radon? Smoking? Both?

Final Verdict

Now the jury must render a verdict! If I were the jury, I would say that the BEIR V risk predictions are an upper range to the actual risks, especially for low dose exposures. I do not feel the risks are zero, but the evidence that the risks are less than those predicted by BEIR V is strong. The possibility that the atomic bomb doses may be too low is another reason to believe that the BEIR V risk values could be too high. I also feel that a threshold is a possibility.

The Health Physics Society has taken a position that because the risk at low dose is unknown, then we should be cautious when using risks projected from high dose. The society issued its Position Statement in March, 1996.

> "In accordance with current knowledge of radiation health risks, the Health Physics Society recommends against quantitative estimation of health risk below an individual dose of 5 rem [50 mGy] in one year or a lifetime dose of 10 rem [100 mGy] in addition to background radiation. Risk estimation in this dose range should be strictly qualitative accentuating a range of hypothetical health outcomes with an emphasis on the likely possibility of zero adverse health effects. The current philosophy of radiation protection is based on the assumption that any radiation dose, no matter how small, may result in human health effects, such as cancer and hereditary genetic damage. There is substantial and convincing scientific evidence for health risk at high dose. Below 10 rem [100 mGy] (which includes occupational and environmental exposures), risks of health effects are either too small to be observed or are non-existent. (Mossman, et al.)

The lung cancer predictions of the BEIR IV report are probably accurate for smokers. The risk values are questionable for nonsmokers; one study on miners shows a correlation, but other studies show none. The risk values for nonsmokers are maximum values; the actual risks should be significantly less than these values.

Of course, 12 people who hear the same evidence can come to different conclusions. Therefore, not everybody who reads the information presented above will arrive at the same conclusion, and we may have a hung jury.

What will the future bring?

As indicated at the beginning of this chapter, we know that we do not have all the information to make a conclusive determination of the risk from radiation. We just need more data. We have seen the risk values increase from the 1980 values in BEIR III to the 1990 values in BEIR V. Will a succeeding analysis cause the risk values to increase more? That is possible. But so is a decrease.

What do we do? Is it time to revoke the bail? Some may say that based on history the next large study will produce higher risk values. If so, do we shut down the nuclear industry altogether or make radiation standards much more restrictive? Would this "margin of safety" be worth the possible lives saved? It might, yet it might not. As I will discuss in later chapters, there is a price to pay for being too cautious.

The only reaction is to accept what we know presently and act accordingly. Trying to react to what might be the case only leads to confusion and possibly to inappropriate actions.

We know that cars are becoming safer, but they're still the cause of many deaths each year. Do we stop manufacturing cars until they are "perfectly safe," whatever that may mean? No, we manufacture cars and each year we make small improvements: seat belts, sturdier side panels for crash protection, air bags, and whatever else the future may bring. We can't hold auto manufacturers responsible for deaths and injuries from past car accidents that might have been prevented in present-day cars.

Likewise, we have to accept what we know about radiation right now but we also need to keep taking data. If we do find that the risks are higher, then we can make the improvements in procedures and standards to reflect this new knowledge.

Chapter 14
The Cost of the Fear of Radiation

We have all crossed a street, and have watched others cross a street. This is an activity that requires some caution, but it is not considered a dangerous activity. I would say that crossing a

street falls in the "safe" category. How safe depends on how the person crossing the street performs this simple maneuver.

I get nervous watching children dart among traffic to cross the street. Will I see an accident right in front of my eyes? Some children seem oblivious to the dangers around them. My heart usually skips a few beats while I watch cars steer and stop to avoid children.

However, most people take a more responsible attitude and cross a street with care and caution. We look both ways to be sure we can get across without causing a driver to stop for us. If the road is slippery or it is night time, we consider these conditions and exercise even more caution. We realize there is a hazard and respect that danger.

Then there could be others who have a fear of crossing the street. They may conjure up images in their mind of stepping off the curb and getting part way across the street when a car comes careening around the corner and hits them.

I might call these three reactions to crossing a street obliviousness, respect, and fear. These are three natural reactions to many situations that involve a risk of injury. I am sure that we all have exercised one of these reactions in some form at some time in our lives.

Never reacted with fear? What about standing at the edge of a cliff? That should be safe if there is no strong wind, yet would you do it? Even with a railing, some will not approach the edge. I have no problem if a railing is there, but without a railing I get a bit nervous if I get too close.

Immediate Rational and Irrational Actions

Obliviousness, respect, and fear are common reactions to danger, but are they rational? That depends on the situation and on our definition of rational.

Let's consider a scenario that involves a toxic substance. Let's call this substance "Agent A," a generic term that represents any of the human produced materials that can cause illness in humans: dioxin, PCB, radioactivity, or pesticides. Reactions to any of these materials tend to be the same because we cannot detect them with our senses, and small amounts can cause harm.

In our scenario, a local industry has an accident in which it releases Agent A into the atmosphere. Your job, as the local health official, is to notify those living downwind that they need to evacuate. What kinds of reactions can you expect as you tell each family they should leave?

Some will say, "I've lived here all my life and have been exposed to Agent A. I see no problem. Why should I disrupt my life because you think it is dangerous?" Others will quietly but efficiently pick up what they need and leave. Yet others will react with absolute fear and immediately run out of the house in a state of panic. Again, examples of obliviousness, respect, and fear.

Are these reactions rational? Most people would say that leaving quickly with no panic is a rational reaction. But what about the other two reactions?

Of the two extremes, obliviousness is probably the most irrational. Being totally oblivious of the potential harm greatly increases the risk that you will suffer some harm. Ignoring a problem doesn't make it go away.

Fear can get you out of dangerous situations quickly, but not always without some risk. Leaving immediately means that you keep your exposure to Agent A to an absolute minimum. If you leave your house in a panic you could trip and fall, drive too fast and have an accident, or suffer a heart attack from the added stress. Any of these consequences could be more harmful than exposure to Agent A you might receive by leaving in a more orderly fashion.

Delayed Reactions

The above scenario described possible immediate reactions. Another reaction could take place days, weeks, or years later. If some people become ill with a disease or malady that may seem strange at the time, they may connect the illness to the release of Agent A. If there is an increase of a common illness (i.e., cancer or leukemia), people may connect that with the release. They may observe a significant number of specific illnesses in a small area near the facility that released Agent A.

Showing such a correlation between an illness and the release of Agent A is easy; if the illness occurs after the release then there is a correlation. However, proving a connection is not so easy. To prove a connection we must first have evidence that Agent A actually causes the observed illness. Then we need to have evidence that the amount of Agent A released was sufficient to cause the illness.

If the illness has some specific characteristics (latency period, appearing only in certain populations, etc.) then those characteristics must match what we observe. For example, suppose we know that Agent A causes a specific type of tumor in the lung, but only in smokers (maybe because of a reaction between cigarette smoke and Agent A). If greater numbers of these tumors appear in the lungs of both smokers and nonsmokers after the release of Agent A, then we would conclude that Agent A is not the cause.

In the following sections I will discuss several cases of reactions to incidents or accidents at or near a nuclear facility. My purpose of these discussions is not to cover all the reactions. Rather, I want to look at a few and discuss why these reactions occurred. I will also look at potential or real consequences of these reactions.

The Cost of the Fear of Radiation

Fear is a good defensive reaction to get someone out of danger, but this reaction can also increase the risk of other dangers. In the following situations, I have outlined some results of the fear of radiation. For many people the result is no more than lost time and money, but for others fear results in physical harm.

Three Mile Island

This is clearly the worst nuclear accident in the United States. Because of several malfunctions at the plant -- some mechanical and some human -- 60 percent of the reactor core of Unit II at Three Mile Island (TMI) melted. Because the reactor is in a concrete containment building with air filters, the only radioactivity released was in the form of gases, mostly noble gases plus some radioactive iodine. No liquids or particulate matter escaped the building.

The estimates for the population dose to the approximately two million people living within 50 miles of TMI range from 20,000 to 35,000 person-mGy (2,000,000 - 3,500,000 person-mrad) (Daniels 88). Using the average risk value of 0.00005 per mGy (Table 10-5, Chapter 10), the maximum number of excess cancer fatalities in this two million population would be about one. This would be above the 400,000 solid tumor and 15,000 leukemia deaths expected normally in these two million people.

However, the immediate health effects were not from the radiation but from the fear of radiation and concern over what might happen. There was much uncertainty of how much radioactivity TMI released and what possible health effects would result from the radiation. People's emotions ranged from mild concern to absolute fear.

Utility and government officials held meetings in an attempt to explain what was happening. These meetings usually ended in shouting matches. Citizens labeled the officials as anything from uncaring people to outright liars. Many news reports were incomplete, both because the media did not know what was important and because utility officials giving information could not communicate effectively to the news media. "Experts" gave widely varying predictions of the health effects, from no deaths to thousands.

Of course, many people could not assimilate all this information and became stressed. This stress not only caused emotional problems, it also caused physical illnesses. For a study of stress after the TMI accident, Houts, et al. did two telephone surveys of over 2,000 people, one three months and one was nine months after the accident. They found that "between ten and twenty percent of the population within 15 miles of TMI had heightened levels of distress (compared to persons beyond 40 miles from TMI)" (Houts et al., 1980 27). There were 400,000 people living within 15 miles of TMI (Ad Hoc Population Assessment Group) and between 40,000 and 60,000 of

these people exhibited symptoms of stress. There are no records of any deaths resulting from this stress, but obviously there was some risk of that happening.

The symptoms of this stress were both behavioral (irritability, fits of anger, sleeplessness, loss of appetite, feeling trembly, trouble thinking, and overeating) and physical (headaches, diarrhea, constipation, abdominal pain, sweating spells, stomach trouble, frequent urination and rash). The data show that the levels of stress decreased with distance from the plant. However, the stress did not decrease with time after the accident. Some data show increased stress with time. (Houts, et al., 1980; Houts et al., 1988)

Whereas most natural disasters have a definite conclusion, this had none. A flood is over when the water recedes, a tornado is gone moments after it appears, and a burning house ends when the last flame dies out.

At TMI, there was no definite end. The source of the threat remained as long as the reactor contained radioactive debris. In addition, the possible effects (i.e., cancer) would not become evident for years after the accident and the public kept hearing frequent assertions that the original danger was greater than they were told (Houts, et. al., 1980). Consequently, stress levels continued for many months after the accident.

People took many different actions to reduce the stress of the accident. Many people evacuated the area near the plant. Considering the uncertainty of what was happening in the plant and what radioactivity was or could be released, this was undoubtedly a rational reaction.

Many may have left out of fear, but there was no mass panic and there apparently were no serious consequences. However, such actions did not come without risks. Anybody who drove a vehicle had an added risk of an accident. Anybody who left by airplane received a radiation dose from the flight. That dose could have been more than what that person would have received from TMI.

Two days after the accident, Pennsylvania Governor Thornburg ordered an evacuation of pregnant women and small children. For those who had no place to go, the Hershey Sports Arena served as an evacuation center. Also on that day a local television station asked me to appear on news program to discuss the radiation surveys we were conducting at Dickinson College. Also appearing on the news program was a physician from the nearby M. S. Hershey Medical Center. After our interviews, we met in the station lobby where the physician's wife asked why he did not mention something. He explained that if he had mentioned this information it could have caused panic among the women at the Hershey Arena.

What was this information? Confining so many people in such a small area greatly increases the possibility of an outbreak of an infectious disease. If a child in the arena had measles, that child could infect pregnant women, and measles has a high probability of causing birth defects to a fetus.

Luckily no measles appeared at the evacuation center, so no harm resulted. However, children and pregnant women being in such an enclosed space greatly enhanced the risk of spreading measles to pregnant women. In fact, by the time the evacuation took place, the residents around the plant had received most of the radiation dose already. Was the risk of measles greater than the risk from the additional radiation dose?

One family on a trip to Minnesota drove through upstate New York to avoid the area around TMI. By the time they reached Minnesota one was nauseous, one had itchy skin and another thought her hair was falling out. They concluded that they were suffering from radiation sickness and sought help at a hospital emergency room.

To make matters worse, a friend had lent them a civil defense pocket dosimeter (an instrument about the size of a short, thick pencil used to measure radiation dose) and the dosimeter readings increased every time they looked at the scale. Using blood tests and testing their thyroid glands for radioactivity, the physician concluded that the family had not received a high enough dose of radiation to cause their symptoms (Ketchum (a) 420) Here, the result was stress over what the family perceived to be great harm from radiation.

Chernobyl

Both the direct effects from radiation and the fear of radiation produced much more disastrous results during the 1986 Chernobyl accident than those at Three Mile Island. During the attempts to contain the accident, 28 died of direct radiation exposure. In addition, one died of thermal burns and one died immediately in the explosion, whether from radiation or from the heat is not known (Nugis & Konchalovskii 363).

One analysis of the radioactivity released puts the total dose to the world population at 600,000,000 person-mGy (United Nations Scientific Committee on the Effects of Atomic Radiation, 1988 30). Based on this dose and using the average risk factor (Table 10-5, Chapter 10) of 0.00005 per person-mGy, the predicted maximum number of cancer deaths from the Chernobyl accident would be about 30,000.

Another analysis puts the total world lifetime dose at 930,000,000 person-mGy, about 50 percent more than the UNSCEAR estimate. A more detailed analysis of these doses to each country puts the estimated death rate at 17,400, about 25 percent less than our rough estimate based on total dose (Anspaugh, et al.).

Of course we will never know how many actually will die. These possible deaths will occur over a period of about 50 years and will not be observable among the normal 500 million cancer deaths. And, the 600,000,000 (or 930,000,000) person-mGy population dose includes many individuals who received small doses. If there is a threshold, or the actual risk is much lower than the risks predicted by Table 10-5, then the above estimates of future deaths are too high.

However, we have already observed a significant number of other deaths because of Chernobyl, not due to radiation but due to the fear of radiation.

> According to the IAEA [International Atomic Energy Agency], an estimated 100,000 to 200,000 wanted pregnancies were aborted in Western Europe because physicians mistakenly advised patients that the radiation from Chernobyl posed a significant risk to unborn children. Several nuclear physicians in Europe said that they were contacted by obstetricians for advice on whether they should recommend abortions for their patients. (Ketchum (b) 938)

Later studies show that there may not have been 100,000 abortions, but there were a significant number. Live births in Greece dropped 23 percent during January 1987, reportedly from an estimated 2100 additional abortions performed in May 1986. Overall, during 1986, an estimated 2,500 women had abortions who otherwise would not have terminated their pregnancies. (Trichopoulos) The calculated average dose in Greece during the first year after the accident was about 0.6 mGy (60 mrad) (United Nations Scientific Committee on the Effects of Atomic Radiation, 1988 30), far below the dose that would cause any harm to the fetus.

In Italy there were 4,000 to 8,000 abortions performed because of fear of radiation from Chernobyl. The study that produced these numbers used four different models; the average of the models gave about 5,000 additional abortions during the five months following the accident (Spinelli and Osborn). In Italy, the average dose over the first year after the accident was about 0.3 mGy (30 mrad). Denmark apparently had about 100 additional abortions (Knudsen).

Studies on four other countries -- Sweden (Odlind and Ericson), Norway (Iregens, et al.), Finland (Harjulehto et al.), and Hungary (Czeizel) -- showed no increase in the abortion rate due to the Chernobyl accident. However, no data are available for countries such as Bulgaria, Austria, Romania, and Yugoslavia, countries in which the average radiation dose was from 0.4 to 0.76 mGy (40 to 76 mrad) (United Nations Scientific Committee on the Effects of Atomic Radiation, 1988 30).

The known number of abortions in Greece, Italy and Denmark is about 7,500. If we estimate an equal number of abortions in the countries in which nobody did studies, then we can estimate that the fear of radiation caused the loss of about 15,000 human lives.

These abortions were real, not just possible, deaths. The doses were much too low to have any effect on the fetus. The abortions were totally unnecessary. The fear of radiation had a cost -- a very high cost in this case.

The release of the report on human radiation experiments (Subcommittee on Energy and Power, 1986) produced outrage that humans could be subjected to such dangerous experiments. But it also produced fear among some people who had been or were to be nuclear medicine patients.

Did this fear produce any tangible effects? According to testimony at the Senate hearings, "medical institutions are receiving calls from patients questioning and even refusing nuclear medicine procedures " (Massé. Even those who were subjects of the original studies reacted: "The expressions of fear and psychological trauma we are now hearing from early nuclear medicine patients and tracer study participants suggests a greater potential for harm due to the way in which this issue has been handled than is likely to have resulted from the original studies or medical procedures" (Massé).

Crying "fire" in a crowded theater can cause panic from the fear of being burned. Crying "fire" when radiation is involved can also have harmful effects, maybe not as drastic as in a theater, but harmful as well. When women have abortions because they fear what radiation might do to their unborn child, that is a disastrous result. When people refuse to have medical procedures for which the benefits greatly exceed any possible harm done by the radiation involved, that is also a disastrous result.

Chapter 15
Clusters

Although most people who live near a nuclear facility may not fear for their lives, many have concerns about what could happen to them. They must trust the operators of the facility to tell them if the public receives any radiation exposure. If this trust is lacking, then the residents will feel they are vulnerable to unknown dangers.

This anxiety about potential dangers leads to increased awareness of health problems. When a group of people in a small area are diagnosed with cancer or leukemia, or the incidence of birth defects increases, people begin to suspect that radiation from the nuclear facility is the cause.

Accusations are easy to make. The news media give clusters of illness coverage because they have all the necessary ingredients of a good story: disaster, helpless victims, and an accused culprit. However, a correlation -- the illnesses happen after the nuclear facility began operation -- does not always mean connection -- the nuclear facility actually caused the illnesses).

Disproving these accusations is a difficult and tedious process that takes a long time. By the time someone disproves the accusations, the public has lost interest. Or, if the public hears these accusations for a long time, they believe them to be true no matter what other information they receive.

In this chapter I will discuss two examples of concern about the effects of radiation: the Pilgrim power plant in Massachusetts and the Sellafield nuclear facility in England. Apparent clusters of leukemia appeared near both facilities. Below I have outlined the problem that existed at each and then I have offered explanations of why the apparent link between the nuclear facility and the leukemia does not seem logical.

These two cases do not represent all possible examples. They are just two I have chosen which have rather clear explanations. Other cases exist for which there are similar explanations and others for which presently there are no easy explanations. The purpose of discussing these examples is to show what kinds of questions to ask to decide if a real causal relationship exists between radiation exposure and observed health effects.

In the last part of this chapter I will discuss clusters in general. They are easy to find and easy to correlate with radiation exposure. But to show a causal connection is quite another story. I will offer reasons why clusters from radiation exposure are not likely.

Pilgrim Study

In the 1980s, citizens near the Pilgrim Nuclear Power Plant in eastern Massachusetts expressed concerns that the incidence of leukemia had increased during the years after the plant

began operation. In 1990 the Massachusetts Department of Public Health issued a study on leukemia in eastern Massachusetts (Morris & Knorr (a)).

The Pilgrim nuclear power plant began operation in 1972. The plant had a history of emissions during the 1970s because of problems with fuel rods. The public was concerned that these emissions might be responsible for an increased number of leukemia cases. Based on the plant's starting time, the early emissions, and the latency period of leukemia, the study only considered leukemia diagnosed from 1978 to 1986. "It was assumed in the analysis that the period of time from exposure to crest of leukemia (i.e., latency period) was five years. As a result, any residence or job held during the five years prior to diagnosis was not counted in estimating an individual's exposure potential" (Morris & Knorr 2).

Because no data were available on the actual radiation doses received by the citizens near the plant, the analysis used a "potential for exposure." This was determined by the distance from the plant to the residence, the time of residence, the proximity of the resident's occupation and the time of employment at this occupation and the frequency downwind of the residence and occupation (Morris & Knorr).

That report contained the following findings:

> 1. Individuals with the highest potential for exposure . . . had almost four times the risk of leukemia as compared with those having the lowest potential for exposure.
>
> 2. An association between radiation released from the Pilgrim plant and leukemia incidence was found only among those cases diagnosed before 1984.
>
> 3. No apparent relationship with the plant was observed for cases diagnosed between 1984 and 1986.
>
> 4. Among those diagnosed before 1984, the dose-response relationship was observed in that the relative risk of leukemia increased as the potential for exposure to the plant also increased. (Morris & Knorr 2)

There it was, increased leukemia near the plant after the plant started operation. Sure sounds like they got the culprit! But let's take a closer look at the evidence to be sure that it fits with what we know about radiation-induced leukemia.

We know that there is a minimum two-year latency period. For people at some ages, the latency period is longer. If radiation from Pilgrim did cause the leukemia, it would show up no sooner than two years after the plant began operation. The study accounted for this latency period by not considering any cases prior to five years after the plant opening.

To prove a connection between the plant operation and the incidence of leukemia, the leukemia should start increasing from two to five years after operation began. Then the number of leukemia cases per year should increase for a few years then remain about constant for a few more years. Finally, about ten years after the start of operations, the leukemia rates should begin to slowly decrease, reaching zero in about 30 years. Here, the time of the study is much less than the 25 to 30 year at-risk period. If radiation were causing leukemia, the incident rate should remain high during the entire rest of the study. Instead, the rates decreased rapidly at the time they should be at a maximum.

By not considering the leukemia rates before the plant opened and during the five years after opening, the study had no information on the change. If the rate were already high before the plant opened and during the first five years of operation, then there would be no change and therefore unlikely that the radiation from Pilgrim would be the cause.

The use of the word "crest" is not in agreement with the findings of the BEIR V studies. Leukemia risk does not "crest" after the latency period. It slowly rises with time, reaching a maximum in about 10 years (the time for the maximum risk depends on the age of those receiving the radiation). Then the risk slowly decreases for the next 15 to 20 years, finally reaching zero 30 to 35 years after the exposure.

If the increased radiation levels during the 1970s were the cause of the leukemia, then the increase in the number of cases should have reached a peak in the mid-to-late 1980s and then slowly

decreased. Reporting the increase and then a decrease in leukemia and then reporting a corresponding increase and decrease in emissions implies that radiation is the cause of the leukemia. However, the summary booklet discounts this correlation: "Although it is not possible to reach definite conclusions regarding cause and effect, the results strongly indicate that the effect of low-level radiation on the occurrence of leukemia should be further investigated . . . " (Morris & Knorr 3).

Then we need to look carefully at what is being compared to what. "four times the risk of leukemia as compared with those having the lowest potential for exposure" (Morris & Knorr 2) does not say that the number of cases was higher than normal, just compared to others with a "lower potential for exposure." In actuality, the leukemia incidence near the plant (i.e., the high potential area) was about normal, and the incidence away from the plant (i.e., the low potential area) was lower than normal. In fact, the entire county surrounding the Pilgrim plant had a lower leukemia incidence rate than the United States as a whole. Therefore the question should not be "why is the rate close to the plant high?" but "why is the rate far from the plant so low?" (Wilson 3 - 4).

Finally, the use of "potential" for exposure leaves a lot to be desired. The actual radiation detected off-site was minimal. "Off-site monitoring of radiation indicated that prior to 1980, radiation levels had occasionally been detected above background" (Morris & Knorr 6) The plant exceeded the EPA limit (established in 1980) of 0.25 mGy (25 mrad) per year in the mid-1970s, but not since 1980 (Morris & Knorr 8). However, these radiation dose -- about 25 percent of the dose from background terrestrial radiation -- are much too low to cause any observable increase in leukemia.

Windscale (now Sellafield), United Kingdom

The second worst nuclear power plant accident (less severe than Chernobyl, more than TMI) occurred in 1957 at the Windscale Nuclear facility which is north of Manchester and Liverpool, England. The accident released over 40 different radionuclides. This release produced a significant radiation dose not only to the people near Windscale, but also to people in the European Continent. The estimated total dose to this population is 1,200,000 person-mGy (120,000,000 person-mrad) (Dunster 243).

Using the average leukemia risk value of 0.0008 percent per mGy (Table 10-2, Chapter 10), we would expect 10 additional leukemia cases throughout this population because of the Windscale accident.

From 1951 to 1991, 11 leukemia cases were reported in Seascale, two miles south of the Sellafield (renamed from Windscale) site. Only 1.12 cases would be expected normally for this population. Just north of the site, from 1968 to 1985 Egremont had four cases where only 0.6 would be expected (Younger).

These numbers seemed to prove that the radiation caused the additional cases of leukemia. However, only children born near Sellafield got leukemia and most of these children were not born until after the 1957 accident. If the radiation from the accident was not causing the leukemia, then maybe, as one researcher proposed, the radiation directly altered the sperm of the fathers and these altered sperm are what caused the leukemia. (Younger)

Now let's see if the data fits what we know about leukemia. First, we need to look at the evidence from past experience. Most of what we know about the effects of radiation comes from the atomic bomb survivors. The children of the men exposed to radiation from the bombs are part of the atomic bomb survivor study. However, no increase in leukemia has been observed in the children of these men. The doses received by these men were much higher than that received by the Sellafield workers.

Then we need to look at all the workers at Sellafield. If the altered sperm theory were valid then we should see an increase in leukemia at the same rate in the children of all the workers. Most workers lived in towns other than Seascale and Egremont. Based on the increase in these two towns, 53 cases would have been expected in the areas where most of the workers lived. However, no additional cases were observed (Younger).

In such a situation, researchers look for other pollutants that might be the cause of this leukemia. One group of researchers (Kinlen et al.) proposed that the cause was a virus. Although viruses are not known to cause leukemia in humans, they are a common cause of leukemia in cats. Cat leukemia appears in small groups of cats (i.e., clusters) mainly in kittens who have not developed an immunity to the virus (Younger).

Before the construction of the nuclear facility, the Sellafield area was rural. When the facility opened, many urban families moved to Sellafield, bringing their children with them. Along with the children came viruses to which the rural children had not been exposed. To verify this hypothesis, the researchers studied cities that grew quickly between 1945 and 1965. Indeed, they found similar excess cases of leukemia in these cities (Kondo 146; Younger). Based on this evidence it appears that the radiation from Sellafield may not be the cause of the excess leukemia.

Although scientists have not closed the books on this case, it shows that determining the real cause of increases of leukemia (or solid tumors) is not an easy task. An increase occurring near a nuclear facility does not mean that the facility is the cause. Correlation does not mean connection. To prove a connection, we must be sure that all the facts of the situation fit the known characteristics of the effects of radiation.

Clusters

The two previous situations involved clusters. In the Pilgrim example, more cases of leukemia appeared in selected areas close to the plant than in areas away from the plant. In the Sellafield example, more-than-normal leukemia cases appeared in two small towns near the facility. In each case, the proximity of the nuclear facility prompted people to suspect that radiation was the cause of the higher incidence of leukemia.

What is the nature of a cluster? Typically clusters are the result of an infectious agent (Cehn and Sagan). Some bacteria or fungus spreads from one person to another in an area where people have lots of contact with one another. Flu spreads rapidly in groups such as college students, residents of nursing homes, and other places where lots of people are confined to a small area. Other areas may not be affected. One child with chicken pox can infect many other children in one school, while children in other schools are not affected.

The study of clusters can lead to important discoveries about the causes of some illnesses. A cluster of children with a high incidence of deafness led an Australian ear specialist to learn that the deafness resulted from mothers contracting measles while pregnant. Another physician observed a high incidence of a rare variety of liver cancer among employees of one manufacturer and traced the cause to vinyl chloride (Cehn and Sagan).

For infectious diseases such as the flu and chicken pox, the time from the infection to the appearance of the disease is only a few days to a week or two. This short incubation or latency period means that the illness appears quickly. With a little detective work, anyone might determine the person who started the cluster.

Suppose that chicken pox had a 15-year incubation period rather than a week or two. Someone brings the disease to a third-grade class in one school and infects 20 students. Fifteen years later these students are living all across the country when they finally show the symptoms of chicken pox. There is no cluster and even the cleverest detective would be hard pressed to detect that these 20 widespread cases originated in one place at one time in the past.

What if the cases appeared over a span of time, say from 10 to 20 years after the initial infection? These 20 cases would appear at a rate of about two per year. Deciding that these all had the same origin would be virtually impossible.

Radiation-induced cancer is much like this example. A small group of people exposed to a large dose of radiation will have a higher incidence of cancer. However, the characteristics of radiation-induced cancer make observable clusters unlikely.

Suppose that 1,000 people are exposed to 500 mGy (50,000 mrad) simultaneously. This is a large dose; no group has ever experienced such a dose other than the atomic bomb victims. According to the average risk values (Table 10-2, Chapter 10), the radiation would cause at most

four of these 1,000 people to get leukemia and at most 40 to develop solid tumors. However, these solid tumors and leukemia will not appear at once. After the two-year latency period, the four leukemia cases would appear over a span of about 30 years. Similarly, the 40 solid tumors would appear over a period of 30 years or longer starting about 10 years after the exposure.

People in the United States move about every five years. Many of these moves are local but when considering clusters, even a short move can take the person out of the area. The first leukemia may appear about five years after the exposure. But this is about the same time that half the people in that area have moved away. Therefore, the first leukemia has a 50 percent chance of appearing inside the area where the people received the radiation dose. In later years the probability decreases so that of the five expected radiation-induced leukemia, only one or two may actually appear in the area where the exposure occurred.

The situation for solid tumors is even more dramatic. About 10 years after the exposure, when the first tumor might appear, only 25 percent of the original people may remain in the area. The probability of all 40 expected solid tumors occurring in the original area is very small.

Then we need to consider the natural incidence of solid tumors and leukemia. There would be eight cases of leukemia appearing naturally in these 1,000 people. Although the number of radiation-induced leukemia is about the same as the natural incidence in this case, the overall low number over 30 years would make statistical analyses virtually impossible. There would be many years with no cases, several years with one case, and maybe one or two years with two cases. This data would be inadequate to make a conclusive argument that radiation caused some of the leukemia.

For solid tumors the situation is different but not much better than that for leukemia. The 1,000 people would have 200 tumor fatalities naturally. These would be spread over the lifetime of the population, but to simplify the argument, let's assume they occur over a period of 40 years, or five per year. The 40 radiation-induced tumors would appear at about one per year over this period of 40 years. A careful collection of data over several years might provide enough statistical evidence that radiation was the cause, but highly unlikely even if all the people stayed in the same area.

Now let's turn the situation around. Suppose that we observe a cluster of cancer. What is the probability that radiation exposure caused them? What is the probability that they are just the result of a random statistical fluctuation? Remember, when trying to decide if the cause as radiation, all the characteristics about radiation-induced cancer must be present. These characteristics include a 10-year latency period and a continual high level of solid tumors for many years after the latency period. For leukemia the latency period is two years and the period at risk is about 30 years. For tumors, the period at risk may be also be 30 to 40 years; that is uncertain at this time.

Let's suppose that 15 solid tumors appear where 10 are expected. First, there is about a 5-percent chance that the 15 is just a statistical fluctuation of the 10 expected cases. That means that there is a 95-percent chance that one or more carcinogens, possibly radiation, caused them.

If the cause were radiation, then the exposure for each could have occurred at some random time between 10 and 50 years before the time the cluster appeared. If there had been an increased dose to these people 30 years before the cluster appeared, what is the probability that this exposure caused the cluster?

Because the dose could have been received any time over the previous 10 to 50 years, the probability is 1/40 that the dose received 30 years previous was the cause of one cancer. The probability that all five exposures occurred in the same year would be (1/40) X (1/40) X (1/40) X (1/40) X (1/40), or one chance in 100 million.

To show that the additional solid tumors or leukemia are radiation induced, the cluster group must be followed for many years. If the additional cases appear each year for many years, then the probability that the cause is a single radiation exposure increases.

Shortly after the TMI accident, claims of cancer clusters appeared in the press. However, the probability that these cancers were caused by radiation from the accident is zero. The latency period was totally ignored. These observed cancers had their origin long before the accident. If they were radiation-induced, the exposure was at least 10 years before the accident. In fact, later studies

showed that the incidence of cancer in people living near TMI was not any higher than the normal rate (Hatch, et al.).

Although radiation-induced leukemia should have been appearing from about 1983 to the present, any radiation induced solid tumors would not be appearing until about the present time (mid 1990s). Therefore, giving the accident a clean bill of health based on past studies is premature at this time. Only studies covering the next 10 to 20 years can show conclusively whether the accident caused any excess cancers.

In summary, because of the nature of radiation-induced-cancer, the probability that any cancer from radiation would be observed in a cluster is very small. The combination of a latency period and long period over which they appear destroys the chance that these solid tumors or leukemia will occur in one small area over a short time.

Chapter 16
Plutonium: Just How Toxic is It?

Plutonium has suffered from bad press. We often hear: "Plutonium is the most toxic element known to exist." This statement appeared early in the World War II years "in part to ensure support for the needed protection measures, in part to secure full cooperation of the workers, and in part because the accumulating data were pointing in that direction" (Stannard 367-368).

But what does "toxic" mean? A material can be toxic from its chemical characteristics (as are the standard poisons such as arsenic and strychnine) or from radiation. The effects from plutonium radiation can be either immediate or delayed. Immediate effects require large doses in a short time, but delayed effects need moderate to large doses over a long time. However, as we learned in earlier chapters, the delayed effects are not 100 percent toxic to the individual receiving the dose. A large dose only increases the risk of cancer, it does not give a 100 percent risk.

Toxicity

First, let's consider chemical toxicity. Several other materials are much more toxic than plutonium. Because data on the toxicity of plutonium for humans is nonexistent, we need to look at animal data. The following numbers are for amounts in the blood stream. For a 100-gram mouse, 130 μg[3] of plutonium would be toxic. Equally toxic would be only 50 μg of strychnine, 5×10^{-7} (0.0000005) μg of botulinus toxin A, or 7×10^{-10} μg of crystalline botulinus toxin (Stannard 368). If human toxicity per mass is comparable, then the toxic amounts for 70 kg person would be 90 mg of plutonium, 36 mg of strychnine, 3.5×10^{-7} mg of botulinus toxin A, or 5×10^{-10} mg of crystalline botulinus toxin.

People are primarily afraid of plutonium's radiation. Plutonium has several different isotopes (same element with the same number of protons but with different numbers of neutrons). The most common isotope, produced in a nuclear reactor as a byproduct of nuclear fission, or produced for the expressed purpose of making nuclear weapons, is plutonium-239. Therefore, I will base the following discussion on plutonium-239.

Plutonium-239 emits alpha particles. It also emits some gamma radiation, but the number of gamma rays is small and of no real consequence when considering the dose from plutonium. As we learned in Chapter 1, alpha radiation does not penetrate material readily. Only living material immediately next to an alpha emitter will receive the alpha energy. Alphas cannot penetrate the dead layer of skin so the only way plutonium can cause harm is if it is inside the body. Alphas do more biological damage per absorbed dose than beta or gamma radiation, so plutonium is more hazardous if taken into the body.

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[3] Because the amounts are very small, we will use fractions of a gram as our unit of measure. A microgram (μg) is one millionth of a gram. Later we will use milligram (mg) which is one thousandth of a gram.

Disregarding injection, there are only two pathways into the body: ingestion or inhalation. Information derived from the human studies shows that the plutonium will concentrate in the bones and liver. However, to get to these sites the plutonium must get to the blood stream first.

Studies show that nearly all of ingested plutonium passes through the gastro-intestinal system with only a small fraction digested and entering the blood stream. Most is excreted. The blood picks up only 0.003 percent or less (International Commission on Radiological Protection). If we want to give a 70-kg person a chemically toxic dose of plutonium (90 mg in the blood stream), that person would have to ingest 3 kg of plutonium.

Terrorist Threats

One concern about plutonium is that terrorists would get hold of some and use it to threaten the population of a city. There are two potential kinds of threats. The terrorists could make a nuclear weapon or they could use the plutonium to poison the people in the city. The threat of making a nuclear weapon is real, but how great is the threat of poisoning?

As we just saw, putting plutonium in drinking water is not an effective way to poison people. To give one person a toxic dose of plutonium, the terrorist would have to get each person to drink water with about 3 kg of plutonium in it. This amount is about the minimum needed for a critical mass for a nuclear explosion! Of course smaller amounts, though not immediately toxic, would produce a later risk of cancer in the population. Such delayed effects would diminish the impact of my terroristic threat.

What about spreading the plutonium in the air over a city? It is very difficult to get significant quantities in the air over a large area so this is not an efficient way to deliver a threatening radioactive dose.

I did a rough calculation to determine the amount of plutonium that would be in the air above a city if a terrorist were to spread 1 kilogram of plutonium over an area of one square mile. That concentration would be about 0.02 μg per cubic foot if there is no wind to blow it away. If each person breathes this air for one hour, each would inhale about 1 μg of plutonium. As we will see shortly, that is significant but would have no immediate effects on the population. This amount of plutonium would increase the risk of cancer, but these cancers would not appear for many years.

This situation is analogous to car exhaust. We all know that running a car in a closed garage can be lethal to anybody in the garage. However, we drive thousands of cars in our cities with no real harm to those in the city. Why is there no harm? The vast amounts of air rapidly disperse the exhaust so the concentration in the air is far from being harmful. Likewise, spreading plutonium over a large volume of air reduces its concentration and its effectiveness as a toxic agent.

Plutonium inhalation; allowed limit of intake

When a person inhales air containing suspended plutonium compounds, some of that plutonium will get into the blood and then to the liver and bones. However, we also need to consider the lungs themselves. If the plutonium sticks in the lungs, then the lungs will also receive a considerable radiation dose and potentially cause lung cancer. But in the lungs, plutonium is not any more dangerous than the same activity of most any other alpha emitter.

The energy of the alpha particle from plutonium decay is more than the energy of the alpha from radium, but less than that from the alphas from radioactive nuclides such as polonium-214, and americium-241. Polonium exists in the air we breathe as a result of radon in the air, and americium is found in smoke detectors. Therefore, plutonium's alpha radiation does less damage than most alpha-emitting radionuclides to which we are exposed or have potential exposure.

The International Commission on Radiation Protection has established limits for workers. These limits are expressed in terms of activity, Bq (or Ci)[4]. These limits are set by determining the amount of a radioactive material that will produce an annual dose of 50 mSv (5,000 mrem)[5] to the total body. For plutonium, the risk from this dose is entirely from cancer of the liver, bones, and lungs. This limit is called the Allowed Limit of Intake (ALI).

Table 16-1 gives the ALI values for plutonium plus three other radionuclides with which we may come in contact. I have also included the conversion from Bq to mass so you can see how much mass is involved.

Radium-226 and polonium are naturally occurring radioactivity while americium-241 is human-produced. Radium-226 is commonly found in water supplies that come from wells. Polonium-210 is found in nature in the soil and, to some extent, in the air. It is also found on tobacco plants and as a result in the lungs of smokers. Americium-241 is the radionuclide used in ionization-type smoke detectors.

Table 16-1
Annual Limits of Intake (ALI)

	ingestion (oral)		inhalation (breathing)	
Radionuclide	Bq	μg	Bq	μg
Plutonium-239	200,000	87	350*	0.15*
Radium-226	70,000	0.92	20,000	0.26
Polonium-210	100,000	0.0006	20,000	0.00012
Americium-241	50,000	0.40	200	0.0016

*Some chemical compounds have different fractions that get to the blood stream from the lungs and some stay in the lungs for different times. The value listed here is an average of the two values listed.

Source: International Commission on Radiological Protection, Publication 30

Plutonium Compared with Three Other Common Radionuclides

We saw that plutonium does not enter the blood readily from ingestion. Therefore we would expect that the limit for ingestion to be higher for plutonium than for elements that more readily enter the blood stream. In fact we do see this in table 16-1. In terms of activity (Bq), the ALI for ingestion of plutonium is twice that for polonium-210, three times that for radium-226, and four times that for americium-241.

When we want to compare the toxicity between different materials, the common unit of measure is mass. If we consider the mass rather than activity, the picture changes considerably. The 87 μg limit for plutonium is about 100 times that for radium-226, 200 times that for americium-241, and 15,000 times that of polonium-210. In terms of mass, plutonium is 100 to 15,000 times less toxic than these other three radionuclides when ingested.

Plutonium is more toxic when inhaled. The ALI, in terms of activity, for plutonium is 60 times less than those of radium-226 and polonium-210. However, the ALI for plutonium is greater than that for americium-241. But again, the picture is considerably different when we look at the masses. The plutonium ALI is still less than radium-226 (by about a factor of two), but is about 100 times greater than the ALI for americium-241 and 1200 times that for polonium-210.

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4 Activity is a measure of the rate at which atoms of a radioactive material emit radiation, or decay. An activity of one Becquerel (Bq) is one decay (and in this case, one alpha) per second. A Curie (Ci) is 37 billion decays per second.

5 Because we are dealing with alpha radiation in this chapter, we will need to use the dose equivalent (Sievert (Sv) or rem, or, to keep some consistency, in terms of mSv or mrem) as our unit of dose. Remember that alpha radiation does more biological damage per dose (Gray of rad) than X- and gamma-radiation.

Radium is almost nonexistent in water supplies that come from rivers and lakes, but can have measurable quantities in well water. Many public water supplies that come from wells can have radium-226 in the five to 36 pCi (0.2 to 1.3 Bq) per liter range (Stannard 1340; Eisenbud 172-173; Kriege and Hahne). However some wells can have considerably higher levels. One study of wells in Iowa found one well with 48 pCi (1.8 Bq) per liter (Kriege and Hahne). One well in Pennsylvania had 17 pCi (0.6 Bq) of radium-226 but 140 pCi (5.2 Bq) per liter of radium-228 (Smith and Delano).

A person who drinks 1.5 liters of water a day with 36 pCi (1.3 Bq) per liter ingests over 700 Bq per year. This is 1 percent of the ingestion ALI for nuclear workers. However, some mineral springs have water with radium concentrations up to 100,000 pCi (3700 Bq) per liter (Eisenbud 198). Nobody would probably drink 1.5 liters of this water each day. However, a person would only have to drink 20 liters in one year to reach the maximum allowed intake for nuclear workers. Twenty liters per year is only 55 milliliters per day, about two ounces per day.

Polonium-210 in air ranges from 0.0002 to 0.0015 Bq per cubic meter (0.005 to 0.04 pCi per cubic meter) (National Commission on Radiation Protection (a) 101). A person breathing an average of 20 cubic meters per day would have an intake of about 6 Bq per year at an average concentration of 0.0008 Bq per cubic meter. This is a minor amount. However, cigarette smokers breathe in considerably more than this.

Smoke inhaled by a smoker contains about 0.007 Bq (0.2 pCi) of polonium-210 per cigarette (Radford and Hunt 248). A two-pack-a-day smoker inhales about 100 Bq of polonium-210 in the cigarette smoke each year which is 0.5 percent of the maximum allowed intake for nuclear workers. Some calculations put the radiation dose to parts of the respiratory system at 160 mSv (16,000 mrem) per year (National Commission on Radiation Protection (b)).

The americium-241 source in a smoke detector has a typical activity of 1 μCi, or 37,000 Bq. The radiation dose from the gamma radiation is small, but there is some risk if someone removes the americium source and either eats or breathes in the americium. The 37,000 Bq activity is less than the annual limit of intake (ALI) of 50,000 Bq for ingestion. If 50,000 Bq gives a whole body dose equivalent of 50 mSv (5,000 mrem) then the 37 kBq will give a dose of about 40 mSv (4,000 mrem). The ALI for inhalation is only 200 Bq, so 37,000 Bq is 185 times that limit. Although it would be difficult to scrape off all the americium and breathe it in, a person could inhale enough to pose a serious risk.

In case of fire, the americium-241 could burn off the detector and get into the air (and smoke) in the house. If a person were to inhale the entire 1 μCi of americium-241, then that would produce a total dose to that person's bone surface of 2,800 mSv (280,000 mrem). (International Commission on Radiological Protection) In a fire, however, the americium would disperse quickly so the hazard from the radioactivity would be of minor concern especially when compared with the hazards of the smoke and fire.

Maybe a better comparison would be with radon progeny. At the radon "safe" level of 4 pCi/L (150 Bq/m^3) each of the progeny has a concentration about half that of radon, or 75 Bq/m^3. At some average breathing rate, an average person would inhale about 500,000 Bq of each of the radon progeny each year. However, not all the progeny stay in the lungs and they have considerably shorter half-lives than plutonium. As a result it is hard to make a direct comparison, but we can see that the radon progeny can (and do) have concentrations in the lungs that produce a radiation dose comparable to that from the ALI of plutonium.

From the above discussions we can see that plutonium has gotten a bad rap; it may be dangerous, but it is far from the "most toxic" element on earth. This does not mean that we can ignore it. Small amounts of plutonium in the lungs, like any alpha emitter, have a probability of causing lung cancer. But the likelihood of anybody getting enough in the lungs to pose a significant risk is small.

Chapter 17
Irresponsibility and Absurdity

Newspapers, magazines, and books contain many claims of harmful effects of radiation not documented in the scientific literature. In this chapter I will present some brief discussions of some these claims. For each I could go on for pages, but the pages would be too hot to handle! I have no objection to people making claims that are not in agreement with the facts generally accepted by the scientific community. However, I strongly object when those making these claims do so with little or no scientific basis. Those who make these claims usually have an agenda and, in my opinion, that does not include reason and responsibility.

Many who make these claims of radiation effects base their claim on some known connections between radiation and effects. However, the amount of radiation to produce these effects is just too small to produce the effects. For example, scientists have evidence that carbon dioxide can cause global warming. If I run my automobile on the morning of a summer day and the temperature subsequently rises during the day, can someone claim that my car exhaust caused the temperature rise? This is the type of connection used in many claims about the effects of radiation.

We should be cautious when dealing with radiation, but being overcautious can lead to wasted funds and increased risks from other causes. I will discuss one example that I feel falls into the category of absurdity.

Increased Death Rates; Use of Percent Changes

In Deadly Deceit, Gould and Goldman make claims that radioactivity from Chernobyl caused a large increase in infant and total deaths in the United States. Using percent changes in death rates and measured levels of radioactive iodine in milk, they found a supra-linear correlation between iodine levels and percent change in death rates across the United States (Gould and Goldman 18).

We know that radiation can harm the fetus, but the levels need to be in the order of tens of mGy. The maximum levels of iodine reported were such that the iodine would produce a thyroid dose of less than 0.02 mGy (2 mrad) and a whole body equivalent dose of less than 0.0005 mGy (0.05 mrad). This dose is a tiny fraction of normal background radiation. Even if other undetected radionuclides were present, they would produce only a small additional dose. Yet Gould and Goldman claim that this radiation dose was enough to produce large increases in the death rates.

The cause of the adult deaths? They do not claim that it was cancer or even acute radiation effects. Instead they claim that certain groups succumbed to normal illnesses, primarily from AIDS-related illnesses, infectious diseases, and pneumonia. They targeted the increase in the death rate from AIDS-related illnesses over the previous year (Gould and Goldman 17). In a time when the incidence of AIDS is increasing rapidly, we would expect an increase in that death rate. Although we do know that radiation affects cells, there is no study in the literature to show an immune-response effect at any dose. Even the atomic bomb survivors did not show this effect.

The data on death rates presented by Gould and Goldman are in terms of percent changes from the corresponding month of the previous year. This type of data gives misleading results. A decrease in the death rate a particular month of one year means that when the death rate returns to normal the following year it appears as an increase. Even if the numbers fluctuate about some average value, when we look at the average change it will always be positive; normal becomes an increase.

For example, suppose the death rate changes from 100 to 90 and then back to 100 in three consecutive months. The percentage changes are a negative 10 percent and then a positive 11 percent. The "net" change is then a positive one percent (i.e., 11 minus 10) even though the death rate returned to the original value.

This type of analysis is similar to the situation with the study at the Pilgrim nuclear plant. When looking for change we must compare the month or location under study with some average value to see if the death rate changes from what would be considered normal. We cannot compare changes to something that fluctuates.

Gould and Goldman also claim that the accidents at the Savannah River nuclear weapons facility in the 1970s and at TMI in 1979 caused increases in death rates (Gould and Goldman

Chapters 4 and 5). They base their claims on percentage changes. Here the comparison is to the expected death rate. The infant and total death rates have been falling so they project what it should be. When the death rate does not decrease as fast as a linear extrapolation suggests, then they correlate that slower decrease with these accidents. Correlation is not connection.

Mutations: AIDS and Lyme Disease

Gould and Goldman claim that Lyme disease and AIDS are the result of human-produced radiation. (Gould and Goldman 132-141) In both cases they claim it is due to mutations. For Lyme disease they claim a bacteria mutated, and for AIDS a retrovirus mutated. The radiation that supposedly created Lyme disease came from a nuclear power plant in Connecticut. For AIDS, the radiation was supposedly from radioactive strontium from fallout.

Radiation can cause mutations. However, how much radiation is needed? To a first approximation, the sensitivity of an organism depends on the organism's size; the smaller the organism, the less sensitive. Insects are more resistant to radiation than large mammals. For example, an acute dose of 6,000 mGy (600,000 mrad) is fatal to humans, but 18,000 Gy (1,800,000 mrad) is needed to kill fish. The dose to kill yeast is about 300,000 mGy (30,000,000 mrad). To kill paramecium, the dose is about 3,000,000 mGy (300,000,000 mrad). A similar relationship exists for mutations.

In both cases the dose from the human-produced radiation is small. The radiation from the nuclear power plant in Connecticut would be undetectable from background radiation. AIDS had its beginnings in equatorial Africa. Weather patterns are such that the regions near and south of the equator received minimal fallout from atmospheric nuclear weapons testing including those in the Pacific Ocean (International Physicians for the Prevention of Nuclear War).

The claim that a particular small increase in radiation above normal background is the particular radiation that caused AIDS or Lyme disease is hard to believe. I would say these claims are ridiculous and irresponsible.

Crying Fire

Although we have a good idea of what radiation can do, there are always unknowns. We should not throw out claims of disease not seen before without some investigation. Scientists have made many important scientific discoveries by observing something different and then following up with a more detailed study. So what is wrong with making claims that go against the mainstream of thought?

Essentially nothing is wrong if that claim is first thoroughly researched before making it public. The cold fusion story is an excellent example of the release of information before thoroughly checking the facts. What were the consequences of that episode? Besides many red faces, research groups around the world interrupted their research to try to get onto the bandwagon. Some researchers diverted precious money to a futile effort that could have been spent on other research. As a minimum, several careers were severely disrupted or ruined.

The day after the accident at TMI, Dr. Ernest Sternglass flew into Harrisburg, Pennsylvania. Based on some quick radiation readings, he predicted "I-131 doses could prove devastating to small children and infants *in utero*" (Wasserman and Solomon 247). Later he held a news conference and announced that his studies showed a three-fold increase in infant deaths at the two Harrisburg hospitals (Harrisburg Evening News). However, in an editorial about Sternglass's claims, the Harrisburg Evening News stated that: "a check at the hospitals -- which Sternglass claimed as his source for these figures -- revealed very different statistics. And hospital officials said the rate of infant deaths apparently has not been affected by the nuclear accident. Either Sternglass is inept at gathering statistics, or worse, he simply fabricated them to fit his own conclusion" (Harrisburg Evening News).

Was this a responsible action? I think not. Dr. Sternglass apparently did not do a proper job of getting facts before making his pronouncement. The amount of iodine-131 released was a minute

fraction of all the radioactivty from the accident (Daniels 86) and would do no harm to anybody. What is wrong with being too cautious? Isn't it better to err on the safe side?

If you were to discover a small fire in a theater, would you stop the film and calmly ask the patrons to leave quickly? Or, would you run out in a panic and shout "Fire"? Think about the consequences of each action.

When Sternglass shouted "infant deaths," what was the reaction? At the minimum, pregnant women became alarmed. Would stress greatly affect the fetus? Possibly. More than the effects of the radiation? Also, possibly. Would shouting "devastating to small children and infants *in utero*" cause an expectant mother to abort the fetus for fear of having a deformed child? No such cases were reported, but who knows?

What happens if the public hears exaggerated claims which turn out to be untrue? After repeated claims the public will become insensitive to cries of harm. When a situation arises in which radiation really does some harm, the public will no longer listen. Crying "wolf" too often is bad news for the chickens.

Is it Safe to Eat This Dirt?

About 30 years ago there were proposals to use nuclear detonations to make artificial harbors for ships. As part of the planning for such a harbor in Alaska, scientist conducted several studies on the movement of radioactive materials in the Alaskan tundra. Although the nuclear explosion never took place, some radioactivity from the environmental study remained. The only radionuclide remaining after 30 years was 0.11 GBq (3 mCi) of radioactive cesium, which has a 30 year half-life. The cesium-137 was in a six-foot mound of dirt that had a volume of about 3,000 cubic feet. The mound was located 35 miles from any human settlement (Gerusky).

To put this amount in perspective, 3,000 cubic feet of average soil in the U.S contain about 8 mCi of natural radioactivity. In 1970, a student and I measured the fallout radioactivity in some soil in Pennsylvania. We measured about 17 GBq (450 mCi) of cesium per square mile (Miller). An area of 4 acres of this soil contained the same amount of radioactive cesium that was in that pile of dirt in Alaska.

The local government with jurisdiction over the site petitioned the federal government to remove the contaminated soil. When the job was finally completed, the soil was packed in steel drums and transported to the Nevada test site for disposal. It even got police escort for part of the trip. The cost: $13 million (Gerusky) .

The risk of leaving this radioactivity in that dirt pile in Alaska was virtually zero. That cesium was not going anywhere. The cesium would not leach into any ground water. Nobody was going to put a garden on top of that soil so no cesium was going to get into any food. Nobody would be building a house on that soil. Nobody would eat the soil either. Was the remediation risk-free? Nobody was hurt, but there were tales of how a plane made two unsuccessful attempts to land at the site but couldn't because of high winds. The passengers on that plane had a much different view of the potential risk from the cesium and the real risk of a plane crash than those who wanted the site cleaned up. (Gerusky, 1997).

Chapter 18
Reason

When you are in an automobile accident, your insurance claim adjuster must decide how much to spend to repair your car. If only a fender is damaged, then the insurance company will pay to have the fender repaired. However, if the costs are more that the car is worth, the car is "totaled" and taken to the junk yard. You receive a settlement equal to the worth of your car before the accident.

Likewise, if your house needed repairs, you would repair the house only if the cost of the repairs didn't exceed the value of the house. Who decides what to spend on what repairs? You do. You are free to make the decision about what to repair. Upon what do you base your decision?

If the roof leaks and water might damage the ceiling below, then you might find that the cost of fixing the ceiling is more than the cost of fixing the roof. On a pure cost basis, you would save money repairing the roof. But what if you can't put a monetary value on the benefits of the repairs?

If the outside doors in your house are wooden and have poor locks, there is a risk that somebody could break into the house. A thief could steal items which may have some monetary value. However, the intruder could also attack your family, causing both physical and psychological damage. How much is that worth? You can't really put a monetary value on feelings so you must make a subjective decision.

If you live in an area where break-ins are common, then the door repair would definitely be worth the money spent. If you live in an area where crime is virtually unknown and people routinely leave their doors unlocked, then the repair may not be worth the money.

What if you do not understand or do not trust the local officials who say that the crime rate is low? You then might decide that protecting your house is very important. You could spend thousands of dollars on new doors and windows, a heavy fence around the yard, an internal and external security system, and then hire someone to guard the house when you are away. That should reduce the risk of anybody intruding to just about zero. Now you are safe.

What are the consequences of all this extra protection? It costs money. Unless you have an inexhaustible supply of money, then the money you spend on the protection measures cannot be spent on other items. If the tradeoff is that you do not buy a pleasure boat, then the money you spent on security does not affect your safety.

But suppose it means that you delay buying a new car and keep your old car longer. Your old car does not have a passenger-side air bag but a new car would. If you are in an accident, anybody on the passenger side may suffer more injuries than they may in a new car. Your extra expenditures to reduce the risk of an intruder increases the risk of injuring a passenger in an accident.

These are the same kinds of decisions and consequences that we face when we deal with the risk from radiation. Which actions are reasonable and which ones are not? In the radiation protection field, there is a phrase: "As Low As Reasonably Achievable" (ALARA). This means that when methods are available to reduce the radiation exposure to a worker, then those methods should be used if the cost is reasonable. The problem is the definition of "reasonable."

If your neighborhood school has high levels of radon then you would want the school administration to reduce the radon levels. If the remediation costs only several thousand dollars to reduce the radon to the EPA recommended level of 4 pCi/L, then that is definitely reasonable. If it costs an additional few thousand dollars to reduce it to 3 pCi/L, that might also be reasonable. However, if the cost is millions of dollars to get the levels down to 1 pCi/L, then that may not be reasonable.

Some parents might protest and say that any reduction is worth the cost. But where would the money come from? Are the parents willing to have the quality of their children's education reduced to pay for the radon remediation? Maybe they will pay additional taxes for the work. As indicated in earlier chapters, we don't really know for sure that there is any significant risk at these low levels. A level of 3 pCi/L may be just as safe as 1 pCi/L.

Some parents might be willing to pay the costs to reduce possible health risks, but what about the taxpayers who do not have children in school? If they perceive the decrease of risk minimal or nonexistent, they may oppose such large expenditures.

These are the same problems we face when we try to remediate radiation levels. Should we clean up an area contaminated by radioactivity? What is the risk if we don't and what is the risk if we do? Is the cost of cleanup reasonable? The answer to these questions depends on how we view the risks.

If we, like the homeowner who spend thousands of dollars on a home security system, have an exaggerated view of the risks, then we could spend money on a cleanup that really does no good. The price we pay is the loss of resources to pay for other measures that would be more effective.

The theme of this chapter is reason; what is reasonable to spend on radiation remediation? In previous chapters, we discussed the effects of radiation and how much radiation is needed to cause these effects. However, some people do not agree with this understanding. First we will look at these

claims and put them into perspective and then explore possible consequence of accepting these claims.

Second we will consider what could happen if we abolished anything that could produce an involuntary radiation dose to the public. Would this produce lower risks or would we just shift the risk from one activity to another? Could the risk increase?

Then we'll consider keeping the present uses of radiation but consider how much we want to spend (and are spending) to keep the risk to some acceptable level. How does this cost compare with the costs to keep other risks low? This will be one way to get a measure of benefit and risk.

Alternatives

One way to reduce radiation dose from human activities is to eliminate these sources. For example, we could close down all nuclear power plants, eliminate X-rays and nuclear medicine, stop using radioactive materials in research, and ban consumer products such as watches that have glow-in-the-dark dials. Except most medical practices and many research methods, there are "non-radioactive" alternatives available for most uses of radiation.

We can use coal, oil, natural gas or even solar, wind, hydro, or geothermal energy as sources of energy for electrical power rather than nuclear power. However, these options come with a price, and that price is risk.

What is the price we would pay for replacing nuclear with coal? First, we would not get rid of radioactive emissions to the environment. A coal-fired generating plant releases natural radioactivity, such as thorium-232 and uranium-238 and a long series of radioactive elements these elements generate, including radium, radon, and radioactive lead and polonium. These radioactive materials produce a radiation dose to the public through inhalation of the radioactivity in the air, ingestion of radioactivity that gets into the food chain, and from direct radiation exposure from radioactivity deposited on the ground.

A typical 1,000-megawatt coal-fired plant will produce a dose of about 5,000 person-mGy (500,000 person-mrad) to the public each year. The estimated annual total population dose from all the coal-fired plants in the United States is between 80,000 and 700,000 person-mGy (8,000,000 and 70,000,000 person-mrad) (National Commission on Radiation Protection). Note that this is considerably more than the 20,000 to 35,000 person-mGy (2,000,000 to 3,500,000 person-mrad) total population dose from the TMI accident.

Besides radioactivity, coal plants release sulfur-dioxide and carbon-dioxide. Sulfur-dioxide, when combined with water in the atmosphere, creates sulfuric acid which falls as acid rain. Carbon-dioxide contributes to the greenhouse effect. Although the technology is available to remove the sulfur from coal plant emissions, not all plants use it because of the cost. Most coal plants have fly-ash removal. Fly-ash is the small black particulate material (soot) that comes from burning coal. However, this fly-ash contains several nasty elements.

Oil and gas combustion also produce carbon-dioxide. However, probably the greatest drawback of using oil or natural gas is that these two fuels are more valuable as fuels for automobiles and for heating homes. In addition, oil is the source of many materials that we use everyday such as plastics, asphalt, and chemicals. Our oil and gas resources are finite. Some scientists estimate that the world oil and gas resources will last only several more decades. We should save these valuable resources for the future rather than burning them up simply to avoid some risk today.

Although solar, wind, hydro, and geothermal energy generate pleasant thoughts of pollution-free energy, they are not benign. The production of solar photovoltaic cells requires manufacturing processes that use some rather nasty chemicals. Solar panels, to be efficient, need to be in areas with lots of sun, mainly deserts. However, these areas are very sensitive to minor alterations so solar energy has the potential to do major environmental harm.

A 500-acre solar power plant in California's Mojave Desert has a peak output of 90 megawatts, less than 10 percent of the normal output of a typical nuclear plant. The average power is considerably less. This plant uses direct heating to produce steam which turns a turbine rather

than using photovoltaic cells to convert sunlight directly to electricity. However, it does produce electricity at a wholesale cost only slightly higher than nuclear (Wolfson 276).

The costs of solar photovoltaic cells has decreased to where they are close to the cost per watt of nuclear power plants. The area needed to produce a significant amount of power is large. However, only about 3 percent of the land area of New Mexico would be needed to power the entire United States (Wolfson 278). The power may be available, but there is always the problem of storage (the sun tends not to shine at night and clouds often appear in New Mexico) and the potential environmental harm to desert areas.

Wind energy is not really polluting unless you want to consider sight pollution. They fall in the category of ugliness comparable to crabgrass, satellite antennas, and highway sound barriers. The manufacture and maintenance of the windmills is not risk-free. Besides sight pollution, the windmills pose a risk to migrating birds. Windmills kill hawks, owls, and falcons when the birds fly into the windmills (Wolfson 261).

Hydropower requires dams, and dams have a large environmental impact on wetlands and migrating fish and they sacrifice valuable farm land. History has shown that people living below a dam have a real risk of death from a dam that breaks.

Geothermal energy uses heated water from the earth. Generally this water contains many toxic materials. In Hawaii, environmentalists and native peoples oppose a proposed geothermal plant. This plant would be built in one of the few remaining rain forests in the United States (Wolfson 266).

There is no such thing as a free lunch. Because each kind of energy has its own set of environmental problems, none is perfect. We need to take a lesson from the environment: diversity. We need to develop all kinds of electrical generating methods and use a mix. This would spread the harm equally among many different areas of the environment.

The elimination of industrial uses of radiation would have various impacts. Eliminating the use of radioactive sources in oil and gas well drilling would mean a lower success rate for finding oil and natural gas. Besides the added environment and human risk of drilling more wells, the major drawback would be higher costs for oil and gas.

Eliminating the use of radioactive sources to inspect welds in pipelines could have disastrous results. Pipelines that carry natural gas must pass though populated areas to get the gas to those areas. Without adequate inspection of the welds, a pipeline could rupture and cause an explosion and fire that would put nearby residents at risk.

Eliminating a risk from radiation by eliminating the sources of radiation exposure will not guarantee a safer world. Often the risk would be greater. Before advocating the elimination of radiation, we need to be sure that the elimination will really reduce risk.

Is Cleaning Up Nuclear Sites a Sound Investment?

Sites used for the nuclear weapon's program in the continental United States are scheduled for cleanup. The cost of this cleanup is in the billions of dollars, not millions. Will we get our money's worth? Does the fear of radiation drive a cleanup that poses less of a risk than other, less-frightening, but more damaging, risks?

An editorial in the June 16, 1994, Washington Post stated:

> Although the nuclear weapons plants have a reputation for toxic pollution, the CBO [Congressional Budget Office] cites EPA studies concluding that hazardous waste sites present less danger to health than many more common threats -- indoor pollution for one, pesticide residues in food for another. The way the federal government is currently allocating its spending on environmental hazards is not closely related to the risks as they are assessed by experts it has consulted. That raises the question about the annual outlay of $6 billion for this nuclear cleanup. It's the right figure only if the money is buying more health

protection than it could if aimed at other kinds of pollution. (Washington Post)

Much like the Alaskan example, we would do better to leave the installations isolated for many years as long as they pose no threat to the public in that form. Cleanup can take place after much of the radioactivity has decayed away. "Sometimes the cleanup itself creates risks. Unless hazardous materials are likely to leak into the atmosphere or water supplies, leaving them alone is often worth considering" (Washington Post).

Then Federal Judge, now Supreme Court Judge, Steven Breyer described another example of excessive cost for trivial results in his book Breaking the Vicious Circle. After a New Hampshire toxic waste site was cleaned up, all but one of the litigating parties agreed that this was sufficient. However, one party successfully litigated further cleanup. This further cleanup cost $9.3 million. What did it get? Instead of a child being able to eat small amounts of this soil for 70 days and suffer no harm, that child could eat dirt for 245 days. How many dirt-eating children do you know? (Breyer 11-12)

In an article in the Washington Post, Robert Samuelson commented on this story: "The Standard Retort is: A rich country like ours can afford absolute safety. No we can't. Regulatory costs raise prices or taxes. Our incomes are lower than they might be. That's okay if we receive lots of benefits -- much cleaner air or healthier food. But its not okay if the benefits are trivial or nonexistent" (Samuelson).

How Much Do We Pay to Save a Life?

How much is the public willing to pay to save a life? Usually, a lot. In one survey in Sweden, when asked what a government or municipality should spend to save a life, "one-half of the respondents replied that no expense was too high" (Bengtsson and Moberg 663). Some Swedish officials expressed opinions of what protection costs would be reasonable. The results ranged from one times the gross national product per person for preventing lung cancer from radon, to one to two for road safety, to five to forty for radiation protection by the government nuclear facility.

From these studies, the "rule of thumb" for radiation protection in Sweden is that if some measure costs less than one million U.S. dollars for one prevented case of serious radiation injury, then that measure is strongly justified. If the cost is between $1 million and $5 million, then the measure is justified. If the cost is more than $5 million, then the measures are not justified unless there are some special reasons (Bengtsson and Moberg).

When the ALARA (As Low As Reasonably Achievable) concept was first introduced years ago, there was no official definition of what was "reasonable." The unofficial definition that spread around the industry was $1,000 per person-rad ($100,000 per person-Gray or $100 per person-mGy). If we assume that the risk is from cancer, then we can use the Table 10-5 average risk value of 0.0049 percent per mGy to determine a cost per life. Converting from percent to numerical risk, this becomes 0.000049 , or rounding, 0.00005 per person-mGy. At $100 per person-mGy, the cost to save one life is $2 million ($100 per person-mGy ÷ 0.00005 per person-mGy). This is in line with the Swedish survey results.

How do these values compare with what we actually spend for other life-saving measures? Table 18-1 lists values for cost per life-year saved for several measures. The cost per year of life saved is how much it costs to save one year of life for one person. For example, suppose that a certain medical test costs $100 and for every 1000 people undergoing this test one potentially fatal case is detected. Without this test that person would have died. And suppose that on the average the person whose life was saved lives an average of 20 years longer. The cost per life would be the total cost of the tests, $100,000 ($100 X 1000), divided by the life-years saved (20), or $5,000.

The values listed in Table 18-1 are just a few of more than 500 lifesaving interventions in the Tengs study. However, you cannot take these values as absolute. Each value is the result of many assumptions by different researchers. Some values listed in Table 18-1 are averages of two or more values that can differ significantly. For example, the $39,000 for periodic motor vehicle inspection is

the average of two values: $21,000 and $57,000. Another estimate not included in the average is $1,300,000.

The costs of medical screening and treatment are generally lower than the cost of toxin control. The costs of medical screening and treatment range in the tens to hundreds of thousands of dollars, while the costs of toxin control range in the millions of dollars per year of life saved. This would suggest that to achieve a higher number of lives saved, we should shift some money from control to screening and treatment.

For example, an industry that uses asbestos would save money from reduced asbestos control. They could use the money saved for a better lung screening program for all the company's workers. A better lung screening program would detect more lung cancers earlier which would mean earlier treatment and more lives saved.

In an article in the Wall Street Journal, David Stipp commented on the excessive cost of some pollution controls listed in this study.

> Still, the study shows many prevention programs are like gold-plated cannons aimed at flies. The estimated 'excess mortality' they avert, especially in pollution control, often is minuscule. Consider the study's most expensive intervention: preventing releases of carcinogenic chloroform at pulp mills, which costs an estimated $99.4 billion for each life-year saved. The chloroform controls at 48 mills costs only $30.3 million annually, says Tammy Tengs, lead author of the study. But researchers estimate that it would be necessary to spend that much on controls for more than 33,000 years to avert a single fatal case of cancer. (Stipp)

Remember that the values in Table 18-1 are for life-years saved, not just lives saved. On a per life basis, the costs are higher. For example, let's assume that an average of 20 years of life are saved for each life saved. The cost per life saved for ALARA radiation control is $2.5 million per year of life saved. For one life saved, the cost is 20 times this, or $50 million. This is far more than the approximate $1 to 5 million based on the Swedish study (Bengtsson and Moberg) and the informal ALARA value.

How Did We Get Into this Quagmire?

Just how did we get into this situation and how can we get out of it? Without going through the history of radiation regulations, let's just say that the public has demanded a super clean environment where toxic materials and radiation are concerned. However, the pendulum may have swung too far. We need to step back and look at what we are really doing by requiring that a nuclear facility be super clean.

In his book describing the "vicious circle" of regulation, Steven Breyer lists the three aspects of this circle: public perception, congressional reaction, and uncertainties of the regulatory process. Each feeds on the other. The public, perceiving that some substance is dangerous pressures Congress to pass stiff regulations on that substance. The regulatory process often uses risk calculations that emphasize the upper bounds of a risk (the "safety-factor") (Breyer 45). This reinforces the public perception of the dangers of the substance. And the circle continues!

The third aspect deals with the process of determining risk. As we have seen for radiation, that task is not easy, especially if the risk calculation depends on many different factors. We, as a public, must become more educated so we can better understand the real risks of radiation. Those making the risk calculations must be clear when they explain the risks so public perception is not

Table 18-1
Costs of Life-Saving Interventions

Measure	Cost per Life-year (1993 dollars)
Immunization of children	$0
Prenatal care for pregnant women	$0
Mandatory seat belt use and child restraint laws	$98

Media campaign to increase voluntary use of seat belts	$310
Mammography for women over 50	$810
Smoking cessation advice (average men & women)	$1,700
Colorectal cancer screening for people over 40	$4,500
Smoke and heat detectors in homes	$8,100
Neonatal intensive care (average all weights)	$9,800
Screen blood donors for HIV	$14,000
Breast cancer: post-surgical chemotherapy	$22,000
Ban asbestos on brake blocks	$29,000
Smoke detectors in airplane lavatories	$30,000
Periodic motor vehicle inspections (average of 2 lowest)	$39,000
Driver air bag vs manual shoulder/lap belts	$42,000
Hypertension screening for asymptomatic people, age 20	$68,000
Radon control for homes with >4 pCi/L	$140,000
Ban amitraz pesticide on pears	$350,000
National 55 mph speed limit on rural interstates	$510,000
Precautions to prevent HIV infection in medical workers	$890,000
Ground fault circuit interrupters	$1,100,000
School bus safety: sensors on buses	$1,400,000
Reduce Working Level from 1.0 to 0.3 in uranium mines	$1,600,000
As low as reasonably achievable (ALARA) control at nuclear plants	$2,500,000
Ban asbestos on clutch facings	$2,700,000
Dioxin emission control at paper mills	$4,500,000
Benzene emission control at service station storage tanks	$91,000,000
Radiation emission standard at nuclear power plants	$140,000,000
Radiation emission control at NRC & non-DOE licensed facilities	$2,600,000,000
Chloroform private well emission standard at 48 pulp mills	$99,000,000,000

Source: Tengs et al.

distorted, either believing the risk is higher or lower than what is really the case. Finally, Congress (and we as citizens in contact with Congress), must carefully look at the data and make intelligent decisions about the most efficient use of limited resources where the public health is concerned.

Congress may be ready to change. Several members of congress "have pledged to offer risk-assessment amendments to every major piece of environmental legislation" (Graham) In the same article in the Wall Street Journal, John Graham, one of the authors of the Tengs study, goes on to say:

> Our elected officials need to ensure that the public is informed of the costs and benefits of new regulation. As they get serious about passing 'risk' legislation, let's grade them on how well they promote sound principles of risk analysis and management. A good report card means that we are taking a first step to cure America's syndrome of paranoia and neglect. (Graham)

Is all this conjecture about spending too much on control really having any effect on the real world? John Graham stated:

> Some communities have already begun to suffer the consequences of uninformed government spending. In Columbus, OH, for example, local officials were forced to cut basic public health services in order to comply with a variety of costly environmental mandates from the U.S. Environmental Protection Agency. Faced with an increasing number of unfunded federal mandates, achievable only at the expense of programs that address health risks prevalent in their communities, state and local officials are starting to cry foul. (Appalachian Compact Users of Radioactive Isotopes)

In the keynote address to the 1994 annual meeting of the Health Physics Society in San Francisco, Nuclear Regulatory Commissioner Dr. E. Gail de Planque stressed risk-based regulations.

> [D]eveloping a program for radiation protection involves philosophy as well as science. One can propose an initial premise: and that is that 'regulatory decisions on health and safety should be risk-based.' There are, of course, alternative bases for regulatory decision making such as 'best available technology.' However, risk-based decision making may be preferable because this mode of decision making acknowledges that the resources available for limiting risk are not inexhaustible and ensure that the resources which are available to society as a whole will be put to the best overall use. (de Planque)

With this statement, Commissioner de Planque has zeroed in on the real reason we are spending excessive amounts on small risks: "the best available technology." It just seems logical that if we have the technology to reduce a risk, we should use it. However, this thinking ignores the fact that the risk reduction may not be very effective.

Using "the best available technology" philosophy, we should armor plate and pad our cars so in an accident, even at high speeds, the occupants would not be injured. However, the cost to society would be tremendous. The cost of a car and the amount of fuel consumed would skyrocket, putting car ownership out of the reach for the average citizen. The use of resources to make these "safe" cars would divert funds from other more effective ways to reduce risks to the public.

Risk-based Decision Making

Commissioner de Planque outlines the three components of risk-based decision making: risk assessment, selection of an acceptable risk level, and risk management. Risk assessment is a scientific endeavor. However, as we saw when we looked at how radiation risks are determined, this process does involve some subjective judgments. The selection of an acceptable risk level requires consideration of the costs to society for reducing the risk as well as the benefits from using the substance. When we finally have an acceptable level of risk, then we can proceed to risk management through the regulatory process (de Planque).

That "acceptable risk level" is the big stumbling block in this process. What are the "normal" risks we face each day? Table 18-2 lists some of these risks.

What would be an acceptable annual risk to the public from radiation? To achieve one chance in a million, assuming a linear dose response, we would have to limit the annual dose to 0.02 mGy (2 mrad); for a risk of one per 100,000, the limit would be 0.2 mGy (20 mrad). In terms of cigarettes smoked, two cigarettes smoked in a lifetime produces a one in a million chance of dying, and 21 cigarettes in a lifetime produce a one in 100,000 chance. (Breyer 5)

But here I would insert a word of caution. Remember that these risk values, even the cigarette ones, assume a linear extrapolation down to zero dose. The 2 cigarettes giving a risk of one in a million is about what I derived in chapter 13 for the risk per person-cig. That simple derivation predicted 90 deaths among the 160,000,000 people who smoked just one cigarette, or 180 deaths if they each smoked two in a lifetime. This is a risk of 180 ÷ 160,000,000, or 1.1 out of a million. But can just one or two cigarettes in a lifetime really cause a real risk? I doubt it. Likewise, is the linear extrapolation for radiation risk valid down to the tiniest dose? I likewise doubt that.

Table 18-2
Risks of dying each year
Values are per million

Risk	Chance per million

hit by lightening	0.5
0.01 mGy (1 mrad) annual radiation dose	0.5
In airline crash in one trip	1.0
0.1 mGy (10 mrad) annual radiation dose	5
In airline crash in ten trips	10
As a pedestrian	20
In home fire	30
Hit by drunken driver	50
Woman having a baby	110
Appendectomy operation	200
Being a police officer	220
Typical 10 mGy (1 rad) annual dose to nuclear power plant worker	500
Being a fireman	800
Age 25-34; all risks	1370
Skydiving	2000
Age 35-44; all risks	2290
50 mGy (5 rad) maximum annual dose to nuclear worker	2500
Smoker (one or more packs a day)	3000
Age 45-54; all risks	5840

Sources: non-radiation values: Breyer 4-5
radiation values:
From Table 10-5 in Chapter 10, an annual dose of 1 mGy (100 mrad) produces a risk of 0.005 percent per mGy, or as a numerical risk, 0.00005 per mGy. A person receiving an annual dose of 0.01 mGy (1 mrad) per year would have a risk of 0.0000005 (0.01 Gy X 0.00005 per mGy = 0.0000005 = 5 X 10^{-7} = 0.5 X 10^{-6}) or 0.5 chances out of a million. For 0.1 mGy (10 mrad) a year, the risk is five out of a million,etc.

What would you argue for an acceptable dose to the public? To recommend a risk of less than one in a million would require some strong arguments why it should be less than just about every other risks we face every day, both voluntary and involuntary. To recommend a risk of more than 100 per million would require strong arguments why an involuntary risk should be so high. Somewhere between one and 100 per million would seem like an acceptable risk. If we as a public could agree on some value, then we could proceed with the business of effective risk management.

Chapter 19
Responsibility

Our Role in Society

Suppose someone proposes to build a nuclear facility near your home. Or, suppose an operating nuclear facility having technical problems is releasing radioactivity. You are on the route of a radioactive waste shipment. What should your role be as an informed, concerned citizen? Should you just pack up and leave? That is not a responsible response. A responsible person would stay, gather information, ask questions, then push to have regulatory agencies act on your behalf if the risk to the public is not acceptable.

Many facilities that use radioactivity are small and the amounts of radioactivity are small. These facilities would include well-logging companies, industrial radiography companies, certain manufacturing companies, and universities with research programs. These facilities do have

accidents and workers do receive unexpected doses of radiation. However, based on experience, these institutions pose a minimal risk of exposing the public to radiation.

However, accidents have happened. Workers at a well-drilling firm in Hebron, Ohio, attempted to remove a cesium-137 source from a damaged lead cover by drilling into the lead cover. They punctured the cesium source releasing the radioactivity. However, they did not follow proper procedures and consequently tracked the contamination outside the building and to several homes. Had they followed proper procedures the contamination would have been contained in the one section of one building. Neither the workers nor the public were exposed to significant radiation doses. The company lost its license to use radioactive materials (Fugate).

The one facility that poses the greatest risk of radiation exposure to the public is a nuclear power plant. No new plants are planned at this time so you should not have to respond to the construction of a new plant. However, an operating plant could have an accident that would pose a risk. What should you do?

The operator of the plant and the regulatory agencies should be prepared to provide information to the public about what is happening. However, no amount of information is going to sooth the nerves of all people; expect some stress and possible panic. Demand answers about what is happening. What radioactivity has the facility released? How much do they expect to be released soon?

You can't calculate the dose yourself, but you can demand this information from utility or public officials. Listen carefully to how the information is presented. Do not accept blanket statements such as “there is no problem” or “it is safe.” Make your own judgments based on what information you can gather yourself.

Then there will be those who will pronounce that doom is coming. Listen carefully to what they have to say but also learn if the facts are in line with what you know about radiation. Ask how they arrived at the conclusion of doom; what doses are they talking about and to whom? As with the details given by the utility and regulatory agencies, also listen carefully to how the information is presented. Do not accept blanket statements such as “you are all going to die” and “the doses are lethal.” Make your own judgments.

You don’t trust the utility or regulatory agencies? Then maybe you should think about setting up an independent monitoring network now, before the need arises. A network of at least 25 citizens with small radiation monitors could provide valuable independent information about the radiation doses in the area. This type of network is presently in place around a few operating nuclear plants. One around TMI has about 50 active participants who report data regularly (Luetzelschwab).

Although this can get boring as long as the plant is operating normally, the background information is invaluable in case of a large release of radioactivity. The cost of each monitor is a few hundred dollars and the regular operating costs are minimal when volunteers are willing to do the work.

The other nuclear facility that is going to cause concern is a low-level radioactive waste (LLRW) facility. If one is planned for your area, you need to gather as much information as possible. The company planning to construct the facility or the state that will license the facility will provide the basic information about construction, waste containment, planned air and water monitoring, and expected radiation doses to the public and workers. You could also ask for information on presently operating LLRW facilities. The best information to get is from those living near an operating LLRW facility. Such a trip would require some cost for travel, but those who are in the situation will undoubtedly provide the best information you can get.

As with nuclear power, there are those who are adamantly opposed to the construction of a LLRW facility. Listen to what they have to say, but again, don’t accept blanket statements about how many people will die. Upon what do they base their conclusions? If their data on radiation levels do not agree with those from the proposed facility operator or the state regulatory agency, then ask both why there is a difference. If you can determine a value for the expected dose to the public, then you can make up your own mind about who is right.

Again, if there is some distrust of the LLRW operators, a citizen’s monitoring network would be helpful. In this situation, simple radiation monitors would be useful but may not be totally adequate. Some radioactivity could be released to the atmosphere, but the most likely pathway to

the environment would be through the ground water. Therefore, the monitoring should include water sampling. However, that is not cheap; you would need some funding for this type of monitoring. Volunteers could do the sample collection, but the analysis would involve equipment that requires some expertise. The samples could be sent to an independent lab for analysis, but that would also cost money.

When a nuclear facility has an accident or if the mere existence of the facility is a cause of continual concern with the public, then a public forum about the operation of the facility may be useful. After the TMI accident, the Nuclear Regulatory Commission (NRC) established an advisory panel. The function of the panel was to listen to the comments from the residents of the Three Mile Island area, to give Pennsylvania government officials an opportunity to participate in the NRC's cleanup plans, and to make recommendations to the NRC commissioners.

Over the years the objectives changed to meet changing situations, but it was a valuable forum for the public. The nuclear facility operator, the regulatory agency, and the public had a chance to voice their proposals and concerns.

> NRC staff members believed that the scrutiny of the Advisory Panel forced the licensee [TMI officials] to think through their plans very carefully before presenting them to either the agency or the panel . . . the existence of the Advisory Panel influenced the way information about the cleanup was delivered and presented. Technical information was prepared by both the NRC and the licensee for wide dissemination and understanding by members of the lay public. (Lach)

The key word in this section has been "responsible." Radiation and its effects are frightening, but that is no reason to act irrationally and irresponsibly. With what you have learned in this book, you can probably ask the right questions and make rational and responsible decisions. We must put personal gains and losses aside and try to determine what is the best for the public as a whole. Unfortunately, this kind of thinking is difficult when we observe many others, both in government and private business, doing less than a stellar job at it.

In an editorial about the TMI Accident, the Harrisburg Evening News took both the utility and anti-nuclear groups to task for their irresponsible activities.

> Up to now, the nuclear industry and Metropolitan Edison Co., in their frenzy to defend themselves from a barrage of criticism, appeared to hold a monopoly on self-serving statements and half truths. Sadly, it seems that the anti-nuclear forces now feel compelled to evoke the Big Lie. For a scientist to present grossly inaccurate data is inexcusable. But to fit the method of analysis to a conclusion makes the scientist's motives suspect. Nuclear opponents must achieve the same level of responsibility that they are demanding from the nuclear industry. Sophistry and statistical sleight of hand only will dilute their credibility. (Harrisburg Evening News)

As much as we would like to have a clean, risk-free place to live and work, that is impossible. Somebody must assume some risk to manufacture and transport the items we need to make life possible and items we desire to make life pleasurable. We all should accept our share of the risks. Why should anybody have the right to shove the risk onto somebody else just because they have the money or clever arguments to keep the risks away? Remember, even if the proposed facility is not built anywhere that does not mean individuals are not at risk. Any alternative most likely will cause risk to somebody.

Some argue that those who generate the risk should keep the risk. But those who work in an industry are not producing the risk because they want to. Medical researchers who use radioactivity in a search for a cure for AIDS are not generating radioactive waste for their own benefit; it is all part of the research. The beneficiaries are the future AIDS victims who might be saved by that research. An electric utility that uses nuclear power to generate electricity is responding to the demands of the public for electricity. In this process they generate LLRW.

If we do not want a low-level radioactive waste facility near us are we saying that our health is more important than the health of those who have to work around the waste in the research facility or nuclear plant? Would an LLRW facility produce a smaller dose to the public (including the

workers) than from hundreds of separate storage facilities where the waste is generated? In addition, what is the risk to the public from an accidental release of waste from hundreds of unmonitored storage sites compared with one well-engineered, controlled waste facility? Is the total population dose from one LLRW facility more or less than the total population dose from many storage sites?

When a shopping center or (non-polluting) industry wants to locate somewhere, local officials seem to desire them for the added tax base and jobs. But these kinds of facilities also bring traffic congestion, demands on utilities, loss of farm and forest land, crime, and other undesirable effects. Why do these kinds of facilities not undergo the same kind of scrutiny required of a nuclear facility?

If a nuclear facility is proposed for your area, be prepared to ask questions. A good hard look may show that it is not as good as some, but not as bad as others, would like you to believe. For example, if someone gave me a list of undesirable facilities to be built next to my house, a low-level radioactive waste facility would be far down on my list, way behind things such as a sanitary landfill, shopping center, super highway, housing development, and any kind of an electrical power plant.

As a final thought before turning you loose with all this information, remember that you are doing science. Science has certain rules that you have to follow. The primary rule is that we must be able to duplicate the facts. When someone claims some response to radiation, ask if this has been observed and documented elsewhere. If someone observes the effect at one location or at some time, did someone else also observe the same effect at other locations or times under the same conditions?

Although peer-reviewed literature may not be readily accessible to you, try to get information from sources that are credible. Newspaper editorials may be good, but, as the name implies, the person writing the editorial may have a point to make; it is not always a statement of fact but an opinion. Newspaper reports can be good, but remember two important facts: One, the facts gathered may suffer from inaccuracies, and two, newspapers emphasize the sensational rather than the mundane. Whatever you read, read with a critical eye. Ask if what you are reading makes sense with what you understand to be true. Talk with people on both sides of the issue.

Finally, be open to change. If you have an opinion before reading something or talking with somebody, be ready to change your mind. A responsible person should make independent conclusions and not just believe anything written or spoken by others. What we think is true about something may not be true in reality. Remember the shark. That commonly perceived, viscous, eat-anything creature turned tail when face-to-face with a person.

Has the Pendulum Swung Too Far?

Let me close this chapter, and the book, with an extensive quote from Merril Eisenbud's book <u>How Clean is Clean? How Safe is Safe?</u> In his book, Dr. Eisenbud discusses the environmental movement and the regulations that it has generated over the past 30 years. In his last chapter he recognizes the important gains made by the environmental movement, but also recognizes that we may be wasting valuable resources to decrease the small risks from some industries.

> The environmental populists have made their point. Their message has been delivered, has been understood clearly. The time has come for a period of collective introspection to permit us to reorder our environmental health priorities. We must reorder the priorities but also preserve the environmental populism that has accomplished so many good things during the past three decades. We must ask whether the vast sums of money being spent for successive reductions in emissions from automobile tailpipes and industrial sources could be better spent on traditional environmental health problems which at the present time are receiving insufficient attention.
>
> Above all, we must permit informed discussion of the pros and cons of proposals that involve environmental protection. If a person does not believe a particular proposal is worthy of financial support he or she should

> have the right to argue against it without being branded 'anti-environmental' -- a modern form of heresy. We must recognize that many environmental decisions involve high degrees of economic and scientific complexity. Politicians should not be accused of being 'anti-environmental' because they do not agree with the popular mood on any given issue.
>
> We must not return to the unrestricted environmental practices of the 1960s, but we must ask whether we have passed the point of diminishing returns in some of our programs of pollution control, while at the same time we have neglected more basic problems of environmental health protection. Environmental populism has reversed the swing of a pendulum that was moving in the wrong direction. History will record that the reversal was a major social development, for which we should be grateful. However, have we gone too far in some directions and not far enough in others. We must take steps to assure that billions of dollars are not wasted on trivial environmental health risks while major environmental health problems are ignored. (Eisenbud 46-47)

Epilog
Three Mile Island Litigation -- The Trial that Wasn't

Shortly after the Three Mile Island accident in 1979, several citizens filed for claims of personal loss and health damage from the accident. General Public Utilities, the corporate owner of TMI, settled these claims out of court rather than go to the expense of a trial. This settlement produced over 2400 additional claims that would have cost the company billions of dollars to settle, so they decided to settle these cases in court. After many years of delay, the trial was set for the summer of 1996.

Nearly half the claims were for cancer (solid tumors and leukemia); the others included ailments such as aching joints, back pain, cesarean, constipation, diarrhea, hernia, and loss of memory. Obviously trying every claim would take centuries to complete, so the judge asked that each side pick six cases (later reduced to five) and the outcome of these test cases would be a guide for the settlement of the other 2400. Then the pretrial arguments began.

What was interesting about the litigation was the tactics used by the two sides. Because cancer is the only ailment documented as a radiation effect, the plaintiffs picked all their five cases as cancers. The defendants might have picked five non-cancer cases and would have had an easy time showing that these were not caused by radiation. However, they also chose five cancers, apparently five litigants who had low radiation doses and therefore would be easy to show that radiation could not have caused these cancers.

As indicated earlier, the average dose to the public, as determined by several studies, was less than a tenth of a mGy. The maximum dose to anybody was only 1 mGy (100 mrem). From the risk factors listed earlier, the risk of anybody getting cancer from a dose of less than 1 mGy is very small. Based on these risk factors, the plaintiffs would have had a hard time proving damage. However, the plaintiffs would take a different approach.

Rather than trying to prove that 1 mGy caused the claimed effects, the plaintiffs attempted to prove that, because the claimed effects were observed, then the radiation dose to the public had to be much more than 1 mGy. They were claiming the dose was 1000 mGy (100 rem). To me this is an interesting tactic: using the claims you are trying to prove as the truth and then use that "truth" to prove that the cause existed.

But how can you explain the fact that all the radiation measurements from the company, state and federal agencies showed doses less than 1 mGy? Simply argue that the radioactivity released by the plant followed narrow paths such that they fell between all the radiation monitors and were never detected. And that is just what they planned to do.

In addition, the plaintiffs had to argue that the emissions from the plant were much higher than the other experts had calculated. The studies done after the accident put the release at 2.5

million Curies. A witness for the defendants calculated the release was 8.6 million Curies; a considerably higher amount than the other studies gave. The plaintiffs had a witness who claimed that the plant released between 25 and 100 million Curies during some large releases called blowouts, but this witness later retracted his testimony (Rambo).

The plaintiffs presented evidence that they claimed showed high radiation levels existed after the accident. These claims included detection of radioactivity in Albany, New York, and Portland, Maine; reddening of skin, hair loss, vomiting, and pet deaths near the plant; a re-analysis (by Wing; later published as Wing, et al.) of a health study (Hatch et al.) that showed higher doses; witness from Russia that studied tree damage; and an analysis of blood samples (taken in 1994 and 1995) of local people that indicated high doses from the accident (Rambo).

The defendants countered with witnesses who testified that only one of the ten test cases received more than 0.25 mGy (25 mrem); that person had a dose of 0.75 mGy (75 mrem). In addition, they introduced other studies that verified their dose calculations, the Pennsylvania Department of Health and other studies that showed no evidence of health effects, and whole body counting studies done at local hospital that showed no evidence of radioactivity in people near the plant (Rambo).

How does a judge handle such a case? The purpose of the pretrial testimony is to give the judge a basis for deciding which witnesses will be allowed to testify before a jury. This means that the judge must have some knowledge of the scientific facts of the case. In this case, Judge Sylvia Rambo, of the United States District Court for the Middle District of Pennsylvania, did her homework. She not only studied previous legal cases involving radiation injury, but she also studied the basic literature on radiation, including the BEIR V report.

To have a valid claim, Judge Rambo specified four factors, based on Pennsylvania law, for the plaintiffs to prove: one. The defendants released radioactivity into the environment in excess of regulatory limits; two. The plaintiffs were exposed to this radiation; three. The plaintiffs have injuries; and four. Radiation was the cause of those injuries. The defendants conceded factor one and did not dispute factor three.

The problem with claiming radiation caused cancer is that there are so many other causes of cancer that it is impossible to state with absolute certainty that radiation was the cause. In order to prove damage, Judge Rambo specified several ways that the plaintiffs could meet this burden of proof: the illness was caused by radiation; the diagnosis occurred within the established latency period; cite evidence that the illness is susceptible to radiation induction at exposures in excess of 100 mGy (10 rem) (based on scientific literature that below 100 mGy the statistical evidence of increased risk is not certain); rule out other potential causes; present studies that there is an expected rise in the illness after radiation exposure; and show indication of radiation effects on plants and animals in the area (Rambo).

The plaintiffs presented evidence to satisfy these conditions. However, Judge Rambo was not convinced their evidence was valid. In her ruling, she had the following comments.

> 1. The plaintiffs had no admissible source term evidence. The testimony of the plaintiff witness amounted "to little more than speculation regarding what might have happened." According to the witness: "I did not see any supporting indications that would lead me to believe that there was a blowout...I do not believe there was evidence of a blowout."
> 2. The plume "testimony was found to be inadmissible during the Daubert hearings based upon the unscientific and unreliable methodology supporting his testimony."
> 3. The evidence for the high doses claimed by the plaintiffs was lacking.
>
> The record presently before the court does not support the fundamental assumptions made by Dr. Wing -- that the doses were significantly higher than originally estimated. In the absence of this assumption, Dr. Wing himself admits that he would be unable to make a causal interpretation based on his findings.
>
> The abnormalities in the blood samples "decrease significantly in the first year following exposure.... Plaintiffs have presented no scientific evidence

> that would support a finding that the ... analysis, performed more than fifteen years after the accident, is more than a minimally accurate means of proving prior exposure to radiation.
>
> The tree damage evidence was found to be lacking in scientific depth. About the witness: "Professor Shevchenko likely has more personal experience making first-hand observations of radiation exposed areas than any other expert involved in this litigation. His credentials are impressive.... Indeed his observations of tree damage in the former Soviet Union were made in conjunction with subcellular analysis of tree tissue. However, Professor Shevchenko has not performed similar studies on the trees he observed in the TMI area. ("No, I have not conducted such investigation (sic). ... I did not have such an opportunity.") To the extent that Professor Shevchenko's confidence in his abilities is warranted, the record nevertheless shows that his observations were cursory (Rambo).

In her final ruling, Judge Rambo stated: "The paucity of proof alleged in support of Plaintiffs' case is manifest. The court has searched the record for any and all evidence which construed in a light most favorable to Plaintiffs creates a genuine issue of material fact warranting submission of their claims to a jury. This effort has been in vain" (Rambo). She then granted a summary judgment for the defendants.

What does all this mean? There is one rather interesting implication from this litigation. The plaintiffs did not argue that the measured doses caused the claimed health damage. They tried to argue that the doses were high enough so their claims would have validity based on data in the scientific literature. This implies that they conceded that low doses cannot cause measurable radiation damage. If this is the case, then those who oppose the construction of low-level radioactive waste facilities, for example, cannot argue that people will suffer health damage. Predictions of drastic health effects from low levels of radiation will still be good fodder for the press, but when taken to court, the claims will not hold water.

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Appendix A
Detailed Listing of Relative Risk for Different Kinds of Solid Tumors

Table A-1
Solid Tumor Incidence and Motality Relative Risks from Radiation
Values in percent per mGy[1]

Study[2]	Group[3]	Esophogus		Stomach		Colon		Liver		Lung		Female Breast		Bladder	
		Inc.	Mort	Inc.	Mort	Inc.	Mort	Inc.	Mort	Inc.	Mort	Inc.	Mort	Inc.	Mort
LSS-1	Male	0.016	0.037	0.012	0.017	0.087	0.047	0.061	0.054	0.036	0.021			0.035	0.083
	Female	0.106	0.016	0.052	0.026	0.048	0.047	0.011	0.027	0.208	0.159			0.180	0.024
	AAE<20	0.332	0.120	0.074	0.022	0.062	0.060	0.139	0.126	0.057	-0.038	0.332	0.569	0.071	0.024
	AAE>20	0.013	0.056	0.024	0.022	0.070	0.047	0.019	0.031	0.106	0.088	0.098	0.055	0.079	0.071
	All	0.029	0.059	0.030	0.022	0.067	0.047	0.041	0.044	0.100	0.076	0.174	0.179	0.076	0.048
	TSE5-19									0.104	0.048	0.119	0.080		
	TSE20-29									0.105	0.204	0.134	0.092		
	TSE30-42									0.085	0.069	0.221	0.287		
	Mass TB		0.001								-0.019	0.040			
	Ank. Spond		0.030		0.001				0.005		0.012	0.124			0.017
	Other				0.028[a]		0.047[a]		-0.05[a]			0.035[d]	0.081[g]	0.007[h]	0.021[a]
	Other				0.009[b]		0.004[b]		0.00[b]		0.039[b]	0.043[e]			
	Other				0.101[c]		0.013[c]		-0.05[c]			0.007[f]			0.054[c]
LSS-2	Male	0.004		0.018		0.076		0.067		0.039					0.048
	Female	0.183		0.051		0.068		0.017		0.169					0.223
	AAE0-9	0.060		0.065		0.188		-0.025		0.056		0.321			-0.025
	AAE10-19	0.000		0.069		0.105		0.085		0.060		0.261			0.100
	AAE20-39	0.035		0.043		0.057		0.092		0.082		0.124			0.084
	AAE≥40	0.037		0.012		0.062		0.044		0.085		0.059			0.123
	All	0.028		0.032		0.072		0.049		0.080		0.159			0.102

Comments for Table A-1:
[1]Values for doses greater than 200 mGy. For doses les than 200 mGy, these values represent maximum relative risks; the actual risks could be considerably less than the values listed here.
[2]LSS-1: Life span study; UNSCEAR, 1994

Ank Spon: Ankylosing Spondylitus (UNSCEAR, 1994)
Mass TB: Massachussetts turbuculus study (UNSCEAR, 1994)
Can TB: Canadian Tuberuculus study (UNSCEAR, 1994)
LSS-2: Life span study; Thompson, et. al., 1994
[3]AAE = Age at exposure; TSE = Time since exposure

[a]Benign gynaecological disease (UNSCESAR, 1994)
[b]Peptic ulcer (UNSCESAR, 1994)
[c]Metropathia haemorrhagica (UNSCESAR, 1994)
[d]Swedish breast irradiation (UNSCESAR, 1994)
[e]New York acute post-partum mastitis (UNSCESAR, 1994)
[f]Contralateral breast; U.S. (UNSCESAR, 1994)
[g]Canada TB fluoroscopy (UNSCESAR, 1994)
[h]Cervical cancer case-control (UNSCESAR, 1994)

The relative risks in Table A-1 may be hard to interpret because they are relative to the natural risk. Table A-2 shows the absolute risk. These values were derived by multiplying Table 10-3 values for the relative risk by the natural risk of each type of cancer.

Table A-2
Absolute Risk per Dose from Various Solid Tumor Cancers
Mortality: values in percent per mGy

Cancer Type	Natural solid tumor rate[1]		Relative risk percent per mGy[2]		Absolute risk[3] percent per mGy	
	Male	Female	Male	Female	Male	Female
Bladder	5.6	1.7	0.097	0.156	0.00027	0.00014
Brain & Nervous System	5.1	3.4	0.015	0.169	0.00004	0.00029
Female Breast		27.4		0.169		0.0023
Colon	20.0	14.1	0.062	0.058	0.00060	0.00041
Esophogus	5.9	1.5	0.022	0.172	0.00007	0.00013
Liver	5.2	3.2	0.061	0.022	0.00016	0.00004
Lung	74.2	30.6	0.035	0.176	0.0013	0.0027
Oral Cavity	4.6	1.7	0.016	0.046	0.00004	0.00004
Ovary		7.9		0.063		0.00025
Pancreas	9.9	7.1	0.022	0.011	0.00011	0.00039
Prostate	25.3		0.030		0.00038	
Rectum	3.5	2.0	0.00	0.051	0.0000	0.00005
Stomach	6.9	3.1	0.034	0.039	0.00012	0.00006
Uterus		3.5		-0.003		-0.000006
All solid tumors[4]	218.0	140.8	0.026	0.072	0.0028	.0051

[1]Rate per 100,000 per year; from American Cancer Society (1994); based on 1988-90 death rates adjusted to 1970 U.S. Population
[2]Average of UNSCEAR (1994) and Thompson et. al. (1994) values in Table 10-3
[3]The values in these columns are derived by multiplying the natural risk (per 100,000 population per year) by the percent relative risk per mGy and then multiplying this by 50 years, the approximate time at risk after a radiation exposure. This gives a risk in percent per mGy.
[4]RERF does not list separate total relative risks for male and females. The values here are from UNSCEAR, Table 9. The percent risk per mGy are lower than those from Table 9-1 because different populations are used for the different risks and different assumptions go into the different calculations.

Appendix B
Mathematics

Percents

A percent is a fraction of 100. Ten percent is 10 parts of 100, or, 10 ÷ 100 = 0.1

To convert a number written in decimal form to a percent, multiply the number by 100. For example, to convert 0.22 to a percent, multiply 0.22 by 100:

0.22 X 100 = 22 percent (22%)

To change a percent to a decimal, divide the percent by 100. For example, to convert 57 percent to a decimal, divide by 100:

57 percent ÷ 100 = 0.57

Risks are given in percent per mGy. This can be converted to a fraction per mGy by dividing the percent per mGy by 100. For example:

0.022 percent per mGy ÷ 100 = 0.00022 per mGy

This means that the fractional risk is 0.00022. This can be converted to a chance per number by taking the inverse of the number: 1 ÷ 0.00022 = 4545. This gives a risk of 1 chance out of 4545, or about one out of 4500.

Percent risk conversion to one chance out of a number

To convert a percent risk to one chance out of a number, add two decimal places (to make the number a fraction) and then invert the number. For example, if the risk is 0.004. Adding two decimal places gives 0.00004. Inverting this (1 ÷ 0.00004) gives 25,000. The table below gives the conversion for a range of percent risks.

Percent risk	One chance out of:
0.0001	1,000,000
0.0002	500,000
0.0005	200,000
0.001	100,000
0.002	50,000
0.005	20,000
0.01	10,000
0.02	5,000
0.05	2,000
0.1	1,000

Scientific notation

Numbers such as 1,198 and 0.024 are easy to read and visualize. However, numbers such as 2,300,000,000 and 0.0000000405 can be hard to visualize and also hard to write. (Counting zero's is not easy!) For very large and very small numbers, we use scientific notation which is based on powers of 10.

We know that the square of a number is the number multiplied by itself. The square of 10 is 10^2 = 10 X 10 = 100. The cube of a number is the number multiplied by itself twice, or: 10^3 = 10 X 10 X 10 = 1,000. We can continue this for any power of 10.

10^4 = 1 followed by 4 zeros = 10,000
10^5 = 1 followed by 5 zeros = 100,000
10^6 = 1 followed by 6 zeros = 1,000,000

10^9 = 1,000,000,000
10^{12} = 1,000,000,000,000

Now we can write any large number with the scientific notation if we just count the number of places. For example, 12,000,000 is 12 followed by 6 places, or it is 1.2 followed by 7 places. Therefore we can write the number either as 12 X 10^6 (12 X 1,000,000 = 12,000,000) or as 1.2 X 10^7 (1.2 X 10,000,000 = 12,000,000). The second way (the number between 1 and 10) is the preferable way.

Likewise, we can write small numbers using the inverse powers of 10. For example, 10^{-1} is 1 divided by 10 (10^{-1} = $1/10^1$ = 1/10 = 0.1). The minus sign in the exponent means that the 10 is in the denominator. Similarly, 10^{-2} = $1/10^2$ = 1/100 = 0.01. We can continue this for any inverse powers of 10.

10^{-3} = $1/10^3$ = 1/1,000 = 0.001
10^{-4} = $1/10^4$ = 1/10,000 = 0.0001
10^{-5} = $1/10^5$ = 1/100,000 = 0.00001

10^{-9} = $1/10^9$ = 1/1,000,000,000 = 0.000000001
10^{-12} = $1/10^{12}$ = 1/1,000,000,000,000 = 0.000000000001

We can abbreviate some of these powers of 10 with prefixes on the units. For example, the prefix for 1000 is kilo, abbreviated as k. A distance of 1 kilometer (1 km) is 1,000 meters. The prefix for 0.001 (one thousandth) is milli, abbreviated as m. A distance of 1 millimeter (1 mm) is one thousandth of a meter. A dose of 1 mGy is a thousandth of a Gray. The prefixes (with abbreviations) from the book are:

10^{-12}: pico (p) [1 pCi = 1 picoCurie = 0.000000000001 Curie]
10^{-9}: nano (n)
10^{-6}: micro (μ) [1 μGy = 1 microGray = 0.000001 Gray]
10^{-3}: milli (m) [1 mrem = 1 millirem = 0.001 rem]
10^{-2}: centi (c) [1 cm = 1 centimeter = 0.01 meter]
10^2: deka (d)
10^3: kilo (k) [1 kg = 1 kilogram = 1000 gram]
10^6: mega (M) [1 Mbq = 1 MegaBecquerel = 1,000,000 Bq]
10^9: giga (G) [1 Gbq = 1 GigaBecquerel = 1,000,000,000 Bq]

Graphs

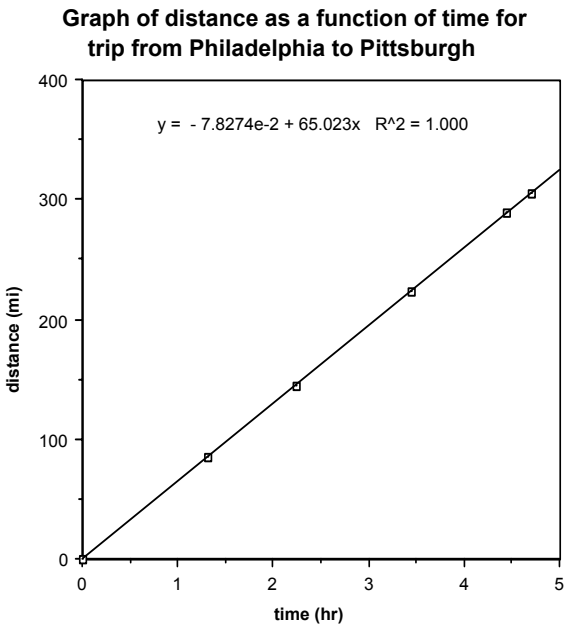
Graphs play an important part in analyzing the risk from radiation. The display of data in graphical form is important to understand.

When scientists takes data to find the dependence of one factor on another, they measure two values and plot one against the other. In the book we plotted risk as a function of radiation dose.

As an example, let's assume that you are driving from Philadelphia to Pittsburgh on the Pennsylvania Turnpike. You start with a full tank of gas so you can make the trip without stopping. You set your cruise control at 65 mph and start out with your trip odometer set to zero and a stopwatch set at zero time.. You want to find your distance as a function of time, so you record the distance at various times. You get a table of data as shown below. In the third column, you convert the time in minute to time in hours by dividing the minutes by 60.

distance (miles)	time (minutes)	time (hours)
0	0	0
85	78	1.31
145	134	2.23
224	207	3.45
289	267	4.45
306	282	4.71

Now, you plot (well, you have your computer do the plotting) the distance as a function of time (distance on the vertical axis, time on the horizontal axis). Your graph looks like the one to the right. You determine the slope of the line and find that it equals 65 (your speed!). The slope gives the ratio of the change of distance to the change of time, which is the speed.



If you had data of risk as a function of dose, the risk would be equivalent to the distance and the dose to the time. Then the slope would tell you how the risk changes with dose. The steeper the slope (i.e., the faster you drive), more the risk increases with dose.

Now, suppose you don't travel at a constant speed. Instead, you do your trip in 60 mile segments. In the first segment, you travel at 10 mph, second at 20 mph, third at 30 mph, etc. until you have traveled 300 miles. You measure the time at the end of each segment and get the following data on the next page

Plotting these data, you get the curve as shown at the right. Note that the computer fit shows a fit that has a linear term (-1.6133x) plus a quadratic term (1.6725x^2) so the fit is linear-quadratic.

distance (miles)	time (hours)
0	0
60	6

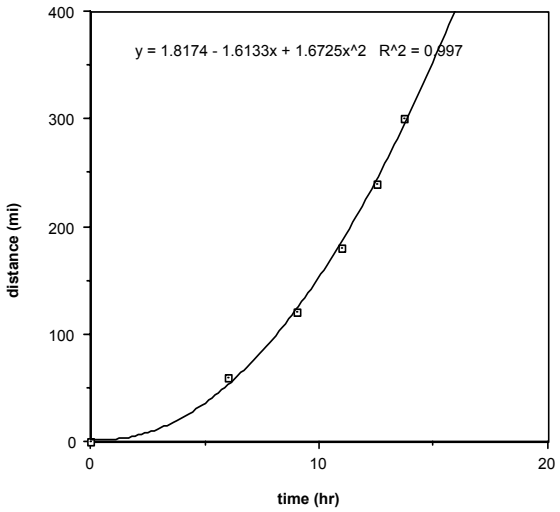
120	9
180	11
240	12.5
300	13.7

What does this mean? During each segment, your speed increases so the distance you travel in a given time increases. This differs from the linear curve. The linear curve is for the situation where your speed is constant and therefore the distance traveled per time is the same throughout the entire trip.

If this were a plot of risk as a function of dose, then we would conclude that as the dose increases, the risk per dose also increases. This compares with the linear curve where the risk per dose is the same for any dose.

To get some idea of how the same situation can give different results, suppose you change the way you take the data. If you measured the time at the mid-point of each segment, you would get the following data and graph.

Graph of distance as a function of time for changing speed: time measured a the end of each 60 mile segment



distance (miles)	time (hours)
0	0
30	3
90	7.5
150	10
210	11.75
270	13.1

Notice that the curve still look the same, and the fit is linear-quadratic, but the fit has changed. The linear term has a coefficient of +0.34934 rather than -1.6133 and the quadratic term has a coefficient of 1.4770 rather than 1.6725. This is one example of how the data from the same situation can give different results.

Graph of distance as a function of time for changing speed: time measured at mid-point of each 60 mile segment